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# Plethysmographic variability index as a predictor of propofol-induced hypotension: A prospective observational study

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## ABSTRACT

**Background:** Propofol as an intravenous (IV) induction agent frequently causes significant hypotension and requires monitoring and prompt intervention. The plethysmographic waveform, obtained from a pulse oximeter, relies on two components of light absorption-red and infrared, representing changes in blood volume and cardiovascular status of the patient. **Aims and Objectives:** The current study aimed to obtain a baseline value and positive predictive value (PPV) of plethysmographic variability index (PVI) to predict hypotension and also compare the PPV of PVI and perfusion index (PI) to predict hypotension induced by propofol. **Materials and Methods:** Seventy patients posted for elective surgery were first given IV crystalloids 2 h before surgery and then induced with propofol IV. Hemodynamic parameters, PI and PVI were recorded from baseline until 3 min post-intubation. Hypotension was defined as a fall in systolic blood pressure >30% or mean arterial pressure (MAP) <60 mmHg. Patients were then grouped into those who developed hypotension (Group H) and those who did not (Group NH). Statistical analysis of MAP, PVI, and PI was done. Receiver operating characteristic (ROC) curves were plotted and analyzed. The PPV of PVI and PI was calculated and compared. **Results:** Hypotension occurred in 56 patients. The mean baseline MAP was lower in Group H (91.3±10.54 mmHg vs. 99.93±3.36 mmHg). The fall in MAP was highest at 3 min post-induction (Group H 59.38±7.09, Group NH 79.36±8.05). The difference in baseline PVI was not statistically significant (Group H 15.59±3.67, Group NH 15.43±5.65). PVI peaked in Group H when MAP was minimum (59.38±7.09 mmHg) at 3 min post-induction. The difference in baseline PI was not significant at any time point (Group H 1.13±1.02, Group NH 0.92±0.47) Area under the ROC curve of 0.534 for PVI and 0.559 for PI were not statistically significant hence showing no correlation between baseline PVI and PI and propofol-induced hypotension. **Conclusion:** Baseline PVI and PI can serve as screening tools and not diagnostic tools for predicting hypotension. Baseline PVI ≥19 is more accurate to predict post-induction hypotension than the values mentioned in previous studies.

**Key words:** Plethysmographic variability index; Perfusion index; Hypotension; Propofol

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## INTRODUCTION

Propofol as an intravenous (IV) induction agent frequently causes significant hypotension and requires monitoring and prompt intervention. It results due to a combination of factors that include drug-induced cardiovascular depression, fall in systemic vascular resistance (SVR) with profound vasodilation, and

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decreased pre-operative volume status of patients due to nil oral intake.

Various static and dynamic techniques are used to predict and determine fluid responsiveness in a patient. These include monitoring central venous pressure, pulmonary capillary wedge pressure, left ventricular end-diastolic area (LVEDA), variations in arterial pulse pressure (ΔPP), vena cava diameter, pulmonary artery occlusion pressure, right atrial pressure, right ventricular end-diastolic volume, stroke volume variation, and aortic velocity time integral.[1] These indicators rely on cardiopulmonary interactions in mechanically ventilated patients and have consistently been shown to be good predictors of fluid responsiveness; however, they need expertise and resources.

Goal-directed fluid therapy decreases overall morbidity and mortality of patients coming for surgery. The plethysmographic waveform, obtained from a pulse oximeter, relies on two components of light absorption-red and infrared, representing changes in blood volume and cardiovascular status of the patient. It is characterized by two waveforms: A fast one, which results from stroke volume, and a second slow frequency waveform which is synchronous with respiration. The plethysmographic variability index (PVI) establishes the maximum and minimum plethysmographic waveform amplitudes and computes the difference of the two, expressed as percentage. PVI ranges from 0 to 100. Pre-load dependence, intravascular volume deficit, high SVR, and fluid responsiveness have been linked to greater plethysmographic waveform variability. In healthy individuals, there exists a correlation between pre-anesthetic PVI and hypotension following induction with propofol.[2] A higher PVI is associated with a greater fall in mean arterial pressure (MAP). Hence, an early prediction of intraoperative hypotension and treatment can improve perioperative outcomes.[3]

