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A Comparative Study of four different induction methods (Thiopentone, Propofol, Etomidate, and Propofol plus Etomidate) in maintaining Cardiovascular and Hemodynamic stability following Endotracheal Intubation in Elective Surgeries

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ABSTRACT

The objective of the study was to compare efficacy of four different induction methods (thiopentone,propofol, etomidate and propofol plus etomidate) in maintaining hemodynamic stabilityfollowing induction and endotracheal intubation in patients scheduled for elective surgeries under general anaesthesia. 120 patients, aged between 15 to 60 years, of either gender, and ASA physical status I and II, were randomized into four equal groups of 30 each. Group I: received Thiopentone (5 mg/kg body weight) intravenously (i.v.); Group II: received Propofol (2.5 mg/kg body weight) i.v.; Group III: received Etomidate (0.3 mg/kg body weight) i.v.; Group IV: received Propofol (1 mg/kg body weight) plus Etomidate (0.2 mg/kg body weight) i.v.Heart rate, systemic blood pressure and oxygen saturation of all patients were monitored and recorded at baseline, before induction, after induction and 1 minute, 2 minute, 3 minute, 5 minute after intubation. Significant difference was found in heart rate among all four groups at different time intervals, except at induction and 5th minute after intubation between group III and group IV. SBP revealed significant difference among various groups at different points of time except at induction, 2nd, 3rd, 5th min after intubation among group III and group IV. DBP had significant difference among all the groups, except group III and IV, at induction, 2nd, 3rd, 5th minute after intubation. Mean blood pressure revealed significant differences among various groups at different points of time, except between group III and group IV, wherethere was significant difference only at 1 min after intubation.The study reveals that co-administration of etomidate with propofoleffectively attenuates intubation reflex thanetomidate, propofol or thiopentone alone.

KEYWORDS; Thiopentone, propofol, etomidate, intubation, hemodynamics

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INTRODUCTION

In general anesthesia, airway management is of utmost importance. It can be achieved with either endotracheal tube or supraglottic airway devices.[1,2] Endotracheal intubation still remains the gold standard in airway management, as it is the safest method of protecting the airway and delivering anesthetic gases.[3,4] Among many available methods of tracheal intubation, direct laryngoscopy is the widely accepted method.[5,6,7,8] The endotracheal tube is introduced into the trachea under direct vision ensuring, thereby, protection against aspiration of gastric contents. The intubation response during direct laryngoscopy, however, may prove to be detrimental in patients with cardiac risk factors such as hypertension and ischemic heart disease.[9] The adverse effects of direct laryngoscopy include cardiac dysrhythmia, hypertension, myocardial ischemia, hypoxia, hypercapnia, laryngospasm, bronchospasm and increased intracranial and intraocular pressure.

In general anesthesia, an intravenous induction agent of choice would be the one that preserves hemodynamic stability during induction and endotracheal intubation, produces minimal respiratory side effects and undergoes rapid clearance.[10,11] No such ideal induction agent, however, exists. Different induction agent used in common clinical practice are thiopentone, propofol, etomidate, ketamine.[12] There are very few published studies in the literature that have compared the physiological effect of various induction agents during laryngoscopy and intubation.Hamzeh et al. compared three methods of induction of anesthesia (propofol, etomidate, and propofol plus etomidate) to study the hemodynamic stability after laryngeal mask airway insertion in elective surgeries.[13]

The objective of this study was to compare the efficacy of different anesthetic induction methods, viz. thiopentone, propofol, etomidate and a propofol plus etomidate in maintaining hemodynamic stability during induction and following endotracheal intubation in patients scheduled for elective surgery.

MATERIALS AND METHODS

After approval of institutional ethical committee, 120 patients aged between 15 to 60 years, of either sex and ASA physical status I and II, scheduled for elective surgery under general anesthesia were enrolled for the study. The exclusion criteria were as follows: patient refusal, ASA physical status III and IV, emergency surgery, patient with history of hypersensitivity to either of the study drugs (i.e. thiopentone, propofol or etomidate), restricted mouth opening (< 2.5 cm), Mallampati grade 3 and 4, presence of any pathology in pharynx or larynx, presence of systemic diseases like ischemic heart disease, hypertension, bronchial asthma, diabetes mellitus and porphyria.

