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Original Article / Özgün Araştırma

Validity of Pulse Pressure Variation (PPV) Compared with Stroke Volume Variation (SVV) in Predicting Fluid Responsiveness

Sıvı Yanıtının Tahmin Edilmesinde Atım Hacmi Değişimi (SVV) ile Karşılaştırıldığında Nabız Basıncı Değişiminin (PPV) Geçerliliği

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Abstract / Öz

Objective: Static monitors for assessing the fluid status during major surgeries and in critically ill patients have been gradually replaced by more accurate dynamic monitors in modern-day anaesthesia practice. Pulse pressure variation (PPV) and systolic pressure variation (SPV) are the two commonly used dynamic indices for assessing fluid responsiveness.

Methods: In this prospective observational study, 50 patients undergoing major surgeries were monitored for PPV and SPV: after the induction of anaesthesia and after the administration of 500 mL of isotonic crystalloid bolus. Following the fluid bolus, patients with a cardiac output increase of more than 15% were classified as responders and those with an increase of less than 15% were classified as non-responders.

Results: There were no significant differences in the heart rate (HR), mean arterial pressure (MAP), PPV, SVV, central venous pressure (CVP) and cardiac index (CI) between responders and non-responders. Before fluid bolus, the stroke volume was significantly lower in responders (p=0.030). After fluid bolus, MAP was significantly higher in responders but there were no significant changes in HR, CVP, CI, PPV and SVV. In both responders and non-responders, PPV strongly correlated with SVV before and after fluid bolus.

Conclusion: Both PPV and SVV are useful to predict cardiac response to fluid loading. In both responders and non-responders, PPV has a greater association with fluid responsiveness than SVV.

Keywords: Fluid management, pulse pressure variation, systolic pressure variation, fluid responsiveness

Amaç: Günümüzde anestezi pratiğinde, büyük ameliyatlarda ve ağır hastalarda sıvı durumunun değerlendirilmesi için kullanılan statik izlem yöntemlerinin yerini, daha doğru sonuçlar veren dinamik izlemler almıştır. Nabız basıncı değişimi (PPV) ve sistolik basınç değişimi (SPV) sıvı yanıtını değerlendirmek amacıyla yaygın bir şekilde kullanılan dinamik indekslerdir.

Yöntemler: Bu prospektif gözlemsel çalışmada, major cerrahi geçirecek 50 hastada anestezi indüksiyonundan ve 500 mL izotonik verildikten sonra PPV ve SPV monitörize edildi. Bolus sıvı uygulamasını takiben, %15'ten fazla kardiyak debisi artışı olan hastalar yanıt verenler olarak, %15'ten daha az artışı olanlar ise yanıt vermeyenler olarak sınıflandırıldılar.

Bulgular: Yanıt verenler ve vermeyenler arasında kalp atım hızı (HR), ortalama arter basıncı (MAP), PPV, SVV, santral venöz basınç (CVP) ve kardiyak indeks (CI) açısından anlamlı bir fark bulunmadı. Bolus sıvı uygulaması öncesinde, atım hacmi yanıt verenlerde anlamlı derecede daha düşüktü (p=0,030). Bolus sıvı uygulaması sonrasında, MAP yanıt verenlerde anlamlı ölçüde daha yüksek bulundu, ancak HR, CVP, CI, PPV ve SVV açısından anlamlı fark gözlenmedi. Bolus sıvı uygulaması öncesinde ve sonrasında, hem yanıt veren hem de yanıt vermeyen hastalarda, PPV değeri ile SVV değeri arasında güçlü bir ilişki saptandı.

Sonuç: PPV ve SVV sıvı yüklenmesine verilen kardiyak yanıtı tahmin etmede yararlıdır. Hem yanıt veren hem de vermeyen hastalarda PPV, SVV ile kıyaslandığında, sıvı yanıtı ile daha fazla ilişkilidir.