Perfusion Index (PI) is the ratio between light absorbed by pulsatile blood flow to light absorbed by non pulsatile blood flow in peripheral extremities. Consequently, PI reflects the amplitude of the plethysmographic waveform and is a measure of SVR. The PVI reflects the degree of change in PI caused by breathing over atleast one respiratory cycle.[4] PI is influenced by cardiac output and the balance of sympathetic and parasympathetic nervous systems. When a sympathetic stimulation occurs, the PI decreases. Thus, it may be used as an indicator of the severity of shock. Low PI is linked to poor outcomes, especially in critically ill patients. An increase in PI after a passive leg-raising test or a fluid bolus may be used as an important guide in directing goal-based fluid therapy.

### Aims and objectives

We aimed to obtain baseline cutoff values and positive predictive values (PPV) of PVI for predicting hypotension following induction of general anesthesia using propofol. We also compared the PPV of PVI and PI as a predictor of hypotension..

## MATERIALS AND METHODS

This study was done at a tertiary hospital after obtaining clearance from the Institutional Ethics Committee and obtaining CTRI registration (CTRI/2021/05/033645). Patients aged 20–59 years, of ASA-PS I and II, posted for elective surgery under general anesthesia, were included in the study. The sample size was calculated by two sided regression method.[5] Required sample size was 70 with correlation coefficient of −0.42, alpha error 1%, and power (1-β) of 90 %. Patients were explained the procedure along with a subject information sheet and written informed consent was obtained. Patients with Hypertension, Peripheral Vascular Disease, Autonomic Neuropathy, Chronic Kidney Disease, Chronic Alcohol Abuse, or a Difficult Airway were excluded from the study.

As per hospital protocols, all fasting patients were started on IV crystalloids at a rate of 1–2 mL/kg/h, 2 h before surgery. In the operating room, standard monitors including electrocardiogram, non-invasive blood pressure, end-tidal carbon dioxide, and pulse oximeter were connected. Baseline hemodynamic parameters such as heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and MAP were noted before induction. PVI and PI were recorded using the Masimo Radical-7 Pulse Oximeter by an anesthesiologist who was not monitoring the hemodynamic parameters of the patient.

Ringer lactate infusion at a rate of 10 mL/kg/h was started through a wide-bore IV cannula. Patients were pre-oxygenated with 100% oxygen and administered IV. Glycopyrrolate 5 mcg/kg, IV Ondansetron 0.15 mg/kg, IV Midazolam 0.03 mg/kg, and IV Fentanyl 2 mcg/kg as premedication. Propofol was given slowly at a rate of 10 mg every 10 s until verbal response to command was lost. Bag and mask ventilation was done and IV Atracurium 0.5 mg/kg was administered. After 3 min, the patient was intubated with an appropriately sized endotracheal tube by direct laryngoscopy. HR, SBP, DBP, MAP, PVI and PI values were noted every minute until 3 min post-intubation. Maintenance of anesthesia was done with isoflurane, oxygen, air, and intermittent doses of muscle relaxant.

Hypotension was defined as drop in SBP >30% of baseline or absolute MAP <60 mmHg.[2] Any decrease in MAP

<55 mmHg was treated with rapid IV fluid administration (10 mL/kg/h) or Ephedrine 6 mg IV boluses. Bradycardia was defined as HR <50 bpm or a fall of more than 30% below baseline value, whichever was lower, and was treated with Atropine 0.6 mg iv boluses. Hemodynamic parameters were noted at defined intervals.

### Statistical analysis of data

After data collection, patients were grouped into those who developed hypotension (Group H) intraoperatively and those who did not (Group NH) as described in Figure 1.

