

**A COMPARATIVE STUDY OF HAEMODYNAMIC EFFECTS OF PROPOFOL AND ETOMIDATE AS AN INDUCTION AGENT IN CORONARY ARTERY SURGERY**

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**ABSTRACT: BACKGROUND:** Patients undergoing coronary artery bypass grafting are susceptible to haemodynamic lability during anaesthesia induction. **AIMS AND OBJECTIVE:** To evaluate the hemodynamic effects of etomidate in comparison to that of propofol during induction of general anaesthesia. **SETTINGS AND DESIGN:** The study was conducted in the department of anaesthesia of a tertiary care medical college setting over a period of one year from October 2013 to October 2014 on patients undergoing elective coronary artery bypass surgery under general anaesthesia. **MATERIAL AND METHODS:** 40 adult patients who are aged 35–65 years belonging to American Society Anaesthesia grades 1 and 2 undergoing elective surgery under general anaesthesia, were divided randomly into two groups of 20 patients each. By using bispectral index of 50 as a goal for induction, group A patients were given intravenous propofol injection, and group B were given etomidate injection. **STATISTICAL ANALYSIS:** Data is presented as mean and standard deviation. The statistical analysis was performed using SPSS 15.0, Stata 8.0, MedCalc 9.0.1 and Systat 11.0. A "p" value of less than 0.05 was taken as significant. **RESULTS:** Our results in both (P and E respectively) groups showed significant reduction in arterial pressure (30%-22%), SVRI (31%-23%), and LVSWI (38%-32%) after anaesthesia induction. However, the heart rate (3%-10%) and cardiac index did not change significantly. SVRI significantly increased in the etomidate group after intubation. **CONCLUSION:** Propofol can produce a larger reduction in contractility, arterial pressure, and after load when compared to etomidate as an induction agent in patients with coronary artery disease. Etomidate is less effective in preventing stress response to intubation.

**KEYWORDS:** Etomidate, propofol, anaesthetic induction agents, coronary artery bypass surgery.

**INTRODUCTION:** Induction of general anaesthesia in patients with coronary insufficiency must ensure haemodynamic stability along with hypnosis. The purpose of this study was to analyse the haemodynamic effects of propofol in comparison with that of etomidate for use in induction of anaesthesia in patients scheduled for CABG.

Propofol, an alkylphenol derivative, provides rapid onset and short duration of action. It causes considerable reduction in systemic vascular resistance and arterial pressure 15% to 40% after iv induction with 2mg/kg.<sup>1,2,3,4,5,6</sup> Its effect on HR is variable (Reset baroreceptor).<sup>7</sup> It causes direct myocardial depression at doses above 0.75mg/kg.<sup>8,9</sup>

Etomidate is a carboxylated imidazole derivative, has a rapid onset (10-12sec) and a brief duration of action, and hydroxylates primarily in liver. It provides hemodynamic stability in both noncardiac and cardiac disease patients after dosage of 0.15 to 0.30 mg/kg.<sup>10,11,12</sup> It directly inhibits 11-beta hydroxylation, which results in temporary reduction in biosynthesis of cortisol and

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aldosterone with serum concentrations in minimum limit of normal range.<sup>13</sup> Myoclonic movements are seen in 30-40% of patients. The drug was reformulated using lipid emulsion and reintroduced in 2007 in India.

**MATERIALS AND METHODS:** It was a prospective, randomized, and double blind comparative study conducted at Vydehi Institute of Medical Sciences and Research centre, Bengaluru, India. The study was conducted after approval from the institute's ethics committee. Forty consecutive patients with a normal LV function undergoing CABG surgery with use of CPB were enrolled into the trial.

The following patients were excluded from the study: those undergoing emergency surgery, ejection fraction less than 50%, having preexisting arrhythmias, with congestive cardiac failure, with coexisting cardiac diseases, with pre-existing bleeding and coagulation abnormalities, renal dysfunction (Serum creatinine > 2 mg/dl), Mallampati 3 and 4, epilepsy, allergy, on mechanical ventilation or on steroid therapy.

Twenty patients were randomly allocated into either the propofol (P) or the etomidate (E) group. The randomization was computer aided and done at admission level of the cardiac surgery department of the hospital.

