Original Research Article

A comparative study of different doses of esmolol to attenuate pressor response of laryngoscopy and endotracheal intubation

Shiva Kumar B S[1], Ravi Kumar[2*]

[1]Assistant Professor, Department of Anaesthesia, Sri Siddartha Medical College, Agalakote, Tumkur, Karnataka, INDIA.
[2]Professor, Department of Anaesthesia, MMCH, Modakkallur, Calicut, Kerala, INDIA.
Email: rknnese@gmail.com, ravinese@yahoo.com

Abstract

Background: Most patients undergoing surgery under general anesthesia invariably needs laryngoscopy and intubation, and is invariably associated with pressor response like cardiovascular changes such as tachycardia, rise in blood pressure and wide variety of cardiac arrythmias. These effects are deleterious in susceptible individuals leading to perioperative myocardial ischemia, acute heart failure and cerebrovascular accidents. **Objectives:** The present study is designed to determine the effective bolus dose of esmolol which would attenuate the pressor response to laryngoscopy and endotracheal intubation. **Methods:** A randomized control study was carried out on 100 patients above 18 years belonging to ASA physical status I or II, scheduled for non cardiac surgery under general anesthesia. Patients were randomly allocated into 4 groups of 25 each. All patients were preoxygenated with 100% oxygen for 3 minutes. Patients in group A received 10 ml normal saline, group B ,group C and group D receiving esmolol 50 mg, 100 mg, and esmolol 150 mg respectively, intravenously 2 minutes before intubation. Anesthesia was induced with Inj Propofol 2 mg kg^{-1} and tracheal intubation facilitated with Inj succinylcholine 1.5mgkg^{-1}. Laryngoscopy and intubation was performed by single investigator. The heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were recorded before induction, after induction and after intubation and thereafter every minute for 7 minutes. **Results:** The maximum rise in heart rate was 45% in control group compared to 35.6% in patients receiving esmolol 50 mg, 13.8% in esmolol 100 mg and 6.4 % in esmolol 100 mg group. The maximum rise in systolic blood pressure in control group was 23.6% compared to 19.9% in those receiving esmolol 50 mg and 6.2% in esmolol 100 mg group. The systolic blood pressure was below the baseline throughout the study period with a decrease of 5.5 % in patients given esmolol 150 mg. **Conclusion:** In this study all the groups in which esmolol was used showed a decrease in mean value of systolic blood pressure, diastolic blood pressure, mean arterial pressure after administration of drug. Esmolol 100 mg group adequately attenuated the heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure.

Key Word: laryngoscopy and endotracheal intubation.

[*]**Address for Correspondence:**
Dr Ravi Kumar, Professor, Department of Anaesthesia, MMCH, MOdakkallur, Calicut, Kerala, INDIA.
Email: rknnese@gmail.com, ravinese@yahoo.com
Received Date: 20/11/2019 Revised Date: 19/12/2019 Accepted Date: 11/01/2020
DOI: https://doi.org/10.26611/101513213

Access this article online	
Quick Response Code:	Website: www.medpulse.in
	Accessed Date: 04 February 2020

INTRODUCTION

Most patients undergoing surgery under general anesthesia invariably needs laryngoscopy and intubation, and is invariably associated with pressor response like cardiovascular changes such as tachycardia, rise in blood pressure and wide variety of cardiac arrythmias[1] .These effects are deleterious in susceptible individuals leading to perioperative myocardial ischemia, acute heart failure and cerebrovascular accidents[2,3] Esmolol is a cardioselective β-1 adrenergic blocking agent. It has rapid onset and short duration of action with a elimination half life of 9 minutes. It is an ester and is rapidly metabolized by esterase in the blood to a free acid metabolite that has beta adrenergic potency of 1/1600 of esmolol. Its kinetics are therefore suited to a relatively short application without causing prolonged bradycardia or hypotension and it has been shown to be effective in attenuating the pressor response to laryngoscopy and endotracheal intubation.[4,5,6,7] Studies have shown that use of intravenous lidocaine controls blood pressure

