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An observational study of haemodynamics with etomidate as an induction agent in patients with coronary artery disease

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Abstract
Etomidate has been widely used in western countries has now become available in India. Etomidate is preferred in patients with CAD, especially with poor left ventricular (LV) function as it provides stable cardiovascular profile but it has less inhibitory effect on pharyngolaryngeal reflex thus may cause less blunting of response to laryngoscopy and intubation. After getting approval from institutional ethics committee, study was carried out in all patients fulfilling the inclusion and exclusion criteria during the study period. Patient diagnosed or known case of CAD either from history, investigations were included in the study after obtaining written valid informed consent. There was no hypertension or hypotension in group A (EF<45%) but in group B (EF>45%) hypertension was noted in just 1 patient and no hypotension noted. Statistically they were non-significant (p value-0.077). There was no occurrence any arrhythmias or significant ST changes in either group at any point of time from induction to 10min after intubation.

Keywords: Haemodynamics, etomidate, coronary artery disease

Introduction

Coronary Artery Disease (CAD) is the most common cardiac condition in cardiac patient presenting for non-cardiac surgery. General anaesthesia induction agent may decrease arterial blood pressure via myocardial depression, vasodilatation and attenuation of autonomic nervous system activity while laryngoscopy and endotracheal intubation can elicit hypertension, tachycardia and dysrhythmia due to sympathetic stimulation [1]. During induction of anaesthesia and intubation in patients with CAD haemodynamic changes are very important as they can lead to Myocardial Ischemia or heart failure or both. Thus concerns during induction and intubation in these patients with CAD include haemodynamic stability, attenuation of stress response and maintenance of balance between myocardial oxygen supply and demand [2, 3].
Etomidate has been widely used in western countries has now become available in India. Etomidate is preferred in patients with CAD, especially with poor left ventricular (LV) function as it provides stable cardiovascular profile but it has less inhibitory effect on pharyngolaryngeal reflex thus may cause less blunting of response to laryngoscopy and intubation [4]. Thus even though induction with etomidate may lead to stable haemodynamics, intubation response may cause tachycardia and hypertension thus increasing myocardial oxygen demand and predisposing patient with CAD to myocardial ischemia or

infarct. These haemodynamic responses may prove detrimental especially in patients with poor left ventricular ejection fraction.

Thus we decided to undertake this study to investigate haemodynamic responses and haemodynamic adverse effects if any at induction and intubation with etomidate as an induction agent in patients with CAD undergoing non cardiac surgery and compare them between patients with non-compromised (EF > 45%) and compromised (EF< 45%) LV function.

Methodology

After getting approval from institutional ethics committee, study was carried out in all patients fulfilling the inclusion and exclusion criteria during the study period. Patient diagnosed or known case of CAD either from history, investigations were included in the study after obtaining written valid informed consent. 2D Echo of patient diagnosed or known CAD was routinely done in our institution mainly to know the ventricular function. CAD patients who were undergoing elective non cardiac surgery and who were given etomidate as an induction agent were observed. Thorough pre anaesthetic check-up was done by routine anaesthetist.
Routine and specific investigations were noted. Preoperative medications taken by the patient were noted. The patient then underwent general anaesthesia as per standard protocol as decided by the anaesthetist incharge. All patients were monitored with electrocardiogram, non-invasive blood pressure (NIBP), pulse oximeter and capnograph. Intra-arterial BP monitoring and central venous pressure monitoring if done were noted. Baseline vital parameters were noted down. IV fluids Ringer Lactate (RL)/Normal saline (NS) was started at 5ml/kg/hr. Pre oxygenation was done with 100% O_2 for 3min. Patients were pre medicated with IV midazolam 0.02 mg/kg and IV fentanyl 2 mcg/kg 5minutes before induction. Anaesthesia was induced with etomidate in dose 0.2-0.3 mg /kg in graded doses till loss of eyelash reflex as our routine practice. Total dose of etomidate required for induction was noted down. After induction, inj rocuronium 1.2 mg/kg was given for intubation. Patients were ventilated with 100% oxygen for 60 seconds before intubation. After intubation, patients were ventilated with 100% oxygen. Endotracheal intubation was confirmed with auscultation and ETCO2. After intubation, all patients were maintained on 50:50 O_2:N_2O and 0.5% sevoflurane on closed circuit on controlled ventilation till the end of 10 minutes which is the end point of our study. Further maintenance of anaesthesia was done by either inhalational or intravenous anaesthetic as per discretion of anaesthetic incharge according to haemodynamics. Haemodynamic parameters heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean blood pressure (MBP) was noted down at various points as follows:
Baseline parameters (T0): immediately after taking patient on OT table (T1): Parameters noted after induction (T2), (T3), (T4) & (T5): Parameters noted immediately after intubation at 1min, 3min, 5min and 10min respectively.
The occurrence of adverse effects were observed and defined as follows

