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Comparative evaluation of Etomidate and Thiopentone on haemodynamic stress response at laryngoscopy and intubation in patients undergoing cardiac surgery.

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Abstract

Background: Etomidate, which contains a carboxylated imidazole, is structurally unrelated to other anaesthetic agents. It has minimal effects on cardiovascular system.

Context: Comparing the effect of Etomidate and Thiopentone on hemodynamic parameters during induction of GA for CABG surgery.

Aim: This study aims to compare etomidate and thiopentone on the following hemodynamic parameters: Heart rate (HR); Blood pressure (BP); Cardiac output (CO); Cardiac index (CI); Systemic vascular resistance (SVR), Pulmonary artery pressure (PAP) and ST-segment changes in Electrocardiogram (ECG).

Subjects and methods: In this study 50 patients aged 18 - 70 yrs. posted for elective coronary bypass grafting (CABG) surgery under cardiopulmonary bypass (CPB) were divided into 2 groups of 25 patients each. After premedication with Inj. Morphine 0.1mg/kg and Inj. Midazolam 0.04mgkg im, Group T (Thiopentone) received Inj. Fentanyl 3mcg/kg + Inj. Thiopentone 3-4mg/kg. Group E (Etomidate) received Fentanyl 3mcg/kg + Inj. Etomidate 0.2 to 0.3 mg/kg. Vecuronium Bromide was used to provide muscle relaxant in both the groups. Heart rate, Arterial Blood pressure, Cardiac Output (CO), Cardiac Index (CI), Systemic Vascular Resistance (SVR), Pulmonary Artery Pressure (PAP), ST- segment changes in ECG were recorded at baseline (before induction), just after intubation (within 1 minute), 3 minutes after intubation, 7 minutes after intubation.

Results: Etomidate provided greater hemodynamic stability as compared to Thiopentone when used as an induction agent.

Conclusion: In patients with compromised myocardial function, Etomidate is a superior drug as compared to

Thiopentone for maintaining hemodynamic stability at induction.

Keywords: Etomidate, Thiopentone Sodium, Coronary Artery Bypass Grafting (CABG), Hemodynamic stress response

Introduction

The deleterious effects of anaesthetic agents in patients suffering from coronary artery disease are well known. The risk increases when a patient has compromised hemodynamics. There is paucity of literature regarding the choice of the suitable agent to avoid deleterious effects in such patients[1-4]

Anaesthetic induction techniques for cardiovascular surgery are usually based on considerations such as hemodynamic stability, effects on myocardial oxygen supply and demand, and minimizing intubation stress response[1-4].

For induction of intravenous anaesthesia thiopentone sodium, Propofol, Ketamine, Etomidate, Benzodiazepines and opioids are being used. Ketamine increases myocardial oxygen demand, Thiopentone sodium, bezodizepams and propofol decreases Perfusion pressor, opioids required prolonged post-operative ventilation. Etomidate though hemodynamically stable suppresses adrenal gland and after a promising start went into disfavor with the reports of increase mortality.

In last decade or so there is rekindled interest in Etomidate for its stable hemodynamic profile as induction agent in cardiac patients. It was found that the prolonged used of Etomidate in intensive care units resulted in mortality but short-term use in Operation Theatre is safe.

In our institution we use thiopentone sodium and propofol as IV induction agent given slowly, with proper hydration (maintaining normal CVP).

With this background of safety and hemodynamic stability of Etomidate, we investigated weather Etomidate can be a better alternative to Thiopentone Sodium.

Thiopentone is a barbituric acid causes a fall in blood pressure and increase in heart rate. Cardiac output is often maintained by a rise in heart rate and increased myocardial contractility from compensatory baroreceptor reflex. Sympathetically induced vasoconstriction of resistance vessels may actually increase peripheral vascular resistance. However, in the absence of an adequate baroreceptor response (e.g. Hypovolemia, congestive heart failure and beta-adrenergic blockade) cardiac output and arterial blood pressure may fall dramatically due to uncompensated peripheral pooling and unmasked direct myocardial depression. Patients with poorly controlled hypertension are particularly prone to wide swings in blood pressure during induction[5]

Etomidate, which contains a carboxylated imidazole, is structurally unrelated to other anaesthetic agents. The imidazole ring provides water solubility in acidic solutions and lipid solubility at physiological pH. It has minimal effects on cardiovascular system. A mild reduction in peripheral vascular resistance is responsible for a slight decline in arterial blood pressure. Myocardial contractility and cardiac output are unchanged[5] It has a stable cardiac profile and is commonly used to induce anaesthesia in adults who have limited hemodynamic reserve.

