Trauma Monthly 2022; 27(4): 543-551

10.30491/tm.2022.318447.1410

Trauma Monthly

Original Article

Comparison of Different Hemodynamic Items in The Prediction of Fluid Responsiveness in Hypotensive Trauma Patients Under Mechanical Ventilation

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Received 2021-12-06; **Accepted** 2022-08-16; **Online Published** 2022-08-30

Abstract

Background: This study aimed to compare pulse pressure and pulse oximetry fluctuations in predicting the response to fluid therapy and find a noninvasive method to monitor intravascular volume in traumatic patients on ventilators.

Method: Forty hypotensive severe multiple trauma patients under mechanical ventilation were enrolled in this cross-sectional study. Based on cardiac index (CI) changes in response to 500-mL normal saline administration, patients were divided into two groups of responders and non-responders (each with 20 subjects). Mean arterial pressure (MAP), diastolic blood pressure (DBP), systolic blood pressure (SBP), heart rate (HR), pulse pressure variation (PPV), and respiratory variations in pulse oximetry plethysmographic waveform amplitude (ΔPOP) were compared before and after the intervention.

Results: The mean age of patients (22 males and 18 females) was 44.8 ± 15.4 years, who were divided into two groups: responder and non-responder to liquid. Following fluid therapy, SBP, DBP, and MAP only significantly increased in the responder group, while PPV and ΔPOP significantly decreased ($P < 0.05$). Also, PPV and ΔPOP significantly correlated before and after the intervention in the study groups ($P < 0.05$).

Discussion: This study demonstrated that ΔPOP, as PPV monitoring, can predict fluid responsiveness and volume status in hypotensive multiple trauma patients under controlled mechanical ventilation.

Keywords: Pulse Oximetry, Trauma, Pressure, Fluid Therapy, Intensive Care

Introduction

Traumatic injuries are a major cause of death.[1] One of the preventable causes of mortality and morbidity in trauma patients is proper and timely fluid resuscitation to maintain hemodynamic balance and avoid over-hydration of patients.[2] only half of the trauma patients can “respond” to fluid therapy, and only half of them are dependent on preload.[3] Fluid responsiveness refers to intravenous administration of fluids to increase heart rate (HR) or blood pressure.[4]

Static hemodynamic monitoring, such as CVP, which is based on measuring cardiac preload, is not accurate enough to predict patients’ response to fluid therapy.[5] Numerous studies have consistently demonstrated that the magnitude of respiratory variation of stroke volume predicts fluid responsiveness accurately in mechanically ventilated patients.[6]

Transmission of positive pressure during controlled mechanical ventilation to the intrathoracic great vessels causes the pulse pressure variation (PPV), which is the physiologic base of predicting fluid responsiveness through PPV.[7] Thus, a high PPV value accurately predicts fluid responsiveness (15% increase in CO after fluid therapy).[8]

Although PPV is accepted as an accurate indicator in monitoring the status of intravascular volume, it requires the installation of an arterial line, which is an invasive and time-consuming procedure and needs skilled staff.

An available, noninvasive, and simple device that can indicate changes in stroke volume would be particularly practical. The pulse oximeter could be a good candidate because the pulse oximetry plethysmographic (ΔPOP) waves demonstrate the peripheral arterial pressure waveform.[9]

Some studies have shown a good relationship between ΔPP and ΔPOP in septic, hypovolemic, and cardiac patients.[10]

Also, ΔPOP may be a useful device to predict fluid responsiveness.[11]

Although some previous studies have suggested that ΔPOP may offer a noninvasive guide for fluid therapy,[12] the correlation between ΔPOP and PPV has not been evaluated before and after fluid challenge in responder and non-responder groups [13] (especially in critically ill trauma patients) because, in these patients, it is vital to make a quick decision about the appropriate amount of intravascular volume to prevent complications.

The aim of this study was to compare the accuracy of pulse oximetry wave changes with PPV in hypotensive, multiple trauma patients under mechanical ventilation in a referral trauma center, Kerman Province, Iran.

Methods

This cross-sectional study was conducted at a single tertiary trauma center (Shahid Bahonar Hospital in Kerman Province) between March 2020 and March 2021.

This study was approved by the Ethics Committee of Kerman University of Medical Sciences (code: IR.KMU.REC.1399.468). Written informed consent was obtained from the patients' legal guardians.

