JHWCR JOURNAL OF HEALTH, WELLNESS AND COMMUNITY RESEARCH 3007-0570

https://doi.org/10.61919/4c510693

Original Article

Anesthesia Drug Response Index in Obese Patients Using Perfusion Index + BIS: A Novel Composite Model

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"Cite this Article" Received: 01 March 2026; **Accepted**: 13 March 2026; **Published**: 30 March 2026

Author Contributions: Concept: SA, AA; Design: SA, MAS, HMFB; Data Collection: SA, MAS, DS; Analysis: AA, HMFB; Drafting: SA, UJA, MAS; Critical Review: AA, HMFB, DS. **Ethical Approval**: DHQ Hospital, Khanewal, Pakistan. **Informed Consent**: Written informed consent was obtained from all participants; **Conflict of Interest**: The authors declare no conflict of interest. **Funding**: No external funding; **Data Availability**: Available from the corresponding author on reasonable request; **Acknowledgments**: N/A.

ABSTRACT

Background: Obese patients undergoing general anesthesia are vulnerable to post-induction hypotension because obesity alters anesthetic drug distribution, autonomic tone, vascular reserve, and cardiopulmonary compensation. Bispectral index (BIS) and perfusion index (PI) are usually interpreted separately, although induction response involves simultaneous hypnotic, vascular, and hemodynamic changes. Objective: To determine whether peri-induction changes in BIS and PI, individually and as part of a composite Anesthesia Drug Response Index (ADRI), are associated with post-induction hypotension in obese adults undergoing elective surgery under general anesthesia. Methods: This prospective cohort study included 128 obese adult patients with BMI ≥30 kg/m². BIS, PI, MAP, and heart rate were recorded at baseline and during the first ten minutes after induction. Post-induction hypotension was defined as MAP reduction ≥20% from baseline, MAP <65 mmHg, or vasopressor requirement. Predictive performance of BIS fall, PI rise, baseline PI, and ADRI was evaluated using ROC analysis. Results: Post-induction hypotension occurred in 39 patients (30.5%). Hypotensive patients had higher BMI, more ASA III status, more hypertension, lower baseline MAP, and higher baseline PI. ADRI showed the strongest internal discrimination, with sensitivity 84.6%, specificity 80.9%, and AUC 0.86 at a cut-off score ≥7. Conclusion: Combined BIS and PI response assessment may improve early peri-induction risk classification in obese surgical patients. ADRI demonstrated promising internal discrimination but requires external validation before clinical implementation. Keywords: Obesity; General Anesthesia; Post-Induction Hypotension; Bispectral Index; Perfusion Index; Anesthesia Drug Response Index; Hemodynamic Monitoring

INTRODUCTION

Obesity is increasingly encountered among adult surgical patients and represents a major perioperative challenge because it alters respiratory mechanics, cardiovascular reserve, autonomic regulation, and the pharmacokinetic and pharmacodynamic behavior of anesthetic drugs. In patients undergoing general anesthesia, excess adiposity is associated with altered lean body mass, increased cardiac workload, variable volume of distribution, and a higher prevalence of comorbid conditions such as hypertension, diabetes mellitus, and obstructive sleep apnea. These physiological changes can make induction of anesthesia less predictable, particularly when standard dosing strategies are applied across patients with different body composition and vascular reserve. In obese patients, the immediate post-induction period is therefore clinically important because vasodilation, reduced sympathetic tone, myocardial depression,

and relative hypovolemia may converge to produce an abrupt fall in mean arterial pressure (MAP), sometimes requiring early vasopressor support (1,2).

Post-induction hypotension is not a benign monitoring event; it reflects a clinically relevant hemodynamic disturbance during the transition from wakefulness to anesthetized state and has been associated with adverse perioperative outcomes in large observational studies. The risk is particularly relevant in obese surgical patients because many have reduced cardiopulmonary reserve and associated vascular dysfunction, which may limit their ability to compensate for anesthetic-induced vasodilation and myocardial depression. In the Pakistani perioperative setting, this issue has practical importance because the burden of obesity and obesity-associated comorbidity is rising, while advanced invasive hemodynamic monitoring is not consistently available across all operating rooms. Local evidence also suggests that body mass index can influence propofol-related cardiovascular depression in Pakistani patients, supporting the need for context-specific assessment of anesthetic response rather than relying entirely on international dosing assumptions (1).