Written informed consent was taken and NPO status maintained. Medications were continued except angiotensin converting enzyme inhibitors. All patients were pre medicated with intramuscular injection of morphine 0.1 mg/kg and promethazine 0.5 mg/kg half hour prior to induction of anesthesia as per institute protocol.

In the operation theatre, an intravenous line was established and preloaded with 5ml/kg lactated ringer. Oxygen supplementation was done by face mask. Intra- arterial cannula and pulmonary artery catheter (Edwards Lifesciences LLC, Irvine,CA, USA) were inserted after local anesthetic infiltration of the insertion site. Patient was monitored with pulse oximetry electrocardiogram (5-lead ECG), end tidal carbon- dioxide (EtCO<sub>2</sub>), invasive blood pressure (IBP) central venous pressure, pulmonary capillary wedge pressure (PCWP), continuous cardiac output(CCO), and bispectral index (Aspect Medical Systems, Cambridge, MA, USA). Anaesthetic agent for induction was prepared by an independent colleague. By keeping BIS below 50, anaesthesia was induced with either propofol (Diprivan, Astra Zeneca, Cheshire, United Kingdom) 0.5mg/kg/min or etomidate (Etomidat-Lipuro 2%, B. Braun, Melsungen, Germany) 0.05mg/kg/min. Endotracheal intubation was facilitated with rocuronium bromide (Roger, Cardilla health care, Mumbai) in the dose of 0.1 mg/kg in a single attempt less than 20 seconds. Mechanical ventilation was instituted to maintain eucapnia. Analgesia was attained with fentanyl up to a total dose of 10µg/kg. Midazolam was administered in divided doses up to a maximum of 0.1mg/kg. Anesthesia was maintained with titrated doses of sevoflurane. Parameters were recorded before induction (T1), after induction (T2), after intubation (T3), and 7 minutes after intubation (T4).

Hypotension (MAP ≤55 mm Hg) was treated with incremental doses of phenylephrine. Hypertension (MAP ≥100 mm Hg) was treated with fentanyl 1 µg/kg up to three times and then with a nitroglycerine infusion (10–100 µg/kg).<sup>21</sup> Bradycardia (HR ≤40 min) was treated with atropine 0.3 mg up to three times, and thereafter with ephedrine 5 mg. Tachycardia (HR ≥90 min) was treated with fentanyl 1 µg/kg up to three times and thereafter with metoprolol 1 mg bolus dose.

**STATISTICAL ANALYSIS:** Study design is of descriptive comparative type. The statistical softwares SPSS 15.0, Stata 8.0, Med Calc 9.0.1, and Systat 11.0 were used for the analysis of the data. Microsoft

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word and Excel have been used to generate graphs, tables etc. The patient characteristics (nonparametric data) were analyzed using “Chi-square test” while the inter group comparison of the parametric data was done using the “unpaired t-test.” The P value was determined to evaluate the level of significance.

### SIGNIFICANT FIGURES:

- + Suggestive significance (P value: 0.05-0.10).
- \* Moderately significant (P value: 0.01- 0.05).
- \*\* Strongly significant (P value: P<0.01).

A power analysis from previous studies revealed that a sample size of 20 patients per group was required to achieve a power of 80% and a 0.05 for detection of the desired hemodynamic changes (20 beats/minute or 20 mmHg difference in heart rate and blood pressure respectively).

**RESULTS:** The objective was to compare haemodynamic variables between the propofol group and the etomidate group during induction and orotracheal intubation, which includes the heart rate, arterial pressures (Systolic, diastolic, and mean), cardiac index, pulmonary artery pressures (Systolic, diastolic, and mean), central venous pressure, stroke volume index, systemic vascular resistance index, pulmonary vascular resistance index, left ventricular stroke work index, and right ventricular stroke work index.

Our hypothesis was to prove that propofol causes less stable haemodynamics as compared to etomidate during anaesthesia induction.

There is no significant difference between the two groups with respect to demographics, comorbid conditions, echo findings, and preoperative drugs (Table 1).

According to clinical observations and analysis of BIS, a satisfactory anaesthesia level was present in all the patients. The mean dose requirement for propofol and etomidate was 1.6(range 1.2-2.2) mg/kg and 0.22(0.16-0.24) mg/kg respectively.

Baseline parameters (T1 of Table 2 and Table 3) of heart rate, systemic arterial pressure, pulmonary arterial pressure, PCWP, systemic vascular resistance index, PVRI, cardiac index, LVSWI, and RVSWI between the two groups were statistically insignificant.