After obtaining written and informed consent, the patients were randomly, but equally placed into four different groups using a random number table.

Group I: Induction with thiopentone (5 mg/kg body weight) i.v.

Group II: Induction with propofol (2.5 mg/kg body weight) i.v.

Group III: Induction with etomidate (0.3 mg/kg body weight) i.v.

Group IV: Induction with propofol (1 mg/kg body weight) plus etomidate (0.2 mg/kg body weight) i.v.

Airway assessment like mouth opening (inter-incisor gap), Mallampati grade, dentition and neck flexion and extension of all patients was done. Baseline (preoperative) heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP) and oxygen saturation(SpO_2) were noted during the pre-anesthetic check-up. All patients were given premedication with tab. alprozolam 0.25 mg, tab. ranitidine 150 mg and tab. metoclopramide 10 mg, the night before surgery and in the morning two hours prior to surgery. The patients were kept nil per oral for 8 hours for solids and 4 hours for clear liquids prior to surgery. On arrival at the operation room, standard anaesthesia monitoring, including continuous electrocardiogram (ECG), non-invasive blood pressure (NIBP) and pulse oximetry were attached and baseline haemodynamic parameters were recorded. An 18 G intravanouscannula was secured on the dorsum of the left hand. Inj. fentanyl 2 µg/kg i.v., and inj. midazolam 0.025 mg/kg i.v. were given 2 minutes before induction. Thereafter, group I received inj. thiopentone 5 mg/kg i.v.; Group II received inj. propofol 2.5 mg/kg i.v.; group III received inj. etomidate 0.3 mg/kg i.v.; and group IV received inj. propofol 1 mg/kg plus inj. etomidate 0.2 mg/kg i.v. as an induction agent. After loss of consciousness, as decided by inability to respond to verbal commands, bag and mask ventilation was continued. Inj. vecuronium (0.1 mg/kg) was administered to facilitate endotracheal intubation. The patients were intubated with appropriate size endotracheal tube (ET). Proper placement of endotracheal tube was confirmed by capnography and bilateral auscultation of the chest. Following successful placement of ET tube, anesthesia was maintained by oxygen and nitrous oxide (40:60) mixture, isoflurane (1-1.5%) and along with intermittent doses of inj. fentanyl and inj. vecuronium. At the end of the surgery, the residual neuromuscular blockade was antagonized with inj. neostigmine (0.05 mg/kg) i.v. and inj. glycopyrolate (0.01 mg/kg) i.v.Extubation was performed when respiration was adequate and patient was able to obey verbal commands.

Heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure and oxygen saturation were continuously monitored and recorded before induction, after induction and at 1 minute, 2 minute, 3 minute, 5 minute after intubation. The obtained data were compared and presented as mean±SD, frequency and percentage. The qualitative variables were compared using

Chi-square test, while the quantitative parameters were compared using one-way analysis of variance (ANOVA) test, followed by post-hoc test. A p-value less than 0.05 was taken as significant.

RESULTS

The demographic data viz. age, weight, basal metabolic index (BMI), gender and ASA physical status of patients of all the groups were comparable and there was no significant difference. (Table 1)

Table 1: Demographic profile

	Group I(n=30)	**Group II**(n=30)	**Group III** (n=30)	**Group IV** (n=30)	**p-value**
Age (years) (Mean ± SD)	37.23 ± 7.70	34.47 ± 6.72	33.90 ± 6.28	37.30 ± 9.39	0.178
Weight (kg) (Mean ± SD)	62.60 ± 4.91	59.93 ± 7.04	58.77 ± 6.38	61.45 ± 5.28	0.115
BMI (kg/m^2) (Mean ± SD)	22.43 ± 1.20	22.46± 2.59	21.99 ± 1.95	22.77 ± 2.73	0.704
Gender					0.241
M	20	14	15	20	
F	10	16	15	10	
ASA grade					0.475
I	17	15	20	20	
II	13	15	10	10	

The heart rates (HR) (mean±SD) at each time interval among the four groups were compared for statistical evaluation. Baseline and pre-induction HR were comparable among all four groups with no statistical significant differences ($p > 0.05$). Inter group comparison showed that there were significant differences ($p < 0.05$) in heart rate among all four groups at time interval (after induction and 1, 2, 3 min after intubation). At 5 min after intubation, there were significant differences among groups, except between group III and group IV. (Table 2a, 2b; Fig 1)

