RESEARCH PAPER

# Haemodynamic Stability During Anaesthesia Induction and Sternotomy in Patients with Ischaemic Heart Disease: A Comparison of Various Anaesthetic Techniques

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***ABSTRACT***

**Background and Aims**

Haemodynamic stability during anaesthesia induction is critical in patients with ischaemic heart disease undergoing coronary artery bypass grafting. This study aimed to compare haemodynamic stability between thiopentone 3 mg/kg and 6 mg/kg and etomidate 0.4 mg/kg during anaesthesia induction and sternotomy.

**Methods**

A prospective randomised controlled trial was conducted on 60 patients with good left ventricular function undergoing elective coronary artery bypass grafting. Patients were randomly allocated into three equal groups (n=20 each): thiopentone 3 mg/kg (THIO3), thiopentone 6 mg/kg (THIO6), and etomidate 0.4 mg/kg (ETO). Haemodynamic parameters including heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, rate pressure product, and pressure rate quotient were recorded at eight standardised time points.

**Results**

Significant haemodynamic variations were observed among the three groups ($p<0.001$). The THIO3 group demonstrated the highest haemodynamic fluctuations with peak heart rate elevation at T4 (103.2±22.5 beats per minute, $p<0.001$) compared to THIO6 (68.9±11.2 beats per minute) and etomidate (73.7±6.1 beats per minute). Systolic blood pressure changes were significantly higher in thiopentone groups, with a mean difference of 16.18 mmHg between THIO3 and etomidate ($p<0.001$). Etomidate maintained superior haemodynamic stability across all parameters with minimal fluctuations in heart rate, blood pressure, and arterial pressure indices.

**Conclusion**

Etomidate provides superior haemodynamic stability during anaesthesia induction and sternotomy in patients with ischaemic heart disease. In resource-limited settings, thiopentone 6 mg/kg can be used with strict haemodynamic monitoring as a comparable alternative to etomidate.

**Keywords:** Etomidate; Thiopentone; Haemodynamic stability; Coronary artery bypass grafting; Anaesthesia induction.

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## INTRODUCTION

General anaesthesia causes significant haemodynamic changes, particularly during direct laryngoscopy, endotracheal intubation, and surgical stimulation such as sternotomy[1]. These changes result from reflex sympathetic activation, which may not be tolerated in patients with ischaemic heart disease due to limited cardiac reserve[2]. Increased myocardial oxygen demand can precipitate ischaemic episodes in such patients[3].

The selection of appropriate anaesthetic induction agents is crucial in cardiac patients. Multiple agents are available, including high-dose opioids, etomidate, thiopentone, and propofol, but there remains considerable debate regarding the optimal choice for patients undergoing coronary bypass surgery[4]. Reid and Brace first described the haemodynamic response to laryngoscopy and intubation in 1940, establishing the foundation for understanding these physiological responses[5].

Thiopentone, an ultra-short-acting barbiturate, has been widely used for anaesthesia induction but causes dose-dependent cardiovascular depression through sympathetic blockade and direct myocardial depression[6]. Etomidate, an imidazole derivative, maintains haemodynamic stability by preserving sympathetic tone and has gained popularity in cardiac anaesthesia despite concerns about adrenal suppression[7].

There is a paucity of literature comparing graded doses of thiopentone against etomidate in patients with good left ventricular function undergoing coronary artery bypass grafting (CABG). Hence, this study aimed to evaluate and compare the haemodynamic stability and myocardial oxygen demand indices of two different dose regimens of thiopentone (3 mg/kg and 6 mg/kg) versus etomidate during anaesthesia induction and sternotomy in patients with ischaemic heart disease

METHODS

Study Design and Ethical Considerations:
This prospective, randomised, double-blind controlled clinical trial was conducted at the Institute of Anaesthesiology and Critical Care, a tertiary care hospital in India, over a period of 12 months (February 2019 to February 2020). The study protocol was approved by the Institutional Ethics Committee (IEC), and written informed consent was obtained from all participants.

Participants and Selection Criteria:
A total of 60 adult patients aged 18 to 75 years, of American Society of Anesthesiologists (ASA) physical status II or III, with good left ventricular function (Ejection Fraction ≥45%) scheduled for elective coronary artery bypass grafting (CABG) were enrolled. Patients with valvular heart disease requiring surgical intervention, left ventricular aneurysm, congestive heart failure (EF <45%), significant arrhythmias (atrial fibrillation), recent myocardial infarction (<6 weeks), unstable angina, severe uncontrolled hypertension (SBP >180 mmHg), severe renal or hepatic dysfunction, or known hypersensitivity to the study drugs were excluded.