Basic characteristics	Group P	Group E	P value
Age in years	58.20±10.96	62.45±7.49	0.094
Height (cm)	164.50±7.82	164.18±7.67	0.926
Weight (kg)	71.15±9.19	67.19±10.01	0.065
Gender(M;F)	15:5	16:4	1.000
Hypertension	12(60.0%)	8(40%)	0.659
DM	8(40.0%)	10(45.5%)	1.000
Pre-MI	8(40.0%)	6(30%)	0.149
RWWA	10(50.0%)	14(63.6%)	0.670
LMCA	2(10.0%)	2(10%)	1.000
LAD	20(100.0%)	20(100.0%)	1.000
LCX	14(70.0%)	14(70%)	1.000
RCA	20(100.0%)	19(81.8%)	1.000
EF: Mean ± SD	56.70±5.14	55.64±5.56	0.686

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Sorbitrate	18(90.0%)	19(81.8%)	1.000
Beta blocker	20(100.0%)	20(100.0%)	1.000
ACE inhibitor	10(50.0%)	8(36.4%)	0.670
Calcium channel blocker	4(20.0%)	5(25%)	0.740

**Table 1: Comparison of basic variables between groups**

	HR					CI					MAP				
	Group A		Group B		p value	Group A		Group B		p value	Group A		Group B		p value
	Mean	SD	Mean	SD		Mean	SD	Mean	SD		Mean	SD	Mean	SD	
T1	76.2	13.12	74.64	11.61	0.775	2.94	0.77	2.57	0.5	0.201	119.2	20.85	114.36	14.47	0.541
T2	78.2	11.76	82.36	11.18	0.416	2.77	0.58	2.47	0.5	0.226	83.3	15.78	89.45	16.99	0.402
T3	82.8	9.72	88.73	13.15	0.259	2.88	0.55	2.44	0.51	0.074+	97.7	25.18	111.45	28.2	0.255
T4	78	9.12	85.27	16.95	0.243	2.71	0.76	2.5	0.55	0.48	99.9	16.67	95	15.63	0.495
<b>% Changes</b>															
T1-T2	2.62	10.34	-	5.78	3.89	-	30.12	21.78	-						
T2-T3	5.88	7.73	-	3.97	1.21	-	17.29	24.59	-						
<b>Significance</b>															
T1-T2	0.355	0.069+	-	0.107	0.226	-	<0.001**	<0.001**	-						
T2-T3	0.032*	0.065+	-	0.046*	0.637	-	0.015*	0.002**	-						

**Table 2: Comparison of heart rate (HR), cardiac index (CI), mean arterial pressure(MAP)**

SVRI	SVRI					PVRI					LVSWI				
	Group P		Group E		p value	Group P		Group E		p value	Group P		Group E		p value
	Mean	SD	Mean	SD		Mean	SD	Mean	SD		Mean	SD	Mean	SD	
T1	3181.3	879.04	3396.18	497.96	0.494	237.2	111	255.45	126.94	0.731	55.1	17.4	48.09	14.78	0.331
T2	2201.3	304.81	2630.82	459.51	0.022*	288.3	119.93	305	126.27	0.76	33.95	12.57	32.47	12.85	0.793
T3	2467.7	348.39	3399.45	793.66	0.003**	258.3	65.7	307.09	126.23	0.288	38.86	20.6	38.05	16.05	0.921
T4	2839.5	706.45	2944.64	665.84	0.729	229.5	95.71	249.64	148.9	0.72	38.73	13.18	33.45	12.22	0.353
<b>% Changes</b>															
T1-T2	30.81	22.54	-	21.54	19.4	-	38.38	32.48	-						
T2-T3	12.1	29.22	-	10.41	0.69	-	14.46	17.19	-						
<b>Significance</b>															
T1-T2	0.003**	0.001**	-	0.155	0.146	-	<0.001**	<0.001**	-						
T2-T3	0.044*	0.006**	-	0.415	0.946	-	0.154	0.015*	-						

**Table 3: Comparison of systemic vascular resistance (SVRI), pulmonary vascular resistance (PVRI) and left ventricular stroke work index (LVSWI)**