Descriptive and inferential statistical analysis was done, and data were analyzed using Statistical Package for the Social Science (SPSS) version 22 software (IBM SPSS Statistics, Somers NY, USA, and R environment version 3.2.2) and the R program.[6,7] Significance was assessed at 5% level of significance. Student t-test was used for comparison between hypotensive and non-hypotensive patients. Leven's test was performed to assess the homogeneity of variance. The Chi-square and Fisher Exact test were used to find the significance of study parameters on a categorical scale between the two groups for qualitative data analysis.

Validity of screening was plotted by the receiver operating characteristic (ROC) curve. Area under ROC curve (AUROC) was plotted for determining the best cutoff, sensitivity, specificity, PPV, and negative predictive value (NPV). The cutoff values of PVI and PI in correlation to hypotension were estimated by the Youden index score. P-value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

## RESULTS

The demographics were comparable between both hypotensive and non-hypotensive groups as noted in Table 1. Out of the 70 patients, 80% (56 patients) had hypotension with a fall in MAP of >30% from baseline or MAP <60 mmHg. The mean baseline MAP was different in both groups as seen in Table 2. It was found to be 91.3±10.54 mmHg in hypotensive patients (group H) and was 99.93±3.36 mmHg in the non-hypotensive group (group NH). MAP decreased from time of induction and the fall in MAP was highest at 3 min post-induction in both the groups (group H-59.38±7.09, group NH-79.36±8.05). Baseline SBP and DBP were also higher in group NH and followed the same pattern as MAP during post-induction period also.

Baseline PVI was noted to be 15.59±3.67 in Group H, whereas in Group NH it was 15.43±5.65. The difference