Table 2a: Heart rate (HR) (beats per minute)

Time interval	Group I	Group II	Group III	Group IV	f-value	p-value
Baseline	76.17±7.231	78.33±6.572	76.40±6.667	77.30±5.466	0.686	0.562
Pre induction	88.23±7.477	89.60±5.975	88.03±6.775	88.33±5.785	0.355	0.786
After induction	101.17±6.539	69.43±5.151	88.27±7.249	82.63±6.780	123.808	0.000
1min after intubation	119.57±6.129	76.57±4.539	99.30±5.926	93.50±6.648	274.713	0.000
2min after intubation	110.20±7.854	80.13±4.747	96.37±6.031	91.17±6.747	112.567	0.000
3 min after intubation	102.70±9.296	83.27±4.863	94.40±5.852	90.17±6.018	43.931	0.000
5 min after intubation	96.30±8.293	85.43±4.337	92.50±6.096	89.83±5.670	15.984	0.000

Table 2b: Group comparison of Heart Rate (HR) (beats per minute)

Time interval	Group I vs. II	Group I vs. III	Group I vs. IV	Group II vs. III	Group II vs. IV	Group III vs. IV
Baseline	0.200	0.890	0.502	0.253	0.540	0.594
Pre induction	0.420	0.906	0.953	0.355	0.455	0.859
After induction	0.000	0.000	0.000	0.000	0.000	0.001
1 min after intubation	0.000	0.000	0.000	0.000	0.000	0.000
2 min after intubation	0.000	0.000	0.000	0.000	0.000	0.002
3 min after intubation	0.000	0.000	0.000	0.000	0.000	0.016
5 min after intubation	0.000	0.020	0.000	0.000	0.008	0.102

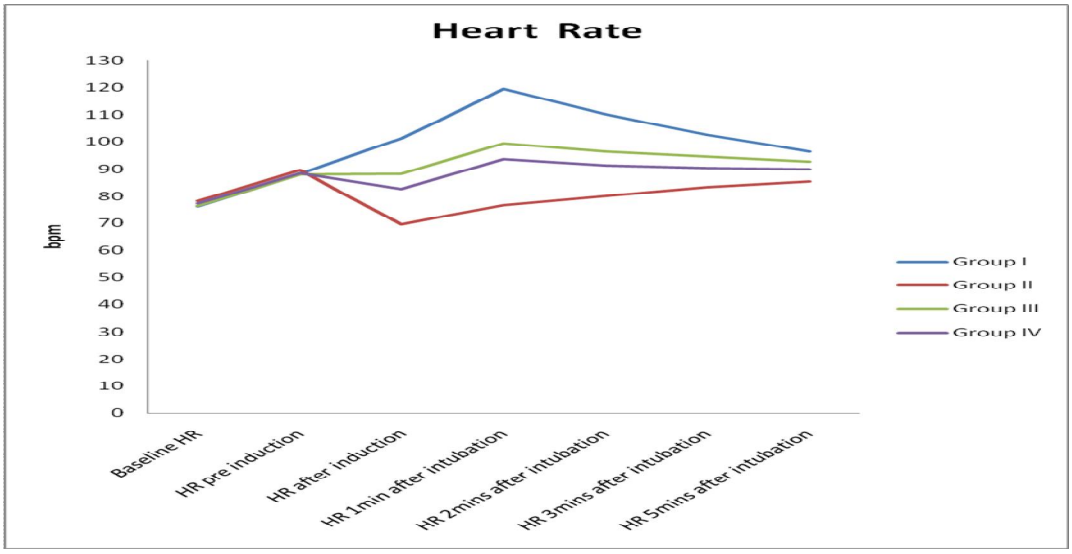

Fig 1: Heart rate (HR) at various intervals

The systolic blood pressures (SBP) (mean±SD) at each time interval among the four groups were compared for statistical evaluation. Baseline and pre-induction SBP were comparable among all the four groups with no statistical significant differences ($p > 0.05$). But SBP of four groups after induction and at 1, 2, 3, 5 minute after intubation were different both clinically and statistically, with p value <0.05. Inter group comparison of SBP (mean±SD) revealed significant differences among various groups at different points of time except that among group III and group IV. Between group III and group IV there was significant difference only at 1 min after intubation (Table 3a, 3b; Fig 2)