Bispectral index (BIS) monitoring provides a processed electroencephalographic estimate of hypnotic depth and is commonly used to reduce excessive anesthetic exposure, improve titration of induction and maintenance agents, and support recovery-oriented anesthetic practice. Previous studies have shown that BIS-guided induction may reduce unnecessarily deep anesthesia in some patient groups, although the effect varies according to patient characteristics, anesthetic agent, and clinical setting (9,10,12-14). In obese and morbidly obese patients, BIS may be especially useful because induction dosing based solely on total body weight can increase the risk of excessive hypnotic effect, whereas underdosing may produce inadequate anesthesia or awareness risk. However, BIS primarily reflects cortical hypnotic response and does not directly quantify peripheral vascular tone or circulatory reserve; therefore, BIS alone may not fully identify patients who are likely to develop hypotension after induction.

Perfusion index (PI), derived from the pulse oximeter waveform, is a non-invasive marker reflecting the ratio of pulsatile to non-pulsatile peripheral blood flow. It is influenced by vascular tone, peripheral perfusion, autonomic activity, and vasodilatory response. Recent prospective studies and meta-analytic evidence suggest that baseline PI and PI-derived changes may help predict hypotension after induction of general anesthesia, including in elderly and major abdominal surgery populations (15-18). The clinical appeal of PI is that it is simple, continuous, non-invasive, and already available on many modern pulse oximeters. In resource-variable settings, this makes PI a potentially valuable peri-induction risk marker, particularly when combined with other routinely available monitoring variables.

The central limitation of interpreting BIS or PI alone is that anesthetic drug response is a multisystem physiological process. Induction of general anesthesia simultaneously affects consciousness, sympathetic tone, vascular resistance, myocardial performance, and arterial pressure. A rapid fall in BIS may indicate strong hypnotic sensitivity, but it does not necessarily imply hemodynamic instability. Similarly, a rise in PI may suggest vasodilation but cannot independently describe the depth of anesthesia or the magnitude of systemic arterial pressure decline. A combined assessment of hypnotic response and peripheral vascular response may therefore provide a more clinically meaningful picture of peri-induction sensitivity than either parameter in isolation. Existing literature has examined BIS-guided dosing and PI-based hypotension prediction separately, but there remains limited evidence on whether an integrated BIS–PI response model can improve early identification of obese patients at risk of post-induction hypotension.

The present study was designed to address this gap by evaluating obese adult surgical patients undergoing elective procedures under general anesthesia in tertiary care hospitals of Punjab, Pakistan. The study focused on the peri-induction behavior of BIS, PI, and MAP during the first ten minutes after induction, with post-induction hypotension defined using clinically relevant MAP decline, absolute MAP threshold, and vasopressor requirement. To improve bedside interpretability, the study also developed an exploratory composite Anesthesia Drug Response Index (ADRI), integrating percentage change in

BIS, percentage rise in PI, and MAP response during the immediate induction period. Because MAP decline is part of both the clinical outcome and the physiological response score, the index was interpreted as an early peri-induction response classification tool, while BIS and PI components were also evaluated individually to assess their non-invasive predictive value.

This study therefore aimed to determine whether peri-induction changes in BIS and PI, individually and as part of a composite ADRI response score, are associated with post-induction hypotension among obese adults undergoing elective surgery under general anesthesia. The study hypothesis was that obese patients demonstrating a rapid fall in BIS, marked rise in PI, and greater early MAP reduction would have a higher probability of post-induction hypotension and vasopressor requirement than patients with more stable peri-induction monitoring trends.

MATERIALS AND METHODS

This prospective cohort study was conducted among obese adult patients undergoing elective surgery under general anesthesia in tertiary care hospitals of Punjab, Pakistan. The study followed an observational design in which all anesthetic decisions were made by the attending anesthesia team according to routine clinical practice, and no additional drug, invasive procedure, or experimental intervention was introduced for research purposes. The design was selected because the objective was to observe natural peri-induction physiological responses and determine whether routinely available non-invasive monitoring variables, particularly BIS and PI, were associated with post-induction hypotension. Reporting was aligned with the principles of the Strengthening the Reporting of Observational Studies in Epidemiology statement for cohort studies (25).