i) Hypertension if increase in baseline systolic BP > 20%.
ii) Hypotension if decrease in baseline systolic BP <20% or <90mmhg.
iii) Tachycardia if increase in baseline heart rate>20% or >100/min.
iv) Bradycardia if decrease in baseline heart rate <50/min.

Other adverse effects observed were arrhythmia, ST segment elevation (new ST segment elevation in atleast 2 anatomically contiguous leads, more than 2mm in V2 V3 and more than 1mm in all other leads) or ST segment depression (combination of two diagnostic criteria in atleast one ECG lead: atleast 1mm depression at J point and either horizontal or downward sloping ST segment depression). Time point of adverse effects and treatment given to treat adverse events as given below noted down:
IV fluids if no response IV ephedrine 3mg increment or IV phenylephrine 50mg increment for hypotension according to pulse rate.
IV fluid if tachycardia accompanied by hypotension.
IV esmolol in 100mcg increment for tachycardia with hypertension IV boluses of etomidate in 2 mg

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per 10s increment for hypertension IV Glycopyrrolate 0.2mg for bradycardia.

IV boluses of etomidate in 2mg per 10 second increment for hypertension.
Drug given and dose required were noted. Patients were grouped as group A and group B for statistical analysis. Group A included patients with EF<45% and group B included patients with EF>45%.

Results

Table 1: Comparison of Dose of Etomidate required for induction in the 2 groups

Parameter	Group		P Value
	EF<45 (group A) (n=40) Mean (SD)	EF >45 (group B) (n=40) Mean (SD)	
Dose of Etomidate	0.21(0.03)	0.22(0.04)	0.209
Unpaired t Test, P Value Not Significant			

This suggests that the dose of Etomidate required for induction in the two groups was comparable with 0.21mg/kg in group A and 0.22mg/kg in group B. The difference between the 2 groups was not significant.

Table 2: Comparison of Heart Rate between 2 Study Groups (N=80)

Heart Rate	Group		P Value
	EF<45 (n=40) Mean (SD)	EF >45 (n=40) Mean (SD)	
Baseline	79.50 (14.04)	83.35 (11.39)	0.182
After Induction	75.12 (12.39)	85.78 (10.96)	<0.001*
1 min after Intubation	84.75 (13.45)	97.83 (12.35)	<0.001*
3 min after Intubation	82.57 (13.25)	94.95 (14.71)	<0.001*
5 min after Intubation	80.12 (12.57)	91.15 (12.26)	<0.001*
10 min after Intubation	77.55 (12.24)	88.60 (12.02)	<0.001*
Unpaired t Test, P Value *Significant			

Table 3: Intra-Group Comparison of Heart Rate for EF <45 (N=40)

Mean Difference Heart Rate	EF<45		P Value
	Mean (SD)	Correlation	
After Induction	-4.38 (5.08)	0.933	<0.001*
1 min after Intubation	5.25 (7.10)	0.867	<0.001*
3 min after Intubation	3.08 (6.71)	0.881	0.006*
5 min after Intubation	0.63 (6.22)	0.896	0.529
10 min after Intubation	-1.95 (6.45)	0.888	0.063

Table 4: Intra-Group Comparison of Heart Rate for EF >45 (N=40)

Mean Difference Heart Rate	EF>45		P Value
	Mean (SD)	Correlation	
After Induction	2.42 (6.53)	0.830	0.024*
1 min after Intubation	14.47 (10.57)	0.687	<0.001*
3 min after Intubation	11.60 (10.03)	0.733	<0.001*
5 min after Intubation	7.80 (8.84)	0.723	<0.001*
10 min after Intubation	5.25 (9.86)	0.647	0.002*

The baseline HR of group A (EF<45%) was compared with HR after induction, after 1min,3min, 5min & 10 min after intubation. There was significant fall in HR after induction (p value<0.001) and the increase in HR after intubation at 1min & 3min was significant (p value <0.001 & 0.006 respectively).