Table 3a: Systolic blood pressure (SBP) (mmHg)

Time interval	Group I	Group II	Group III	Group IV	f-value	p-value
Baseline	129.27±5.420	129.87±6.146	127.83±5.376	127.80±7.208	0.876	0.456
Preinduction	123.57±5.456	125.50±6.067	123.67±5.839	124.97±7.117	0.730	0.536
After induction	114.07±5.930	100.53±8.905	117.73±5.705	118.40±6.750	43.148	0.000
1 min after intubation	145.00±6.742	111.77±6.474	133.87±5.758	130.57±4.826	169.731	0.000
2 min after intubation	135.93±4.323	115.33±7.906	129.10±3.836	126.97±3.891	79.327	0.000
3 min after intubation	132.07±4.177	121.73±4.586	125.30±4.473	125.20±3.995	30.153	0.000
5 min after intubation	130.87±4.869	126.83±3.270	122.47±5.457	123.50±4.431	20.563	0.000

Table 3b: Group comparison of systolic blood pressure (SBP) (mmHg)

Time interval	Group I vs. II	Group I vs. III	Group I vs. IV	Group II vs. III	Group II vs. IV	Group III vs. IV
Baseline SBP	0.703	0.363	0.352	0.198	0.191	0.983
SBP preinduction	0.226	0.950	0.380	0.251	0.738	0.415
SBP after induction	0.000	0.043	0.017	0.000	0.000	0.710
SBP 1min after intubation	0.000	0.000	0.000	0.000	0.000	0.035
SBP 2 min after intubation	0.000	0.000	0.000	0.000	0.000	0.120
SBP 3 min after intubation	0.000	0.000	0.000	0.002	0.002	0.929
SBP 5 min after intubation	0.001	0.000	0.000	0.000	0.006	0.384

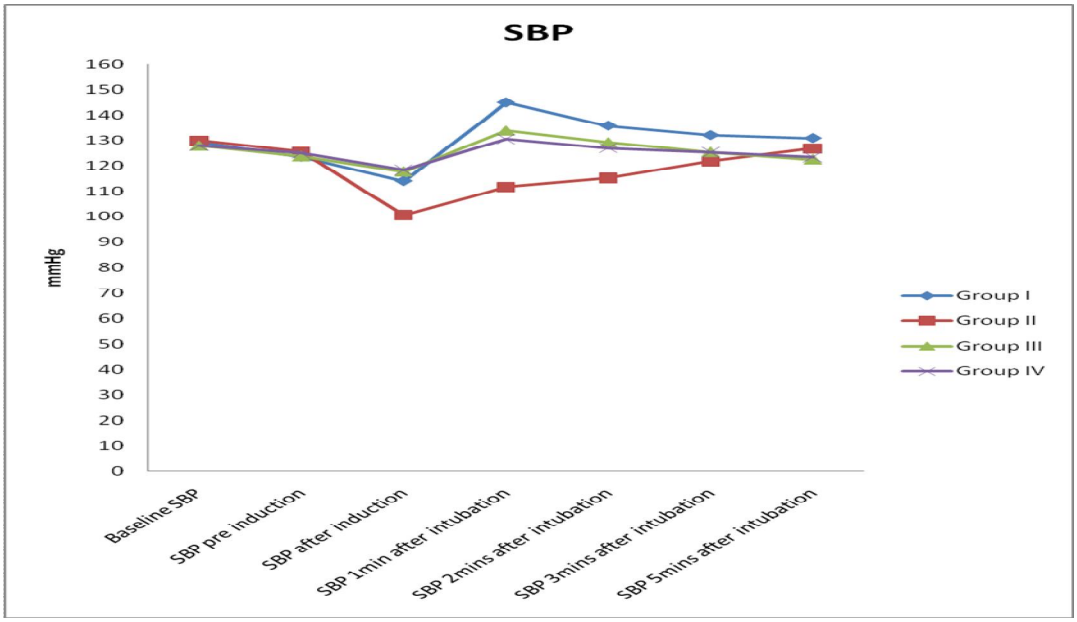

Fig 2: Systolic blood pressure at various intervals

Table 4a: Diastolic blood pressure (DBP) (mmHg)

