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Perfusion index as a predictor of hypotension after induction of general anesthesia in elderly patients – a prospective observational study

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ABSTRACT

Introduction: The elderly patients are at a greater risk for hypotension after induction of anesthesia. Perfusion index (PI) predicted patients vascular responses in many situations. This study hypothesized that baseline value of PI would predict hypotension in geriatric patients after anesthetic induction as population at risk for postinduction hypotension.

Methods: This observational, prospective study involved 30 patients aged >65 years who were scheduled for elective procedures under general anesthesia. From baseline to 15 min after propofol induction of anesthesia, the PI, heart rate, and blood pressure (BP) were recorded every minute. Hypotension was defined as a decrease greater than 30% in systolic BP from baseline.

Results: Early hypotension within 3 min after induction was experienced in 53.3%, while 36.7% had late hypotension within 15 min. Receiver operating characteristic (ROC) curves for PI as a predictive test of hypotension at 3 min showed good predictability of the baseline PI for early postinduction hypotension with area under receiver operating characteristic curve (AUC) 0.97, sensitivity 88%, specificity 93%, positive predictive value (PPV) 93%, negative predictive value (NPV) 87%, and a cutoff value of \leq 1.3. After intubation, the predictive value decreased, with PI > 0.91 to predict late hypotension with sensitivity 91%, specificity 58%, PPV 56%, NPV 92%, and AUC 0.66.

Conclusions: Pl could predict early hypotension following propofol induction in the geriatric population. So, it can be considered a valuable tool in monitoring this vulnerable population after induction of anesthesia.

1. Introduction

Hypotension is common during the time period postinduction of anesthesia and prior to surgical stimulation onset [1]. Predictors of hypotension following anesthetic induction at 0–10 min included the American Society of Anesthesiologists' Physical Status (ASA) III–V, age \geq 50 years, mean arterial pressure (MAP) with baseline <70 mmHg, increasing the fentanyl induction dosage and the usage of propofol for induction of anesthesia [2].

With the induction of anesthesia, elderly patients are inclined to sensitivity to volume status and have higher hemodynamic instability. Additionally, aging may diminish functional brain reserve and impair cerebral autoregulation, rendering the organ more susceptible to minute hemodynamic alterations [3,4].

The perfusion index (PI) assesses the pulsatility of peripheral blood. It is calculated as part of the processing

of the plethysmography waveforms using infrared wavelengths. It is a noninvasive and simple method for evaluating peripheral perfusion [5].

In intraoperative and critically ill patients, PI is beneficial for monitoring the level of anesthesia and responsiveness to fluid treatment [6,7] and predicting patient response during hypotensive anesthesia [8]. The PI predicted hypotension in adults following anesthesia induction with a baseline PI of <1.05 [9].

There is, however, no study that specifically examines its usage in patients older than 65 years postinduction of anesthesia using fentanyl and propofol as a multivariate risk factor for postinduction hypotension. So, in this study, the authors hypothesized that based on patient's preinduction systemic vascular resistance, a threshold point for PI that reliably predicts postinduction hypotension in the elderly may be determined.

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2. Methods

This observational prospective study was conducted in the general surgery theater at Kasr Al-Ainy Hospital between December 2020 and March 2022. This work was done to detect the capability of PI to predict postinduction hypotension following anesthetic induction with propofol and fentanyl in geriatric patients.

Thirty subjects were enrolled after approval from the Faculty of Medicine, Cairo University ethics committee (ID: N-12/2020) and after registration at clinicaltrials.gov (ID: NCT04682717). Written informed consents were used to document patients' consent to participate.

The inclusion criteria were patients aged >65 years, volume nonresponder patients (with inferior vena cava [IVC] diameter of >1.5 cm + collapsibility index < 50%), fasting for 6 h without food and 2 h without clear fluids, and ASA I to II having elective surgery while under the effect of general anesthesia.

The exclusion criteria were patients taking vasoactive medications, having hypertension, decompensated heart failure, hypoxia, renal failure, preoperative mean blood pressure (BP) (MAP) <70 mmHg, difficult airway, emergency surgeries, and body mass index \geq 30.