Inclusion criteria were patients with multiple trauma due to severe road accidents (based on trauma score), hypotension (systolic blood pressure [SBP] <90 or mean arterial pressure [MAP] <65), and admission to the intensive care unit (ICU) during 12 hours of trauma.

Exclusion criteria were refusal of the legal guardian to participate, dysrhythmia, hypoxemia, cardiac abnormalities, chronic obstructive airway disease/intra-abdominal hypertension, sepsis, thoracic surgery/spinal cord injury, obvious bleeding, need for a high dose of vasopressors (more than 10 μ/kg/min), pregnancy, liver trauma, and retroperitoneal hematoma.

Adult patients (18 to 70 years old) who were hospitalized in Shahid Bahonar Hospital were included in the study according to the inclusion criteria.

Initially, patients were monitored by electrocardiogram (ECG), noninvasive blood pressure, and pulse oximetry. Patients' demographic information was collected, and the cardiac index (CI) was measured continuously using the Doppler probe implantation in the esophagus of the patients. After performing the Allen test, an arterial line was inserted in the radial artery of the non-dominant hand, and pulse pressure was recorded.

All patients were under mechanical ventilation in the supine position with a 30° angle of head elevation. Ventilator mode was the volume assist-control mode, and the tidal volume was 6-8 mL/kg of body weight, positive end-expiratory pressure (PEEP) ≤8 cm H_2O, RR = 10-12/min. All of them were sedated by fentanyl infusion (Richmond score of -4 to -5 and no respiratory effort).

Before fluid therapy, HR per minute, SBP, diastolic blood pressure (DBP), and MAP were measured and recorded. Data on arterial pulse rate waves were transferred to a computer and recorded using TrendFaceSolo version 1.1 (Ixellence GmbH, Wildau, Germany) and calculated using the following formula:

$$PPV= (PPmax-PPmin)/ [(PPmax+PPmin)/2]$$

At the same time, ΔPOP was continuously measured through the forefinger of the dominant hand (which did not have an arterial line) using a pulse oximeter probe and an impermeable cover to the surrounding light. The computer analysis of pulse oximeter plethysmograph waves was performed using PhysioLog version 1.0.1.1 (Protolink Inc., Richardson, TX, USA) and based on the following formula:

$$\Delta POP=(POPmax-POPmin)/ [(POPmax+POPmin)/2]$$

All hemodynamic data, including CI, HR, MAP, DBP, SBP, PPV, and pulse oximeter plethysmograph changes (ΔPOP), were recorded by an observer who was unaware of the study. Then, 500 mL of normal saline (0.9%) was infused within 15 minutes, and CI, HR, MAP, DBP, SBP, PPV, and ΔPOP were recorded after fluid therapy. Then, those with more than a 15% increase in CI after volume recovery were categorized as responders.[14] PPV >13% [15] and ΔPOP >11% [16] were considered as intravascular volume deficiency. If, after receiving 500 crystalloids, the levels of PPV and ΔPOP decreased to less than 13% and 11%, respectively, the patient was categorized as a responder to fluid therapy. Also, changes in PPV and ΔPOP were compared before and after fluid therapy to determine whether there was a significant association between these 2 indices.

Sample Size

Using MedCalc version 20.113 and considering the value of 0.8 below the receiver operating characteristic (ROC; curve) as the desired value, the type I error was 0.05, and the power was 90% (the type II error was 0.10). The sample size in each group required 19 people. Finally, to increase the power to 95%, the sample size in each group was determined to be 22 patients.

To compare the mean of hemodynamic parameters (such as HR, SBP, DBP, MAP, and CI before and after fluid therapy in 2 groups), the student *t* test (if normally distributed) or Wilcoxon test (if non-normally distributed) were used. The linear correlation between ΔPP and ΔPOP was calculated using the Pearson correlation coefficient (*r*) after normality evaluation and correction of repeated measurements. In addition, PPV and ΔPOP were compared using Bland-Altman analysis. The findings are presented using mean and SD. *P* values less than 0.05 were considered statistically significant. Data were analyzed using SPSS version 21 (SPSS Inc., Chicago, Ill, USA).

Result

Of the 44 patients, 2 were excluded due to previous cardiac disease, 1 due to cervical spinal cord injury resulting in spinal shock, and 1 due to chronic obstructive pulmonary disease; finally, 40 patients were included in the study. According to the criteria described in the Materials and Methods Section, 20 subjects were in the responder group, and 20 subjects were in the non-responder group.