Anahtar Sözcükler: Sıvı yönetimi, nabız basıncı değişimi, sistolik basınç değişimi, sıvı yanıtı

Introduction

Managing intraoperative fluid therapy in major surgeries can be challenging. Preoperative fasting and general anaesthesia reduce intravascular volume, blood pressure as well as tissue perfusion in patients undergoing surgeries. Hypovolaemia increases the risk of vital organ dysfunction, but excessive intravenous fluid administration can also have deleterious effects. Thus, judicious intravenous fluid supplementation to achieve optimum cardiac performance is one of the most important haemodynamic goals in patients undergoing major surgeries. Objective quantification of the intravascular fluid status can be very difficult and erroneous. Central venous pressure (CVP) monitoring and pulmonary capillary wedge pressure (PCWP) have been traditionally used to estimate the circulating blood volume, but studies have shown that these monitors cannot reliably estimate preload (1) or predict responsiveness to fluid therapy (2-4).

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On the other hand, analysis of arterial pressure contour is a very effective way to assess the haemodynamic status during major surgeries (5). Several studies have reported that dynamic variables obtained from arterial pressure waveform analysis, such as pulse pressure variation (PPV) and stroke volume variation (SVV), are appropriate indicators to assess fluid responsiveness in patients under mechanical ventilation. SVV is a reliable predictor of fluid responsiveness (6). However, the assessment of SVV requires special monitors such as Vigileo monitors with FloTrac transducers (Edwards Lifescience, USA), which may not be widely available. The Vigileo-FloTrac system, which is based on analysis of arterial pulse contour, does not need external calibration, dye dilution, or thermodilution. This system provides a nearly beat-to-beat estimate of stroke volume (SV) and SVV. The device is accurate in assessing the cardiac output and SVV, which has been tested in several settings.

Pulse Pressure Variation (PPV) is a derivative of the arterial pulse waveform integrated in monitors of most anaesthesia workstations. The aim of the study was to validate the accuracy and effectiveness of PPV (measured using standard anaesthesia monitors integrated with workstations) compared with those of SVV (measured using a FloTrac transducer and Vigileo monitor) in predicting fluid responsiveness in patients undergoing major surgeries.

Methods

Institutional Ethics Committee approval was obtained prior to conducting this prospective observational study. The participants were provided a detailed explanation about the purpose of the study and were assured about the confidentiality of the information and that their participation was entirely optional. Written informed consent was obtained from 50 patients undergoing major non-cardiac surgery in a tertiary care hospital. Patients who hadAmerican Society of Anesthesiologists (ASA) physical status 1-3, were aged between 18-60 years, had undergone surgery and required invasive arterial pressure and CVP monitoring at the discretion of the attending anaesthesiologist were included in the study. Patients with any history of arrhythmias, significant valvular diseases, pulmonary hypertension, left ventricular ejection fraction less than 40%, or right ventricular dysfunction respiratory disorders that would result in elevated peak airway pressures wereexcluded from the study. After patient's arrival to the operating room, standard ASA monitors were placed. Anaesthesia was induced using propofol, and vecuronium was used to facilitate tracheal intubation. Patients were ventilated withan inspired oxygen fraction of 0.50 with a tidal volume of 8 mL kg^{-1} ideal body weight and with no positive end expiratory pressure (PEEP). The respiratory rate was adjusted to maintain an end-tidal carbon dioxide concentration of 35-40 mmHg. After the induction of anaesthesia, a 20-gauge arterial cannula was placed in the radial artery. Arterial pressures were measured using a FloTrac transducer and Vigileo monitor, and PPV was calculated using a standard anaesthesia workstation. Newer anaesthesia workstationshave the features of measuring PPV in response to fluid replacement therapy. This feature can be used with standard arterial pressure contour analysis. Clinicians can freeze a pressure waveform and identify the maximum and minimum pressure pulses, which coincide with the respiration cycles, and can estimate PPV. The machine automatically calculates PPV and displays it.Both PPV and arterial blood pressure values were considered as the average of three consecutive values at a 1-minute interval. A triple-lumen (7 Fr) central venous catheter was inserted in the right internal jugular vein or right subclavian vein and used for CVP monitoring and the administration of vasopressors, if required. During measurements and fluid trial, any manipulation such as tilting the operating table, urinary catheter insertion or any surgical intervention was strictly avoided. After establishing the apparatus, the first set of readings of both variables was recorded and the patient was infused with two boluses of 250 mL isotonic electrolyte solution (Sterofundin ISO; B Braun Medical, Switzerland) over a period of 10 minutes. After each bolus, SVV and PPV were recorded. Cardiac output (CO) was calculated from stroke volume (SV) and heart rate (HR) (CO=SV×HR). The values were recorded at baseline and after each bolus of fluid infusion, and this was used to classify patients as responders and non-responders. Following the crystalloid bolus, patients with a cardiac output increase of more than 15% were classified as responders and those with an increase of less than 15% were classified as non-responders. Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), HR, Mean Arterial Pressure (MAP), CO, PPV and SVV were simultaneously recorded at each time point.