Adult patients aged 18 to 65 years with body mass index (BMI) of 30 kg/m² or higher who were scheduled for elective surgery under general anesthesia were eligible for inclusion. Patients of either sex and ASA physical status I, II, or III were included. Patients were excluded if they had severe heart failure, uncontrolled arrhythmia, shock, sepsis, pregnancy, neurological disease likely to affect BIS interpretation, peripheral vascular disease likely to affect PI measurement, emergency surgical indication, or refusal to provide consent. Consecutive eligible patients were approached during the study period, and recruitment continued until the required sample size was achieved. Written informed consent was obtained before enrollment, and patient participation did not alter the planned anesthesia technique or perioperative management.

Preoperative assessment was performed before transfer to the operating room. Demographic and clinical variables included age, sex, weight, height, BMI, ASA physical status, comorbid conditions, current medications, and type of elective surgical procedure. Baseline hemodynamic and monitoring values were recorded after the patient had rested quietly, including systolic and diastolic blood pressure, MAP, heart rate, oxygen saturation, PI, and BIS. BMI was calculated as weight in kilograms divided by height in meters squared, and obesity was operationally defined as BMI ≥30 kg/m². Hypertension and diabetes mellitus were recorded according to documented medical history or ongoing medication use.

In the operating room, standard monitoring was applied before induction and included electrocardiography, non-invasive blood pressure monitoring, pulse oximetry, capnography, temperature monitoring, BIS monitoring, and pulse oximetry with PI display. Baseline values were recorded before administration of induction drugs. General anesthesia was induced by the attending anesthetist using standard induction agents, opioid analgesia, and neuromuscular blockade according to the patient's clinical condition and institutional practice. The researcher did not modify drug selection or dosing. BIS, PI, MAP, and heart rate were recorded at baseline, immediately after induction, and at one, three, five, and ten minutes after induction. Vasopressor administration was documented with the drug used, dose, and time of administration when given by the anesthesia team.

The primary outcome was post-induction hypotension occurring within ten minutes after induction of general anesthesia. It was defined as any one of the following: reduction in MAP of 20% or more from baseline, absolute MAP below 65 mmHg, or administration of vasopressor support for post-induction blood pressure reduction. Secondary outcomes included vasopressor requirement, induction sensitivity pattern, and early recovery profile. Induction sensitivity was assessed using the magnitude of BIS reduction, PI rise, and MAP reduction during the first ten minutes after induction. Recovery variables included time to eye opening, response to verbal command, extubation time, and early postoperative sedation assessment.

For reproducibility, percentage change variables were calculated using baseline values as the reference. Percentage BIS fall was calculated as: [(baseline BIS − lowest BIS within ten minutes after induction) / baseline BIS] × 100. Percentage PI rise was calculated as: [(highest PI within ten minutes after induction − baseline PI) / baseline PI] × 100. Percentage MAP fall was calculated as: [(baseline MAP − lowest MAP within ten minutes after induction) / baseline MAP] × 100. The composite ADRI was constructed as an exploratory peri-induction response score by assigning points to BIS fall, PI rise, and MAP fall, with higher values representing a stronger combined hypnotic, vascular, and hemodynamic response. Because MAP fall was included in the composite response score and also contributed to the hypotension endpoint, the ADRI was interpreted as a peri-induction response classification index rather than a purely pre-event prediction model. To avoid overstatement, BIS fall, PI rise, and baseline PI were also analyzed separately as non-invasive predictive markers.

Patients were categorized into hypotension and non-hypotension groups according to the primary outcome definition. ADRI risk categories were explored using receiver operating characteristic curve analysis, and the cut-off with the best balance of sensitivity and specificity was identified using diagnostic discrimination parameters. In the reported analysis, an ADRI score of 7 or higher was used to identify patients with a high peri-induction response pattern. Predictive performance was compared across BIS fall, PI rise, baseline PI, and composite ADRI using sensitivity, specificity, area under the receiver operating characteristic curve, and statistical significance.