Considering that Kerman Shahid Bahonar Hospital is a tertiary referral center for traffic accidents in the southeast of Iran, to homogenize the population under study, only patients who were in the severe group due to traffic accidents and based on the injury severity score (ISS) criteria were included in the study. In total, the average injury severity score in all patients was 7.8 ± 23; in detail, it was 22.6 ± 7.3 in the responder group and 8.1 ± 23.4 in the non-responder group, and no statistically significant difference was observed in this regard (P= 0.65).

Another criterion investigated was the type of traffic accidents, i.e., vehicle rollover (30%), car to car (25%), motor to car (20%), motor to motor (7.5%), car to obstacle (7.5%), car to person (5%), motor to person (5%); in this regard, no significant difference was observed between the 2 groups (P = 0.08; Table 1).

Table 1. The prevalence of different types of traffic accidents and average ISS in patients

Variables		Group			*P* value
		Total	Responder	Non-responder	
ISS±SD•		23±7.8	22.6±7.3	23.4±8.1	**0.65**
Type of traffic accident	Vehicle rollover	12 (30%)	7 (35%)	5 (25%)	
	Car to car	10 (25%)	6 (30%)	4 (20%)	
	Motor to car	8 (20%)	2 (10%)	6 (30%)	
	Motor to motor	3 (7.5%)	1 (5%)	2 (10%)	**0.08**
	Car to obstacle	3 (7.5%)	2 (10%)	1 (5%)	
	Car to person	2 (5%)	2 (10%)	0	
	Motor to person	2 (5%)	0	2 (10%)	

Out of 40 included patients, 22 were male, and 18 were female, with a mean age of 14.3 ± 82.44 years (18-68 years), 45 ± 15.34 years in the responder group and 44.6 ± 17.32 years in the non-responder group. There was no significant difference between the study groups regarding age (P = 0.810). The mean value of body mass index (BMI) was 8.69 ± 25.22 Kg/m2, and there was no significant difference between the 2 groups (P = 0.981), 25.22 ± 3.31 Kg/m2 in the responder group and 25.25 ± 3.04 Kg/m2 in the non-responder group (Table 2).

Table 2: Basic characteristics and demographic indicators of participants in the 2 groups.

Variable		Total	Responder	Non-responder	Result
Age (year)		44.8±15.4	34.15±45	32.17±6.44	***0.810**
BMI (kg/m^2)		25.24±3.12	31.3±22.25	04.3±25.25	***0.981**
Gender	Male	22	11	11	****0.992>**
	Female	18	9	9	