Subjects and methods

The study was conducted after approval from the hospital medical ethics committee. Fifty patients aged less than 70 years belonging to American Society of Anaesthesiologists physical status II or III, of either sex, scheduled to undergo Coronary Artery Bypass

Grafting (CABG) under general anaesthesia, were included in the study. Patients with Ejection Fraction (EF) less than 40%, Congestive Heart Failure, emergency surgery, respiratory/ hepatic/ renal/ thyroid/ neurologic diseases, morbid obesity (BMI more than 35 kg/sq. Meter), adrenal disease, difficult intubation (mallampati grade III and IV), uncontrolled diabetes mellitus were excluded from the study.

The study was conducted in a randomized prospective double blinded manner. The patients were divided in two groups of 25 each. Patient were premedicated with Inj. Morphine 0.1mg/kg and Inj Midazolam 0.04mgkg IM. Anaesthesia was induced with Inj. Fentanyl 3mcg/kg ± Inj. Thiopentone 3-4mg/kg (Group T) IV/ Inj. Etomidate 0.2-0.3mg/kg IV (Group E) or till loss of eyelash reflex (given 30-45 seconds). Inj. Vecuronium Bromide 0.1mg/kg used as Muscle relaxant in both the groups.

Parameters under study

Heart rate, Arterial Blood pressure, Cardiac Output(CO), Cardiac Index (CI), Systemic Vascular Resistance (SVR), Pulmonary Artery Pressure (PAP), ST- segment changes in ECG were recorded at baseline (before induction), just after intubation (within 1 minute), 3 minutes after intubation, 7 minutes after intubation.

Stastical analysis

Data of hemodynamic parameters was presented in terms of mean ±SD under each group separately and the overall Stastical significance of difference between groups was compared by analysis of variance with repeated measurements. The stastical significance of intergroup comparison of various hemodynamic parameters was carried out by student 't' test and non parametric tests (man whittney test and Leveny test). The stastical significance of various hemodynamic parameters in each group was determined by paired 't' test. The P value of ≤ 0.05 was taken as statically significant.

Observation and results.

Demographic profile of all the patients were comparable in both the study groups so that the p value was not significant.

Group	Age	Weight	Height	Bmi	Bsa
Group E	55.44± 8.79	65.12±6.716	164.96± 7.541	24.0091±2. 76	1.71±0. 11
Group T	57.64± 7.460	60.56±10.654	163.20± 9.260	22.86±4.41	1.63±0. 14
P Value	0.345	0.077	0.465	0.278	0.056

Heart Rate (HR)

Mean heart rate increased significantly one minute after intubation in Thiopentone group (16%) as compared to Etomidate group (4%).

Group	PB	PI- 1	PI -3	PI -7
Group E	76.48±- 15.714	79.44±16.018	73.44±16.553	73.16±18. 29576
Group T	83.84±16.2 24	89.92±11.233	78.24±20.228	75.80±19. 826
P Value	0.11	0.031	0.363	0.627

We also found wide fluctuations in arterial blood pressures (systolic, diastolic, mean) in patients receiving thiopentone as compared to etomidate group the difference however was not found significant in any of the three arterial pressures i.e. systolic, diastolic and mean arterial pressures.