Data were recorded on a structured proforma containing demographic characteristics, clinical status, comorbidities, baseline monitoring values, peri-induction BIS, PI, MAP and heart rate values, vasopressor use, hypotension status, and recovery findings. Data were checked for completeness and consistency before entry into the analysis sheet. Continuous variables were assessed for distributional pattern and summarized as mean ± standard deviation for normally distributed data or median with interquartile range for non-normally distributed data. Categorical variables were summarized as frequencies and percentages. Group comparisons between patients with and without post-induction hypotension were performed using independent-sample t-test or Mann–Whitney U test for continuous variables, and chi-square test or Fisher's exact test for categorical variables, according to distribution and expected cell counts.

Associations between BIS change, PI change, MAP fall, and hypotension status were assessed using correlation and regression methods appropriate to variable type and distribution. Logistic regression analysis was used to identify independent predictors of post-induction hypotension. Clinically relevant variables and variables showing meaningful univariable association were considered for adjusted analysis, including BMI, ASA physical status, hypertension, baseline MAP, baseline PI, BIS fall, PI rise, and ADRI score. Adjusted estimates were interpreted using odds ratios with confidence intervals where available. A p-value less than 0.05 was considered statistically significant. Receiver operating characteristic curve analysis was used to compare the discrimination of BIS fall, PI rise, baseline PI, and ADRI for identifying post-induction hypotension. The composite model was considered clinically useful only if it demonstrated higher discrimination than the individual monitoring parameters and remained interpretable within the limitations of an internally evaluated observational cohort.

RESULTS

A total of 128 obese adult patients undergoing elective surgery under general anesthesia were included in the final analysis. The cohort had a mean age of 43.8 ± 10.9 years and a mean BMI of 35.7 ± 4.8 kg/m². There were 72 male patients and 56 female patients. Post-induction hypotension occurred in 39 patients, corresponding to 30.5% of the total cohort, while 89 patients remained hemodynamically stable during the first ten minutes after induction. Vasopressor support was required in 34 patients, representing 26.6% of the study population. Most hypotensive episodes were observed within the first five minutes after induction.

Table 1. Baseline and Clinical Characteristics According to Post-Induction Hypotension Status

Variable	Total Patients n=128	No Hypotension n=89	Hypotension n=39	Mean Difference / OR	95% CI	p-value
Age, years	43.8 ± 10.9	42.9 ± 10.7	45.7 ± 11.2	2.80	-1.43 to 7.03	0.182
Male sex	72 (56.3%)	48 (53.9%)	24 (61.5%)	1.37	0.63 to 2.95	0.427
BMI, kg/m²	35.7 ± 4.8	34.9 ± 4.3	37.4 ± 5.2	2.50	0.60 to 4.40	0.006
ASA II	81 (63.3%)	61 (68.5%)	20 (51.3%)	0.48	0.22 to 1.04	0.061
ASA III	36 (28.1%)	20 (22.5%)	16 (41.0%)	2.40	1.07 to 5.39	0.031
Hypertension	44 (34.4%)	25 (28.1%)	19 (48.7%)	2.43	1.12 to 5.30	0.023
Diabetes mellitus	31 (24.2%)	19 (21.3%)	12 (30.8%)	1.64	0.70 to 3.82	0.244
Baseline MAP, mmHg	94.6 ± 11.8	96.1 ± 11.4	91.2 ± 12.1	-4.90	-9.46 to -0.34	0.030
Baseline perfusion index	3.1 ± 1.4	2.7 ± 1.1	4.0 ± 1.6	1.30	0.74 to 1.86	<0.001
Baseline BIS	95.8 ± 2.9	96.1 ± 2.7	95.2 ± 3.2	-0.90	-2.07 to 0.27	0.098

Footnote: Continuous variables are presented as mean ± SD. Categorical variables are presented as n (%). Mean differences were calculated as hypotension group minus no-hypotension group. Odds ratios were calculated for categorical variables with post-induction hypotension as the outcome. ASA, American Society of Anesthesiologists physical status; BIS, bispectral index; BMI, body mass index; CI, confidence interval; MAP, mean arterial pressure; OR, odds ratio.

