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Original Research Article

Perfusion Index A Probable Indicator of Hypotension following Propofol Induction: A Prospective Observational Study

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Abstract

Background and Aims: Hypotension with the induction of propofol is a common problem. It is claimed that the perfusion index (PI) can predict hypotension after subarachnoid block. Our theory, which suggests that PI may predict hypotension after propofol induction, states that there is a cutoff value for the perfusion index below that hypotension becomes more likely.

Methods: 55 participants in this prospective, observational research with ASA physical status I & II underwent planned general anesthesia-based surgery. After induction of anaesthesia with titrated dose of propofol, all the baseline vital parameters and PI were evaluated every minute from baseline to 10 minutes after induction. Hypotension was characterized as a fall in mean arterial pressure (MAP) below 60 mm Hg or a reduction in systolic blood pressure (SBP) of over 30% from baseline.

Results: Within the first five minutes following induction, the incidence of hypotension utilizing SBP and MAP standards was 29% and 36%, respectively, whereas severe hypotension was 20%. Baseline PI 1.03 had a sensitivity of 87.5% & a specificity of 92.3% for predicting the occurrence of hypotension at 5 minutes. Area under the curve (AUC)) for the ROC curve was 0.913 with a 95% CI ranging from 0.806 to 0.972. (P <0.001).

Conclusion: Following propofol induction, hypotension can be predicted using the perfusion index.

Introduction

The most popular intravenous induction drug is propofol. Propofol's popularity is due to the fact that it possesses many of the qualities of the elusive optimal anaesthesia drug, including a quick onset of hypnosis and a speedy awakening with minimum excitation [1-4]. Hypotension is frequently linked to propofol induction [5]. Propofol's sympatholytic action, which is amplified in geriatrics, hypovolemic, and cardiac patients, results in hypotension and increases the risk of myocardial ischemia

[6]. In high risk cardiac patients, predicting hypotension during the pre-induction interval utilising non-invasive monitoring can reduce complications [7].

The perfusion index (PI), which is calculated using a pulse oximeter based on the quantity of Infrared light absorbed (940 nm) [8,9], is the proportion of pulsatile to non-pulsatile blood flow within peripheral vascular tissues. The perfusion index is a number that represents the proportion (AC/DC%) of the infrared light absorption of the pulsatile arterial flow signal (AC) to the non-pulsatile signal in the blood and tissue (DC). PI represents the state of the microcirculation, which is extensively innervated by sympathetic nerves and is consequently influenced by a variety of variables that might cause the microvasculature to contract or dilate. It is an indicator of Systems vascular resistance (SVR) [9].

Numerous studies [8,10] have employed the Perfusion index as a means of predicting of hypotension. Though not linearly, the value of PI is inversely correlated with vascular tone. Blood pressure (BP) decreases during spinal anaesthesia have been linked to vasodilatation indicating increased baseline PI [11]. The occurrence and extent of hypotension after subarachnoid block in pregnant women can be affected by the resting SVR. Similarly hypotension following propofol anaesthesia induction might be linked to the baseline SVR.

PI may be used to evaluate perfusion dynamics and is being studied as a non-invasive approach to determine the possibility of developing hypotension after inducing anaesthesia with propofol.

Material and Method

A sample size of 55 cases calculated at 95% confidence interval to predict hypotension following propofol induction on the basis of perfusion index.[3] After approval from institute ethical committee (no:1106/MC/EC/2021, Dated 01/12/2021) & CTRI registration (CTRI/2022/07/044383 Dated 27/07/22) all 55 patients with informed written consent with inclusion/exclusion criteria enrolled for study.

Inclusion criteria: Patients of either sex, between the ages of 18 and 60 of ASA grades I and II who are undergoing planned elective surgery under general anaesthesia after providing written informed permission.

Exclusion criteria: Patients who are either unwilling to get involved in the trial, have hypertension, are using a vasoactive medicine, are pregnant or with an anticipated difficult airway, or are allergic to propofol and its components.