The change in HR at 5min & 10min after intubation was not significant (p value 0.529 & 0.063 respectively) and gradually decreased towards baseline at 5min.
The baseline HR in group B (EF>45%) was compared with HR after induction and at 1min, 3min, 5min & 10min after induction. A slight increase in HR after induction (p value 0.024) and an increase post intubation (p value <0.001) were noted. The HR decreased around baseline at 10min.
On comparing HR between the two groups, baseline heart rate of group A and group B was comparable (p value 0.182, not significant). After induction there was decrease in HR in group A and slight rise in HR in group B, the difference was statistically significant (p value <0.001). After intubation the increase in HR in group B was more than the increase in HR in group A, the difference was statistically significant (p value<0.001). Then there was gradual decrease in heart rate towards baseline in group A at 5min and in group B the decrease was relatively less, reaching around baseline at 10min.

Table 5: Comparison of SBP between 2 Study Groups (N=80)

SBP	Group		P Value
	EF<45 (n=40) Mean (SD)	EF >45 (n=40) Mean (SD)	
Baseline	126.78 (15.13)	131.50 (14.79)	0.162
After Induction	117.07 (15.52)	119.28 (15.77)	0.531
1 min after Intubation	134.92 (17.76)	136.70 (15.58)	0.636
3 min after Intubation	128.90 (23.97)	132.85 (13.93)	0.470
5 min after Intubation	126.98 (14.45)	127.47 (13.00)	0.896
10 min after Intubation	123.40 (13.42)	122.75 (14.50)	0.836
Unpaired t Test, P Value Not Significant			

Table 6: Intra-Group Comparison of SBP for EF <45 (N=40)

Mean Difference SBP	EF<45		P Value
	Mean (SD)	Correlation	
After Induction	-9.60 (4.26)	0.962	<0.001*
1 min after Intubation	8.15 (9.55)	0.843	<0.001*
3 min after Intubation	2.12 (17.25)	0.697	0.441
5 min after Intubation	0.20 (9.30)	0.803	0.893
10 min after Intubation	-3.37 (9.41)	0.789	0.029*

Table 7: Intra-Group Comparison of SBP for EF >45 (N=40)

Mean Difference SBP	EF>45		P Value
	Mean (SD)	Correlation	
After Induction	-12.22 (8.62)	0.825	<0.001*
1 min after Intubation	5.20 (12.35)	0.670	0.011*
3 min after Intubation	1.35 (11.42)	0.685	0.459
5 min after Intubation	-4.02 (10.19)	0.738	0.017*
10 min after Intubation	-8.75 (12.90)	0.612	<0.001*

The baseline SBP of group A was compared with SBP after induction and 1min, 3min, 5min & 10min after intubation. The fall in SBP after induction was significant (p value <0.001). The increase in SBP was significant at 1 min after intubation (p value <0.001). The change in SBP at 3min & 5min after intubation was not significant (p value 0.441 & 0.893 respectively) and decreased towards baseline at 5min.
The baseline SBP in group B was compared with SBP post induction and 1min, 3min, 5min & 10min post intubation. There was significant decrease in SBP after induction (p value <0.001). After intubation at 1 min there was increase in SBP that was statistically significant (p value 0.011). At 5min & 10min post intubation there was decrease in SBP below the baseline value which was statistically

significant.

On comparing the SBP of two groups, the baseline SBP of group A and group B was comparable (p value 0.162). After induction there was fall in SBP in both groups and the difference was not significant (p value 0.531). At 1 min after intubation there was increase in SBP in both groups and the difference was not significant (p value 0.636). There was fall in SBP towards baseline in both groups at 5min after intubation and the difference in fall was not significant (p value 0.896).