Patients who developed post-induction hypotension had a higher BMI than those who remained stable, with a mean difference of 2.50 kg/m² and a 95% CI of 0.60 to 4.40. ASA III status was more frequent in the hypotension group, with an odds ratio of 2.40 and a 95% CI of 1.07 to 5.39. Hypertension was also more common among hypotensive patients, with an odds ratio of 2.43 and a 95% CI of 1.12 to 5.30. Baseline MAP was lower in the hypotension group by 4.90 mmHg, while baseline perfusion index was higher by 1.30 units. Age, sex, diabetes mellitus, ASA II status, and baseline BIS did not show clear group separation based on the reported estimates and confidence intervals.

After induction, BIS declined in both groups, but the decline was greater among patients who developed post-induction hypotension. At three minutes after induction, the mean BIS was 40 in the hypotension group and 48 in the non-hypotension group. Perfusion index increased after induction in both groups, with a sharper rise among patients who developed hypotension. This combined pattern of deeper early hypnotic response, greater peripheral perfusion change, and MAP reduction was reflected in the composite Anesthesia Drug Response Index.

Table 2. Internal Discrimination of BIS, Perfusion Index, and Composite ADRI for Post-Induction Hypotension

Predictor	Cut-off Value	Sensitivity	Specificity	AUC	p-value
BIS fall after induction	>45% fall	71.8%	69.7%	0.74	<0.001
Perfusion index rise	>38% rise	76.9%	72.0%	0.78	<0.001

Predictor	Cut-off Value	Sensitivity	Specificity	AUC	p-value
Baseline perfusion index	>3.5	69.2%	74.2%	0.75	<0.001
Composite ADRI score	≥7	84.6%	80.9%	0.86	<0.001

Footnote: ADRI, Anesthesia Drug Response Index; AUC, area under the receiver operating characteristic curve; BIS, bispectral index. Reported values represent internal discrimination within the study cohort. AUC confidence intervals, predictive values, likelihood ratios, Youden index, and pairwise AUC comparison statistics were not available from the supplied data.

The composite ADRI showed the highest internal discrimination for post-induction hypotension, with an AUC of 0.86, sensitivity of 84.6%, and specificity of 80.9% at a cut-off score of ≥7. Perfusion index rise demonstrated stronger discrimination than BIS fall alone, with an AUC of 0.78 compared with 0.74. Baseline perfusion index also showed meaningful discrimination, with an AUC of 0.75 at a cut-off value of >3.5. These findings indicate that the combined peri-induction response pattern captured by ADRI performed better than single-parameter monitoring measures in this cohort.

Table 3. Early Recovery Profile According to Post-Induction Hypotension Status

Recovery Variable	No Hypotension n=89	Hypotension n=39	Mean Difference	95% CI
Time to eye opening, minutes	7.4 ± 2.8	8.9 ± 3.1	1.50	0.35 to 2.65
Extubation time, minutes	9.2 ± 3.1	10.8 ± 3.6	1.60	0.27 to 2.93

Footnote: Values are presented as mean ± SD. Mean differences were calculated as hypotension group minus no-hypotension group. p-values were not available from the supplied data.

Early recovery was modestly slower in patients who developed post-induction hypotension. Time to eye opening was longer in the hypotension group by 1.50 minutes, with a 95% CI of 0.35 to 2.65. Extubation time was also longer in the hypotension group by 1.60 minutes, with a 95% CI of 0.27 to 2.93. No severe immediate postoperative complication was reported in the recovery area.