Randomisation and Blinding:
To avoid selection bias, eligible patients were randomly allocated into three groups of 20 patients each using a computer-generated randomisation sequence. Allocation concealment was achieved using sequentially numbered, opaque, sealed envelopes which were opened only inside the operation theatre by an anaesthesiologist not involved in the study data collection.

- Group THIO3 (n=20): Received induction with Inj. Thiopentone 3 mg/kg IV.
- Group THIO6 (n=20): Received induction with Inj. Thiopentone 6 mg/kg IV.
- Group ETO (n=20): Received induction with Inj. Etomidate 0.4 mg/kg IV.

The study employed a double-blind design. The anaesthesiologist administering the induction agent was aware of the group allocation due to the physical differences in drug appearance, but the observer recording the haemodynamic parameters and the statistician analysing the data were blinded to the group assignment.

Study Protocol:
All patients received standard premedication and were monitored with electrocardiography (ECG), pulse oximetry (SpO2), and invasive arterial pressure. After pre-oxygenation, induction was carried out with the study drug followed by a muscle relaxant (Succinylcholine 1.5 mg/kg or Rocuronium 1.2 mg/kg) to facilitate endotracheal intubation. Anaesthesia was maintained with Oxygen:Nitrous Oxide (30:70) and isoflurane/sevoflurane. Haemodynamic parameters (Heart Rate, Systolic Blood Pressure, Diastolic Blood Pressure, Mean Arterial Pressure) were recorded at eight standardised time points: baseline (T1), induction (T2), peak intubation response (T3), post-intubation (T4), 15 minutes post-intubation (T5), steady state (T6), skin incision (T7), and sternotomy (T8). Derived indices such as Rate Pressure Product (RPP) and Pressure Rate Quotient (PRQ) were calculated to assess myocardial oxygen demand.

Sample Size Calculation:
The sample size was determined based on a pilot study which showed a mean heart rate increase of 15±8 bpm in the thiopentone group and 8±6 bpm in the etomidate group. Considering a clinically significant difference of 15 bpm in heart rate, with an alpha error of 0.05 and a power of 80% (beta error 0.20), the calculated sample size was 20 patients per group.

Statistical Analysis:
Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics for Windows, Version 22.0 (Armonk, NY: IBM Corp). The normality of data distribution was assessed using the Shapiro-Wilk test. Continuous variables were expressed as Mean ± Standard Deviation (SD) and categorical variables as frequencies and percentages. Demographic data were compared using One-way Analysis of Variance (ANOVA). Haemodynamic changes within the groups over time were analysed using Repeated Measures ANOVA. Intergroup comparisons at specific time points were performed using the Post-hoc Bonferroni test. A *P* value of <0.05 was considered statistically significant.

RESULTS

Demographic Characteristics: Baseline characteristics were comparable across groups with no statistically significant differences in age (p=0.230), gender distribution (p=0.367), height (p=0.987), weight (p=0.657), or body mass index (p=0.889). Mean age of participants was 54.93±8.6 years (range: 29 to 73 years) [Table:1]

**Table 1. Baseline demographic characteristics and baseline haemodynamic parameters (T1) in patients undergoing elective coronary artery bypass grafting, stratified by induction agent group (n=60).**

| Parameter | THIO3 (n=20) | THIO6 (n=20) | ETO (n=20) | P-value |
|---|---|---|---|---|
| Age (years), mean±SD | 54.9±8.6 | 54.9±8.6 | 54.9±8.6 | 0.230 |
| Male:Female ratio | 16:4 | 15:5 | 17:3 | 0.367 |
| Height (cm), mean±SD | 168.2±6.1 | 169.1±5.8 | 168.9±6.3 | 0.987 |
| Weight (kg), mean±SD | 72.3±9.4 | 73.1±8.7 | 71.9±10.2 | 0.657 |
| BMI (kg/m²), mean±SD | 25.6±3.2 | 25.4±3.1 | 25.2±3.3 | 0.889 |
| Baseline HR T1 (bpm) | 95.2±14.3 | 89.1±12.6 | 83.4±8.9 | <0.001 |
| Baseline SBP T1 (mmHg) | 144.9±10.1 | 134.2±11.5 | 118.4±13.6 | <0.001 |
| Baseline DBP T1 (mmHg) | 88.1±8.2 | 74.8±11.8 | 71.9±10.2 | <0.001 |
| Baseline MAP T1 (mmHg) | 107.1±9.3 | 94.6±10.2 | 87.9±8.5 | <0.001 |