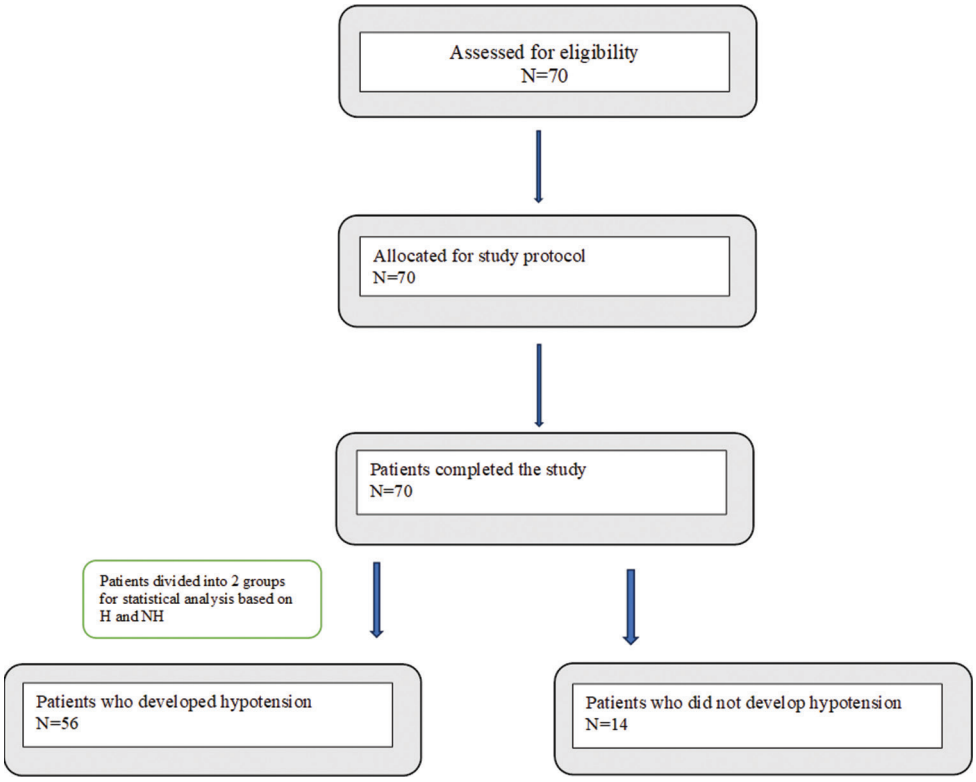


**Figure 1:** CONSORT diagram

in the baseline PVI in both the groups was not statistically significant as noted in Table 3. In Group H, PVI gradually increased after induction with propofol and rose to 19.5±3.12 at 3 min post-induction, whereas the change in PVI was not significant in NH patients at that time point also. PVI peaked at nadir of MAP in Group H where MAP was found to be minimum (59.38±7.09 mmHg) at 3 min post-induction.

Baseline PI was 1.13±1.02 in Group H, whereas in Group NH it was 0.92±0.47 as seen in Table 4. The difference in baseline PI between the groups was not significant at any time point (P=0.468). PI increased gradually post-propofol induction and peaked at 3 min post-induction in both the groups, that is, 2.35±1.4 in Group H and 2.4±1.22 in Group NH.

Correlational analysis between PI and PVI in both H and NH group at different time intervals showed that in Group H there was a major distortion in relationship between PI and PVI, whereas in Group NH the distortion was noted only at 3 min post-intubation as seen in Table 5.

ROC curves were plotted to show the validity of baseline PVI in predicting hypotension and the results showed no significant correlation between baseline PVI and post-induction hypotension as noted in Figure 2. Area under the ROC curve was 0.534 and was not statistically significant. PVI ≥15 had a sensitivity of 87.50% and a specificity of 42.86%. The PPV was noted to be 51.53% and NPV 77.41%. ROC curves were plotted again at peak hypotension at 3 min post-induction and PVI at that point was found to be 19.5±3.12 in Group H as noted in Figure 3. It showed a sensitivity of 92.66%, PPV of 92.69%, NPV of 89.8% and AUROC was 0.732.

Similarly, ROC curves were plotted to study the validity of baseline PI <0.67 in predicting propofol-induced hypotension as shown in Figure 2. AUROC at baseline PI was 0.559 with sensitivity of 82.14% and specificity of 42.86. The PPV was 29.91% and the NPV was 70.58%. AUROC results showed no correlation between baseline PI and propofol-induced hypotension. AUROC was statistically not significant even at 3 min post-induction.

**Table 1: Demographics**

| Demographic variables | Hypotensive group-H (%) | Non hypotensive group-NH (%) | P-value |
|---|---|---|---|
| Age (years) | | | |
| 21–30 | 12 (21.4) | 5 (35.7) | 0.334 |
| 31–40 | 15 (26.8) | 3 (21.4) | Not significant |
| >40 | 29 (51.8) | 6 (42.9) | |
| Total | 56 (100) | 14 (100) | |
| Mean±SD | 40.29±10.51 | 37.14±11.97 | |
| Gender | | | |
| Female | 28 (50) | 6 (42.9) | 0.632 |
| Male | 28 (50) | 8 (57.1) | Not significant |
| Total | 56 (100) | 14 (100) | |
| ASA | | | |
| I | 38 (67.9) | 10 (71.4) | 0.797 |
| II | 18 (32.1) | 4 (28.6) | Not significant |
| Total | 56 (100) | 14 (100) | |

**Table 2: MAP between the two groups**

| MAP (mm Hg) | Hypotensive group-H | Non-hypotensive group-NH | P-value |
|---|---|---|---|
| Baseline | 91.3±10.55 | 99.93±3.36 | 0.004** |
| Post-premedication | 91.18±9.55 | 98.14±7.12 | 0.013* |
| Post-induction-1 min | 73.73±9.7 | 89.29±6.89 | <0.001** |
| Post-induction-2 min | 65.32±11.65 | 83.57±7.65 | <0.001** |
| Post-induction-3 min | 59.38±7.09 | 79.36±8.05 | <0.001** |
| Pre-intubation | 60.05±6.64 | 79.71±9.29 | <0.001** |
| Post-intubation-1 min | 82.66±13.53 | 95.93±13.53 | 0.002** |
| Post-intubation-2 min | 88.57±12.16 | 93.57±17.02 | 0.210 |
| Post-intubation-3 min | 90.14±11.95 | 89.07±12.21 | 0.766 |