Procedure: One day prior to surgery, pre-anaesthetic assessments were performed on all patients. Written consent was procured from each patient after being fully informed about the surgery and the study's methodology. When patients arrived at the operation table, a pulse oximeter, non-invasive blood pressure (NIBP), and ECG monitor was attached to the patient, and baseline measurements (perfusion index (PI), PR, BP, and SpO_2) were taken. With the help of an 18-gauge cannula, intravenous access was established, and lactated Ringer's solution injections started at a rate of 10 mL/kg/hour.

For 3 to 5 minutes, the patient received pre-oxygenation with 100% O_2. IV premedication followed by Fentanyl 2 ug/kg IV were given first, followed by IV propofol slowly administered at a rate of 10 mg in every 5 seconds, titrated to loss of verbal response. An intravenous succinylcholine dose of 1.5 mg/kg was given to aid tracheal intubation after bag and mask ventilation was confirmed. A consultant anaesthesiologist then intubated the patient's trachea using an appropriate-sized endotracheal tube.

After propofol induction, the parameters were kept track every minute. General

anesthesia was maintained with 50% N_2O in oxygen, sevoflurane/isoflurane, and iv atracurium loading (0.5mg/kg) and intermittent doses (0.1mg/kg). Up to 5 minutes after intubation, hemodynamic parameters were monitored at 1-min intervals. The incidence of hypotension was estimated in two sets: the first set, 5 minutes after anaesthesia induction (effect of the induction agent), and the second set, the first 5 minutes after intubation (impact of the induction procedure and endotracheal intubation). After the procedure, the patient was moved to the recovery room and postoperative vitals were noted.

Statistical Analysis

SPSS version 28.0 was used to evaluate the data after it had been gathered and calculated using Microsoft Excel 2013. For quantitative variables, the data were presented as the mean (standard deviation), and for qualitative factors, as percentages. The relationship between the baseline PI and the occurrence of hypotension was investigated using the point-biserial correlation. Binomial logistic regression was used to find independent prediction for forecasting hypotension if bivariate correlation was discovered to approach statistical significance. All haemodynamic variables were correlated with the PI using Spearman's, and if univariate connection was discovered, linear regression was performed to determine independent prediction. For values of baseline PI, receiver operating characteristic (ROC) curves have been generated to predict hypotension (hypotension: SBP < 30% under baseline, MAP <60 mmHg, and severe hypotension: MAP <55 mmHg). Statistics were considered significant at P <0.05.

Observations and Results:

All patients who participated in this prospective observational study were scheduled for different operative procedures under general anaesthesia; of them, 27 (49.09%) were male and 28 (50.91%) were female. The selected patients varied in age from 18 to 60 years, with 13 (23.6%) being under 30 and 42 (76.4%) being 30 years of age or older. A large percentage of the patients fell within the age range of 41 to 50. The average age was 39.75 ±12.78. The average weight was 63.49±11.29 kg.

Patient taken in operating room with consent from the patient, baseline vitals recorded for all 55 patients. Mean values of PI, SBP, DBP, MAP, HR and SPO_2 were 1.78±0.82, 126.75±19.25, 86.13±9.89, 100.07±12.65, 93.73±9.39, and 98.87±0.94, respectively.

After propofol induction until the fifth minute and after intubation until the tenth minute, the perfusion index was measured every minute along with other data. The perfusion index trend first increased until the fifth minute and subsequently decreased until the tenth minute, although the shift was not statistically significant (p=0.206). Up to the fifth minute after propofol induction, systolic blood pressure fell. However, following intubation, there was a modest rise in SBP. SBP dropped by 19.5% from the baseline at the tenth minute. At various times, the alteration in systolic BP was found to be statistically significant (p<0.001). Similar to SBP, the trend of change in diastolic and mean blood pressure fell following propofol induction until the fifth minute and then slightly increased after intubation. At the 10th minute, DBP and MAP had decreased by 21.43% and 24.85%, respectively, compared to their baseline values. At various time periods, the changes in DBP and MAP were statistically significant (p<0.001). Heart rate was elevated with propofol induction for initial 6 minutes, and it then slightly decreased following intubation. At the tenth minute, the HR increased by 6.30%. At various times, the heart rate changed in a statistically significant way (p<0.001). SpO2 was

98.87±0.94 at baseline, and there was no significant change over time.