Time interval	Group I	Group II	Group III	Group IV	f-value	p-value
Baseline	75.93±5.105	75.80±6.228	74.70±4.757	75.23±5.184	0.336	0.799
Pre induction	72.27±4.160	73.23±6.447	72.17±4.340	71.90±5.498	0.377	0.770
After induction	65.47±2.933	60.30±4.236	68.00±4.307	68.30±5.338	22.357	0.000
1 min after intubation	85.80±7.752	65.63±3.728	77.00±4.299	73.13±4.183	76.835	0.000
2 min after intubation	82.77±8.046	67.37±3.285	73.00±3.833	72.27±3.805	47.669	0.000
3 min after intubation	78.57±5.374	68.43±3.191	72.37±3.023	71.43±3.598	35.550	0.000
5 min after intubation	76.43±4.710	72.40±2.943	71.43±2.269	70.27±4.093	16.330	0.000

The diastolic blood pressures (DBP) (mean ± SD) at each time interval among the four groups were compared for statistical evaluation. Baseline and pre-induction DBP were comparable among all the four groups with no statistical significant differences ($p > 0.05$). But DBP of four groups after induction and at 1,2,3,5 minute after intubation were different both clinically and statistically, with p value <0.05. There were significant differences ($p<0.05$) in inter group comparison of DBP (mean±SD) among the groups except group III and IV. But there was significant difference between group III and IV only at 1 min after intubation. At 5 min after intubation there were no significant differences between group II versus III and group III versus IV. (Table 4a, 4b; Fig 3)

Table 4b: Group comparison of diastolic blood pressure (DBP) (mmHg)

Time interval	Group I vs. II	Group I vs. III	Group I vs. IV	Group II vs. III	Group II vs. IV	Group III vs. IV
Baseline	0.923	0.374	0.613	0.427	0.682	0.700
Pre induction	0.473	0.941	0.785	0.428	0.322	0.843
After induction	0.000	0.024	0.012	0.000	0.000	0.787
1 min after intubation	0.000	0.000	0.000	0.000	0.000	0.005
2 min after intubation	0.000	0.000	0.000	0.000	0.000	0.580
3 min after intubation	0.000	0.000	0.000	0.000	0.004	0.357
5 miafter intubation	0.000	0.000	0.000	0.305	0.025	0.216

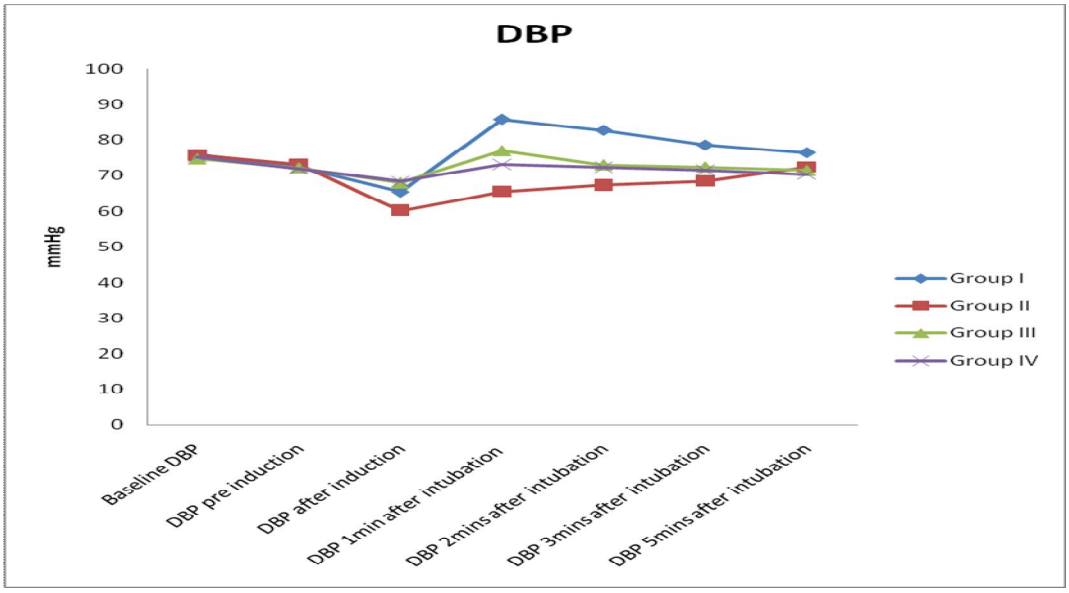

Fig 3: Diastolic blood pressure (DBP) at various intervals

The mean blood pressures (MBP) (mean±SD) at each time interval among the four groups were compared for statistical evaluation. Baseline and pre-induction MBP were comparable among all the four groups with no statistical significant differences (p >0.05). But MAP of four groups after induction and at 1,2,3,5 minute after intubation were different both clinically and statistically, with p value <0.05.