Table 8: Comparison of DBP between 2 Study Groups (N=80)

DBP	Group		P Value
	EF<45 (n=40) Mean (SD)	EF >45 (n=40) Mean (SD)	
Baseline	76.28 (9.94)	76.97 (9.62)	0.750
After Induction	69.10 (8.57)	73.65 (7.86)	0.016*
1 min after Intubation	80.25 (10.42)	86.80 (9.70)	0.005*
3 min after Intubation	77.83 (10.04)	83.03 (8.29)	0.014*
5 min after Intubation	75.75 (9.37)	81.20 (7.83)	0.006*
10 min after Intubation	73.95 (8.60)	77.58 (9.19)	0.072
Unpaired t Test, P Value *Significant			

Table 9: Intra-Group Comparison of DBP for EF <45 (N=40)

Mean Difference DBP	EF<45		P Value
	Mean (SD)	Correlation	
After Induction	-7.17 (3.91)	0.921	<0.001*
1 min after Intubation	3.97 (7.15)	0.754	0.001*
3 min after Intubation	1.55 (8.78)	0.613	0.271
5 min after Intubation	-0.52 (8.44)	0.620	0.696
10 min after Intubation	-2.32 (8.40)	0.598	0.088

Table 10: Intra-Group Comparison of DBP for EF >45 (N=40)

Mean Difference DBP	EF>45		P Value
	Mean (SD)	Correlation	
After Induction	-3.32 (5.25)	0.838	<0.001*
1 min after Intubation	9.82 (7.16)	0.725	<0.001*
3 min after Intubation	6.05 (9.78)	0.411	<0.001*
5 min after Intubation	4.22 (7.88)	0.608	0.002*
10 min after Intubation	0.60 (9.23)	0.519	0.683

The baseline DBP in group A was compared with DBP after induction and 1min, 3min, 5min & 10min after intubation. The fall in BP after induction was significant (p value <0.001). The rise in DBP after intubation at 1 min after intubation was statistically significant (p value 0.001). The changes in DBP after 3min, 5min & 10min after intubation was not significant (p value 0.271, 0.696 & 0.088 respectively) and decreased slightly below baseline at 10min.

The baseline DBP in group B was compared with DBP post induction and 1min, 3min, 5min & 10min post intubation. A decrease in DBP after induction was noted (p value <0.001). After intubation there was increase in DBP at 1min and 3min and was statistically significant (p value <0.001 & <0.001 respectively). DBP returned to baseline at 10min after intubation.

The baseline DBP values of the two groups were comparable (p value- 0.750). After induction the fall in DBP was statistically significant in group A as compared with group B (p value 0.016). After intubation at 1 min the increase in DBP in group B was significant as compared to group A (p value 0.005). Thereafter there was gradual decrease in DBP in both groups towards baseline by 10min after intubation.

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Table 11: Comparison of MAP between 2 Study Groups (N=80)

Map	Group		P Value
	EF<45 (n=40) Mean (SD)	EF >45 (n=40) Mean (SD)	
Baseline	93.10 (11.11)	95.15 (10.76)	0.407
After Induction	85.12 (10.29)	88.85 (9.37)	0.094
1 min after Intubation	98.47 (12.09)	103.43 (10.79)	0.057
3 min after Intubation	94.85 (13.01)	99.30 (10.07)	0.091
5 min after Intubation	92.82 (10.12)	95.90 (9.67)	0.169
10 min after Intubation	90.43 (9.40)	91.92 (11.14)	0.520
Unpaired t Test, P Value Not Significant			

Table 12: Intra-Group Comparison of MAP for EF <45 (N=40)

Mean Difference MAP	EF<45		P Value
	Mean (SD)	Correlation	
After Induction	-7.98 (3.57)	0.947	<0.001*
1 min after Intubation	5.36 (7.14)	0.814	<0.001*
3 min after Intubation	1.74 (9.36)	0.710	0.246
5 min after Intubation	-0.28 (8.01)	0.719	0.824
10 min after Intubation	-2.67 (8.08)	0.701	0.043*

Table 13: Intra-Group Comparison of MAP for EF >45 (N=40)

Mean Difference MAP	EF>45		P Value
	Mean (SD)	Correlation	
After Induction	-6.29 (5.27)	0.872	<0.001*
1 min after Intubation	8.28 (8.12)	0.716	<0.001*
3 min after Intubation	4.15 (10.02)	0.538	0.013*
5 min after Intubation	0.75 (8.36)	0.669	0.574
10 min after Intubation	-3.22 (10.52)	0.539	0.060

The baseline MAP in group A (EF<45%) was compared with MAP after induction and 1min, 3min, 5min & 10min after intubation. The fall in MAP after induction was significant (p value <0.001). The increase in MAP at 1min after intubation was significant (p value <0.001). The change in MAP at 3min & 5min after intubation was not significant (p values 0.246 & 0.824 respectively) and reached around baseline at 5 min.