* Independent *t* test; ** Chi-square test

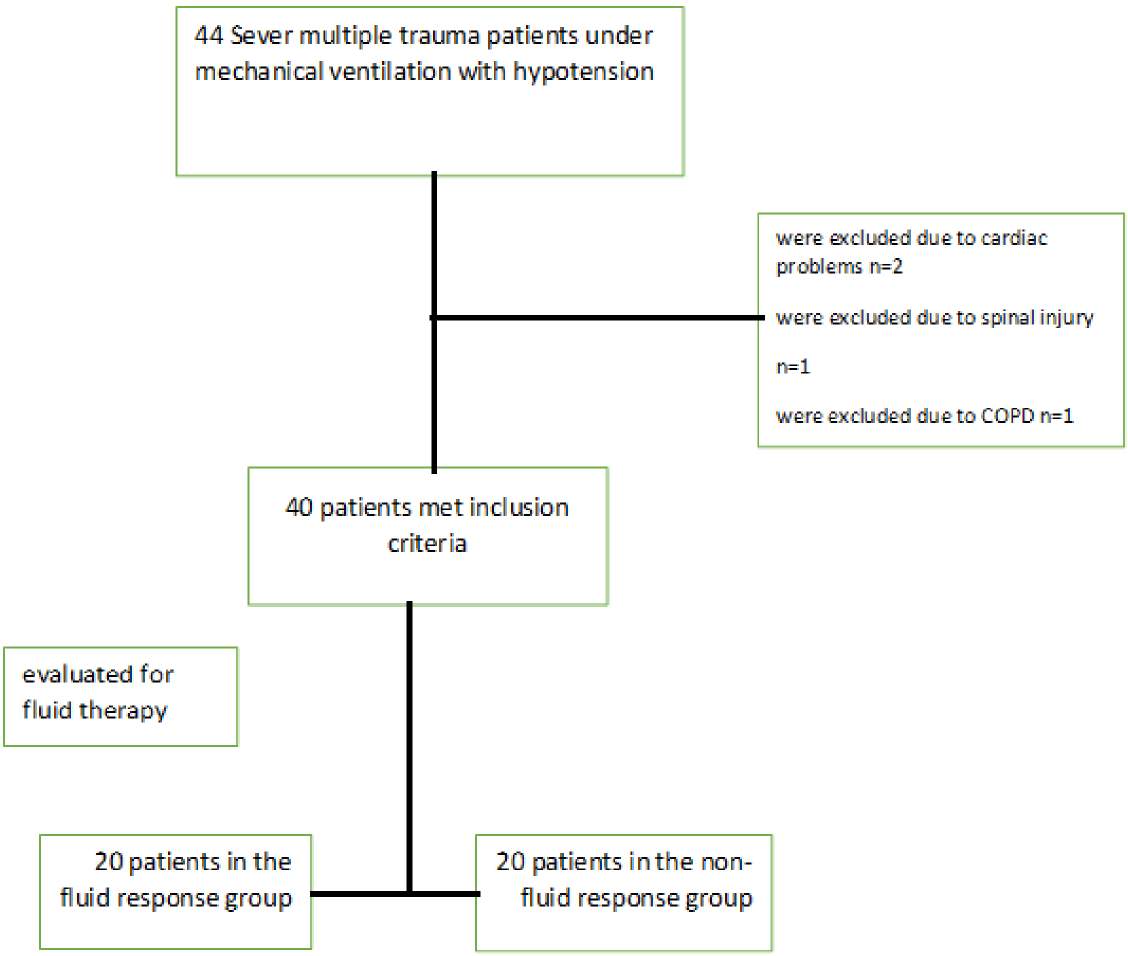

Figure 1. CONSORT Flow Diagram

The mean value of CI before fluid therapy was 3 ± 0.17 L/min/m2, which increased from 3 ± 0.9 to 3.5 ± 0.066 in the responder group (16.6% increase) while, for those in the non-responder group, increased from 3 ± 0.17 to 3.1 ± 0.44 (less than 15% increase; P = 0.001). For those in the responder and non-responder groups, the mean HR of patients before the intervention was 113.75 ± 13.47 and 112.9 ± 17.7, respectively, and there was no difference between the study groups (P = 0.904). However, after the intervention, these values were 97.1 ± 12.25 and 109.3 ± 15.28, respectively. As it is obvious, HR decreased in both groups, with no significant difference between the study groups (P = 0.803).

In the responder group, SBP was 76.3 ± 13.13 mm Hg before the intervention, while in the non-responder group, SBP before and after the intervention were 72 ± 11.41 and 86.1 ± 7.04, respectively; these values were significantly higher in the responder group (P < 0.001). Also, DBP in the responder group before and after the intervention was 50.45 ± 5.02 and 63.65 ± 12.65, respectively, while in the non-responder group, these values were 49.75 ± 5.25 and 55.4 ± 5.76 before and after the intervention, respectively; DBP was not significantly different before the intervention between the study groups (P = 0. 071). However, an increase in mean blood pressure after the intervention was higher in the responder group, and the difference was statistically significant (P < 0.001).

The mean MAP was 60.87 ± 4.45 and 59.07 ± 3.89 mm Hg in the responder and non-responder groups, respectively, and there was no significant difference between the study groups (P = 0.073). After fluid therapy, MAP was 78.98 ± 8.04 and 65.87 ± 4.45 mm Hg in the responder and non-responder groups, respectively, which was significantly higher in the responder group (P < 0.001).