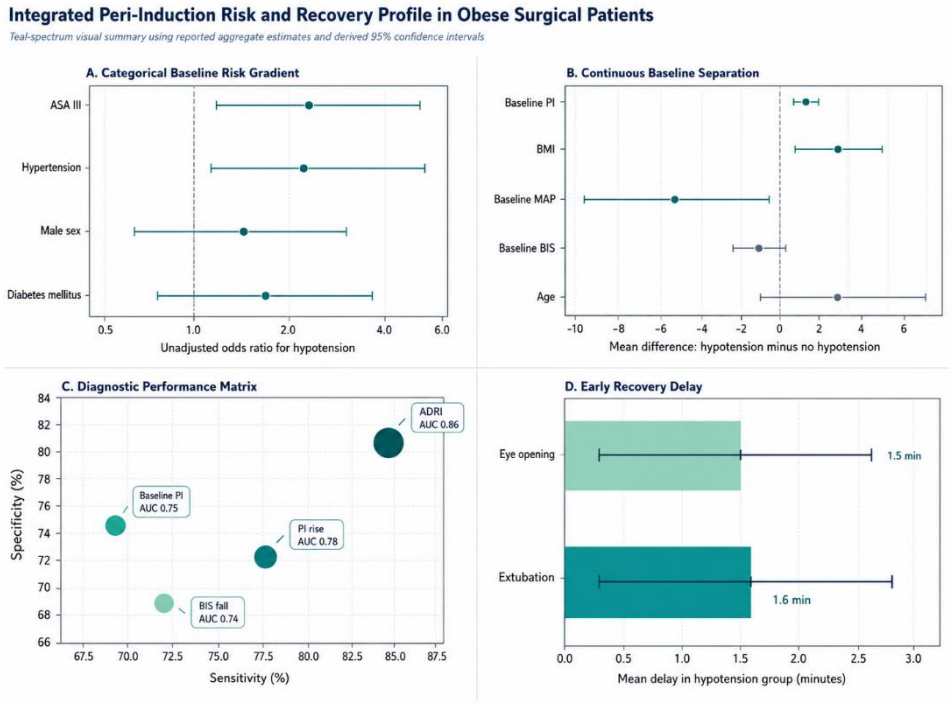

Figure 1. Integrated Peri-Induction Risk and Recovery Profile in Obese Surgical Patients

The panelled figure summarizes the relationship between baseline clinical risk, peri-induction monitoring discrimination, and early recovery delay among obese patients undergoing general anesthesia. Categorical baseline risk gradients showed that ASA III status and hypertension were associated with higher unadjusted odds of post-induction hypotension, with odds ratios of 2.40 and 2.43,

respectively, while male sex and diabetes mellitus showed wider confidence intervals crossing unity. Continuous baseline separation demonstrated that patients who developed post-induction hypotension had higher BMI by 2.50 kg/m², higher baseline perfusion index by 1.30 units, and lower baseline MAP by 4.90 mmHg compared with patients who remained stable. The diagnostic performance panel showed that the composite ADRI score had the strongest internal discrimination, with sensitivity of 84.6%, specificity of 80.9%, and AUC of 0.86, exceeding BIS fall, PI rise, and baseline PI alone. The recovery panel showed modest prolongation of immediate recovery among hypotensive patients, with time to eye opening delayed by 1.50 minutes and extubation time delayed by 1.60 minutes, indicating that early hemodynamic instability was accompanied by a small but measurable recovery burden.

The manuscript states that logistic regression identified BMI, baseline perfusion index, and composite ADRI score as important predictors of post-induction hypotension, while the association of hypertension became weaker after adjustment. However, the supplied results did not include crude or adjusted odds ratios, 95% confidence intervals, regression coefficients, model covariates, calibration statistics, or internal validation estimates. Therefore, a formal multivariable regression table could not be reconstructed from the available aggregate data and should be added from the original statistical output.

Overall, post-induction hypotension occurred in approximately one-third of obese adult patients undergoing elective surgery under general anesthesia. Patients who developed hypotension had higher BMI, more frequent ASA III status, more hypertension, lower baseline MAP, and higher baseline perfusion index. BIS declined more rapidly and PI increased more sharply after induction among hypotensive patients. The composite ADRI showed stronger internal discrimination than BIS fall, PI rise, or baseline PI alone, supporting its use as an exploratory peri-induction response classification index requiring further validation before routine clinical application.

DISCUSSION

This prospective cohort study evaluated peri-induction monitoring responses in obese adult patients undergoing elective surgery under general anesthesia and found that post-induction hypotension occurred in 30.5% of the cohort. Patients who developed hypotension had a distinct clinical and physiological profile, including higher BMI, more frequent ASA III status, higher prevalence of hypertension, lower baseline MAP, and higher baseline perfusion index. They also demonstrated a sharper early decline in BIS and a greater rise in perfusion index after induction. The composite Anesthesia Drug Response Index demonstrated stronger internal discrimination for post-induction hypotension than BIS fall, perfusion index rise, or baseline perfusion index alone, with an AUC of 0.86 at a cut-off score of ≥7. These findings support the clinical premise that obese patients who show a combined pattern of deeper hypnotic response, peripheral vasodilatory change, and early hemodynamic decline represent a higher-risk peri-induction phenotype.