	BP- B			BP- 1			BP-3			BP-7		
	SYS	DYS	MEAN	SYS	DYS	MEAN	SYS	DYS	MEAN	SYS	DYS	MEAN
GROUP E	128.68±28.79	70.36±11.32	92.08±17.25	130.04±26.9	73.04±14.90	92.16±18.22	119.52±18.36	65.72±15.28	86.52±14.55	121.4±22.08	68.48±12.25	87.92±15.89
GROUP T	128.4±19.08	74.72±7.84	95.32±8.65	136.48±25.55	79.96±13.28	101.92±18.31	121.64±29.01	70.72±15.96	90.08±21.71	117.2±18.106	68.24±10.98	86.48±13.16
P VALUE	0.969	0.121	0.406	0.39	0.9	0.065	0.759	0.264	0.5	0.466	0.942	0.729

Cardiac output (CO)

The baseline mean cardiac output in group E (Etomidate) remained almost same at 1 min after intubation (3.5% decrease) while mean cardiac output in group T (Thiopentone) was increased by 20% from baseline. However, the difference was not found to be significant statistically but overall variation in cardiac output was more (20% +15%=35%) in group T as compared to group E (18%)

GROUP	CO-B	CO- 1	CO -3	CO -7
GROUP E	5.632±1.78	5.428±1.994	4.476±0.812	4.66±1.25
GROUP T	5.328±1.62	6.424±3.065	4.672±1.943	4.63±1.767
P Value	0.676	0.232	0.312	0.593

Cardiac Index (CI)

The baseline mean cardiac index in group E (Etomidate) was 3.428±1.102L/min/m^2 which remained almost same at 3.28±1.17L/min/m^2 (4% decrease) at 1 min and eventually decrease to 2.848±0.837 L/min/m^2 7 min after intubation (18% decrease from baseline) whereas in group T(Thiopentone) cardiac index increased to at 1 min from a baseline (a21% increase) and eventually decreased to (9% decrease from baseline). The difference was not to be statistically significant but overall variation in cardiac index was more (21%+9%=30%) in T as compared to group E (18%)

GROUP	CI-B	CI- 1	CI -3	CI -7
GROUP E	3.428±1.102	3.28±1.117	2.772±0.507	2.848±0.837
GROUP T	3.268±0.89	3.97±1.824	2.888±1.056	2.916±1.086
P Value	0.502	0.167	0.734	0.676

Pulmonary Artery Pressure (Mean)

The mean of mean pulmonary artery pressure was 21.84±7.543 mm Hg in Group E (etomidate) while it was 22.08±6.150 mm Hg in Group T (thiopentone) at baseline which increased by 14% to 25.20±8.406 mm Hg 1 min post intubation but remained almost same at 21.92±6.934 mm Hg in group E. The mean PAP in group E was more stable than group T throughout the study period but the difference was not statistically significant.

GROUP	P-MB	PM- 1	PM -3	PM -7
GROUP E	21.84±-7.543	21.92±6.934	20.40 ±6.39	21.20±5.859
GROUP T	22.08±6.150	25.20±8.406	22.84±7.663	21.40±5.867
P Value	0.902	0.139	0.227	0.905

ST-Segment elevation

The mean ST segment elevation in Group E (Etomidate) was 0.32±0.36 and 0.196+0.5358 in T (Thiopentone) at baseline. ST segment turned towards normal more sharply in group E (34%) as compared to T (8%), 7 min post intubation. However, no statistically significant differences were seen in both the groups throughout the observation period.

GROUP	ST-B	ST- 1	ST -3	ST -7
GROUP E	0.32±0.36	0.236±0.359	0.156±0.3267	0.212±0.3778
GROUP T	0.196±0.5358	0.1±0.4992	0.08±0.5439	0.181±0.3933
P Value	0.166	0.340	0.471	0.418

Systemic Vascular Resistance (SVR)

The SVR was after 3 minutes increased by about 10% in group E whereas it increased by about 12%. SVR remained more stable in group E as compared to group T during the study period but the difference was not significant statistically.

GROUP	SB	S1	S2	S7
GROUP E	1241+226.345	1313.20±240.431	1362±230.561	1367.64±221.130
GROUP T	1318.68±274.520	1313.60±444.728	1481.24±376.825	1439.76±474.390
P Value	0.283	0.997	0.187	0.496

In our study, Cardiac Output and Cardiac index changes were greater in thiopentone group as compared to etomidate group. Patients receiving etomidate had no significant changes in PAP whereas patients who received thiopentone had more fluctuations in PAP. ST segment returned more towards normalization in etomidate group**.** In our study, SVR remained lower in patients receiving etomidate as compared to patients receiving thiopentone.