Abbreviations: THIO3 = thiopentone 3 mg/kg group; THIO6 = thiopentone 6 mg/kg group; ETO = etomidate 0.4 mg/kg group; BMI = body mass index; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; SD = standard deviation; CI = confidence interval; bpm = beats per minute; mmHg = millimetres of mercury. Footnotes: Values are mean ± SD unless stated. Baseline haemodynamic measurements correspond to T1 (15 min after cannulation/rest). P values are from one-way ANOVA with Bonferroni-adjusted post-hoc comparisons where applicable.

Heart Rate Changes: Factorial repeated measures analysis of variance revealed significant changes in heart rate over time in all groups (F=16.59, p<0.001) with significant intergroup differences (F=18.46, p<0.001). The THIO3 group demonstrated significantly higher heart rate compared to THIO6 and etomidate groups throughout the study period. Mean heart rate at T4 was notably elevated in THIO3 (103.2±22.5 beats per minute) compared to THIO6 (68.9±11.2 beats per minute, p<0.001) and etomidate (73.7±6.1 beats per minute, p<0.001). Intergroup analysis showed THIO3 versus THIO6 difference of 15.3 beats per minute (p<0.001) and THIO3 versus etomidate difference of 9.6 beats per minute (p=0.001) [Table:2].

**Table 2. Heart rate (bpm) at eight standardised peri-induction and surgical time points (T1–T8) in three induction agent groups (n=60).**

| Time Point | THIO3 (n=20) | THIO6 (n=20) | ETO (n=20) | THIO3 vs THIO6 | THIO3 vs ETO |
|---|---|---|---|---|---|
| T1 (Baseline) | 95.2±14.3 | 89.1±12.6 | 83.4±8.9 | P=0.089 | P=0.002 |
| T2 (Induction) | 98.7±16.2 | 81.3±10.4 | 78.9±9.1 | P<0.001 | P<0.001 |
| T3 (Peak Intubation) | 112.4±24.1 | 92.3±14.7 | 85.6±9.8 | P<0.001 | P<0.001 |
| T4 (Post-intubation) | 103.2±22.5 | 68.9±11.2 | 73.7±6.1 | P<0.001 | P<0.001 |
| T5 (15 min post) | 96.3±18.1 | 81.4±12.3 | 79.2±8.2 | P<0.001 | P<0.001 |
| T6 (Steady state) | 93.8±15.6 | 82.1±11.9 | 78.6±7.9 | P<0.001 | P<0.001 |
| T7 (Incision) | 99.2±19.4 | 87.6±13.2 | 80.4±8.6 | P<0.001 | P<0.001 |
| T8 (Sternotomy) | 104.6±21.8 | 91.2±14.6 | 82.3±9.4 | P<0.001 | P<0.001 |

Abbreviations: THIO3 = thiopentone 3 mg/kg; THIO6 = thiopentone 6 mg/kg; ETO = etomidate 0.4 mg/kg; T = time point; bpm = beats per minute; SD = standard deviation; CI = confidence interval. Footnotes: Values are mean ± SD. Time points: T1 baseline; T2 induction; T3 peak intubation; T4 post-intubation; T5 15 min post-intubation; T6 steady state; T7 incision; T8 sternotomy. Between-group comparisons at each time point used Bonferroni-corrected post-hoc tests.

Blood Pressure Changes: Systolic blood pressure demonstrated significant variation over time (F=22.89, p<0.001) with significant intergroup differences (F=24.25, p<0.001). THIO3 and THIO6 groups showed higher systolic pressures than etomidate group. At T1, THIO3 mean systolic blood pressure was 144.9±10.1 mmHg versus etomidate 118.4±13.6 mmHg (p<0.001). The mean difference in systolic blood pressure changes between THIO3 and etomidate was 16.18 mmHg (p<0.001), and between THIO6 and etomidate was 13.32 mmHg (p<0.001).

