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Pre-induction Stroke Volume Variation as a Predictor of Early Post-induction Hypotension in Non-Cardiac Surgery: a Cross-Sectional Observational Study

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Abstract

Introduction: General anesthesia induction-related hypotension is common and is associated with postoperative organ injury. Dynamic indices derived from echocardiography may help identify patients at risk, although stroke volume variation (SVV) is conventionally more robust under controlled mechanical ventilation than during spontaneous breathing. We investigated whether pre-induction transthoracic echocardiography-derived SVV was associated with arterial pressure 10 min after induction in adult elective non-cardiac surgical patients.

Patients and Methods: This single-centre cross-sectional observational study enrolled consecutive adult patients undergoing elective non-cardiac surgery with general anesthesia at a tertiary hospital (August–September 2022). Pre-induction SVV was measured in the supine position using the left ventricular outflow tract method during standardized spontaneous breathing. Anesthesia was induced with propofol 1.5 mg kg−1, fentanyl 2 μg kg−1, and atracurium 0.5 mg kg−1. Non-invasive arterial pressure was recorded for 10 min after induction. The prespecified primary outcomes were systolic blood pressure (SBP) and mean arterial pressure (MAP) at 10 min. Spearman's rank correlation was used.

Results: Sixty-four patients were analysed (mean age 48.4 yr; 57.8% male; 62.5% ASA physical status II). Mean pre-induction SVV was 13.4% (SD 4.3). Mean systolic blood pressure (SBP) decreased from 116.1 (7.9) mmHg pre-induction to 93.3 (6.3) mmHg at 10 min; mean arterial pressure (MAP) from 93.4 (12.3) to 76.8 (6.1) mmHg. Higher pre-induction SVV correlated with lower SBP at 10 min (Spearman r = −0.494; 95% CI −0.660 to −0.282; P < 0.001) and lower MAP at 10 min (r = −0.676; 95% CI −0.790 to −0.516; P < 0.001).

Conclusion: Pre-induction transthoracic echocardiography-derived SVV was associated with lower arterial pressure 10 min after induction. Because the study was observational and measurements were obtained during spontaneous breathing, the findings should be interpreted as hypothesis-generating and warrant confirmation in prospective studies.

Keywords: Echocardiography; General Anesthesia; Hemodynamics; Hypotension; Stroke Volume; Surgical Procedures, Operative

Introduction

General anesthesia induction–related hypotension (GAIH) is a common and clinically significant event in routine anesthetic practice, often occurring before surgical incision.[1] Even brief episodes of mean arterial pressure (MAP) below widely accepted thresholds have been associated with adverse postoperative outcomes, including myocardial injury and acute kidney injury. These findings underscore the importance of early hemodynamic optimization during the peri-induction period. However, predicting which patients will develop hypotension immediately after induction remains challenging, particularly in elective non-cardiac surgery. Early identification of at-risk patients may enable clinicians to tailor pre-induction optimization strategies and select appropriate induction techniques to mitigate hemodynamic instability.

Stroke volume variation (SVV) is a dynamic hemodynamic parameter that reflects heart–lung interactions and serves as an indicator of preload responsiveness.[1,2] Traditionally, SVV is derived from arterial waveform analysis during controlled mechanical ventilation, where cyclic changes in intrathoracic pressure are predictable and reproducible. In recent years, transthoracic echocardiography (TTE) has emerged as a non-invasive alternative for estimating stroke volume using the left ventricular outflow tract (LVOT) method. This approach offers the potential to assess hemodynamic status at the bedside before induction of anesthesia. Pre-induction TTE-derived SVV may therefore represent a practical tool for anticipating post-induction hypotension. Nevertheless, its physiological validity outside controlled ventilation settings remains uncertain, as spontaneous breathing introduces variable intrathoracic pressure fluctuations that may affect measurement reliability.[1–3]

Given these considerations, further evaluation of pre-induction SVV obtained using TTE is warranted to determine its clinical utility in predicting early hemodynamic changes after induction of anesthesia. Therefore, this study aimed to investigate the association between pre-induction TTE-derived SVV (LVOT method) and arterial pressure 10 minutes after induction of general anesthesia in adult patients undergoing elective non-cardiac surgery. We hypothesized that higher pre-induction SVV would be associated with lower systolic blood pressure (SBP) and mean arterial pressure (MAP) following induction.

Patients and Methods

This single-centre cross-sectional observational study was conducted in the operating theatre of a tertiary general hospital from August to September 2022. The study protocol was approved by the Research Ethics Committee of the Faculty of Medicine, Udayana University, Denpasar, Indonesia (No. 2284/UN14.2.2.VII.14/LT/2022; approved on 22 August 2022). Written informed consent was obtained from all participants prior to enrolment.

Consecutive adult patients scheduled for elective non-cardiac surgery under general anesthesia were screened for eligibility. Eligibility criteria were designed to ensure reliable left ventricular outflow tract (LVOT) Doppler assessment and appropriate hemodynamic interpretation. Detailed inclusion and exclusion criteria are provided in Online Supplementary File S1.