The primary objective was to measure PPV and SVV before and after fluid infusion to the patient undergoing major surgery and to classify patients as responders and non-responders based on the percentage change in CO and the secondary objectives were to compare and validate the accuracy and predictability of fluid responsiveness measured using PPV and SVV.

Statistical analysis

All statistical analyses were performed using IBM Statistical Package for the Social Sciences (IBM SPSS Statistics, Armonk, NY, USA) version 20. The clinical profile of patients was analysed using chi-square test for qualitative

Table 1. Demographic data (n=50)

Patient characteristic	Responders (n=25)	Non-responders (n=25)
Age (years) (range)	42.53 (22–60)	45.34 (27–60)
Sex (male/female)	17/8	15/10
(% Male/female)	68/32	60/40
Weight (mean and in years)	62.0 (43–86)	60.61 (47–75)
ASA class I	0	6
II	21	18
III	4	1

ASA: American Society of Anaesthesiologists

Table 2. Haemodynamic variables before and after fluid loading

	n	Mean		SD
	Statistic	Statistic	SE	Statistic
PFB SBP	50	115.0600	1.55430	10.99055
PFB DBP	50	69.1800	1.13342	8.01450
PFB MAP	50	84.4800	1.06339	7.51934
PFB HR	50	73.6800	0.89417	6.32275
PFB SVV	50	16.0400	0.39275	2.77717
PFB PPV	50	24.2800	0.57003	4.03070
PFB CVP	50	9.8000	0.16903	1.19523
PFB SV	50	66.7200	0.83644	5.91449
PFB CI	50	2.6560	0.05607	0.39649
PFB CO	50	4916.4400	85.83716	606.96038
Post 250 mL SBP	50	117.8200	1.30890	9.25531
Post 250 mL DBP	50	73.2600	1.07354	7.59111
Post 250 mL MAP	50	88.2000	0.97729	6.91051
Post 250 mL HR	50	72.0800	0.65704	4.64600
Post 250 mL SVV	50	9.9600	0.38857	2.74761
Post 250 mL PPV	50	16.2000	0.45175	3.19438
Post 250 mL CVP	50	11.4200	0.10725	0.75835
Post 250 mL SV	50	75.1400	0.89215	6.30843
Post 250 mL CI	50	3.4740	0.07204	0.50943
Post 500 mL SBP	50	119.0600	1.26859	8.97027
Post 500 mL DBP	50	74.3800	1.03091	7.28961
Post 500 mL MAP	50	89.2800	0.94201	6.66100
Post 500 mL HR	50	72.8800	0.65630	4.64072
Post 500 mL SVV	50	9.4600	0.37927	2.68184
Post 500 mL PPV	50	15.0200	0.45400	3.21025
Post 500 mL CVP	50	11.6400	0.12041	0.85141
Post 500 mL SV	50	76.0800	0.92044	6.50852
Post 500 mL CI	50	3.5640	0.06944	0.49105
CO	50	5549.4400	90.10411	637.13229
% change in CO	50	13.4284	1.44432	10.21288

PFB: Prefluid Bolus; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; MAP: mean blood pressure; HR: heart rate; SV: stroke volume; SVV: stroke volume variation; CVP: central venous pressure; PPV: pulse pressure variation; SD: standard deviation; SE: standard error; CI: cardiac index; CO: cardiac output

variables and Student's t-test for quantitative variables. The correlation between quantitative outcomes was assessed using Pearson's correlation. A p value less than 0.05 was considered statistically significant.