MAP: Mean arterial pressure, * Moderately significant (P value: p <= 0.05) ; ** Strongly significant (P value: p <= 0.01)

**Table 3: PVI between the groups at different time points**

| PVI | Hypotensive group-H | Non-hypotensive group-NH | P-value |
|---|---|---|---|
| Baseline | 15.59±3.67 | 15.43±5.65 | 0.897 |
| Post-premedication | 16.29±3.38 | 15±3.28 | 0.205 |
| Post-induction-1 min | 17.52±3.45 | 15.71±4.51 | 0.105 |
| Post-induction-2 min | 18.73±3.14 | 16.5±4.35 | 0.032* |
| Post-induction-3 min | 19.5±3.12 | 16.29±4.29 | 0.002** |
| Pre-intubation | 20.27±3.42 | 15.93±4.57 | <0.001** |
| Post-intubation-1 min | 20.45±4.09 | 17.36±4.63 | 0.016* |
| Post-intubation-2 min | 21.11±4.33 | 17.14±4.47 | 0.003** |
| Post-intubation-3 min | 21.25±4.14 | 18.29±5.31 | 0.027* |

PVI: Plethysmographic variability index, * Moderately significant (P value: p <= 0.05); ** Strongly significant (P value: p <= 0.01)

**Table 4: Perfusion index between the groups at different time points**

| PI | Hypotensive group-H | Non-hypotensive group-NH | P-value |
|---|---|---|---|
| Baseline | 1.13 ± 1.02 | 0.92 ± 0.47 | 0.468 |
| Post-premedication | 1.32 ± 1.42 | 1.01 ± 0.6 | 0.426 |
| Post-induction-1 min | 1.81 ± 1.46 | 1.61 ± 0.97 | 0.626 |
| Post-induction-2 min | 2.06 ± 1.44 | 1.88 ± 1.11 | 0.665 |
| Post-induction-3 min | 2.35 ± 1.4 | 2.4 ± 1.22 | 0.888 |
| Pre-intubation | 2.53 ± 1.2 | 2.29 ± 1 | 0.501 |
| Post-intubation-1 min | 2.98 ± 1.73 | 2.87 ± 1.33 | 0.835 |
| Post-intubation-2 min | 3.23 ± 1.82 | 3.11 ± 1.44 | 0.825 |
| Post-intubation-3 min | 3.47 ± 2.01 | 3.65 ± 1.92 | 0.761 |

PI: Perfusion index

**Table 5: ROC analysis**

| Variables | ROC results to predict hypotension | | | | Cut-off | AUROC | SE | P-value |
|---|---|---|---|---|---|---|---|---|
| | Sensitivity | Specificity | PPV | NPV | | | | |
| Baseline | | | | | | | | |
| PVI | 87.50 | 42.86 | 51.53 | 77.41 | ≥15 | 0.534 | 0.110 | 0.754 |
| PI | 82.14 | 42.86 | 29.91 | 70.58 | <0.67 | 0.559 | 0.097 | 0.543 |
| 3 min post-induction | | | | | | | | |
| PVI | 92.66 | 71.43 | 92.69 | 89.8 | >15 | 0.732 | 0.105 | 0.0269* |
| PI | 78.57 | 35.71 | 71.27 | 62.42 | ≤2.9 | 0.538 | 0.093 | 0.6857 |

ROC: Receiver operating characteristic, PVI: Plethysmographic variability index, PI: Perfusion index, PPV: Positive predictive value, SE: Standard error, * p <= 0.05, NPV: Negative predictive value, AUROC: Area under receiver operating characteristic. * Moderately significant ( P value: p <= 0.05) ; **Strongly significant (P value: p <= 0.01)