The baseline MAP in group B (EF>45%) was compared with MAP post induction and 1min, 3min, 5min & 10min post intubation. A fall in MAP after induction and rise in MAP after intubation were noted and were statistically significant (p value <0.001 & <0.001). There after MAP gradually decreased towards baseline at 5min after intubation.

The baseline values of MAP were comparable in two groups (p value-0.407). The fall in MAP after induction was comparable in two groups (p value 0.094). The increase in MAP at 1min after intubation was comparable in two groups (p value 0.057). There was decrease in MAP towards baseline at 5min in both groups.

Table 14: Association between Study Group and Adverse Outcomes/Treatment (N=80)

Hypotension/Hypertension	Group		P Value
	EF <45 (group A) (n=40) n (%)	EF >45 (group B) (n=40) n (%)	
Hypotension	0(0%)	0(0%)	NS
Hypertension-NTG	0(0%)	1 (2.5)	0.314

Arrhythmia	0(0%)	0(0%)	NS
ST segment changes	0(0%)	0(0%)	NS
Chi-Square Test, P Value Not Significant			

There was no hypertension or hypotension in group A (EF<45%) but in group B (EF>45%) hypertension was noted in just 1 patient and no hypotension noted. Statistically they were non-significant (p value-0.077). There was no occurrence any arrhythmias or significant ST changes in either group at any point of time from induction to 10min after intubation.

Discussion

The first parameter that we studied was heart rate, baseline heart rate was noted down of all patients in both groups. The changes in HR after induction and various intervals after intubation were compared with baseline and between the two groups.

In patients with EF < 45% there was significant decrease in heart rate after induction. After intubation at 1min & 3 min there was increase in heart rate that was statistically significant. Then the heart rate gradually decreased towards the baseline and the change in HR was not significant at 5min & 10min post intubation.

In patients with EF > 45% there was significant increase in heart rate after induction. After intubation significant increase in HR was observed. Thereafter HR decreased at 3min & 5min after intubation, but the difference from baseline was significant. These findings show that the decrease in HR after induction and increase in HR after intubation were transient and not clinically significant, and the HR returned to baseline around 10min after intubation, thus indicating stable haemodynamics.

When the heart rates in the two groups were compared the baseline values were comparable (p value-0.182). After induction there was decrease in heart rate in patients with EF<45% and increase in heart rate in patients with EF >45%, the difference between the two groups was significant (p value<0.001). After intubation at 1 min the increase in heart rate in patients with EF > 45% was significant as compared to patients with EF<45% (p value<0.001). After 3min, 5min & 10min of intubation there was gradual decrease in heart rate towards the baseline, the decrease in patients with EF >45% was significant as compared to patients with EF < 45% (p value <0.001, <0.001 & <0.001 respectively).

The above observations suggest that variation in heart rate at any time in both the groups were not more than 20% of baseline. The decrease in heart rate post induction in patients with EF < 45% and increase post intubation though statistically significant, was not clinically significant.

Similarly the increase in heart rate in patients with EF > 45% was only statistically significant and there was no tachycardia clinically noted. In both groups the rate returned towards baseline within 10min of intubation. When compared between the 2 groups the changes in heart rate was slightly more pronounced in patients with EF > 45%. The heart rate variability did not have any adverse clinical effect and patients were stable in both the groups, with EF<45% as well as in patients with EF>45%. Laryngoscopy did not produce any tachycardia and thereby not increasing the oxygen demand of myocardium. Similar were the findings in other studies.

RAVEEN SINGH *et al.* [5] did RCT of anaesthetic inducing agent in patients with CAD with LVEF<45% and observed that Etomidate was the least effective in minimizing the stress response, with a statistically significant increase from baseline in heart rate (P = 0.001) one minute after intubation. They recorded a decrease in HR from a baseline of 74.0±15.9bpm to 68.5±10.9bpm 3minutes post induction which then increased upto 84.9±12.5bpm immediately post intubation and returned below baseline within 5minutes. Similar trend was noted in our present study where there was a statistically significant rise in HR immediately post intubation with a lower HR post induction and 5 minutes post intubation in our patients with EF<45%.