The mean PPV before fluid infusion was 12.62 ± 4.45 and 13.45 ± 1.15 in the non-responder and responder groups, respectively, and there was no significant difference between the 2 groups (P = 0. 900), i.e., it was higher than 13% in the responder group and less than 13% in the non-responder group. After fluid therapy, PPV decreased in both groups, i.e., 12.22 ± 1.62 and 11.68 ± 2.45 in the responder and non-responder groups, respectively. In addition, PPV considerably decreased in the responder group, while in the non-responder group, it was less than 13%; this difference was both clinically and statistically significant (P = 0.021). Before fluid therapy, ΔPOP was 13.7 ± 1.27 and 12.37 ± 1.74 in the responder and non-responder groups, respectively; the difference was statistically significant (P = 0.017) (Table 3). After infusion, ΔPOP was 11.77 ± 1.2 and 12.2 ± 2.59 in the responder and non-responder groups; while it decreased in both groups, the observed decrease was significantly higher in the non-responder group *(P* = 0.042; Table 3). There was a significant, direct association between PPV and ΔPOP in the responder group before (r_s = 0.65 *and P* = 0.002) and after (r_s = 0.54 and *P* = 0.010) the intervention (Table 4; Charts 1 and 2). It can be argued that a change in PPV or ΔPOP can change the other variable, which is true for both groups.

Table 3: Evaluation of vital indicators and fluid therapy before and after the intervention in the 2 groups

Variable		Responder	Non-responder	Result
CI (L/min/m^2)	Before	3±0.9	3±0.17	*0. 701
	After	3.5±0.66	3.1±0.44	*0.001
SBP (mm Hg)	Before	11.41±3.72	13.13±76	*0. 908
	After	29.5±15.106	04.7±1.86	*0.001 <
DBP (mm Hg)	Before	02.5±45.50	25.5±75.49	*0. 071
	After	65.12±65.63	55.4±5.76	*0.001 <
MAP (mm Hg)	Before	45.4±87.60	89.3±07.59	*0. 073
	After	04.8±98.78	17.5±41.65	*0.001 <
HR (beat/min)	Before	47.13±75.113	7.17±9.112	**0.904
	After	97.1±12.25	109.3±15.28	**0.803
PPV%	Before	15.1±45.13	45.4±62.12	**0.900
	After	62.1±22.12	45.2±68.11	*0.021
ΔPOP%	Before	27.1±7.13	74.1±37.12	**0.017
	After	1.2±77.11	59.2±2.12	**0.042
* Mann-Whitney ;***t* test				

Table 4: The association between PPV and ΔPOP before and after the intervention.

Group	Intervention time	Variable	ΔPOP
Responder	Before	PPV	0.65=r_s 0.002=*P*
	After		0.54=r_s 0.010=*P*
Non-responder	Before	PPV	0.980=r_s 0.001<*P*
	After		0.920=r_p 0.001<*P*

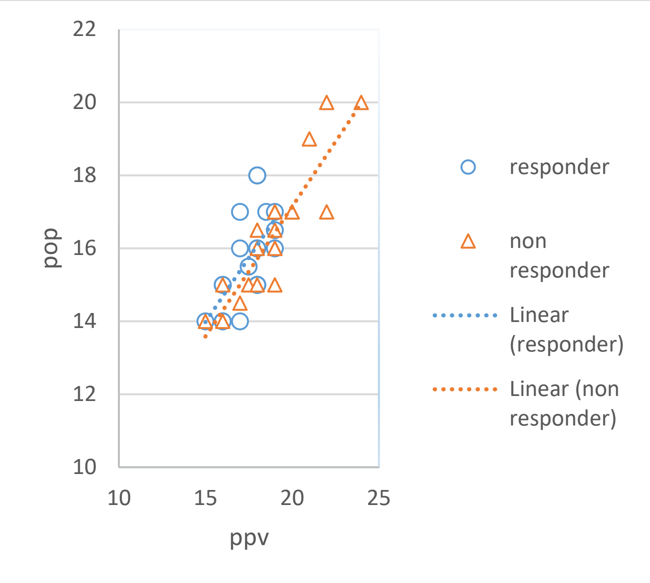

Chart 1: The association between PPV and ΔPOP before fluid therapy.