Table 5a: Mean blood pressure (MBP) (mmHg)

Time interval	Group I	Group II	Group III	Group IV	f-value	p-value
Baseline	93.43±4.256	93.70±5.383	92.17±4.379	91.73±5.638	1.119	0.344
Pre induction	89.37±3.792	89.57±4.783	88.57±4.321	89.53±5.686	0.300	0.826
After induction	81.66±3.051	73.71±4.876	84.57±4.192	85.00±5.425	41.019	0.000
1 min after intubation	105.53±6.433	81.67±3.695	95.95±4.082	92.77±4.066	143.549	0.000
2 min after intubation	100.48±5.439	83.35±3.927	91.70±3.081	90.50±3.555	88.266	0.000
3 min after intubation	96.40±3.778	86.20±2.919	90.01±2.484	89.35±3.504	53.174	0.000
5 min after intubation	94.57±3.305	90.54±2.453	88.44±2.528	88.01±3.830	28.420	0.000

Inter group comparison of MAP (mean±SD) revealed significant differences among various groups at different points of time except that among group III vs group IV. Between group III and group IV, there was significant difference only at 1 min after intubation. (Table 5a, 5b; Fig 4)

Table 5b: Group comparison of mean blood pressure (MBP) (mmHg)

Time interval	**Group I vs. II**	**Group I vs. III**	**Group I vs. IV**	**Group II vs. III**	**Group II vs. IV**	**Group III vs. IV**
Baseline	0.835	0.324	0.186	0.233	0.127	0.735
Pre induction	0.869	0.511	0.891	0.411	0.978	0.427
After induction	0.000	0.013	0.005	0.000	0.000	0.715
1 min after intubation	0.000	0.000	0.000	0.000	0.000	0.003
2 min after intubation	0.000	0.000	0.000	0.000	0.000	0.259
3 min after intubation	0.000	0.000	0.000	0.000	0.000	0.431
5 min after intubation	0.000	0.000	0.000	0.009	0.002	0.587

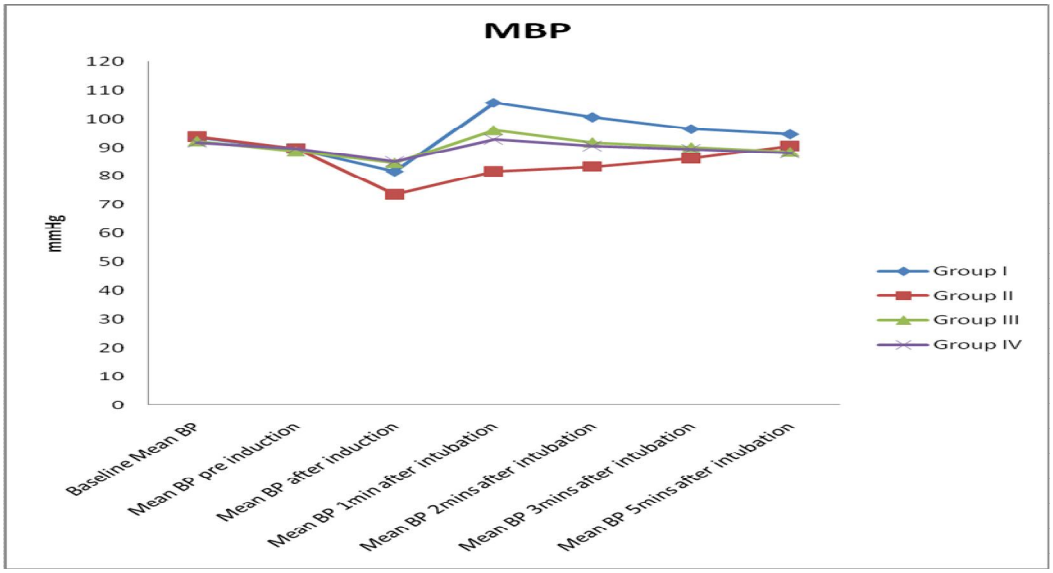

Fig 4: Mean blood pressure at different intervals

There were no significant differences in terms of oxygen saturation among four groups at baseline, preinduction, after induction and 1,2,3,5 minutes after intubation. (p-value>0.05.) (Table 6a, 6b)