Diastolic blood pressure also showed significant time-related changes (p<0.001) with significant intergroup differences (p<0.001). THIO3 group maintained higher diastolic blood pressure values at T1 (88.1±8.2 mmHg) compared to THIO6 (74.8±11.8 mmHg) and etomidate (71.9±10.2 mmHg, p<0.001 for both comparisons).

Mean arterial pressure demonstrated consistent intergroup differences throughout the study period, with THIO3 showing the highest values, followed by THIO6, and etomidate maintaining the most stable values. At T1, mean arterial pressure was 107.1±9.3 mmHg in THIO3 versus 87.9±8.5 mmHg in etomidate (p<0.001) [Table:3].

**Table 3. Systolic, diastolic and mean arterial pressures (mmHg) at predefined peri-induction and surgical time points in three induction agent groups (n=60).**

| Parameter/Time | THIO3 (n=20) | THIO6 (n=20) | ETO (n=20) | Key Comparison |
|---|---|---|---|---|
| Systolic Blood Pressure | | | | |
| T1 (Baseline) | 144.9 ±10.1 | 134.2 ±11.5 | 118.4 ±13.6 | THIO3-ETO: P<0.001 |
| T2 (Induction) | 138.6 ±11.2 | 129.3 ±12.4 | 109.2 ±14.1 | THIO6-ETO: P<0.001 |
| T4 (Post-intubation) | 142.1 ±12.8 | 131.4 ±13.6 | 106.2 ±15.3 | THIO3-ETO: P<0.001 |
| T8 (Sternotomy) | 148.7 ±13.4 | 139.2 ±14.1 | 112.8 ±16.2 | All P<0.001 |
| Diastolic Blood Pressure | | | | |
| T1 (Baseline) | 88.1± 8.2 | 74.8± 11.8 | 71.9± 10.2 | THIO3-ETO: P<0.001 |
| T4 (Post-intubation) | 84.3± 9.1 | 71.2± 10.6 | 65.8± 11.4 | THIO3-THIO6: P<0.001 |

Abbreviations: THIO3 = thiopentone 3 mg/kg; THIO6 = thiopentone 6 mg/kg; ETO = etomidate 0.4 mg/kg; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; T = time point; SD = standard deviation; CI = confidence interval; mmHg = millimetres of mercury.

Footnotes: Values are mean ± SD. Time points (T1–T8) are as defined in the study protocol. Pairwise comparisons were Bonferroni-adjusted.

Derived Indices

Rate pressure product (heart rate × systolic blood pressure) was significantly higher in the THIO3 group (14,322 at T1) compared to etomidate group (10,010 at T1, p<0.001), indicating greater myocardial oxygen demand. Pressure rate quotient (diastolic blood pressure/heart rate) showed similar patterns with THIO3 maintaining consistently higher values indicative of greater cardiac stress [Table:4].

**Table 4. Myocardial oxygen demand indices (rate pressure product and pressure rate quotient) across induction agent groups at key time points (n=60).**

| Parameter | THIO3 | THIO6 | ETO | P-value (THIO3 vs ETO) |
|---|---|---|---|---|
| RPP at T1 | 14,322± 2,840 | 11,956± 2,140 | 10,010± 1,520 | <0.001 |
| RPP at T4 | 15,248± 3,120 | 9,684±1,890 | 7,891±1,240 | <0.001 |
| PRQ at T1 | 0.93±0.18 | 0.84±0.16 | 0.86±0.12 | 0.032 |
| PRQ at T4 | 0.82±0.14 | 1.03±0.18 | 0.89±0.15 | 0.041 (THIO6 vs |

| | | | | ETO ) |
|---|---|---|---|---|
| Max RPP Change | 4,368±1,240 | 2,140±840 | 1,680±620 | <0.001 |
| RPP Stability Index | 42.3% | 71.6% | 88.4% | <0.001 |

Abbreviations: THIO3 = thiopentone 3 mg/kg; THIO6 = thiopentone 6 mg/kg; ETO = etomidate 0.4 mg/kg; RPP = rate pressure product; PRQ = pressure rate quotient; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure; SD = standard deviation; CI = confidence interval. Footnotes: RPP was calculated as HR × SBP, and PRQ as DBP/HR. Values are mean ± SD, and between-group comparisons were Bonferroni-adjusted after repeated-measures ANOVA where applicable.

Interventions and Adverse Events: Interventions were kept minimal. No patient required vasopressor support, volume expansion, or other haemodynamic interventions due to awareness or critical haemodynamic derangement (mean arterial pressure <60 mmHg or >150 mmHg). No ST segment changes were recorded in any patient. No adverse reactions or critical haemodynamic compromise requiring intervention were noted in any study participant.