Table 1: Mean value of various parameters at different time interval

Time	PI	SBP	DBP	MAP
Baseline	1.78	126.75	86.13	100.07
1min	2.00	118.98	83.04	93.85
2min	2.10	113.29	78.96	90.36
3min	2.20	107.69	73.33	85.00
4min	2.32	101.02	68.78	79.27
5min	2.43	93.76	65.07	73.20
6min	2.33	96.65	67.71	76.00
7min	2.22	99.07	68.91	77.45
8min	2.08	100.53	69.00	76.80
9min	1.97	101.56	68.25	76.67
10min	1.85	102.04	67.67	75.20

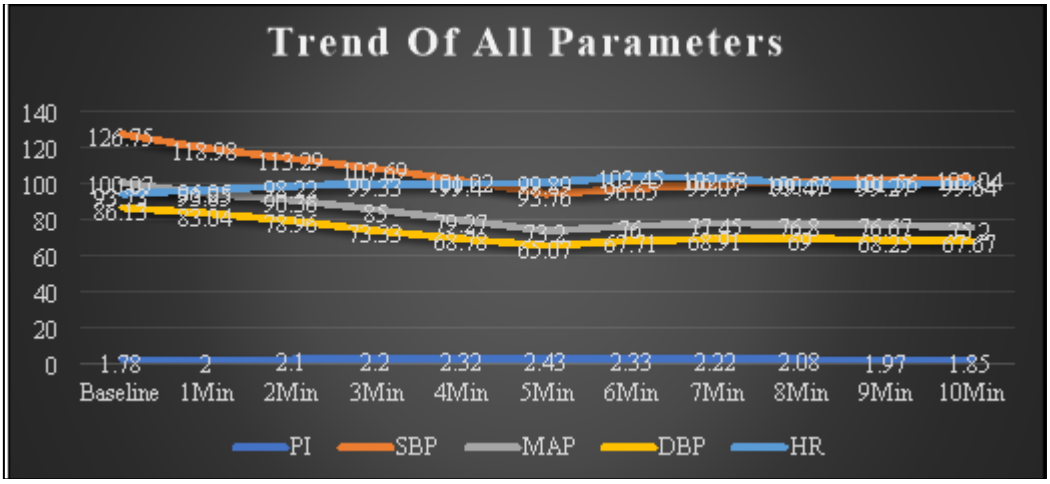

Figure 1: Trend of all parameters at different time interval

Using the spearman rank correlation coefficient (ρ), the relationship between the perfusion index and all the hemodynamic parameters such as SBP, DBP, MAP, and HR was calculated. For SBP, DBP, and MAP, respectively, the spearman correlation coefficient values were -0.448, -0.757, and -0.674. As the PI value was low and the relative blood pressure was high, the negative number indicates an inverse relationship between the two parameters. There was statistical significance in these relationships ($p<0.001$).

The correlation coefficient between pulse rate and PI was 0.052, which was not statistically significant ($p=0.207$).