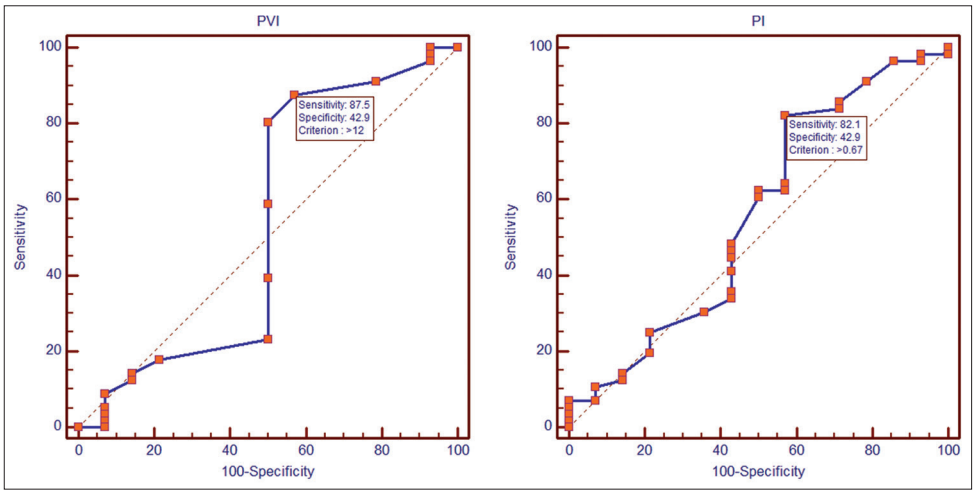


**Figure 2:** ROC at baseline

On comparison of baseline PVI >15 and baseline PI <0.68 in predicting hypotension, PPV of PVI is better than PI though both are not good tests to predict propofol-induced hypotension. Accuracy of tests was same for both, whereas at 3 min post-induction PVI was correlating well with a drop in MAP.

## DISCUSSION

Propofol as an IV anesthetic agent frequently causes significant hypotension. The severity of hypotension depends on the patient's intravascular volume status and SVR. We hypothesized that pre-induction baseline PI and PVI can be useful in predicting propofol-induced hypotension and PVI is a better predictor than PI. There is a huge variation in baseline cut-off values for PVI and PI defined in various studies. Hence, this study was done to find the closest possible baseline cut-off value of PI and PVI to predict propofol-induced hypotension.

In our study, we found a statistically significant drop in MAP, SBP, and DBP after induction with propofol in 80% of our patients. Baseline SBP, DBP and MAP were also lower in these patients. Out of 56 patients that developed