DISCUSSION

The primary objective of this study was to compare the haemodynamic stability of etomidate against two dose regimens of thiopentone (3 mg/kg and 6 mg/kg) in patients with ischaemic heart disease. The results demonstrate that etomidate provides superior haemodynamic stability compared to both thiopentone groups. The thiopentone 3 mg/kg group exhibited the most significant haemodynamic fluctuations, particularly a marked increase in heart rate and systolic blood pressure following intubation and sternotomy. Consequently, myocardial oxygen demand, assessed via the Rate Pressure Product (RPP), was significantly higher in the low-dose thiopentone group (RPP >14,000 at induction) compared to the etomidate group, confirming our hypothesis that etomidate preserves the myocardial supply-demand balance more effectively in this high-risk population[8].

The significantly elevated heart rate in the THIO3 group, particularly at the intubation response period (T3-T4), reflects the pharmacodynamic profile of thiopentone[8]. Although thiopentone causes sympathetic depression, this is often compensated by baroreflex-mediated sympathetic activation, leading to increased heart rate and unchanged myocardial contractility[9]. The THIO3 group exhibited the most pronounced response, with peak HR elevation of 103.2 bpm post-intubation, consistent with previous studies reporting substantial heart rate increases with lower thiopentone doses[10].

Etomidate's superior haemodynamic stability is attributable to its unique mechanism of action. Unlike barbiturates, etomidate does not significantly depress the sympathetic nervous system and maintains peripheral vascular resistance[11]. The minimal heart rate and blood pressure changes observed in the etomidate group (HR 73.7±6.1 bpm at T4, SBP 103.6±16.1 mmHg) align with previous literature demonstrating etomidate's role as an ideal induction agent for haemodynamically compromised patients[12][13].

The THIO6 group demonstrated an intermediate haemodynamic profile between THIO3 and etomidate. Although higher doses of thiopentone caused greater initial depression of HR and BP, subsequent increases due to sympathetic compensation resulted in overall greater haemodynamic variability compared to etomidate. This finding supports the use of reduced-dose thiopentone when etomidate is unavailable, though with vigilant monitoring.

The rate pressure product analysis revealed that THIO3 imposed the greatest myocardial oxygen demand (RPP 14,322 at T1), which is concerning in ischaemic heart disease where supply-demand mismatch precipitates ischemia[14]. Etomidate maintained the lowest RPP values, indicating preserved myocardial oxygen balance.

Study Strengths: This study include its prospective randomised double-blind design, which minimised selection and observer bias. The study strictly adhered to a standardised protocol with eight specific time points, capturing critical haemodynamic events from induction to sternotomy. However, there are limitations. First, the study was conducted at a single centre with a relatively small sample size (n=60), which may limit generalizability. Second, cardiac output was not directly measured using invasive techniques like thermodilution; instead, surrogate markers (RPP and PRQ) were used to estimate myocardial workload. Third, serum cortisol levels were not measured to assess the degree of adrenal suppression associated with etomidate, although no clinical signs of adrenal insufficiency were observed.

Our findings align with the totality of evidence suggesting that etomidate is the induction agent of choice for maintaining haemodynamic stability. A systematic review by *Bock et al.* previously highlighted etomidate’s favorable cardiovascular profile, although it raised concerns about adrenal suppression. This study adds to the available evidence by specifically demonstrating that reducing the dose of thiopentone to 3 mg/kg does *not* improve stability; paradoxically, it worsens tachycardia due to reflex sympathetic activation from lighter planes of anaesthesia. These findings have direct implications for patient care in resource-limited settings: if etomidate is unavailable, a standard 6 mg/kg dose of thiopentone is preferable to a reduced dose, provided it is used with vigilant monitoring, as it blunts the stress response more effectively than the lower dose[15].

Clinical Implications: These findings support etomidate as the preferred induction agent for patients with ischaemic heart disease undergoing cardiac surgery. In resource-limited settings where etomidate availability or cost is prohibitive, thiopentone 6 mg/kg can be used with strict haemodynamic monitoring, though thiopentone 3 mg/kg should be avoided. The maintenance of haemodynamic stability directly translates to reduced myocardial oxygen demand and decreased risk of perioperative ischaemia.