Table 3. Correlation between different parameters in non-responders

		PFB PPV	Post 250 mL SVV	Post 250 mL PPV	Post 500 mL SVV	Post 500 mL PPV
PFB SVV	r	0.875*	0.553*	0.764*	0.579*	0.655*
	p	<0.001	0.004	<0.001	0.002	<0.001
PFB PPV	r		0.367	0.808*	0.408*	0.602*
	p		0.071	<0.001	0.043	0.001
Post 250 mL SVV	r			0.685*	0.874*	0.772*
	p			<0.001	<0.001	<0.001
Post 250 mL PPV	r				0.599*	0.798*
	p				0.002	0
Post 500 mL SVV	r					0.769*
	p					<0.001

*Significant change. PFB: Prefluid Bolus; SVV: stroke volume variation; PPV: pulse pressure variation;

Results

A total of 50 patients were included in this study. Of them 64% were males and 36% were females and most of them belonged to ASA physical status II (78%). The mean age of the patients was 44.36 (SD±10.8) years. Patient characteristics and preoperative findings are presented in Table 1. We observed no technical failure in either device. After anaesthesia induction and endotracheal intubation, baseline haemodynamic parameters were as follows: 115±10 mmHg (SBP), 69±8 mmHg (DBP), 84±7 mmHg (MAP), 16±2 (SVV), 73±6 beats per min (HR) and 2.6±0.3 m^{-2} min^{-1} (cardiac index, CI). There were 25 (50%) responders, defined by an increase in the cardiac output (CO) of >15% after volume expansion of 500 mL. There were no significant differences in HR, MAP, PPV, SVV, CVP and CI between responders and non-responders (p=0.05, 0.13, 0.21, 0.42, 0.81 and 0.08, respectively) at baseline. The increase in CO was at least 15% (range: 15.10%-35.42%) in 25 patients (responders) and less than 15% (range: 10.37%-12.79%) in 25 patients (non-responders). Haemodynamic variables in responders and non-responders before and after fluid challenge are outlined in Table 2. Before fluid infusion, SV was significantly lesser in responders than in non-responders (p=0.030). After fluid infusion, MAP was significantly higher in responders than in non-responders (p=0.07), while there were no significant changes in HR, CVP, CI, PPV and SVV (p=0.08, 0.74, 0.49, 0.89 and 0.56, respectively) between responders and non-responders. Correlations between different parameters in responders and non-responders are outlined in Tables 3-10. In responders, PPV before and after fluid loading was strongly correlated with SVV before fluid load-

Table 4. Correlation between different parameters in non-responders

		PFB PPV	PFB SBP	PFB DBP	PFB MAP	PFB HR	PFB CVP	PFB SV	PFB CI	PFB CO
PFB SVV	r	0.875*	0.005	-0.144	-0.105	0.376	-0.540*	-0.312	-0.443*	0.076
	p	<0.001	0.981	0.492	0.618	0.064	0.005	0.129	0.027	0.717
PFB PPV	r		0.082	-0.182	-0.093	0.480*	-0.590*	-0.446*	-0.515*	0.059
	p		0.698	0.385	0.658	0.015	0.002	0.025	0.008	0.78
PFB SBP	r			0.36	0.743*	-0.194	0.079	-0.307	0.348	-0.338
	p			0.077	<0.001	0.353	0.709	0.136	0.088	0.098
PFB DBP	r				0.891*	-0.139	0.261	0.128	0.387	-0.041
	p				<0.001	0.507	0.207	0.543	0.056	0.848
PFB MAP	r					-0.193	0.238	-0.042	0.460*	-0.183
	p					0.355	0.251	0.842	0.021	0.382
PFB HR	r						-0.216	0.02	-0.185	0.758*
	p						0.3	0.924	0.377	<0.001
PFB CVP	r							0.572*	0.565*	0.217
	p							0.003	0.003	0.298
PFB SV	r								0.562*	0.666*
	p								0.003	<0.001
PFB CI	r									0.232
	p									0.265

*Significant change. PFB: Prefluid Bolus; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; MAP: mean blood pressure; HR: heart rate; SV: stroke volume; SVV: stroke volume variation; CI: cardiac index; CO: cardiac output; CVP: central venous pressure