In the preparation room, after history taking and examination, IVC diameter was measured by ultrasound for assessment of volume status, and if the patient is a volume responder (with an IVC of <1.5 cm + collapsibility index > 50%), the patient was given 250 ml crystalloid over 10 min and was reexamined. This was repeated until the patient had at least just beyond an IVC diameter of >1.5 cm + collapsibility index < 50% for safety of the patients, as the aim of the study was not the ability of PI to predict fluid responsiveness but the ability of PI to predict hypotension induced by induction of general anesthesia as PI reflected changes in the vascular tone. After ensuring that the patients had the targeted IVC diameter and collapsibility index, the baseline BP (systolic BP [SBP], diastolic BP [DBP], and mean BP [MAP]) was measured in three readings from both arms with 2-min interval and their average was taken. PI is automatically calculated by Masimo pulse oximetry (Masimo Radical 7; Masimo Corp., Irvine, CA, USA); it is calculated by measuring the constant amount of light absorbed by nonpulsatile blood and tissues direct current (DC) and the variable amount of light absorbed by pulsating arterial inflow alternating current (AC) with the following equation: $PI = (AC/DC) \times 100\%$. The baseline PI from the index finger was documented.

On arriving the operation room, PI, noninvasive BP, pulse oximeter, and electrocardiography were connected, and their readings were recorded. The patients

had preoxygenation with 100% oxygen for 2 min before induction of anesthesia. Intravenous fentanyl at a dosage of 2 μ g/kg was delivered; propofol came after with a dose of 1–2 mg/kg at a slow rate of 10 mg every 5 s, dripped to loss of responsiveness to verbal communication; and atracurium at a dose of 0.5 mg/kg IV was injected. Postinduction parameters were documented every minute until intubation. For 4 min prior to tracheal intubation, 100% O₂ was used to ventilate the lungs with the proper sized endotracheal tube.

Anesthesia was maintained with oxygen along with isoflurane 1%, and end-tidal CO₂ was maintained at 35–40 mmHg. For 10 min postintubation, hemodynamic parameters were documented at 1-min intervals, and the surgery was not allowed to start except after this time. Hypotension was defined as a decrease in SBP of >30% of baseline value and was treated immediately by ephedrine sulfate 5 mg IV boluses or 10 mg IV boluses followed by 5 mg boluses in case of SBP of >50% of baseline value and titrate to effect every minute. Bradycardia was defined as a decrease in heart rate (HR) <50 beat/min, and atropine 0.6 mg IV boluses was used for the treatment. The recording anesthesiologist was blinded to the baseline values.

The incidence of hypotension was measured in two categories: 3 min after induction of anesthesia and prior to intubation (induction agent effect) and 15 min after induction and prior to skin incision (effect of intubation process and positive pressure ventilation), and then patients were divided into patients with early hypotension at 3 min after induction (H) and patients without early hypotension (Hout).

3. Outcomes

3.1. The primary outcome

A cutoff value of baseline PI below which at 3-min postinduction hypotension could be estimated.

3.2. Secondary outcomes

The relationship between the degree of hypotension and baseline PI, trend of PI and relation of change from baseline to degree of hypotension, negative and positive predictive values (PPVs) of baseline PI, incidence of hypotension at 10 min after intubation, number of patients needed ephedrine, and total ephedrine dose.

3.3. Sample size and statistical analysis

A minimum of 24 patients was calculated as the whole sample size (raised to 30 patients to compensate for the dropouts) using MedCalc software with type 1 error of 0.05, type 2 error of 0.2, and area under the ROC curve of 0.816 based on the result of a previous study taking the correlation of PI and alteration in the MAP post-propofol induction as the primary outcome [9].

SPSS version 20 was used to analyze the data (IBM Corporation, New York, 2014). Numbers and percentages were used to express qualitative data, while quantitative data were expressed as mean \pm standard deviation (SD). The normality of the PI distribution was tested (P = 0.001). A binary logistic regression was used to find independent hypotension predictors if the bivariate correlation was statistically significant. For assessing correlation between PI and all hemodynamic variables, Spearman's was employed; if univariate correlation was detected, linear regression was utilized to find independent predictability. ROC curves were built for baseline PI levels to predict hypotension (SBP 30% lower than baseline). Statistical significance was defined as a P < 0.05.