The association between higher BMI and post-induction hypotension is clinically plausible because obesity modifies anesthetic drug distribution, cardiopulmonary reserve, venous return, and vascular responsiveness. In obese patients, induction dosing based on total body weight may increase the risk of excessive hypnotic or cardiovascular depression, while conservative dosing may risk inadequate depth of anesthesia. Local Pakistani evidence has also shown that BMI can influence propofol-induced cardiovascular depression, supporting the need for population-relevant perioperative monitoring strategies (1). Broader anesthetic literature similarly emphasizes that obese and morbidly obese patients require careful dose selection, airway planning, and hemodynamic surveillance because obesity is often accompanied by altered respiratory mechanics, increased cardiac workload, and comorbid metabolic disease (2,4,5). The present findings are consistent with this framework, as patients in the hypotension group had higher BMI and more frequent ASA III status, suggesting that both body composition and systemic disease burden contributed to peri-induction instability.

Hypertension was more frequent among patients who developed post-induction hypotension, although its adjusted association was reported to weaken after accounting for other variables. This pattern is clinically important because hypertensive obese patients may have altered vascular tone, impaired baroreflex responsiveness, endothelial dysfunction, and exposure to antihypertensive medications that influence peri-induction blood pressure behavior. Prior work has shown that patients receiving antihypertensive medication may be vulnerable to hypotension during anesthetic induction, particularly when sympathetic tone is rapidly reduced (22). Large observational studies have also identified patient-related and induction-related factors as contributors to hypotension after induction of general anesthesia (19,21). In the current cohort, the combination of hypertension, lower baseline MAP, and higher baseline PI may reflect a subgroup with limited vascular compensation at the time of induction.

The observed BIS pattern adds an important hypnotic-depth dimension to the findings. BIS decreased in both groups after induction, but patients who developed hypotension showed a deeper early BIS decline, with a mean BIS of 40 at three minutes compared with 48 among patients without hypotension. Previous studies have demonstrated that BIS-guided induction can help titrate anesthetic depth and reduce excessive hypnotic exposure in selected populations (9,10). In obese and morbidly obese patients, the challenge of selecting an appropriate propofol induction dose has been repeatedly emphasized, particularly because dosing scalars such as total body weight and lean body weight may produce different hypnotic and hemodynamic responses (6-8). The present findings suggest that a rapid BIS fall may identify patients with greater early hypnotic sensitivity, but BIS alone did not provide the strongest discrimination for hypotension. This supports the interpretation that hypnotic depth is only one component of the peri-induction response and should be considered alongside vascular and hemodynamic markers.

Perfusion index was strongly related to hypotension in this cohort. Patients who developed hypotension had a higher baseline PI and a greater PI rise after induction. PI is derived from the pulse oximeter waveform and reflects the relationship between pulsatile and non-pulsatile peripheral blood flow, making it sensitive to changes in peripheral vascular tone. A higher baseline PI or a marked post-induction rise may indicate greater vasodilatory tendency or reduced sympathetic vascular tone. Recent prospective evidence has supported PI-derived variables as predictors of hypotension after induction of general anesthesia, and a systematic review and meta-analysis has further strengthened the evidence for PI as a non-invasive marker of post-induction hypotension risk (15-18). The present results extend this concept to obese surgical patients in a Pakistani tertiary care context, where continuous non-invasive monitoring strategies may be particularly useful because advanced invasive hemodynamic monitoring is not routinely available for all elective surgical cases.

The main contribution of the study is the evaluation of a combined ADRI model that integrates BIS fall, perfusion index rise, and MAP response during the peri-induction period. The ADRI achieved higher sensitivity, specificity, and AUC than BIS fall, PI rise, or baseline PI alone. This suggests that the combined physiological response after induction may be more informative than isolated monitoring parameters. From a clinical perspective, this is logical because induction of general anesthesia simultaneously affects cortical activity, autonomic tone, systemic vascular resistance, venous return, myocardial performance, and arterial pressure. A patient may show a rapid BIS fall without substantial vascular instability, or a PI rise without excessive hypnotic depth, but the convergence of BIS decline, PI rise, and MAP reduction may represent a more complete response phenotype. However, the interpretation of ADRI requires caution because MAP fall contributed both to the composite score and to the definition of post-induction hypotension. Therefore, ADRI should not be presented as a fully independent pre-event prediction model in its current form. It is more accurately described as an exploratory peri-induction response classification index that demonstrated promising internal discrimination and requires refinement into a pre-hypotension version using baseline PI and early BIS/PI trends before clinically significant MAP decline.