How to cite this article: Shiva Kumar B S, Ravi Kumar. A comparative study of different doses of esmolol to attenuate pressor response of laryngoscopy and endotracheal intubation. *MedPulse International Journal of Anesthesiology.* February 2020; 13(2): 104-112.

better than heart rate.[8,9] Opioids[10,11,13], and vasodilators[12,13] have also been used and are not equally effective in controlling blood pressure and heart rate.. Hence the present study is designed to determine the effective bolus dose of esmolol which would attenuate the pressor response to laryngoscopy and endotracheal intubation

METHODOLOGY

The randomized prospective study to compare the attenuation of pressor response to laryngoscopy and endotracheal intubation with different doses of esmolol was undertaken. The Institutional Ethical Committee approved the study and written informed consent was obtained from all the patients before being included in the study. Study population consisted of 100 patients who were randomly divided into 4 groups of 25 patients each. Group A received normal saline. Group B received esmolol 50 mg. Group C received esmolol 100 mg. Group D received esmolol 150 mg.

INCLUSION CRITERIA: All patients above 18 years. Patients belonging to American Society of anesthesiologists' physical status I and II.

EXCLUSION CRITERIA: Patients with conduction block, cardiac arrythmias. Congestive cardiac failure, bronchial asthama, and on beta blocker treatment

- Patients with anticipated difficult airway.

Patients satisfying the above said inclusion and exclusion criteria were subjected to study. All patients received alprozalam 0.5mg and ranitidine 150 mg orally on the night before surgery. They were randomly allocated into 4 groups. All patients were premedicated with Inj glycopyrrolate 0.2mg and Inj fentanyl 1 µgm kg^{-1} intravenously 30 minutes before surgery. Baseline reading of heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were recorded. All patients were preoxygenated with 100% oxygen for 3 minutes. The study group received either 10 ml normal saline, 50 mg esmolol , 100 mg esmolol, and 150 mg esmolol made to 10 ml with normal saline intravenous bolus over 15 seconds. The heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were recorded and anesthesia was induced with Inj Propofol 2 mg kg^{-1} and tracheal intubation facilitated with Inj succinyl choline 1.5mg kg^{-1} . Laryngoscopy and intubation performed by single investigator after 2 minutes of study drug administration. Laryngoscopy was done using rigid laryngoscope with standard Macintosh blade. Intubation was done with appropriate sized, disposable, high volume low pressure cuffed endotracheal tube. Oral intubation was done for all surgical procedures. Laryngoscopy and intubation was done within 15 to 20 seconds. The heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were recorded after induction, after intubation and thereafter every minute for 7 minutes. Anesthesia was maintained with O_2 (33%),N_2O(67%), and intermittent vecuronium 0.05mg kg^{-1} IV and intermittent positive pressure ventilation (IPPV) using Bain's circuit. All surgical stimuli, analgesics supplements and inhaled anesthetics were avoided during the study. At the end of surgery patients were reversed with neostigmine 0.05 mg kg^{-1} IV and glycopyrrolate 0.01 mg kg^{-1} IV. The data was analysed using ANOVA for intergroup comparison and all statistical methods were carried out through the SSPS for Windows (version 16.0). The results were considered significant when p value <0.05.

RESULTS

TABLE 1: AGE DISTRUBUTION

GROUP	N	Mean	Std. Deviation	Minimum	Maximum
A	25	33.28	10.110	20	54
B	25	35.04	11.149	20	60
C	25	39.24	11.099	19	62
D	25	38.16	12.209	20	65
Total	**100**	**36.43**	**11.254**	**19**	**65**

There was no significant difference between four groups in age distribution. (P = 0.214).

TABLE 2: SEX DISTRUBUTION

		GROUP				Total
		A	B	C	D	
SEX	M	14	11	12	12	49
		56.0%	44.0%	48.0%	48.0%	49.0%
	F	11	14	13	13	51
		44.0%	56.0%	52.0%	52.0%	51.0%
Total		**25**	**25**	**25**	**25**	**100**
		100.0%	**100.0%**	**100.0%**	**100.0%**	**100.0%**

There was no significant difference between four groups in sex distribution. (P = 0.859).