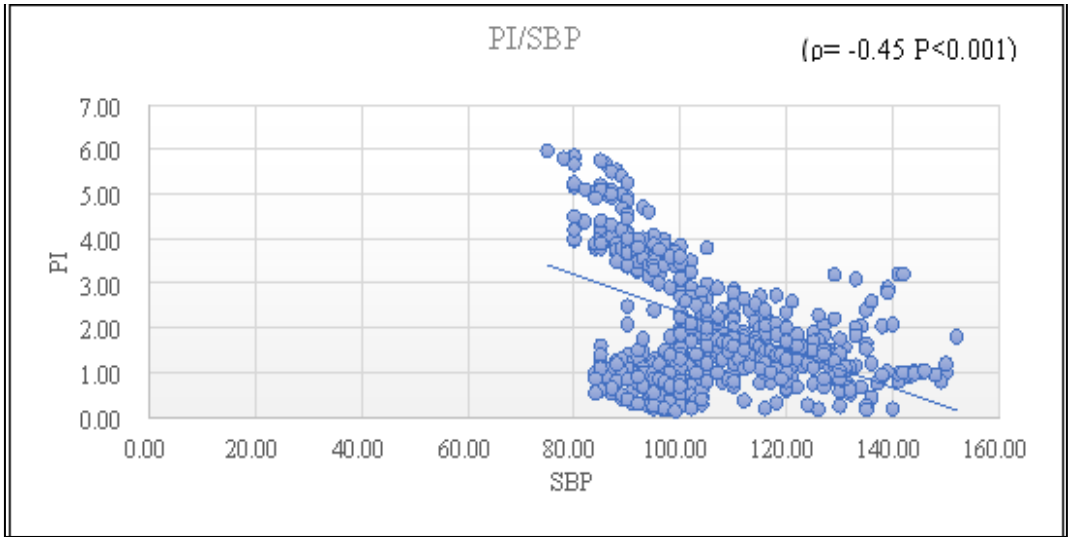

Figure 2: Spearman correlation between PI and SBP

With the aid of baseline perfusion index, regression analysis was carried out to predict hypotension following propofol induction. It was discovered that perfusion index significantly predicted the decrease in SBP ($p<0.001$) ($R2= 0.689$).

Within five and ten minutes after propofol induction, we estimated the incidence of hypotension. Within 5 minutes of the administration of propofol, 29% of patients exhibited hypotension according to SBP criteria, 36% according to MAP criteria, and 20% of patients suffered severe hypotension for which mephentermine 6mg was given as a vasopressor. Within 10 minutes of the first injection of propofol, 36% of subjects demonstrated hypotension as per SBP criteria, 45% as per MAP criteria, and 23% exhibited severe hypotension.

Point bi-serial correlation coefficient was used to calculate the correlation between baseline perfusion index and hypotension according to various criteria, and it was found that there was a significant correlation between baseline perfusion index and hypotension according to SBP criteria both within 5 minutes (rpb -0.594, $p<0.001$) and 10 minutes (rpb -0.383, $p<0.05$) of propofol induction. Negative values indicate that the greater the likelihood of hypotension according to SBP criterion, the lower the perfusion index value. There was no connection between hypotension as defined by MAP criteria and severe hypotension & baseline perfusion index.

Table 2: Point biserial correlation

	Within 5 min			Within 10 min		
	SBP criteria	**MAP criteria**	**Severe hypotension**	**SBP criteria**	**MAP criteria**	**Severe hypotension**
r_{pb}	-0.5938	0.181	0.0604	-0.3831	0.1094	0.0583
P value	<0.001	0.186	0.6613	0.0039	0.4268	0.6721

In order to anticipate hypotension within 5 and 10 minutes based on SBP criteria, PI ROC curves were generated. Area under the ROC curve (AUC) was 0.913 at 5 min, with a 95% confidence interval (CI) ranging from 0.806 to 0.972 and a P value of < 0.001 [Figure 3]. With a sensitivity of 87.5% and a specificity of 92.3%, the baseline PI of 1.03 anticipated a likelihood of intraoperative hypotension at 5 min following propofol induction.

The AUC reached 0.764, 95% CI (0.631 to 0.868), P <0.001 at 10 min [Figure 4].

With sensitivity 70% and specificity 91.43%, baseline PI of 1.03 predicted any development of intraoperative hypotension at 10 min.