Discussion

Our study showed significant changes in heart rate in patients receiving thiopentone which shows 6% increase at 1min post intubation and then decrease by 16% of baseline value at 7mins after intubation whereas etomidate group showed 4% increased after 1min and after 7mins it came nearer to basal level. Our results are consistent with the study of Tarnow et al[16] which showed thiopentone to increase heart rate by 10bpm whereas etomidate failed to alter heart rate significantly. However, Criado A et al[17] while studying the effects of etomidate only found significant increases in heart rate with etomidate which we could not demonstrate. This may be attributed to use of beta blockers, premedication with morphine, midazolam, and use of fentanyl in our patients. Jeffrey et al[24] whop compared etomidate versus thiopentone for induction of anesthesia in 83 ASA class I & II patients concluded that there is no difference between etomidate and sodium thiopental as an anesthetic agent in healthy patients. They also found that pre-treatment with a small dose (100mcg) of fentanyl attenuated increases in heart rate associated with induction of anesthesia and tracheal intubation with either etomidate or sodium thiopental. But our study results showed that there were decreased presser response in both the groups but thiopentone group showed peak end value i.e. a 6% increase after intubation and drop of 16% at 7mins. This was not the case in etomidate group which showed remarkable stability. This may be due to our different types of patients i.e. suffering from heart disease, use of beta blockers, use of higher dose of fentanyl and morphine.

We also observed that systolic arterial blood pressure values changed minimally at 1 min in etomidate group whereas it increases by 6% in thiopentone group and then decreased by 5% from baseline at 7mins after intubation in etomidate group and 7% decrease in thiopentone group. Between 1min and 7min after intubation the variation was 6% in etomidate group and

14% in thiopentone group. Overall swing of blood pressure in thiopentone group was 13% in thiopentone group whereas it was 6% in etomidate group from baseline so etomidate group patients have stable systolic arterial pressures as compared to thiopentone group. These results are concomitant with study conducted by Todd MM, Drummond JC et al[7] who concluded that there were significantly decreased in arterial pressure (to 37% of control) in patients receiving's high dose thiopental anesthesia. Our results are also consistent with the study conducted by Gooding et al[15] who found that etomidate causes minimal changes in arterial blood pressure after with etomidate. Gooding and corssen et al[13] in a similar study on 11 patients found minimal changes in systolic arterial pressure in patients receiving etomidate as an induction agent.

We also found that there were more fluctuations in diastolic blood pressure in patients receiving thiopentone (6% increase at 1min post intubation and then decrease by 9% from baseline at 7mins after intubation) whereas in etomidate group the change is only 4% increase 1 min post intubation and 2% decrease by 7min post intubation and 2% decrease 7mins post intubation. These results were consistent with a study conducted by Famewo CE et al [14], & Gooding et al[13] where they found minimal changes in blood pressure in patients receiving etomidate as an induction agent. Todd MM, Drummond JC et al[7] also found similar results in a study of hemodynamic consequences of high dose thiopental anesthesia.

We also found mean arterial pressure increase of 6% from baseline at 1min after intubation in patients receiving thiopentone but there was no change in etomidate group at the same time after intubation and then it decreased by 5% from baseline at 3mins and further decrease of 9% from baseline at 7mins in patients group, the decrease was only 5% from baseline at 7mins post intubation. Similarly, results were found by Framewo CE, Odugbesan CO[14] who found no significant difference in patients receiving etomidate. Our results are also consistent with the study conducted by Gooding et al[15] who found no significant changes in blood pressure after anesthetizing patients with etomidate. Todd MM, Drummond JC et al[7] also conducted a similar study in which they found significant decrease in blood pressure in patients receiving thiopentone.