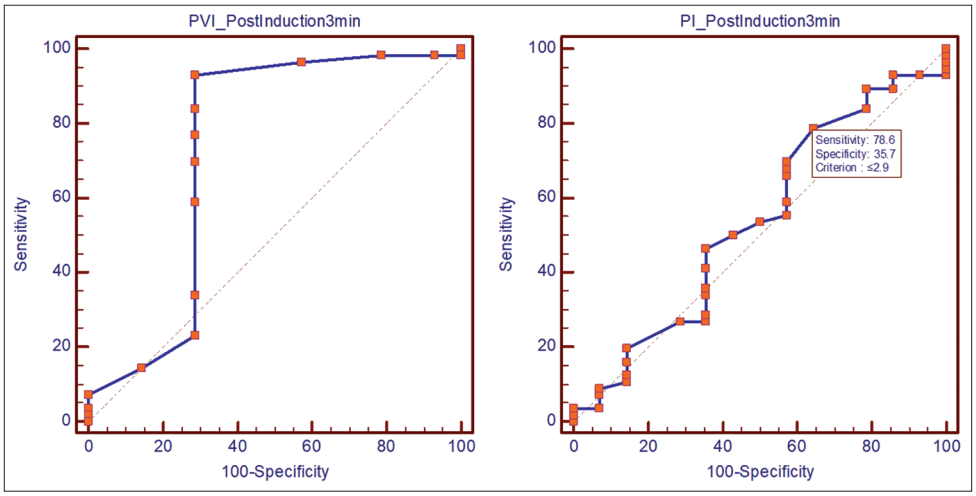


**Figure 3:** ROC at 3 min post-induction

hypotension, 23 patients (41.07%) had PVI >15 and 39 of them (69.6%) had PI <1.05. Out of the 33 patients that did not develop hypotension 50% of them had PVI >15 and 50% had PI <1.05. These baseline cutoff values of PVI and PI were taken from previous studies. For statistical analysis, we divided the patients into two groups-those who developed hypotension (Group H) and those who did not (Group NH).

We found a significant drop in SBP, DBP, and MAP after propofol induction but there was no significant change in HR. Mehendale and Rajasekhar[2] as well as Thirunelli and Nanjundaswamy[8] also found similar results, noting a significant drop in blood pressure (BP) but no change in HR. Abdelhamid et al.,[9] in their study found that 48.4% of patients developed hypotension following propofol induction though they defined hypotension as a drop in MAP 75% from baseline. In our study, 80% of patients had hypotension, the difference probably being due to our different definitions of hypotension. Yuksek[3] defined hypotension as a drop of ≥20% in SBP in geriatric patients undergoing laparoscopic surgeries. They found only 25% developed significant hypotension. Their study group was different from ours with respect to age.

Several studies have been done to predict hypotension induced by subarachnoid blocks by pre-spinal PI and PVI.[10,11] The cause of hypotension in spinal anesthesia is sympathetic blockade, whereas in general anesthesia it is due to the cardio-depressant effects of the IV anesthetic agents. Paul et al.,[11] found PI values ≥2.5 at baseline and ≥4.5 at 1 min had a greater incidence of hypotension while our cutoffs were lower (baseline PI 1.13±1.02 and at 1 min post-induction 1.81±1.46).

There was a correlation between pre-induction BP readings and a drop in BP post-induction. In our study, we found that baseline pre-induction SBP, DBP and MAP were lower in patients who developed hypotension than in those who did not as noted in Table 2. Our results differed from Abdelhamid et al.,[9] and Yuksek[3] who could not find significant differences in baseline hemodynamic parameters between hypotensive and non-hypotensive patients.

Mean value of baseline PVI was 15.59±3.67 in Group H in our study compared to 15.43±5.65 in Group NH and the difference was not significant. Baseline PI values were similarly not significant (1.13±1.02 in H group and 0.92±0.47 in NH group).

In our study, we found that baseline PVI was similar in both groups. We assume that this is due to non-uniformity in pre-operative fasting hours, age groups studied, and pre-operative IV fluid administration. It also differed due to different threshold values of BP in defining hypotension.

Mean baseline PI values were 1.05 in the study by Mehandale and Rajasekhar[2], whereas we found it to be 1.13±1.02. The difference we assume could be due to our institutional protocol of starting IV fluids at 1–2 mL/kg in the ward 2 h before surgery. Our results were similar to Thirunelli and Nanjundaswamy[8] as they had also preloaded their patients with IV fluids at 10 mL/kg after 6 h of fasting.

In our study, we noted a significant difference in the mean PVI between group H and group NH at post-induction at 1 min and at 3 min. At both these time points there was a significant fall in the MAP from baseline. ROC curves were plotted to validate the diagnostic accuracy of PVI and PI in predicting propofol-induced hypotension. AUROC for baseline PVI was 0.534 (sensitivity 87.5% and specificity 42.86%) and baseline PI was 0.559 (sensitivity 82.14% and specificity 42.86%) which was not statistically significant (P=0.754 and 0.543, respectively).