Table 5. Correlation between different parameters in non-responders

		Post 250 mL PPV	Post 250 mL SBP	Post 250 mL DBP	Post 250 mL MAP	Post 250 mL HR	Post 250 mL CVP	Post 250 mL SV	Post 250 mL CI
Post 250 mL SVV	r	0.685*	-0.14	0.118	0.024	0.374	-0.249	0.131	-0.023
	p	<0.001	0.504	0.574	0.911	0.066	0.231	0.534	0.912
Post 250 mL PPV	r		-0.008	0.187	0.141	0.411*	-0.36	0.136	<0.001
	p		0.969	0.369	0.503	0.041	0.077	0.517	0.998
Post 250 mL SBP	r			0.520*	0.816*	-0.059	0.304	-0.024	0.326
	p			0.008	<0.001	0.781	0.139	0.909	0.112
Post 250 mL DBP	r				0.917*	0.014	0.067	-0.137	0.035
	p				<0.001	0.947	0.749	0.515	0.868
Post 250 mL MAP	r					-0.009	0.178	-0.108	0.173
	p					0.966	0.396	0.608	0.407
Post 250 mL HR	r						-0.401*	0.066	-0.032
	p						0.047	0.755	0.88
Post 250 mL CVP	r							0.428*	0.664*
	p							0.033	<0.001
Post 250 mL SV	r								0.701*
	p								<0.001

*Significant change. PPV: pulse pressure variation; SVV: stroke volume variation; SBP: Systolic Blood Pressure; MAP: mean blood pressure; HR: heart rate; CVP: central venous pressure; SV: stroke volume; CI: cardiac index; DBP: Diastolic Blood Pressure

Table 6. Correlation between different parameters in non-responders

		Post 500 mL PPV	Post 500 mL SBP	Post 500 mL DBP	Post 500 mL MAP	Post 500 mL HR	Post 500 mL CVP	Post 500 mL SV	Post 500 mL CI
Post 500 mL SVV	r	0.769*	-0.186	-0.422*	-0.371	0.437*	-0.410*	0.206	-0.019
	p	<0.001	0.372	0.036	0.068	0.029	0.042	0.322	0.93
Post 500 mL PPV	r		-0.136	-0.159	-0.161	0.349	-0.578*	0.34	0.007
	p		0.518	0.448	0.442	0.088	0.002	0.097	0.972
Post 500 mL SBP	r			0.440*	0.782*	0.079	0.237	-0.159	0.234
	p			0.028	<0.001	0.707	0.253	0.447	0.26
Post 500 mL DBP	r				0.903*	0.107	-0.132	-0.415*	-0.094
	p				<0.001	0.612	0.53	0.039	0.656
Post 500 mL MAP	r					0.13	0.012	-0.353	0.054
	p					0.537	0.953	0.084	0.796
Post 500 mL HR	r						-0.295	0.032	-0.008
	p						0.153	0.879	0.969
Post 500 mL CVP	r							0.354	0.602*
	p							0.083	0.001
Post 500 mL SV	r								0.733*
	p								<0.001

*Significant change. PPV: pulse pressure variation; SVV: stroke volume variation; SBP: Systolic Blood Pressure; MAP: mean blood pressure; HR: heart rate; CVP: central venous pressure; SV: stroke volume; CI: cardiac index; DBP: Diastolic Blood Pressure; CI: cardiac index; CO: cardiac output

Table 7. Correlation between different parameters in responders

		PFB PPV	Post 250 mL SVV	Post 250 mL PPV	Post 500 mL SVV	PFB 500 mL PPV
PFB SVV	r	0.31	0.554*	0.107	0.527*	0.513*
	p	0.132	0.004	0.611	0.007	0.009
PFB PPV	r		0.177	0.522*	0.03	0.294
	p		0.396	0.007	0.887	0.153
Post 250 mL SVV	r			0.429*	0.828*	0.782*
	p			0.032	<0.001	<0.001
Post 250 mL PPV	r				0.261	0.670*
	p				0.208	<0.001
Post 500 mL SVV	r					0.811*
	p					<0.001

*Significant change. PFB: Prefluid Bolus; PPV: pulse pressure variation; SVV: stroke volume variation

ing (Pearson's correlation coefficient=0.875, 0.685 and 0.769, respectively, p<0.001). A similar significant positive correlation was observed in non-responders. SVV and PPV were found to have a direct correlation with the degree of fluid responsiveness, expressed as CI. PPV and SVV showed better correlation with CI in responders than in non-responders, but the results were not significant. This may be due to a small sample size. Our results demonstrate the efficacy of SVV and PPV in predicting cardiac response to intravenous fluid loading in the given clinical setting. In both responders and non-responders, PPV has a greater association with fluid responsiveness than SVV.