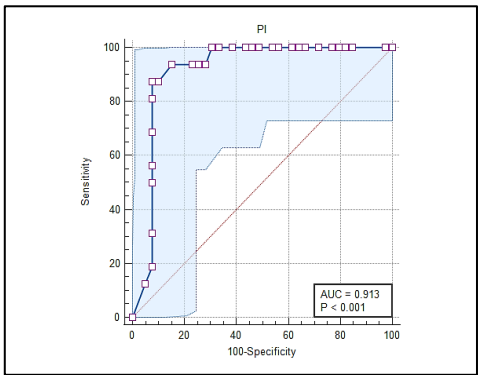

Fig: 3 ROC curve within 5 min

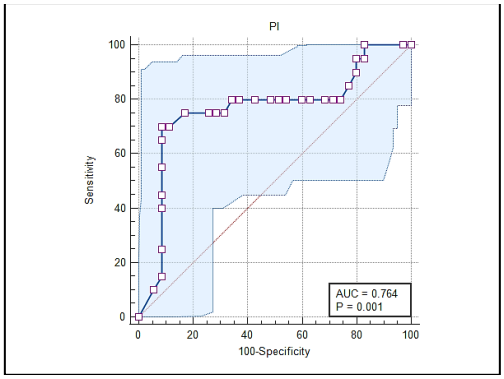

Fig: 4 ROC curve within 10 min

Discussion

We hypothesized in the current study that baseline PI would be helpful in predicting hypotension after propofol induction and investigated for a baseline PI cut off value that predicted hypotension. This hypothesis is supported by our data, which show a statistically significant relationship between PI and the occurrence of hypotension as defined by SBP criteria. The incidence of hypotension was greater when the PI was less than 1.03. SBP, DBP, and MAP were negatively correlated with PI but there was no relationship between PI and HR. With the aid of baseline perfusion index, regression analysis was carried out to predict hypotension following propofol induction. It was discovered that perfusion index strongly predicted the decrease in SBP (R2= 0.689) ($p<0.001$). Incidence of hypotension as measured by SBP was strongly correlated with baseline PI value both within 5 minutes (rpb -0.594, $p<0.001$) and 10 minutes (rpb -0.383, $p<0.05$) after propofol induction. This might be as a result of the emergence of the PI variable, which represents the ratio of the pulsatile component (an indication of SBP) of the plethysmographic waveform to the non-pulsatile component. The nonlinear connection between PI and SBP can be used to explain weak linear association[9]. The baseline PI scatter plot displays a bell-shaped curve, with extremely high as well as low SBP values being related with low PI. Low PI and low SBP are simply explained by the decrease of pulsatility that results from hypovolemia and vasopressor usage.[16] High SBP in the presence of low PI, however, may result from concurrent rises in the non-pulsatile component. Although baseline PI did not meet absolute MAP threshold for hypotension, it significantly correlated negatively with SBP criteria. Since PI is a derivation of vascular contractile status and absolute MAP does not simply take into account the patient's pre-operative vasomotor tone, describing hypotension as a consequence of relative drop in baseline SBP is likely more natural in this situation. Other researchers discovered a negative correlation between low baseline PI and higher MAP decreases following propofol induction, and they concluded that this correlation was causal.[14]

Low PI levels may be caused by compensatory vasoconstriction brought on by relative hypovolemia, and vasodilatation due to propofol may result in hypotension in such individuals. The degree of the correlation between the first 5 and 10 min is likely different because of the haemodynamic impact caused by

endotracheal intubation, which causes BP to normalise and lowers the likelihood of hypotension. The ROC analysis supported these findings, demonstrating that a baseline PI cut-off of 1.03 was strongly predictive of SBP-based hypotension after propofol (5 min) with an AUC of 0.913 and a 95% confidence interval (CI) of 0.806 to 0.972, P< 0.001 [Figure 3]. and 87.5% sensitivity and 92.3% specificity. Following intubation, the predictive power decreased, with PI of 1.03 indicating hypotension with sensitivity of 70%, specificity of 91.43%, and AUC of 0.764 with a 95% confidence interval of (0.631-0.868), P< 0.001 [Figure 4]. Similar to Mehandale et al.[15], this will be useful in clinical circumstances to rule out the possibility of hypotension occurring 5 minutes after propofol induces anaesthesia.