The recovery findings provide additional clinical context. Patients who developed post-induction hypotension had modestly longer time to eye opening and extubation time, with mean delays of 1.50 and 1.60 minutes, respectively. These differences were not large, but they suggest that early hemodynamic instability may be accompanied by a small prolongation of immediate recovery. This may reflect deeper early hypnotic response, higher drug sensitivity, greater vasopressor requirement, or residual anesthetic effect. BIS-guided anesthesia has been associated with improved early recovery outcomes in some studies, although the magnitude and consistency of benefit vary across surgical and anesthetic contexts (10,14). In obese patients, recovery is also influenced by respiratory mechanics, airway obstruction risk, opioid exposure, neuromuscular recovery, and sleep apnea risk, making recovery time a multifactorial endpoint rather than a direct consequence of hypotension alone (2,4,5).

These findings have practical relevance for anesthesia services in Punjab and comparable resource-variable settings. Routine monitoring commonly includes non-invasive blood pressure, ECG, pulse oximetry, and capnography, while BIS and PI may be available but are often interpreted separately. A combined monitoring approach may help anesthetists identify high-risk obese patients earlier during induction, prepare vasopressors more proactively, individualize induction dosing, and intensify short-interval MAP surveillance during the first five to ten minutes after induction. The clinical utility of such an approach would be greatest if a simplified score could be applied before severe hypotension occurs, using baseline PI and early BIS/PI changes rather than incorporating established MAP decline into the risk score.

The study has several limitations that should be considered when interpreting the findings. First, the observational design can identify associations but cannot establish causality. Second, induction drugs and doses were selected by the attending anesthetist according to routine practice, which improves real-world applicability but introduces treatment heterogeneity and potential confounding. Third, the study did not provide detailed drug-dose stratification, antihypertensive medication timing, fluid status assessment, or surgical category analysis, all of which could influence post-induction hemodynamics. Fourth, ADRI was internally evaluated in the same cohort in which the cut-off was explored, and no external validation, bootstrapping, or cross-validation was reported. Fifth, because MAP fall was part of both the ADRI response score and the hypotension outcome definition, the current ADRI should be regarded as a response classification tool rather than a fully independent predictive model. Finally, adjusted logistic regression estimates, AUC confidence intervals, calibration statistics, and pairwise AUC comparisons were not available in the supplied results, limiting assessment of model precision and comparative superiority.

Future research should validate these findings in larger multicenter cohorts with standardized induction protocols, detailed drug-dose recording, and prespecified ADRI scoring. A refined version of the index should test whether baseline PI and early BIS/PI changes can predict hypotension before clinically significant MAP decline occurs. Further work should also evaluate performance across obesity classes, diabetic status, obstructive sleep apnea risk, antihypertensive medication exposure, and high-risk cardiac subgroups. If externally validated, a simplified BIS–PI-based response model may become a practical bedside adjunct for safer induction in obese surgical patients.

CONCLUSION

This prospective cohort study found that post-induction hypotension occurred in approximately one-third of obese adult patients undergoing elective surgery under general anesthesia and was associated with higher BMI, ASA III status, hypertension, lower baseline MAP, higher baseline perfusion index, sharper early BIS decline, and greater PI rise after induction. The composite Anesthesia Drug Response Index showed stronger internal discrimination than BIS fall, PI rise, or baseline PI alone, suggesting that combined assessment of hypnotic depth, peripheral perfusion response, and hemodynamic change may better characterize peri-induction sensitivity than single-parameter monitoring. However, because MAP

reduction contributed to both the composite response score and the hypotension endpoint, ADRI should be interpreted as an exploratory peri-induction response classification index rather than a fully validated independent prediction model. Further multicenter validation using standardized anesthetic protocols and a pre-hypotension BIS–PI-based scoring approach is required before routine clinical implementation.

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