4. Results

From 42 patients enrolled in the study, only 30 patients completed the study, 6 patients did not complete the inclusion criteria, 4 patients refused to be consented, and 2 patients had regional anesthesia instead of general anesthesia. Demographic data are represented in Table 1. Type of surgeries included seven orthopedic trauma, seven laparoscopic, six spine, four joint arthroscopic, two ventriculoperitoneal shunt, one Endoscopic retrograde cholangeopancreatography (ERCP), one thyroidectomy, one hernioplasty, and one pituitary surgery.

Sixteen patients (53.3%) experienced early hypotension (at 3 min), while 11 patients (36.7%) had late hypotension (at 15 min) after induction. ROC curves were constructed for PI as a predictive test of hypotension at 3 min and 15 min after induction. It showed a good predictability of the baseline PI for early postinduction hypotension (Figure 1 and Table 2).

The degree of early hypotension from baseline had a mean percentage of 38.85% and 15.65% for H and Hout groups, respectively. It correlated significantly with the baseline PI with r = -0.363 and P value of 0.048. The relation of baseline PI with changes in HR, SBP, DBP, and MAP was only significant with SBP r =-0.418 and P value of 0.021. No patients required atropine. As regards ephedrine, the H group received 6.15 ± 3.63 mg, which showed a significant correlation with the baseline PI with r = -0.411 and P value of 0.02.

Trends of SBP, MAP, and PI in the first 3 minutes after induction in patients with (H) and without (Hout) early hypotension showed a significant difference between H and Hout groups. As regards the trends during 15 min after induction, HR, SBP, and PI showed significant difference between H and Hout groups (Figure 2)(Table 3). As regards correlation of trends of PI with HR, SBP, DBP, and MAP, there was an insignificant correlation between PI trends and the trends of the other parameters in all patients.

5. Discussion

This prospective observational research investigated the ability of baseline PI to predict postinduction hypotension in geriatrics. What distinguishes this study from other studies is that although PI was previously explored as a marker for response of patient after induction by propofol in adults, it was discovered that a baseline PI < 1.05 predicted the likelihood of hypotension, and no one was concerned about its use in hypotension in patients >65 years old postinduction of anesthesia with fentanyl and propofol as a population at multiple risks for postinduction hypotension and vulnerable to morbidity and mortality due to hypotension.

This study hypothesized that it is feasible to determine a cutoff value for PI that anticipates hypotension in these individuals. The findings of this work supported the hypothesis that the correlation of baseline PI was significant with changes in SBP, the degree of early hypotension, and the dose of ephedrine given. Also, ROC curves showed good predictability of the baseline PI as a predictive test of hypotension at 3 min for early postinduction hypotension with AUC 0.97, sensitivity 88%, specificity 93%, PPV 93%, negative predictive value (NPV) 87%, and a cutoff value of \leq 1.3. This result gives a valuable alarm to the anesthesiologist for the risky elderly patient to develop postinduction hypotension and hence to avoid the possible adverse consequences, and this is the new thing that this work adds to science. After intubation, PPV decreased.

The relationship between baseline PI and changes in HR, SBP, DBP, and MAP was significant only for SBP. Because PI is a derivative of vascular contractile state, stating hypotension as a baseline SBP function is likely more realistic, whereas absolute MAP does not take into consideration the patient's preoperative vasomotor tone [10]. The significance of the difference in correlation between the first 3 min and 15 min is most likely attributable to the hemodynamic impact of endotracheal intubation, which causes BP to stabilize, minimizing the risk of hypotension.

There was a statistically significant as regards SBP, MAP and PI difference between the two groups as regards the trends within the first 3 min after induction of anesthesia. There was an insignificant correlation between PI trends and the trends of the other parameters.