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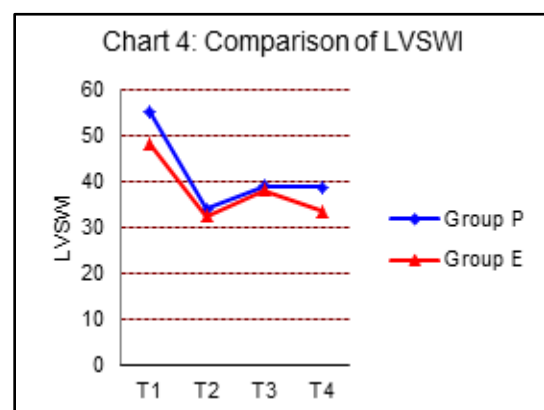
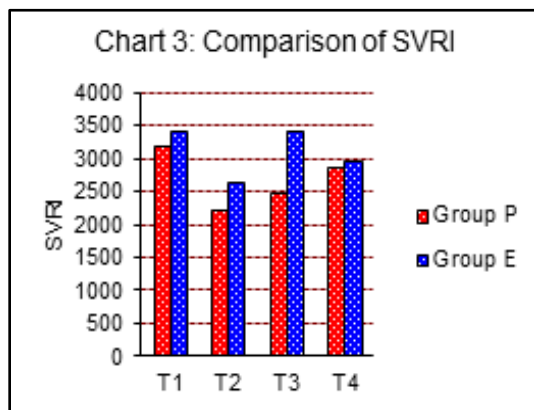
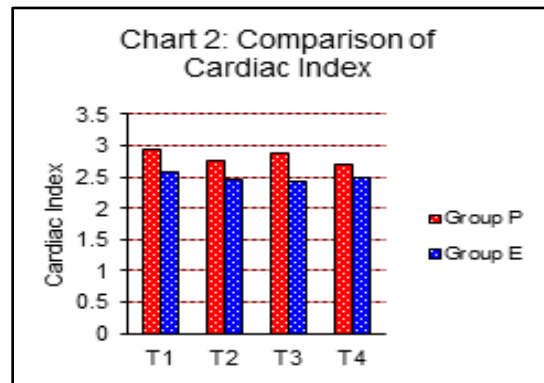
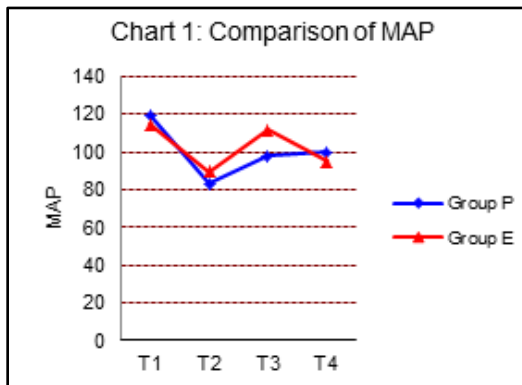


Table 2 shows a comparison of mean heart rate (BPM) between the two groups at selected intervals. Heart rate increased by 3% ( $p>0.1$ ) in group P and 10% ( $p<0.1$ ) in group E after induction and is statistically not significant.

Comparison of systemic arterial pressure changes are shown in table 2 and chart 1. After induction, mean MAP reduced by 30% in group P ( $p<0.001$ ) and 22% in group E ( $p<0.001$ ). Reduction is more in group P, though statistically not significant ( $p=0.402$ ). After intubation, mean MAP continued to be less than the baseline mean MAP. However, it has significantly increased in both groups compared to post induction levels.

Comparison of cardiac index between two groups shown in table 2 and chart 2. CI reduced in both groups after induction ( $p=0.2$ ) and intubation ( $p=0.2$ ) but was statistically not significant.

Comparison of SVRI between the two groups is shown in table 3 and chart 3. After induction, SVRI has reduced significantly in both group P ( $p=0.003$ ) and group E ( $p=0.001$ ). Reduction was more in group P, and it was moderately significant ( $p=0.022$ ) between groups. After intubation, SVRI significantly increased more in group E (30%) as compared to group P (12%) with a  $p$  value of 0.003. Comparison of PVRI is shown in table 3. It showed that after induction and intubation, PVRI slightly increased in both groups, but were statistically not significant ( $p>0.05$ ).