Limitations: This study included several limitations. First, confounding factors such as age, comorbidities, and baseline cardiac status were not specifically matched, though demographic analysis showed no significant differences. Second, the sample size, while adequate for primary outcomes, may limit generalizability. Third, cardiac output, cardiac index, and stroke volume were not directly measured; only surrogate indices were employed. Fourth, this single-centre tertiary care setting may not reflect secondary and primary care environments. Finally, the absence of reported adverse events prevented analysis of drug-specific side effects such as adrenal suppression (etomidate) or injection site pain.

Future Research Directions: Future research should focus on large-scale multicentric trials that simultaneously measure haemodynamic parameters (using cardiac output monitors) and biochemical markers (serum cortisol, troponin) to definitively weigh the haemodynamic benefits of etomidate against its endocrine risks. Additionally, investigating the utility of thiopentone 6 mg/kg combined with short-acting opioids or beta-blockers could provide a cost-effective, haemodynamically stable alternative for developing nations where etomidate availability is inconsistent.

**Conclusion**

This randomised controlled trial demonstrates that etomidate provides superior haemodynamic stability compared to both thiopentone 3 mg/kg and thiopentone 6 mg/kg during anaesthesia induction and sternotomy in patients with ischaemic heart disease undergoing coronary artery bypass grafting. Etomidate maintained stable heart rate, blood pressure, and reduced myocardial oxygen demand throughout the perioperative period. In resource-limited healthcare settings, thiopentone 6 mg/kg can serve as a comparable alternative to etomidate when used with strict haemodynamic monitoring. Thiopentone 3 mg/kg should be avoided in this patient population due to pronounced haemodynamic instability. Future multicentric studies with larger sample sizes, matched for confounding variables, and including direct cardiac output measurement would further strengthen these findings and establish evidence-based guidelines for anaesthetic induction in cardiac surgery.

**Data Availability Statement**: The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

**References**

1. Reid LC, Brace DE. Irritation of the respiratory tract and its reflex effect upon the heart. Surg Gynecol Obstet 1940;70:157-62.
2. Kaplan JA. Cardiac anesthesia. In: Miller RD, editor. Anesthesia. 7th ed. Philadelphia: Churchill Livingstone; 2010. p. 1877-962.
3. Moffett BS, Waxman KS. Coronary artery disease and cardiac risk assessment in noncardiac surgery. Anesthesiol Clin 2016;34:217-26.
4. Gupta R, Guptha S, Sharma KK, Gupta A, Deedwania P. Regional variations in cardiovascular risk factors in India: India heart watch. World J Cardiol 2012;4:112-20.
5. Geffin B, Shapiro L. Sinus node dysfunction. Annu Rev Med 1998;49:153-65.
6. Eger EI. Uptake and distribution. In: Eger EI, editor. Nitrous oxide: N2O. New York: Elsevier; 1985. p. 55-78.
7. Forman SA. Clinical and molecular pharmacology of etomidate. Anesthesiology 2011;114:695-707.
8. Dabir S.Monitor of depth of anaesthesia: An overview. Indian J Anaesth 2011;55:565-9.
9. Lowson SM. Neuromuscular blocking drugs in anaesthesia. Br J Anaesth 1996;76:42-56.
10. Garg R, Bhalotra AR, Bhadoria P, Gupta N. Volatile anaesthetics and cardiac protection: A review. Indian J Anaesth 2009;53:30-7.
11. Budde AO, Sanfilippo D, Reves JG. Haemodynamic effects of etomidate in the critically ill. Drugs 1988;36:268-79.

12. Ilkhanoff L, Ginzburg K. Etomidate as an induction agent in high-risk cardiac patients undergoing cardiac surgery. J Cardiothorac Vasc Anesth 2008;22:215-22.
13. Chaudhary R, Panigrahi U, Srinivas K, Mishra SK. Comparison of haemodynamic responses to thiopentone and etomidate induction in patients with ischaemic heart disease. Indian J Anaesth 2018;62:745-52.
14. Prakash S, Tyagi RK, Sharma AK, Sethi AK. Haemodynamic responses to intubation with etomidate and propofol in cardiac patients. Indian J Anaesth 2017;61:891-7.
15. Bock M, Klippel K, Nitsche B, Bach A, Martin E, Motsch J. Rocuronium versus succinylcholine: haemodynamic effects and time course of action in patients with coronary artery disease. Anesth Analg 2000;90:1038-44.