Table 6a: Oxygen saturation (SpO_2) (%)

Time interval	Group I	Group II	Group III	Group IV	f-value	p-value
Baseline	99.60±0.498	99.63±0.490	99.57±0.504	99.50±0.509	1.744	0.762
Pre induction	99.80±0.407	99.73±0.450	99.73±0.450	99.73±0.450	0.173	0.915
After induction	99.77±0.430	99.73±0.450	99.77±0.430	99.73±0.450	0.057	0.982
1 min after intubation	99.93±0.254	99.97±0.183	100.00±0.000	100.00±0.000	2.109	0.295
2 min after intubation	99.90±0.305	99.90±0.305	99.90±0.305	99.90±0.305	0.000	1.000
3 min after intubation	99.90±0.305	99.90±0.305	99.93±0.254	99.93±0.254	0.141	0.935
5 min after intubation	100.00±0.000	100.00±0.000	100.00±0.000	100.00±0.000	0.000	1.00

Table 6b: Group comparison of oxygen saturation (SpO_2)

Time interval	**Group I vs. II**	**Group I vs. III**	**Group I vs. IV**	**Group II vs. III**	**Group II vs. IV**	**Group III vs. IV**
Baseline	0.797	0.797	0.440	0.607	0.304	0.607
Pre induction	0.558	0.558	0.558	1.000	1.000	1.000
After induction	0.770	1.000	0.770	0.770	1.000	0.770
1minafterintubation	0.410	0.101	0.101	\0.410	0.410	1.000
2minafterintubation	1.000	1.000	1.000	1.000	1.000	1.000
3minafterintubation	1.000	0.646	0.646	0.646	0.646	1.000
5minafterintubation	1.000	1.000	1.000	1.000	1.000	1.000

DISCUSSION

The common cardiovascular response to intubation is increase in heart rate and arterial blood pressure due to an increase in sympathetic activity;[14] although bradycardia associated with increased parasympathetic activity, are also known.[15] Myocardial oxygenation in patients with coronary insufficiency may be severely compromised under these circumstances and ischaemic changes and actual infarction have been reported.[16,17,18] Hypertension and tachycardia during laryngoscopy and intubation may lead to dysrhythmias,[15] and reduction in ejection fraction.[19] Cases of frank left ventricular failure have been described.[20] Cerebral haemorrhage may also occur and convulsions may be precipitated in mothers with pre-eclampsia. Hypertensive patients, even if they receive therapy, are prone to tachycardia and dysrhythmia.[21]

Several methods have been used in an attempt to attenuate this response. Topical anaesthesia of the larynx and pharynx with lignocaine spray has been proven to be unsuccessful because of the need to perform laryngoscopy with resultant stretching and pressure on the tissues of the larynx and pharynx.[21,22] It is interesting that blind nasal intubation without laryngoscopy, did not result in any cardiovascular sequel. However, intravenous lignocaine in a dose of 1.5 mg/kg effectively attenuates the hypertensive response and prevents tachycardia and dysrythrmia.[21,23] Deeper level of anaesthesia reduces the cardiovascular effects of laryngoscopy and intubation,[14] although volatile agents appear to control the changes in arterial pressure more effectively than the changes in heart rate. However, deeper level of anaesthesia does not allow rapid sequence intubation and may unduly delay recovery after short surgical procedures; the associated hypotension may also be undesirable, particularly in patients with coronary insufficiency.