Our results were supported by NAHID *et al.* [6] who in their study of effect of etomidate on haemodynamic in CAD patients with EF<45% and found similar results with etomidate- midazolam group as there was decrease in HR after induction and increase in HR after intubation. Thereafter gradual decreased at 3min post intubation.

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MOHAMMAD REZA HABIBI *et al*. [7] the effects of etomidate in CAD patients with EF<45% and similar to our study observed decrease in HR after induction and increase in HR after intubation. Thereafter gradual decrease in HR was noted at 3 min.

AFSHIN GHOLIPOUR BARADARI [8] studied the haemodynamic effects of etomidate in patients with LV dysfunction (EF<40%) and observed transient decrease in HR after induction and increase in HR after intubation but was not statistically significant as noted in our study. There was decrease in HR towards baseline at 3min.

The study conducted by KAUSHAL *et al*. [9] demonstrated an increase in HR post intubation similar to present study although no decrease in HR was observed post induction or 5minutes post intubation.

OZGUR *et al*. [10] too noted a decrease in HR after induction contrary to increase in HR in our study and significant rise in HR immediately post intubation that decreased to about baseline by 10minutes post intubation as observed in our study in patients with EF>45%.

SHAGUN BHATIA SHAH [11] studied the haemodynamic effects of etomidate as an induction agent and observed that there was transient decrease in HR after induction whereas there was increase in our study and was statistically significant. There was rise in HR after intubation similar to our study but on the contrary was not statistically significant.

VIKRAM SINGH RATHORE *et al*. [12] in support of our study observed fall in HR after induction and increase in HR after intubation but was not significant statistically. They observed a gradual decrease in HR thereafter at 3min & 5min towards baseline similar to our study.

Zed *et al*. studied the intubating conditions and haemodynamics with etomidate and observed that there was increase in HR after intubation as observed in our study and was significant. At 5min & 10min there was gradual decrease in HR below similar to our observation.

Supriya Aggarwal compared the effects of etomidate vs. propofol. They observed a transient increase in HR after induction and intubation, which decreased towards baseline at 10min after intubation as observed in our results.

Baseline SBP was noted down of all patients in both groups. The changes in SBP after induction and various intervals after intubation were compared with baseline and between the two groups.

In patients with EF<45% there was significant decrease in SBP after induction. After intubation at 1min there was significant increase in SBP. At 3min & 5min there was decrease in SBP towards baseline but was not significant. At 10 min there was fall in SBP below the baseline value.

In patients with EF > 45% after induction there was significant fall in SBP (p value<0.001). There was significant rise in SBP at 1min after intubation (p value 0.011). There was decrease in SBP at 3min, 5min and 10min but was significant at 5min & 10min (p value 0.459, 0.017 & <0.001 respectively).

On comparison of SBP between the 2 groups, there was decrease in SBP in the 2 groups and the difference was not significant. After intubation at 1 min there was comparable increase in SBP in both groups. There was decrease in SBP at 3min, 5min & 10min but the difference between the two groups was not statistically significant.

Our results suggest that there was statistically significant fall in SBP after induction in both the groups though these changes in both groups were comparable. Similarly there was also statistically significant rise in SBP in both the groups after intubation which was comparable. The SBP returned to baseline values within 3-5 min of intubation. In neither groups there was any hypotension or hypertension noted clinically, suggesting that response to laryngoscopy was minimal. Patients were stable with respect to systolic blood pressure and did not require any medical intervention.

AFSHIN GHOLIPOUR *et al*. [8] studied the effects of etomidate in CAD patients with EF<40% and observed that etomidate produced a significant decrease in SBP after induction and a similar rise in SBP after intubation. In support of our findings there was gradual fall in SBP at 3min post intubation.

Aghdaii N *et al*. studied the haemodynamic effects of etomidate and midazolam in patients with compromised LV function(EF<45) and observed that there was decrease in SBP after induction and increase after intubation, there was a gradual decreases in SBP thereafter as observed at 5min similar to the observations in our study.