Pre-induction echocardiographic measurements were performed using transthoracic echocardiography. Participants were instructed to breathe slowly and deeply (approximately 6–8 breaths min^{-1}) and to avoid talking or movement during image acquisition. Although respiratory rate was standardised, tidal volume was not quantified and may have influenced stroke volume variation (SVV) measurements.

Using a parasternal long-axis view, the LVOT diameter was measured in mid-systole, 1 cm proximal to the aortic valve, and averaged over three measurements. LVOT velocity–time integral was obtained using pulsed-wave Doppler with the sample volume positioned 1 cm proximal to the aortic valve and aligned parallel to blood flow. Values were averaged over three consecutive beats, or five beats in cases of irregular rhythm. Stroke volume was calculated by the ultrasound system, and SVV was computed as: SVV (%) = [(SVmax − SVmin) / SVmean] × 100.

SVmax and SVmin were derived from Doppler-based stroke volume measurements across at least three consecutive respiratory cycles during spontaneous breathing. Each acquisition was repeated three times, and the mean SVV value was used for analysis. Measurements were obtained after a 2-minute stabilisation period.

All echocardiographic examinations were performed by a single trained operator with experience in more than 50 focused transthoracic echocardiography examinations. Standardised acquisition settings were applied, and consistent LVOT landmarks were used. If image quality was inadequate, measurements were repeated; patients were excluded if adequate imaging could not be obtained. LVOT diameter and velocity–time integral were measured three times and averaged to reduce intraobserver variability. Formal reproducibility metrics were not assessed. [4]

Standard intraoperative monitoring, including non-invasive blood pressure, pulse oximetry, and heart rate, was applied. Baseline hemodynamic parameters were recorded before induction. Preoxygenation was performed with 100% oxygen at 6 L min^{-1} for 3 minutes. Anesthesia was induced with propofol 1.5 mg kg^{-1} (administered via syringe pump at 200 mL h^{-1}), fentanyl 2 μg kg^{-1}, and atracurium 0.5 mg kg^{-1}. Blood pressure was recorded for 10 minutes after induction.

Hypotension was defined as mean arterial pressure <65 mmHg, systolic blood pressure <90 mmHg, or a ≥30% decrease from baseline. Management followed a standardised protocol consisting of crystalloid administration (200 mL over 2 minutes, repeatable up to 400 mL), followed by intravenous ephedrine 5 mg if hypotension persisted. Potential confounders, including baseline blood pressure, ASA physical status, comorbidities, medication use, and pre-induction fluid status, were considered.[5]

The primary outcomes were systolic and mean arterial pressure measured 10 minutes after induction. The primary explanatory variable was pre-induction SVV. This time point was selected to capture early hemodynamic effects before surgical stimulation.

Data are presented as mean (standard deviation). Normality was assessed using the Kolmogorov–Smirnov test. The association between pre-induction SVV and arterial pressure was analysed using Spearman's rank correlation with a two-

sided α of 0.05. Given the exploratory design and limited sample size, analyses were restricted to bivariable correlations without multivariable adjustment or receiver operating characteristic analysis. Statistical analyses were performed using SPSS version 25.

Results

A total of 64 participants were included in the final analysis. Baseline characteristics are summarised in Table 1. The mean (SD) age was 48.39 (6.37) years, 57.8% of participants were male, and 62.5% were classified as ASA physical status II. Mean body mass index was 22.35 (2.05) kg m^{-2}, and mean pre-induction stroke volume variation (SVV) was 13.39% (4.30).

Hemodynamic parameters decreased after induction of general anesthesia. Mean systolic blood pressure decreased from 116.07 (7.95) mmHg before induction to 93.26 (6.31) mmHg at 10 minutes after induction. Mean arterial pressure similarly decreased from 93.37 (12.30) mmHg to 76.80 (6.06) mmHg. Because the analysis focused on hemodynamic values at the 10-minute time point, cumulative burden metrics, such as the duration of mean arterial pressure below 65 mmHg, were not assessed.

Correlation analysis showed a significant inverse association between pre-induction SVV and post-induction arterial pressure.

Table 1. Baseline characteristics (n=64)

Variable	Value
Age, yr	48.39 ± 6.37
Male sex, n (%)	37 (57.8)
ASA physical status I, n (%)	24 (37.5)
ASA physical status II, n (%)	40 (62.5)
Body mass index, kg m−2	22.35 ± 2.05
Pre-induction SVV, %	13.39 ± 4.30
Pre-induction SBP, mmHg	116.07 ± 7.95
Pre-induction MAP, mmHg	93.37 ± 12.30
SBP at 10 min, mmHg	93.26 ± 6.31
MAP at 10 min, mmHg	76.80 ± 6.06

**Values are presented as mean ± standard deviation unless otherwise indicated. ASA = American Society of Anesthesiologists; SVV = stroke volume variation; SBP = systolic blood pressure; MAP = mean arterial pressure.*

Pre-induction SVV was moderately negatively correlated with systolic blood pressure at 10 minutes after induction (Spearman r = −0.494; 95% CI −0.660 to −0.282; P < 0.001) and lower MAP at 10 min (r = −0.676; 95% CI −0.790 to −0.516; P < 0.001), as shown in Table 2.