The incidence of early hypotension was 53.3%, while late hypotension occurred in 36.7% of patients as with the induction of anesthesia, older individuals are prone to higher hemodynamic instability and

	All patients (no. 30)	H group (no. 16)	Hout group (no. 14)	P	value
Age (years)	70.62 ± 6.42 (95%Cl: 68.33 to 72.91)	70.13 ± 7.00 (95%Cl: 66.70 to 73.55)	70.5 ± 5.88 (95%Cl: 67.42 to 73.58)	*	0.95
•				**	
				0.64	
ASA (I/II)	7/23	3/13	4/10)*	0.67
				**	
				0.73	
Sex (M/F)	19/11	10/6	9/5	*	0.73
				**	
				0.72	
SBP (mmHg)	161.15 ± 13.82 (95%Cl: 156.22 to 166.08)	154.06 ± 18.16 (95%Cl: 145.17 to 162.95)	157.86 ± 14.90 (95%Cl: 150.05 to 165.65)	*	0.44
				**	
				0.53	
DBP	85.54 ± 7.19 (95%Cl: 82.95 to 88.11)	85.63 ± 9.49 (95%Cl: 81.97 to 90.27)	$86.79 \pm 7.64 (95\%$ Cl: $82.79 \text{ to } 90.79)$	*	0.73
(mmHg)				**	
•				0.98	
MAP	$109.85 \pm 8.28 \ (95\% Cl: 106.87 \ to \ 112.81)$	104.13 ± 10.04 (95%Cl: 99.22 to 109.04)	113.79 ± 9.02 (95%Cl: 109.03 to 118.53)	*	0.05
(mmHg)				**	
5				0.25	
HR	76.15 ± 10.71 (95%Cl: 72.33 to 79.97)	83.63 ± 14.48 (95%Cl: 76.54 to 90.72)	77.5 ± 11.96 (95%Cl: 71.24 to 82.76))*	0.10
(beat/min)				**	
				0.92	
Ы	2.07 ± 2.78 (95%Cl: 1.07 to 3.07)	0.78 ± 0.40 (95%Cl: 0.59 to 0.97)	2.62 ± 2.38 (95%Cl: 1.38 to 3.86)	*	0.02
				**	
				0.06	

nioou pressure; mar, mean Data are presented as means ± standard deviation. 95% Cl. 95% confidence interval, numbers; M, males; F, females; ASA, the American Society of Anesthesiology; SBP, systolic blood pressure; DBP pressure; HR, heart rate; PI, perfusion index. *P value for H nout, ** P value for all patients and H, ***P value for all patients and Hout.



Figure 1. The AUC analysis for the ability of baseline PI to predict early postinduction hypotension.

sensitivity to volume status. This is related to increased sympathetic tone at rest and modified beta receptor sensitivity, diminished intravascular volume due to decreased thirst response to hypovolemia, preload dependency due to increased myocardial stiffness, increased arterial stiffness, decreased sinus node efficiency, and baroreceptor responsiveness, with the result of a marked reduction in cardiovascular reserve beside myocardial inhibitory and vasodilatory effects of anesthetics and other patients comorbidities. Aging may diminish functional brain reserve and impair cerebral autoregulation, making the organ more susceptible to minute hemodynamic changes. So, they are at increased risk of complications such as myocardial infarction, stroke, and delirium [3,4].

Propofol-induced hypotension is caused by its influence on the autonomic nervous system, which affects both parasympathetic and sympathetic components [11]. Propofol exerts a direct impact on the endothelium via endothelium-dependent and -independent mechanisms [12]. Both lead to vasodilation. If the patient has substantial peripheral vasoconstriction or a low PI, they are more likely to have hypotension. Likewise, if the patient already has a low vascular tone (vasodilated and partially compensated blood volume), as shown by a higher PI, hypotension is less likely, as demonstrated in recent research.

Pl is a fairly recent metric used in plethysmography waveform processing to estimate the pulsatility of blood in the extremities. Pl is a measure of systemic vascular resistance because it reveals microcirculation state, which is heavily innervated by sympathetic nerves. Though not in a linear way, the Pl value is inversely proportional to the vascular tone. As a result, vasodilatation indicating higher baseline Pl has been linked to BP reduction [10].