Mohammad Reza Habibi *et al*. studied the effects of etomidate in patients with compromised LV function (EF<45%) and observed that there was decrease in SBP after induction and increase in SBP

after intubation similar to our results. At 3 min after intubation there was decrease in SBP below the baseline whereas in our study there was decrease in SBP but not below baseline.

Kaushal *et al*. [9] observed that etomidate caused a fall in SBP after induction. After intubation there was rise in SBP as observed in our study also. On the contrary there was no gradual decrease in SBP thereafter as observed in our study but maintained a similar value at 5min post intubation.

In the study by Shah *et al*., comparison of propofol and etomidate showed a fall in SBP following induction with etomidate followed by an increase after intubation and return to near baseline values 5minutes after intubation. This trend was similar to our study.

As per Zed *et al*., studying the characteristics of etomidate during intubation observed a statistically significant ($p<0.0001$) rise in SBP after intubation which declined towards baseline within 5min post intubation which was comparable to our study results. The haemodynamic stability of etomidate was attributed to the fact that etomidate does not cause histamine release and also its cerebroprotective property which made it ideal for patients with haemodynamic instability and head injury.

Baseline DBP was noted down of all patients in both groups. The changes in DBP after induction and various intervals after intubation were compared with baseline and between the two groups.

In patients with EF<45% significant decrease in DBP post induction was noted. At 1 min after intubation there was significant rise in DBP. At 3min & 5min after intubation there was decrease in DBP towards baseline but was not statistically significant. At 10 min after intubation there was fall in DBP below baseline but was not significant.

In patients with EF>45% there was significant decrease in DBP after induction. After intubation at 1 min significant rise in DBP was observed. At 3min & 5 min there was gradual decrease in DBP and returned towards baseline at 10min post intubation.

On comparing the changes in DBP in the 2 groups, it was observed that the baseline values in both groups were comparable. After induction the decrease in patients with EF<45% was significant as compared to patients with EF>45% (p value 0.016). After induction at 1min the rise in BP in patients with EF>45% was significant as compared to rise in patients with EF<45% (p value 0.005). At 3min & 5min there was fall in DBP in both groups and the difference between the two groups was statistically significant (p value 0.014 & 0.006 respectively). The decrease in DBP at 10min was not significant.

This suggests that fall noted after induction in both patients with EF<45% and EF>45% was statistically significant but was within the 20% baseline values clinically. Similarly the increase in DBP following intubation did not cause hypertension nor did it require any intervention. The changes were comparable between the 2 groups. These changes in DBP returned towards baseline in 5-10min of intubation. This suggests that the changes were transient and not clinically significant. As with SBP laryngoscopy did not cause clinically significant rise in DBP.

AFSHIN GHOLIPOUR *et al*. [8] studied the haemodynamic effects of etomidate in patients with LV dysfunction with EF<40%, similar to the results in our study they observed that there was decrease in DBP after induction and increase in DBP after intubation, but contrary to our results there was increase in DBP at 3min rather than a fall in DBP as observed in our results.

KAUSHAL *et al*. [9] studied the effect of etomidate on haemodynamics in coronary artery disease patients, in support of our results they observed decrease in DBP after induction & increase in DBP after intubation, but after intubation at 5 min there was no fall in DBP but was maintained at post intubation level.

Our results are consistent with the results of SHAH *et al*. who in their study observed a significant decrease in DBP following induction with etomidate with an increase in the same following intubation and a return to baseline values 5min post intubation. Haemodynamic stability may be due to its lack of effect on sympathetic nervous system and baroreceptor function. Also, its capacity to stimulate peripheral alpha 2b adrenergic receptors leads to vasoconstriction limiting its hypotensive action.

According to ZED *et al*. there was rise in DBP following intubation which returned to baseline at about 15min post intubation and was not statistically significant whereas in our study the rise in DBP was transient but statistically significant which returned to baseline within 5 min if intubation (p value 0.007).

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Conclusion

Etomidate provided haemodynamic stability during induction with minimal changes in HR and BP. It also showed minimal response during intubation. There were no adverse events such as myocardial ischemia and arrhythmias. This haemodynamic stability was noted irrespective of the degree of cardiac dysfunction. This helps in preserving myocardial oxygen balance and thereby improving the peri-operative outcome in patients with Coronary artery disease (CAD) undergoing non-cardiac surgeries.

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