Table 2. Correlation between pre-induction SVV and arterial pressure at 10 min after induction

Outcome	Spearman r	P value
SBP at 10 min	−0.494 (95% CI −0.660 to −0.282)	<0.001
MAP at 10 min	−0.676 (95% CI −0.790 to −0.516)	<0.001

**CI calculated using Fisher z transformation.*

Discussion

In this single-centre observational study of adult patients undergoing elective non-cardiac surgery, higher pre-induction TTE-derived SVV was associated with lower SBP and MAP at 10 minutes after induction of general anesthesia. These findings suggest that greater preload dependency prior to

induction may be linked to a more pronounced early post-induction decrease in arterial pressure. However, because of the observational design, causality cannot be inferred.

This association is biologically plausible and consistent with established hemodynamic principles. Higher stroke volume variation reflects greater preload dependency and positioning on the steeper portion of the Frank-Starling curve, where cardiac output is more sensitive to changes in venous return. During induction of anesthesia, vasodilatation and myocardial depression caused by hypnotic agents and opioids may reduce venous return and cardiac output, thereby precipitating hypotension. In patients with higher pre-induction SVV, this reduction in preload may be less well tolerated, resulting in a greater decline in arterial pressure following induction.[6,7]

These findings complement previous literature highlighting the clinical relevance of early post-induction hypotension and support the potential role of non-invasive hemodynamic assessment in peri-induction risk stratification. Compared with conventional predictors such as baseline blood pressure, age, and comorbidity burden, TTE-derived SVV provides a physiological measure of preload responsiveness, although it remains operator dependent. This approach may be particularly useful in resource-limited settings where invasive hemodynamic monitoring or advanced predictive tools, such as the Hypotension Prediction Index, are not readily available.[6,8,9]

This study has several strengths. The use of a standardised anesthetic induction and hypotension management protocol reduced treatment variability and improved internal validity. In addition, the echocardiographic method used to estimate stroke volume variation was clearly defined and applied consistently across participants. These features support the reliability of the observed associations within the study context.

Several limitations should also be acknowledged. The single-centre design and relatively homogeneous population, including a restricted ASA physical status range, may limit generalisability. Blood pressure was measured intermittently using non-invasive monitoring, which may have underestimated the incidence and duration of transient hypotensive episodes. In addition, SVV was measured during spontaneous breathing prior to induction, whereas dynamic preload indices are classically validated under controlled mechanical ventilation. Variability in intrathoracic pressure and unmeasured differences in respiratory depth may therefore have affected measurement accuracy. Echocardiography-derived stroke volume calculations are also subject to variability in LVOT diameter and VTI measurements, despite efforts to standardise acquisition and average repeated measurements. Formal intraobserver reproducibility metrics were not assessed, which may have further influenced measurement precision. The statistical analysis was limited to bivariable correlation and did not adjust for potential confounders, including age, baseline hemodynamics, comorbidities, ASA classification, and preoperative medication use. Moreover, the study assessed arterial pressure at a single time point and did not evaluate cumulative hypotension burden or postoperative clinical outcomes.

Future studies should validate these findings in larger multicentre cohorts with

broader patient populations and use multivariable models to adjust for confounding factors. Prospective interventional studies are also needed to determine whether stroke volume variation-guided pre-induction optimization strategies, such as targeted fluid administration, early vasopressor use, or modified induction techniques, can improve clinically meaningful outcomes, including reduced hypotension burden and postoperative organ dysfunction.

Conclusion

This study demonstrates that higher pre-induction TTE–derived SVV is associated with lower systolic and MAP shortly after induction of general anesthesia. These findings highlight the potential role of pre-induction hemodynamic assessment in identifying patients at risk of early post-induction hypotension. The use of non-invasive, bedside echocardiographic parameters may support more individualised anesthetic planning, particularly in settings where advanced monitoring is limited. However, given the methodological limitations and absence of multivariable adjustment, these results should be interpreted with caution. Further prospective, adequately powered studies are required to validate the predictive value of pre-induction SVV and to determine whether SVV-guided optimization strategies can improve clinically relevant outcomes.

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Conflict of Interest

The author(s) report no conflict of interest.

Data Availability Statement

No new data were generated or analyzed in this study.

Author's Contributions

Conceptualization: MLK. Methodology: PAS. Investigation: MLK. Data curation: MLK. Formal analysis: PAS. Writing – original draft: IKWN. Writing – review & editing: MLK. Supervision: PAS, IKWN. All authors approved the final manuscript.

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