Discussion

Determination of the intravascular volume status based on clinical parameters can be difficult as well as misleading in critically ill patients and in patients undergoing major sur-

Table 8. Correlation between different parameters in responders

		PFB PPV	PFB SBP	PFB DBP	PFB MAP	PFB HR	PFB CVP	PFB SV	PFB CI	PFB CO
PFB SVV	r	0.31	-0.27	-0.014	-0.147	0.158	-0.522*	-0.259	-0.588*	-0.116
	p	0.132	0.192	0.945	0.484	0.451	0.008	0.211	0.002	0.581
PFB PPV	r		0.231	-0.044	0.089	0.349	-0.352	0.01	-0.449*	0.246
	p		0.268	0.836	0.673	0.087	0.084	0.961	0.024	0.236
PFB SBP	r			0.309	0.729*	0.001	0.277	-0.063	0.159	-0.035
	p			0.132	<0.001	0.995	0.18	0.766	0.449	0.867
PFB DBP	r				0.876*	-0.073	-0.101	-0.104	-0.119	-0.128
	p				<0.001	0.728	0.632	0.621	0.571	0.541
PFB MAP	r					-0.038	0.067	-0.107	-0.006	-0.101
	p					0.858	0.75	0.612	0.976	0.631
PFB HR	r						-0.024	-0.156	-0.348	0.552*
	p						0.911	0.455	0.088	0.004
PFB CVP	r							0.582*	0.804*	0.474*
	p							0.002	<0.001	0.017
PFB SV	r								0.536*	0.736*
	p								0.006	<0.001
PFB CI	r									0.202
	p									0.333

*Significant change. PPV: pulse pressure variation; SVV: stroke volume variation; SBP: Systolic Blood Pressure; MAP: mean blood pressure; HR: heart rate; CVP: central venous pressure; SV: stroke volume; CI: cardiac index; DBP: Diastolic Blood Pressure; CI: cardiac index; CO: cardiac output

Table 9. Correlation between different parameters in responders

		PFB 250 mL PPV	PFB 250 mL SBP	PFB 250 mL DBP	PFB 250 mL MAP	PFB 250 mL HR	PFB 250 mL CVP	Post 250 mL SV	Post 250 mL CI
Post 250 mL SVV	r	0.429*	-0.514*	-0.431*	-0.588*	0.268	-0.368	-0.207	0.148
	p	0.032	0.009	0.032	0.002	0.196	0.07	0.32	0.481
Post 250 mL PPV	r		-0.109	-0.173	-0.188	0.458*	0.017	0.36	0.179
	p		0.603	0.407	0.368	0.021	0.934	0.077	0.392
Post 250 mL SBP	r			0.198	0.673*	-0.135	0.352	-0.048	-0.281
	p			0.344	<0.001	0.519	0.084	0.821	0.173
Post 250 mL DBP	r				0.857*	0.162	-0.004	0.151	-0.01
	p				<0.001	0.438	0.984	0.47	0.962
Post 250 mL MAP	r					0.047	0.178	0.103	-0.15
	p					0.822	0.393	0.625	0.474
Post 250 mL HR	r						0.031	0.015	-0.035
	p						0.883	0.945	0.869
Post 250 mL CVP	r							0.418*	0.173
	p							0.038	0.409
Post 250 mL SV	r								0.615*
	p								0.001

*Significant change. PPV: pulse pressure variation; SVV: stroke volume variation; SBP: Systolic Blood Pressure; MAP: mean blood pressure; HR: heart rate; CVP: central venous pressure; SV: stroke volume; CI: cardiac index; DBP: Diastolic Blood Pressure; CI: cardiac index