TABLE 3: WEIGHT DISTRUBUTION

GROUP	N	Mean	SD	Minimum	Maximum
A	25	56.44	4.62	43	65
B	25	56.40	3.68	45	65
C	25	55.96	5.57	45	66
D	25	56.76	5.79	46	68
Total	**100**	**56.39**	**4.92**	**43**	**68**

There was no significant difference between four groups in weight distribution. (P = 0.955).

TABLE 4: COMPARISON OF HEART RATE

Time of Assessment	Group A		Group B		Group C		Group D		ANOVA `F'*	P value
	Mean± SD	%Diff	Mean± SD	%Diff	Mean± SD	%Diff	Mean± SD	%Diff		
Base	85±9.26	-	81.76± 9.19	-	80.28± 9.12.	-	78.92 ± 12.84	-	1.630	0.188
Drug	87.36± 9.85	2.7	83.76± 9.33	2.4	81.28± 11.30	1.2	78.52 ± 10.94	-0.4	3.268	0.025
Ind	92.36± 13.15	8.6	87.04± 12.21	6.4	82.84± 8.97	3.1	79.44 ± 10.01	0.6	6.174	0.001
2 min	119.08± 10.35	40	108.04± 11.93	32.1	91.40± 8.84	13.8	83.28 ± 9.07	5.5	63.478	0.000
3 min	123.28± 9.70	45	110.88± 13.6	35.6	91.40± 7.58	13.8	84.04 ± 9.84	6.4	74.244	0.000
4 min	122.72± 9.16	44.3	108.56± 14.16	32.7	89.84± 8.69	11.9	82.48 ± 10.20	4.5	71.550	0.000
5 min	117.52± 8.93	38.2	103.12± 13.44	26.1	85.76± 9.61	6.8	81.12± 9.68	2.7	62.426	0.000
6 min	111.24± 11.79	30.8	99.96± 13.45	22.2	83.08± 9.99	3.4	77.96± 8.90	-0.2	47.103	0.000
7 min	107.68± 12.11	26.2	96.64± 13.18	18.1	81.44± 9.94	1.4	77±7.8 7	-2.4	41.320	0.000

-ve sign indicates decrease, * One way ANOVA, P <0.05, P<0.01 are significant, P <0.001 is highly significant, P > 0.05 is not significant.

One way ANOVA shows no significant difference among all the groups in baseline heart rates (P-0.188). A significant difference is observed in all groups at subsequent assessments upto 7 minutes. Maximum increase in heart rate in group D was 6.4% at 3 minutes which was less compared to 13.8% seen in group C, 35.6% seen in

group B and 45% seen in group A. The heart rate response between groups was very significant at all times starting from injection of drug till 7 minutes (p<0.001) with group C and group D showing a favourable response towards attenuation of heart rate.

Table 5: COMPARISON OF SYSTOLIC BLOOD PRESSURE

Time of assessment	Group A		Group B		Group C		Group D		ANOVA`F'*	P value
	Mean± SD	% Diff	Mean± SD	% Diff	Mean± SD	% Diff	Mean± SD	% Diff		
Base	129.44 ±8.90	-	128± 9.01	-	129.68± 8.60	-	131.44± 11.53	-	0.541	0.655
Drug	128.72 ±7.78	-0.5	127.60± 8.98	-0.3	128.56± 8.16	-0.8	128.64± 12.95	-2.1	0.073	0.974
Ind	128.72 ±10.19	-0.5	128.28± 12.12	0.2	127.88± 9.70	-1.3	124.20± 13.45	-5.5	0.818	0.487
2 min	155.96 ±8.41	20.4	151.32± 8.23	18.2	137.80± 9.30	6.2	129.20± 15.45	-1.7	32.516	0.000
3 min	160.04 ±6.15	23.6	153.56± 8.30	19.9	137.64± 11.34	6.1	129.20± 15.41	-1.8	42.936	0.000
4 min	155± 5.59	19.7	148.72± 8.52	16.1	134.64± 10.19	3.8	127.72± 15.61	-2.8	34.778	0.000
5 min	149.48 ±6.39	15.4	143.76± 8.39	12.3	132.40± 9.23	2	126.16± 16.14	-4	24.526	0.000
6 min	144.72 ±6.94	11.8	139.84± 8.32	9.2	130.44± 8.86	0.5	125.96± 13.65	-4.7	19.177	0.000
7 min	140.88 ±7.23	8.8	135.92± 7.26	6.1	129.56± 9.02	-0.1	125.16± 12.78	-4.7	13.698	0.000