Comparison of Left Ventricular Stroke Work Index (LVSWI) between two groups is shown in table 3 and chart 4. The LVSWI reduced 38% in group P ( $p<0.001$ ) and 32% in group E ( $p<0.001$ ) after induction but were statistically not significant ( $p>0.05$ ). After intubation, the mean LVSWI was

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less than baseline levels but slightly increased after induction. These changes were statistically not significant ( $P > 0.05$ ) between groups.

Pulmonary arterial, systolic, diastolic, and mean pressures increased slightly during induction and intubation in both groups but were statistically insignificant among the groups.

Comparison of Right Ventricular Stroke Work Index (RVSWI) between two groups demonstrate that induction resulted in 16% reduction in group P ( $P > 0.1$ ) and a 23% fall in group E ( $P = 0.025$ ). After intubation, further reduction was noted in both groups, which was statistically insignificant. On the other hand, in group E, there were slightly increased levels ( $p = 0.09$ ). Statistical comparison between two groups was not significant.

**DISCUSSION:** In relation to steroid synthesis suppression, etomidate use was found to be of less clinical significance. There is a renewed interest in use of etomidate in elective cardiac surgeries. This prompted us to study its effect on haemodynamics in good functioning cardiac elective surgical patients in comparison to that of propofol.<sup>13</sup> the recommended bolus induction dose for propofol is 1.5–2.5 mg/kg and for etomidate is 0.15–0.4 mg/kg. We targeted BIS value of 50, which is in the lower third of the recommended range for general anaesthesia (BIS of 45–60) for intubation<sup>14,15,16,17</sup>

The requirement of propofol dose was 1.6 (Range 1.2–2.2)mg/kg and that of etomidate was 0.22 (0.16–0.24)mg/kg. Speed of infusion of both drugs were based on described pharmacokinetics and pharmacodynamics and reports found in the literature (0.50 and 0.75 mg/kg min for propofol and 0.1 mg/kg min for etomidate).<sup>18,19,20,21</sup> We have used morphine 0.1mg/kg as a premedication agent in all the cases. Concurrently, fentanyl 0.02 µg/kg was also administered before induction.

Our study showed no significant differences regarding variables such as gender, age, demographics, co-morbid conditions, echo findings, preop drugs, and baseline hemodynamic parameters; hence, the confounding effect of these variables has probably been neutralized.

Analysis of both groups demonstrated a slight but statistically not significant reduction in cardiac index. This may have been caused by a decrease in arterial pressure accompanied by direct decrease in contractility on induction. These changes may be attributed partially to the depression of sympathetic activity.<sup>22</sup> Propofol causes the impairment of baroreceptor mechanisms. The decrease in arterial pressure and contractility is significantly more in propofol group. The preservation of cardiac index may be attributed to the concurrent reduction in systemic vascular resistance. Analysis of both groups after intubation demonstrated relative preservation of cardiac output due to the return of variables to the baseline values. SVRI has significantly increased in etomidate group after intubation. The change in heart rate was less significant in our study in both groups. Both agents showed no significant difference on right ventricular contractility or pulmonary vascular resistance.

Criado A et al<sup>10</sup> studied the haemodynamic effects of etomidate induction in 36 patients. Their results showed SV, MAP, and LVCW significantly reduced but the heart rate increased significantly. They concluded that although etomidate has a negative inotropic effect, the variables remained within acceptable limits. Our study is consistent with their observations.

Haessler R et al<sup>23</sup> studied the haemodynamic effects of propofol and etomidate in patients with aortic insufficiency or CAD. After induction, they observed that there was a decrease in APs, CI, SV, and LVSWI in both groups. There was a decrease in APD and SVR in group P, and an increase in SVR and APs/SV in group E. There was a decrease in heart rate in both groups. After intubation, baseline values of APs, APD, and heart rate were not exceeded in any group. They concluded that



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propofol induction was associated with a reduction in AP of 30% after induction. This was due to a decrease in after load in combination with systolic and diastolic myocardial dysfunction. Our study is consistent with their observations.

Singh R et al<sup>24</sup> compared the haemodynamic effects etomidate, thiopentone, propofol, and midazolam in patients with coronary artery disease and left ventricular dysfunction.