Following spinal anaesthesia, hypotension in pregnant individuals with a baseline PI >3.5 was predicted. The association between PI and vascular sympathetic tone can be used to explain this finding that is in contrast to ours [11]. In contrast to our work, where the lower PI was likely suggestive of hypovolemia and compensatory vasoconstriction, the high PI was caused by an overall low sympathetic tone, and post-spinal sympathetic block led to hypotension. As a result of compensatory vasoconstriction in non-anesthetized dermatomes, they detected a decrease in PI after spinal anaesthesia during times of hypotension.[11] Similar findings to ours have been reported for critically sick patients with acute renal damage whose baseline PI was less than 0.82 and who had hypotension in response to gradual fluid removal by continuous veno-venous hemofiltration.[10], but when circulatory volume gradually decreased in healthy volunteers, a progressive drop in PI from 2.2 to 1.2 was seen without any change in blood pressure. The authors stated that a median value of 1.4 as normality for healthy people when hypotension developed after PI settled around 1.2.[9] In our investigation, ROC analysis predicted hypotension under a threshold value of 1.03, which is comparable to the results from Mehandale et al. [15] and Van Genderen et al.[9], but the cut-off value is larger than that reported to anticipate hypotension during fluid removal by continuous venovenous hemofiltration in critically ill patients. These victims suffered from acute renal damage, a serious condition.[10] Action on the autonomic nervous system, affecting both the sympathetic and parasympathetic components, is a key component of the process underlying propofol-induced hypotension. Also supported by research are the direct effects of propofol via endothelium-dependent and -independent mechanisms.[18] Vasodilatation is the result of both. The likelihood of the patient experiencing hypotension increases if they have severe peripheral vasoconstriction due to hypovolemia, which leads to low PI. The results of our study also show that the likelihood of hypotension is reduced in patients who already have lower vascular tone (vasodilation and comparatively compensated blood volume), as shown by greater PI.

Propofol usage in individuals with high PI is probably less likely to result in hemodynamic challenges than it would be in those with PI <1.03. Even though the frequency of hypotension was greater during this time, the predictability/non-predictability of hypotension by PI was not as precise when the entire duration of induction and intubation has been taken into account.

Hypotension might develop far past 10 minutes after induction, according to a multicenter investigation of 25000 individuals' hemodynamic responses to propofol.[12] Ten minutes after induction, more over 20% of hypotensive events took place. Our results support this because the observation period extended up to 10 min after induction, highlighting the need of

maintaining awareness well into the maintenance phase. Despite this, there is evidence correlating post-induction hypotension to an increased risk of post-operative morbidity and mortality.[13] Therefore, it is essential to predict, avoid, and effectively treat any haemodynamic instability, particularly hypotension, in order to provide the best possible patient outcome following surgery. Propofol induction is not likely to lead to in hypotension if the PI is larger than 1.03. As a result, it is appropriate to say that PI relies on SBP and that there is a bimodal relationship between the two (low PI has been associated with both high as well as low SBP).

The current study has sufficient power to detect the changes emphasized, therefore more research using a bigger sample size is probably not necessary. With the knowledge we now have about baseline PI and the mechanism of hypotension brought on by propofol induction, we are able to appropriately explain the link between fluctuations in BP and baseline PI. We rapidly treated severe hypotension (MAP <55 mmHg) since it has been shown that this type of hypotension, even if just momentary, can significantly impair organ function.[19] For tissue perfusion, a MAP of 70 mmHg or higher is regarded as normal. Earlier, hypotension was defined as a MAP <60 mmHg.[12] Similarly, others categorized hypotension as MAP <60 mmHg and tackled it whenever it was below 55 mmHg.[20] Even if a MAP falls below 55 mmHg for a brief period of time, it is known to have detrimental consequences. As a result, we used the same techniques to conduct our research.

Limitation of the Study

The absence of direct evidence for the hypothesized reasons, which form the basis of this study, limits its scope. The total dose of propofol, which would have provided information on the entire quantity of medication needed by the individual, was not reported. To validate or disprove the theories and further improve our knowledge in this area, a more thorough investigation involving cardiac output monitoring, invasive blood pressure monitoring, and dynamic markers of hypovolemia would be necessary.

Conclusion

As a result, we concluded from the aforementioned prospective observational research that perfusion index might forecast hypotension after propofol induction. PI has a very high sensitivity for predicting hypotension after propofol induction, particularly before endotracheal intubation.

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