-ve sign indicates decrease, * One way ANOVA, P <0.05, P<0.01 are significant, P <0.001 is highly significant, P > 0.05 is not significant.

One way ANOVA shows no significant difference among all the groups in baseline systolic blood pressure, after injection of drug and after induction of anesthesia. Maximum rise in systolic blood pressure was seen in group A was 23.6% after 3 minutes 19.9% in group B. A significant difference is observed in all groups at subsequent assessments after 2 minutes upto 7 minutes. In group C maximum rise was 6.1% seen after 2 minute. The systolic blood pressure response between groups was very significant at all times starting from 2 minutes till 7 minutes (p<0.001) with group C and group D showing a favourable response towards attenuation of systolic blood pressure.

TABLE 6: COMPARISON OF DIASTOLIC BLOOD PRESSURE

Time of assessment	Group A		Group B		Group C		Group D		AN-OVA` F'*	P value
	Mean ± SD	% Diff	Mean ±SD	% Diff	Mean ±SD	% Diff	Mean± SD	% Diff		
Base	76.68± 5.66	-	75.24± 4.59	-	79.08± 6.18	-	79.76± 8.32	-	2.745	0.057
Drug	75.28± 5.22	-1.8	74.24± 4.29	-1.3	77.92± 6.94	-1.46	77.2± 7.49	-3.2	1.918	0.132
Ind	76.08± 6.89	-0.8	74.92± 7.07	-0.4	77.76± 6.28	-1.66	76.6± 7.39	-4	0.725	0.539
2 min	89.92± 5.35	17.2	88.12± 5.37	17.1	81.72± 7.28	3.3	80.04± 8.96	0.3	12.091	0.000
3 min	91.32± 4.76	19.1	88.84± 5.36	18	82.16± 6.88	3.8	77.84± 10.45	-2.5	18.258	0.000
4 min	89.76± 4.50	17	87.56± 5.97	16.3	80.96± 5.61	2.3	75.92± 11.48	-4.8	18.159	0.000
5 min	86.68± 4.70	13	84.62± 5.44	12.4	79.44± 6.27	0.5	76.84± 9.66	-4	11.202	0.000
6 min	84.16± 5.32	9.7	82.04± 5.30	9	78.2± 6.84	-2	76± 8.66	-4.7	7.601	0.000
7 min	82.2± 5.92	7.1	80.28± 4.68	6.6	77.32± 6.20	-2.2	74.88± 7.49	-6.1	6.860	0.000

-ve sign indicates decrease, * One way ANOVA, P <0.05, P<0.01 are significant, P <0.001 is highly significant, P > 0.05 is not significant

One way ANOVA shows no significant difference among all the groups in baseline diastolic blood pressure, after injection of drug and after induction of anesthesia. Among the groups, maximum rise in diastolic blood pressure was seen in group A which was 19.1% seen after 3 minutes and 18% seen in group B, 3.8% rise in group C and 0.3% rise seen after 2 minutes in group D. The diastolic blood pressure response between groups was very significant at all times starting from 2 minute till 7 minutes of study period(p<0.001) with group C and group D showing a favourable response towards attenuation of diastolic blood pressure.