ROC analysis at 3 min post-induction at nadir of hypotension and PVI at >19 we noted AUROC for PVI was 0.732 (sensitivity of 92.66% and a specificity of 71.43%). This was noted to be statistically significant. However, AUROC of PI was 0.538 and was not significant. The PPV of PVI at 3 min post-induction was 92.69 and is statistically significant (P=0.0269). Analysis of ROC at this time point was not found in other studies. Thirunelli and Nanjundaswamy[8] found results similar to us. In their study, baseline cutoff value of PVI was >17.5 with a sensitivity of 38.3%, specificity of 84%, PPV of 88.6%, NPV of 29.6%. The baseline cutoff value of PI was <0.76 had specificity of 84%, sensitivity of 81.5%, PPV of 80.5%, and NPV of 37.5%.

Gunashekar et al.,[12] in their study, compared the predictive potential of PVI, PI and Pulse Pressure Variability and noted at 5 min, the PVI AUROC of 0.717 with a cut-off>11.5 and a PI AUROC of 0.647 with a cutoff 3.5. While their time points of analysis are different from ours, they also noted, that PVI was slightly more accurate in predicting hypotension compared to PI.

Yuksek[3] with a cutoff PVI >15.45 with a specificity and NPV of 80% found that baseline PVI was a good predictor of hypotension. Abdelhamid et al.,[9] constructed ROC curves and found that PVI and PI were good predictors of propofol-induced hypotension and AUROC was statistically significant. Cutoff PVI >17 had a sensitivity of 82.2% and PPV of 74.5% and PI ≤3.03 had a sensitivity of 77.8% and PPV of 74.5%. Mehandale and Rajasekhar[2] plotted ROC curves for PI as predictor of hypotension and AUROC curve was 0.816 with P<0.001 and baseline PI <1.05 was determined as cut-off in their study with high NPV of 98%. They found that baseline PI was a good predictor of propofol-induced hypotension.

PVI has higher accuracy for mechanically ventilated patients with a regular rhythm and is affected by cardiopulmonary exercise rather than in spontaneously breathing patients. Liu et al.,[4] in their met-analysis noted that PVI in mechanically ventilated patients without any cardiothoracic interventions reliably predicted pre-load responsiveness, provided that the pressure changes in the chest cavity were obvious enough and the cardiopulmonary interaction between different respiratory cycles was stable.

We focused on pre-anesthesia and post-induction values of PVI and PI to evaluate their predictive nature and use these tools to presumptively treatment hypotension. We recorded baseline PVI and PI in patients who were spontaneously breathing and ready to undergo surgery. Anxiety, movements of the patient, shivering and depth of breathing could have affected the results. To minimize such effects, we gave Tablet Alprazolam 0.25 mg to our patients the night before surgery as antianxiety medication and allowed our patients to lie down comfortably on the operation table, covered them with blankets for 3–5 min, and reassured them before starting our procedure.

Baseline PVI can be used as a screening tool to predict hypotension post-propofol induction but these parameters cannot be used as a diagnostic tool. Baseline PVI ≥19, the hypotension peaked, and hence we infer that a new baseline PVI to predict hypotension should be 19 or more. Pre-operative PI is not a sensitive tool to predict propofol-induced hypotension as PI did not correlate with hypotension at any time point in our patients.

### Limitations of the study

More studies with larger sample sizes in age-specific groups need to be done to obtain uniform pre-operative values to come to a valid conclusion. Further, the definition of hypotension is not uniform in all studies. We used the most followed criteria for defining post-propofol hypotension. Continuous BP monitoring may have produced more accurate data. As per our hospital protocol, we could not place an invasive arterial line when not mandated and hence used non-invasive BP monitoring.

## CONCLUSION

Baseline PVI and PI can serve as screening tools and not diagnostic tools to predict hypotension. Baseline PVI ≥19 is more accurate to predict post induction hypotension.

## ACKNOWLEDGMENT

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