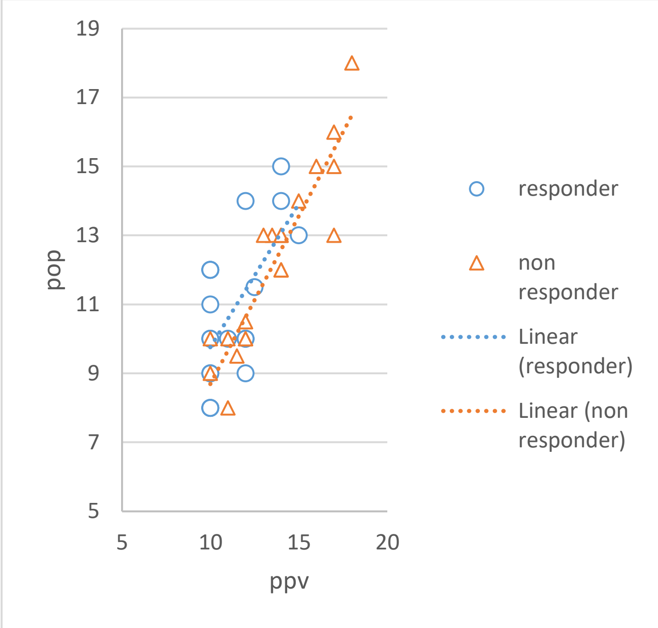

Chart 2: The association between PPV and ΔPOP after fluid therapy.

Discussion

Many recent studies have demonstrated the superiority of dynamic indicators (such as PPV) over static ones (such as CVP and pulmonary artery occlusion pressure [PAOP]) for predicting preload status and response to fluid challenges in patients under mechanical ventilation.[17, 18] Also, it has been shown that monitoring of fluid therapy in mechanically ventilated patients by ΔPP has the potential to decrease the hospital stay time and period of mechanical ventilation, as well as perioperative morbidity in patients undergoing high-risk surgery.[19]

Although PPV measurement is known as accurate monitoring for evaluation of volume status in mechanically ventilated patients, it is an invasive technique and time-consuming.[9] Thus, in some studies, the pulse oximetry plethysmographic signal variation due to positive pressure ventilation has been considered a useful and noninvasive alternative to PPV.[20, 21]

The present study showed a strong direct, linear correlation between the changes of the plethysmographic wave amplitude, which was measured with a skin pulse oximeter, and arterial pulse pressure fluctuations caused by positive pressure ventilation in hypotensive multiple trauma patients under mechanical ventilation. In addition, ΔPOP changes can be used to predict preload status and responsiveness to fluid therapy as an accurate, available, and noninvasive monitoring tool. Also, we found a good correlation between PPV and ΔPOP before and after volume challenge in the responder and non-responder groups. A strong agreement between PPV and ΔPOP was shown in mechanically ventilated patients by Cannesson et al. in 2005.[22] Unlike our study, the patients in this survey were hemodynamically stable. In 2007, the same authors also observed a significant relationship between ΔPOP in the anti-Trendelenburg position and change in MAP after volume expansion as a predictor of changes in heart preload.[23] In our study, pop changes were introduced as a suitable criterion for predicting fluid therapy and intravascular volume status of patients.

Contrary to our results, Bendjelid and Romand concluded that PPV and PPVfina were good predictors of fluid responsiveness during major hepatic surgery. They suggested that intraoperative ΔPOP was not accurate in monitoring fluid responsiveness as a simple and noninvasive tool.[24] The difference between our results and Bendjelid K can be due to sudden and numerous hemodynamic changes during major liver surgery based on the low abdominal compliance, high airway pressures, low aortic compliance, and low