Fentanyl 5µg/kg at induction of anaesthesia effectively prevents the haemodynamic effects of tracheal intubation, while smaller doses attenuate it.[24,25]Alfentanil 30 µg/kg[26] and sufentanil 0.5-1.05µg/kg[27] are also effective. However, the respiratory depression associated with these drugs may be a problem in short procedures, although less so with alfentanil. Beta-adrenoceptor blockade has been advocated as a method to protect against the effects of laryngoscopy, particularly in patients with co-existing hypertension,[28] but may, at times, produce hypotension and bradycardia. Vasodilators may also result in profound hypotension once the stimulus of laryngoscopy is removed. Sodium nitroprusside, due to its short-lived action, has been recommended,[29] but requires intensive monitoring and may itself cause a tachycardia.Harris et al.[30] compared the haemodynamic response to tracheal intubation in 303 patients in whom anaesthesia was induced with either thiopentone 4 mg/kg, etomidate 0.3mg/kg or propofol 2.5mg/kg with or without fentanyl 2µg/kg. After induction with propofol alone, there was a significant reduction in arterial blood pressure, which did not rise

above control value after intubation. On the other hand, significant increase in arterial pressure followed intubation in patients induced with thiopentone or etomidate alone. Increase in heart rate was observed with all agents after laryngoscopy and intubation. The use of fentanyl resulted in decreased systemic blood pressure than those after the induction agent alone. Similar results were observed in the present study. While significant decrease in systemic blood pressure occurred after induction with propofol, which did not increase above baseline value after intubation, there was significant increase in arterial pressure following intubation in the thiopentone or etomidate groups,. Also, rise in heart rate followed laryngoscopy and intubation in all groups. Hug et al. indicated that propofol would lead to bradycardia and hypotension in 4.2% and15.7% of patients respectively.[31] Furthermore, Reves et al[32] and Hiller et al[33] have shown that induction with propofol at the dose of 2-2.5 mg/kg of body weight reduced blood pressure by 20-40% irrespective of presence or absence of any underlying condition. Schmidt et al.[34] found that propofol-induced hypotension is due to the reduction of preload and afterload, which are not synchronized with heart's compensatory responses such as increased cardiac output and increased HR. The hemodynamic instability is further aggravated by high doses of the drug and greater speed injection of the drug.

Brohonet al. studied the effect of propofol or etomidate in combination with alfentanil or sulfentanil on lumbar spinal surgeries and observed that systemic blood pressure decreased in etomidate group in combination with sulfentanil or alfentanil, but remained unchanged in propofol group in combination with either of them.[35] The study of Boisson-Bertrand et al. showed that propofol is suggested for patients who need good post-operative cooperation and etomidate for those who are hemodynamically compromised.[36] Fuchs T. et al. concluded that etomidate induction along with alfentanil and rocuronium, attenuated the reaction to intubation to a greater extent than thiopentone[37]. Likewise, our study had less haemodynamic variation after induction and endotracheal intubation in etomidate group as compared to thiopentone anaesthesia induction. Similarly Scott Jelish W. et al concluded that high-dose etomidate induction, titrated to electroencephalograhy (EEG) burst suppression, preserved stable haemodynamics stability during laryngoscopy and intubation as compared with lower dose, more classic induction with etomidate and thiopentone.[38]NareshDhawanet al concluded that the etomidate at 0.3 mg/kg produces very minimal changes in hemodynamic parameters and shunt fraction in children with congenital shunt lesion.[39] Mehrdad et al. too concluded that patients receiving etomidate have more stable hemodynamic condition, and hence it could be preferred to propofol for induction of general anaesthesia provided there are no contraindications to etomidate.[40] Hamzeh H et al. concluded that Etomidate plus propofol is an effective and alternative to propofol and etomidate alone for facilitating LMA insertion (in terms of number of attempts and insertion ease) with the added advantage of lack of

cardio-vascular depression.[11] The results of the present study also show that the combination of etomidate with propofol produces better haemodynamic stability than etomidate alone at 1 min after intubation, though there was no significant difference at other points of time. Moreover the combination of propofol and etomidate produces haemodynamic stability significantly better than either propofol or thiopentone alone.

CONCLUSION

To summarise, induction with thiopentone alone is not satisfactory as the hemodynamic responses to tracheal intubation need to be attenuated in patients with cardiac diseases. Induction with propofol alone is acceptable in patients with stable haemodynamics; however, it may cause hypotension in volume depleted patients. The combination of etomidate plus propofol has better haemodynamic stability than etomidate alone at 1 min after intubation, though etomidate alone produce comparable haemodynamic stability at other points of time. The combination of propofol and etomidate proved to be significantly better than either propofol or thiopentone alone.

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