PI was investigated as a predictor for patient's vascular response in many situations. A study done by Mehandale and Rajasekhar [9] on 50 adults showed that with specificity 71%, sensitivity 93%, NPV 98%, and PPV 68%, a baseline PI of 1.05 anticipated hypotension occurrence 5 min post-propofol administration. The area under the

Table 2. The AUC analys	sis for the ability	y of baseline PI to	predict early a	and late p	postinduction hypotension.
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	AUC	Sensitivity	Specificity	PPV	NPV	Cutoff value
Early hypotension (no. 16)	0.97	88	93	93	87	≤1.3
Late hypotension (no. 11)	0.66	91	58	56	92	>0.91

AUC, area under receiver operating characteristic curve; CI, confidence interval; NPV, negative predictive value; PI, perfusion index; PPV, positive predictive value.



Figure 2. Trends of mean SBP and PI in patients with early hypotension (H) and patients without (Hout).

		F	P value
HR (beat/min)	First 3 min	4.11	0.11
	Overall 15 min	12.74	0.001
SBP (mmHg)	First 3 min	31.52	0.005
	Overall 15 min	6.51	0.016
DBP (mmHg)	First 3 min	4.79	0.094
	Overall 15 min	0.17	0.682
MAP (mmHg)	First 3 min	1.30	0.033
	Overall 15 min	4.18	0.050
PI	First 3 min	5.86	0.002
	Overall 15 min	48.21	< 0.0001

Table 3. Trends of heart rate, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure and perfusion index in patients with early hypotension (H) and patients without early hypotension (Hout).

Note: P < 0.05 is significant. F = variation between sample means/variation within the samples.

ROC curve was 0.816, with a *P* value of 0.001, which matched our findings. However, we focused on the older group because they are at a higher risk of hypotension.

A baseline PI of 0.82 in patients with acute renal injury predicted the hypotensive outcome to progressive fluid clearance by constant veno-venous hemofiltration in severely sick patients [13]. When circulatory volume was gradually lowered in healthy volunteers, PI gradually fell from 2.2 to 1.2 without BP change. When the PI reached 1.2, hypotension occurred, and a median value of 1.4 was regarded as normal for healthy adults [3]. In our study, under a PI threshold value of 1.3, ROC analysis predicted hypotension, which is similar to the results reported in the previous study.

Ahmed et al. investigated the predictability of PI for hypotension during deliberate hypotension in endoscopic sinus surgery. They identified a significant positive correlation between the baseline PI and both the time necessary to achieve the target MAP and the rate of nitroglycerin infusion. The baseline PI was the only predictor for requirements of nitroglycerin with an AUC 0.85 and a cutoff value of 2.8 [8].

In a multicenter investigation of propofol's hemodynamic effects on 25,000 individuals, hypotension was found much beyond 10 min after induction. More than 20% of hypotensive events occurred more than 10 min after introduction [14]. Our data support this, given the observation period was 15 min after induction, with a 36.7% incidence of hypotension. This disparity in incidence might be attributable to the study's geriatric group, which is more susceptible to postinduction hypotension.

6. Limitations of the study

Although the sample size was calculated using the area under the ROC curve of a similar study done on adults in general, it is essential to confirm the PI cutoff value for predictability in elderly patients

by testing a larger number of patients, as well as subcategories of patients such as hypertensive, obese, and other patients, in order to trust the results of the study for the use in clinical applications. The activity of the patients (ambulant or bedridden) was not targeted which may affect the hemodynamics; however, this did not affect the results as the authors targeted volume nonresponder patients while the PI value reflected the systemic vascular resistance. Furthermore, a comparison of the PI and the pleth variability index may have revealed that one index was better than the other. Due to the fact that the drug was dosed via titration, the amount of drug utilized was accurate to the level desired by each patient, allowing for more generalizable results; however, the total propofol dosage was not documented, which would have provided an indication of the entire quantity of drug necessary for an individual. More meticulous research utilizing monitoring of cardiac output, invasive BP monitoring, and dynamic hypovolemia indicators would be necessary to validate or contradict the findings of this research.

7. Conclusion

The correlation of baseline PI was significant with changes in SBP, the degree of early hypotension, and the dose of ephedrine given. Also, ROC curves for PI as a predictive test of hypotension at 3 min showed good predictability of the baseline PI for early postinduction hypotension; however, the predictive value decreased after intubation. So, it can be concluded that PI may be used to predict early hypotension in the elderly following propofol induction prior to endotracheal intubation.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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