peripheral resistance in these patients.
In contrast to our results, Le Guen et al. showed a poor correlation between ΔPOP and PPV compared to esophageal Doppler in patients requiring renal transplantation.[25] This discrepancy may be due to the pathological endothelial changes affecting arterial compliance, which led to poor capillary distribution. Cannesson et al. concluded that PVI, as automatic and continuous noninvasive monitoring of ΔPOP, could predict fluid responsiveness in mechanically ventilated patients during general anesthesia.[26] Consistent with our results, it showed the value of this monitoring in assessing the status of intravascular volume and response to fluid therapy. Chandler, J. R., et al. demonstrates the good correlation between PPV and ΔPOP indices in children under various physiological stresses. These data show a similarly strong correlation to that described in adults.[27] However, Ji S-H et al. demonstrated no significant correlation between PVI and PPV in children undergoing surgery in the prone position.[28] Given the controversy in the results of studies on children, more studies are needed in this age group.
The study of Cannesson et al. demonstrated a significant relationship between PPV and ΔPOP ($r = 0.92$; $P < 0.05$), showing the ability of ΔPOP that is derived from the pulse oximeter waveform; this ability has the potential for noninvasive fluid responsiveness monitoring in hemodynamically stable patients under general anesthesia and mechanical ventilation.[29] Although the study population differed from ours, it was surprisingly consistent with our results.
Besides showing a direct relationship between ΔPOP and PPV, Coeckelenbergh et al. demonstrated that goal-directed fluid therapy in patients undergoing low-to-moderate risk abdominal surgery reduced hospital stay, amount of fluid, and incidence of postoperative complications.[30]
Further, Warnakulasuriya et al. conducted a study on patients requiring colorectal resection; they showed no significant difference between the ΔPOP and esophageal Doppler groups in mean fluid administered or mean fluid balance.[31]
We evaluated only the correlation between ΔPOP and PPV in hypotensive patients under mechanical ventilation. However, the above studies demonstrated different results of fluid therapy guided by ΔPOP monitoring. In other complementary studies, the results of fluid therapy can be examined based on the guidance of various monitors in multiple hypotensive multiple trauma patients.
Bartels and Thiele concluded that respiratory changes in POP waves correlated only/modestly with PPV, but fluid responsiveness could be assessed using both tools.[32] Also, Hengy et al. conducted a study on patients under anesthesia for major abdominal surgery; they suggested that the use of ΔPOP, instead of PPV, was not appropriate to determine the need for fluid therapy during anesthesia in patients scheduled for major abdominal surgeries.[33]
In contrast with the abovementioned studies, we found a direct, strong association between these 2 variables, which can be attributed to the differences in the study population, method of fluid therapy, and the goal of investigations.
Consistent with our results, Westphal et al. concluded that ΔPpleth ≥11%, with 100% sensitivity and 91% specificity, had a powerful association with PPV >13%; they recommended it as a simple, noninvasive method for evaluating response to fluid therapy after heart surgery.[34] We also found an acceptable linear association between PPV and ΔPOP in both responder and non-responder mechanically ventilated patients to fluid therapy.
Our findings also showed that CI, SBP, DBP, and MAP only increased significantly in the responder group.
There were no differences in HR, MAP, or PPV between the responder and non-responder groups. After fluid bolus, MAP was significantly higher in responders, but there were no significant changes in HR and PPV.
Similar to our results, Radinovic et al. showed a direct relationship between PPV and MAP changes before and after fluid therapy.[35] Rathore et al. concluded that after fluid bolus, MAP significantly increased in responder patients, but there were no significant changes in HR, CVP, CI, PPV, and stroke volume variation (SVV) in both responder and non-responder groups; they mentioned that PPV was strongly associated with SVV before and after liquid bolus.[36]
The observed differences may be due to the study population, sample size, and methodology.
Qi et al. reported that fluid challenge caused a significant increase in DBP, SBP, and MAP in septic shock patients; however, they reported no change in HR.[37] In our study, MAP, SBP, and DBP increased in both responder and non-responder groups; however, it was only significant in the former. In addition, there was

no significant difference in HR between the 2 groups. Further, Ranjit et al. reported no significant association between MAP and CI changes in children with post-fluid septic shock following fluid therapy.[38] In our study, the responder group was characterized by a 15% increase in CI and showed a significant increase in MAP, whereas, in the non-responder group, there was no significant increase in MAP. The observed difference can be attributed to the different populations: Our patients were traumatic and hypotensive, while their patients were children with septic shock.

Limitations

The authors did not assess the effect of pre-hospital admission care, emergency staff care, vasopressor administration, acid-base disturbance, and electrolytes abnormality on the results of this study.

Conclusion

We conclude that ΔPOP ≥11% accurately correlates to the PPVs of more than 13% in severe multiple trauma patients under mechanical ventilation, and ΔPOP has the potential to determine the response to fluid therapy in these patients.

Acknowledgments

The authors sincerely thank the Clinical Research Database of Shahid Bahonar Hospital.

Authors' contributions

M. Ahmadinejad conceived of the presented idea, developed the theory, performed the computations, and wrote and submitted the article.

M. Sharifzadeh K. verified the analytical methods and provided study materials and equipment.

M. Ahmdipour assessed and recorded the hemodynamic parameters.

Y. Arefi M. conducted the study and collected the data.

Conflict of interest

The authors declare no conflicts of interest.

Funding/support

There were no funding sources for the current study.

Ethical consideration

This study was approved by the Ethics Committee of Kerman University of Medical Sciences (code: IR.KMU.REC.1399.468). Written informed consent was obtained from the patients' legal guardians before entering the study and after a comprehensive introduction to the study protocol. In addition, they were informed that they could withdraw from the study at any time. Also, they were ensured about the confidentiality of their information.

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