After induction, there was a significant decrease in the variables compared to the baseline such as the heart rate (-7 to-15%,  $P = 0.001$ ), mean arterial pressure (-27 to-32%,  $P = 0.001$ ), cardiac index (-36 to-38%,  $P = 0.001$ ), and stroke volume index (-27 to-34%,  $P = 0.001$ ). In the etomidate group, there was a significant increase from baseline in both heart rate ( $P = 0.001$ ) and mean arterial pressure ( $P = 0.001$ ) at 1 minute after intubation. All the four agents were acceptable for induction in patients with coronary artery disease and left ventricular dysfunction despite a 30- 40% decrease in the cardiac indices. Our study is similar to this study.

A similar study was done by Petrun M et al<sup>17</sup> in which they noticed that there was no significant differences between the two groups regarding the haemodynamics before intubation. After intubation, MAP ( $P=0.019$ ) was significantly higher in the E group. CI was significantly higher in the Group E after intubation. Kaur S et al<sup>25</sup> studied the effects of propofol and etomidate in cardiac patients in noncardiac surgery. They found lesser decrease in heart rate and blood pressure in the etomidate group. Pandey AK et al<sup>13</sup> compared the effects of propofol and etomidate induction on haemodynamic parameters and serum cortisol levels in patients undergoing coronary artery bypass graft. It was found that propofol group had a significant reduction in SAP, DAP, and SVRI. On the other hand, there was no change in cardiac output following induction of anaesthesia ( $P <0.05$ ) with etomidate. Our study is consistent with their observations. Geeta Karki et al<sup>26</sup> studied thiopentone, propofol and etomidate as induction agents in general surgical patients. They found no significant change in heart rate and mean arterial pressure in the etomidate group. Bendel et al<sup>27</sup> measured the anaesthesia depth with the BSI monitor in their study in patients with aortic stenosis. They found that propofol is twice as likely to cause hypotension during induction when compared to etomidate. They also observed a decrease in CI and no change in heart rate during the induction in both groups. In our study, the findings were similar to this study.

Vermeyen KM et al<sup>1</sup> studied the effects of propofol (1.5mg/kg) and fentanyl in 15 patients with good LV function. Results showed that SAP (-28%), DAP (-23%), SVR (-25%), and LVSWI (-32%). PAWP had insignificant changes. Our observations in the propofol group are consistent with their results. Aun C et al<sup>2</sup> studied the haemodynamic effects of propofol in 10 patients. The results showed that there was a reduction of MAP (-19%), HR (-10%), and LVSWI. In CI and SVI, there was slight reduction. Patrick MR et al<sup>3</sup> compared the effects of propofol (1.5mg/kg) and thiopentone (2mg/kg) in patients with CAD. The propofol group was associated with a reduction in MBP, which was largely due to decrease in SVR. Lepage JM et al<sup>4</sup> studied the effects of propofol and fentanyl in CAD patients using radionuclide. Propofol alone showed a reduction in MAP (-15%). SVR and heart rate remained unchanged. With the addition of fentanyl (0.005mg/kg), there was a reduction of MAP (-35%), HR (-15%). CI and SVRI were significantly decreased.

Kaplan JA et al<sup>5</sup> compared the haemodynamic effects of propofol (2.5 mg/kg) and thiamylal (4 mg/kg) on myocardial revascularization patients. Administration of propofol in their study resulted in a significant decrease in MAP, SVR, and LVSWI as well as increase in heart rate. Mulier JP et al<sup>9</sup> compared the cardiac effects of propofol with thiopentone. Propofol significantly reduced the cardiac

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output, stroke volume, and SAP. They concluded that propofol reduces SAP mainly through its negative inotropic properties.

Although variations in arterial pressure and contractility demonstrated, cardiac output reduced less significantly in both groups. Myocardial ischemic ST-T changes were not appeared during study period. We did not measure cortisol levels.

**CONCLUSION:** Our study showed that propofol produces a larger reduction in contractility, arterial pressure, after load, and preload when compared to etomidate as an induction agent in patients with coronary artery disease. Etomidate is less effective in preventing stress response to intubation. Although no ischemic changes occurred in our study, caution is required while using propofol as an induction agent. Further studies are required for these drugs when used synergistically with adequate doses of opioids and benzodiazepines.

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**LEGENDS:**

- HR-Heart rate.  
 CABG-Coronary artery bypass surgery.  
 MAP- Mean arterial pressure.  
 SVRI- Systemic vascular resistance index.  
 CPB- Cardio pulmonary bypass.  
 LVSWI- Left ventricular stroke work index.  
 PVRI- Pulmonary vascular resistance index.  
 CI- Cardiac index.

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