Cardiac Output and Cardiac index changes were far more in thiopentone group (20% increase from baseline at 1min and then 15% decrease at 7mins with an overall swing of 35%) as compared to etomidate group (where there was only 18% decrease from baseline). These results are in accordance with the study conducted by Cariado A observed that CO and CI had decreased nonsignificantly in patients receiving etomidate. Similar results were also found by Tarnow et a1[16] who found more fluctuations in thiopentone group compared to etomidate group, though the difference was nonsignificant. Todd MM, Drummond JC et a1[7] in a study on thiopentone also found significant reduction in CO.

No significant change in PAP was observed during the study period in etomidate group but thiopentone produces fluctuations in PAP as was also found by Tarnow et a1[16] also found etomidate cause virtually no change in PAP.

Elevated ST segment in both etomidate and thiopentone group returned towards normalization but more in etomidate group as compared to thiopentone group though the ST segment changes were nonsignificant in both the groups. Lischke et a1[19] in a study of ST

segment changes after induction with etomidate found that patients with prior ST changes remained unchanged, but one other patient developed ST segment deviation after induction which we could not find. This may be due to our patients receiving cardioprotective cover of beta blockers and/or they have their myocardium already conditioned to ischemic insults due to coronary artery disease.

SVR remained lower in patients receiving etomidate as compared to patients receiving thiopentone in our study. It decreased by 10% in etomidate group whereas it.

Decrease by 12% in thiopentone group, 7min after intubation. In a similar study, Boer et al[18] found that SVR decreased to 78% of control value after thiopentone and 72% after etomidate. The fall is more acute in the study by Boer et al[18] as the observation in this study is after induction whereas in our study it is after intubation which resulted in increased in SVR as an intubation response.

The annoying side effects of etomidate like pain during injection and myoclonus were not observed in our study as the drug was premixed with 2% Xylocard, a larger peripheral vein was used, patient was premeditated with morphine and midazolam and fentanyl was given immediately prior to induction. Tarnow et al[16] also found similar results in their study.

In our study, there were 4 patients in thiopentone group and 2 patients in etomidate group who had higher blood pressure, heart rate and CO at baseline. The possible reason could be noncompliance with the beta blocker drugs which resulted in higher baseline values in these patients. But in these patients also the fluctuation in hemodynamic variable were far greater in thiopentone group as compared to etomidate group.

There were 3 patients in etomidate group who had very low baseline hemodynamic parameters, but in them also all the values remained within 15% of the baseline values showing etomidate to be a hemodynamically stable drug.

Conclusion

Induction of anesthesia in patients undergoing coronary artery bypass operations is a hazardous procedure because these patients are less tolerant to decrease in coronary artery perfusion pressure and increase in myocardial oxygen demand. Since cardiovascular surgery is expanding, the need for an induction agent which lacks cardiovascular side effects is well appreciated. Consequently, it is useful to consider what effects some of the recently recommended as alternatives induction agents may have on important determinants of myocardial oxygen supply and demand.

Etomidate is a new non-barbiturate agent dissolved in propylene glycol. Some of the features which are important for clinical use include short duration of action, lack of analgesic properties and freedom from histamine release. Preliminary reports have suggested cardiovascular stability following intravenous administration in dogs and in physically fit human volunteers.

Our study showed significant changes in heart rate in patients receiving thiopentone which shows 6% increase at 1min post intubation and then decrease by 16% of baseline value at 7mins after intubation whereas etomidate group showed 4% increased after 1min and after 7mins it came nearer to basal level. Our results are consistent with the study of Tarnow et al[16] which showed thiopentone to increase heart rate by 10bpm whereas etomidate failed to alter heart rate significantly. However, Criado A et al[17] while studying

the effects of etomidate only found significant increases in heart rate with etomidate which we could not demonstrate. This may be attributed to use of beta blockers, premedication with morphine, midazolam, and use of fentanyl in our patients. Our study results showed that there were decreased pressure response in both the groups but thiopentone group showed peak end value i.e. a 6% increase after intubation and drop of 16% at 7mins. This was not the case in etomidate group which showed remarkable stability. This may be due to our different types of patients i.e. suffering from heart disease, use of beta blockers, use of higher dose of fentanyl and morphine.

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