Table 10. Correlation between different parameters in responders

		Post 500 mL PPV	PFB 500 mL SBP	Post 500 mL DBP	PFB 500 mL MAP	Post 500 mL HR	Post 500 mL CVP	Post 500 mL SV	Post 500 mL CI
Post 500 mL SVV	r	0.811*	–0.539*	–0.356	–0.528**	0.007	–0.495*	–0.139	0.008
	p	<0.001	0.005	0.08	0.007	0.972	0.012	0.507	0.97
Post 500 mL PPV	r		–0.459*	–0.231	–0.392	0.32	–0.507*	0.063	0.119
	p		0.021	0.267	0.052	0.119	0.01	0.765	0.57
Post 500 mL SBP	r			0.245	0.671*	0.049	0.333	0.042	0.058
	p			0.237	<0.001	0.816	0.104	0.843	0.783
Post 500 mL DBP	r				0.882*	0.393	–0.007	0.053	–0.068
	p				<0.001	0.052	0.972	0.802	0.747
Post 500 mL MAP	r					0.319	0.146	0.081	–0.01
	p					0.12	0.486	0.7	0.962
Post 500 mL HR	r						0.089	0.323	0.166
	p						0.671	0.115	0.427
Post 500 mL CVP	r							0.401*	0.323
	p							0.047	0.115
Post 500 mL SV	r								0.779*
	p								<0.001

*Significant change. PPV: pulse pressure variation; SVV: stroke volume variation; SBP: Systolic Blood Pressure; MAP: mean blood pressure; HR: heart rate; CVP: central venous pressure; SV: stroke volume; CI: cardiac index; DBP: Diastolic Blood Pressure; CI: cardiac index

gery. Traditionally, estimation of cardiac filling pressure to guide fluid therapy have been done with central venous and pulmonary artery catheters. However, several studies performed in recent times have challenged this traditional concept and have demonstrated that cardiac filling pressures are inaccurate in predicting fluid responsiveness. In addition, several dynamic tests of intravenous fluid responsiveness have been reported. These tests essentially monitor the change in SV after any manoeuvre that either increases or decreases the left ventricular preload. These tests commonly monitor the change in SV during mechanical ventilation to assess the intravascular volume status and predict fluid responsiveness. Several studies have demonstrated that PPV and SVV, which are derived from pulse contour analysis, and plethy smographic variation, which is derived from the change in the amplitude of the pulse oximetry waveform, are highly predictive of fluid responsiveness (7).

Stroke volume variation occurs because of a cyclical change in intrathoracic pressure caused by positive pressure mechanical ventilation. SVV has been recognised as a concept for guiding intravenous fluid therapy more than 20 years ago (8). This variable is the result of decreased venous return to the heart during positive pressure inspiration. SVV results in a concomitant change in arterial pressure and its objective estimation is possible by systolic pulse variation (SPV) and PPV. Both these variables have been used to assess fluid responsiveness in a number of clinical studies and have been shown to be sensitive in predicting the ventricular response to fluid loading (9-11). However, Michard et al. (12) found PPV to be superior to SPV because it reflects changes in transmural pressures more accurately and is less affected by extramural pressures changes such as pleural pressure. Another study found that SPV cannot be explained by only left ventricular volume changes and other factors such as intrathoracic and airway pressure changes affect SPV (13). Both these variables may be affected by changes in the vasomotor tone (14).

The current PiCCOplus monitoring system displays PPV values automatically in real time. In one study, SVV was found to be useful to assess the fluid responsiveness in postoperative patients with preserved as well as diminished left ventricular function (15), whereas in another study, no strong correlation was observed between SVV and changes in SV during a preoperative fluid bolus trial (16). Contradictory findings from a number of published studies may be the result of significant differences in designing these studies, e.g. adopting different ventilatory strategies and fluid therapy protocols and differences in the cardiovascular reserve of the studied patient population. Some authors have even questioned the importance of SVV in accurately assessing fluid responsiveness (14).

There are very few studies that have directly compared SVV with other estimates of SV variation. One such study found a close relationship between SVV and SPV (17), and both these variables can predict fluid responsiveness. Again, in an-

other study, both were found to SVV and PPV correlate well with each other, but the prediction of fluid responsiveness was not studied (18).

Conclusion

Stroke volume variation assessed by a FlowTrac transducer and Vigileo monitor and PPV assessed by anaesthesia workstation-integrated monitors showed comparable performance in predicting fluid responsiveness in patients undergoing major surgeries. PPV monitoring is cost-effective because the transducer used to estimate SVV is more expensive. Therefore, if the appropriate monitor is available, PPV could be preferred for preload estimation in patients undergoing major surgeries.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Institutional Ethics Committee Army Hospital (R&R), Delhi Cantt, India (Date: 23.10.2013).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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References

1. Kumar A, Anel R, Bunnell E, Habet K, Kanotti S, Marshall S, et al. Pulmonary artery occlusion pressure and central venous presuure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects. Crit Care Med 2004; 32: 691-9. [CrossRef]
2. Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. Chest 2008; 134: 172-8. [CrossRef]
3. Michard F, TeboulJL. Predicting fluid responsiveness in ICU patients; a critical analysis of evidence. Chest 2002; 121: 2000-8. [CrossRef]
4. Osman D, Ridel C, Ray P, Monnet X, Anguel N, Richard C, et al. Cardiac filling pressures are not appropriate to predict Haemodynamic response to volume challenge. Crit Care Med 2007; 35: 64-8. [CrossRef]
5. Ellis JE, Roizen MF, Mantha S, Schware ML, Lubarsky DA, Kennan CA. Anesthesia for vascular surgery. Clinical Anaesthesia, 5th ed. Edited by Barash PG, Cullen BF, Stoelting RK: Pheiladelphia; Lippincott Williams& W ilkins.2006; pp 933-73.
6. Biais M, Nouette-Gaulain K, Cottenceau V, Revel P, Sztark F. Uncalibrated pulse contour-derived stroke volume variation predicts fluid responsiveness in mechanically ventilated patients undergoing liver transplantation. Br J Anaesth 2008; 101: 761-8. [CrossRef]
7. Marik PE, Cavallazzi R, Vasu T, Hirani A. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients. A systematic review of the literature. Crit Care Med 2009, 37: 2642-7. [CrossRef]
8. Jardin F, Farcot JC, Gueret P. Cyclic changes in arterial pulse during respiratory support. Circulation 1983; 68: 266-74. [CrossRef]
9. Raphael J, Regali LA, Thiele RH. Hemodynamic monitoring in thoracic surgical patients. Curr Opin Anaesthesiol 2017: DOI: 10.1097/ACO.0000000000000408. [CrossRef]
10. Preisman S, DiSegni E, Vered Z, Perel A. Left ventricular preload and function during graded hemorrhage and retransfusion in pigs: analysis of arterial pressure waveform and correlation with echocardiography. Br J Anaesth 2002; 88: 716-8. [CrossRef]
11. Perel A. Assessing fluid responsiveness by the systolic pressure variation in mechanically ventilated patients: systolic pressure variation as a guide to fluid therapy in patients with sepsis-induced hypotension. Anesthesiology 1998; 89: 1309-10. [CrossRef]
12. Michard F, Boussart S, Chemla D, Anguel N, Mercat A, Lecarpenteia Y, et al. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. Am J RespiCrit Care Med 2000; 162: 134-8. [CrossRef]
13. Denault AY, Gasior TA, Gorcsan J III. Determinants of aortic pressure variation during positive-pressure ventilation in man. Chest 1999; 116: 176-86. [CrossRef]
14. Pinsky MR. Probing the limits of arterial pulse contour analysis to predict preload responsiveness. Anesth Analg 2003; 96: 1245-7. [CrossRef]
15. Reuter DA, Felbinger TW, Schmidt C,Kilger E, Goedje O, Lamm P. Stroke volume variation for assessment of cardiac responsiveness to volume loading in mechanically ventilated patients after cardiac surgery. Intensive Care Med 2002; 28: 392-8. [CrossRef]
16. Wiesenack C, Prasser C, Rodig G. Stroke volume variation as an indicator of fluid responsiveness using pulse contour analysis in mechanically ventilated patients. Anesth Analg 2003; 96: 1254-7. [CrossRef]
17. Reuter DA, Felbinger TW, Kilger E. Optimizing fluid therapy in mechanically ventilated patients after cardiac surgery by on-line monitoring of left ventricular stroke volume variations: comparison with aortic systolic pressure variations. Br J Anaesth 2002; 88: 124-6. [CrossRef]
18. Reuter DA, Goresh T, Goepfert MS. Effects of mid-line thoracotomy on the interaction between mechanical ventilation and cardiac filling during cardiac surgery. Br J Anaesth 2004; 92: 808-13.[CrossRef]