TABLE 7: COMPARISON OF MEAN ARTERIAL BLOOD PRESSURE

Time of assessment	Group A		Group B		Group C		Group D		AN-OVA` F'*	P value
	Mean± SD	%Diff	Mean± SD	%Diff	Mean± SD	%Diff	Mean± SD	%Diff		
Base	94.13± 5.65	-	92.68± 4.78	-	95.46± 7.14	-	95.72± 7.88	-	1.161	0.329
Drug	93.04± 4.95	-1.15	92.21± 4.78	-0.5	94.25± 6.40	-1.26	93.20± 7.80	-2.63	0.468	0.705
Ind	93.59± 7.32	-0.5	92.66± 7.95	-0.02	93.96± 6.95	-1.5	91.10± 9.13	-4.8	0.653	0.583
2 min	111.92 ±5.19	18.8	101.16 ±27.46	9.1	99.30± 8.61	4	94.99± 11.59	-0.7	5.237	0.002
3 min	114.18 ±4.21	21.3	110.39 ±4.87	19.1	99.89± 8.88	4.6	93.40± 13.11	-2.4	31.101	0.000
4 min	111.57 ±3.65	18.5	107.96 ±5.31	16.4	98.18± 7.38	2.8	92.18± 13.81	-3.69	27.545	0.000
5 min	107.59 ±4.12	14.2	104.32 ±4.80	12.5	96.74± 7.10	1.3	92.05± 11.94	-3.8	21.432	0.000
6 min	104.47 ±4.78	11.1	101.37 ±5.00	9.3	94.75± 7.25	-0.75	91.57± 11.04	-4.3	15.737	0.000
7 min	101.47 ±5.21	8	98.67± 4.25	6.4	93.36± 7.08	-2.19	90.68± 9.88	-5.3	12.966	0.000

-ve sign indicates decrease, * One way ANOVA, P <0.05, P<0.01 are significant, P <0.001 is highly significant, P > 0.05 is not significant.

One way ANOVA shows no significant difference among all the groups in baseline mean arterial pressure, after injection of drug and after induction of anesthesia. Among the groups, maximum rise in mean arterial pressure was seen in group A which was 21.3 % seen after 3 minutes and 19.1% seen in group B 4.6% rise in group C. The mean arterial pressure response between groups was very significant at all times starting from 2 minute till 7 minutes of study period(p<0.001) with group C and group D showing a favourable response towards attenuation of mean arterial pressure blood pressure.

DISCUSSION

The sequence of induction anaesthesia, laryngoscopy and tracheal intubation are associated with marked haemodynamic changes and autonomic reflex activity which may be a cause of concern in many high risk patients.[14] Laryngoscopy and intubation is associated with rise in heart rate, blood pressure and incidence of cardiac arrhythmias There is a potential for life threatening complications due to these changes in patients with coronary artery disease, systemic arterial hypertension, leading to myocardial ischaemia, heart failure and cerebrovascular catastrophies.[3,15,16,17] It is known that the effect on heart rate after esmolol bolus dose comes on after 1 minute, where as the effect of blood pressure comes after 2 minutes. [18] Ebert *et al* 1989 observed that maximum cardiovascular response occurred 2 minutes after intubation. They also noted that the responses (hypertension, tachycardia) are proportional to the duration of laryngoscopy. These responses start showing up at 15 seconds and peak up at 45 seconds. Hence, they recommend that the duration of laryngoscopy and tracheal intubation should be limited to 30 seconds. These potentially dangerous changes disappear within 5 minutes of onset of laryngoscopy. Although these responses of blood pressure and heart rate are transient and short lived, they may prove to be detrimental in high risk patients especially in those with cardiovascular disease, increased intracranial pressure or anomalies of the cerebral blood vessels. In our study, we have excluded cases with anticipated difficult intubation.

Laryngoscopy and intubation was done at 2 min after injection of study drug. An increase in mean arterial pressure of 26.5 mm Hg and 20 to 40 torr when compared with awake control levels and 35 to 60 torr when compared with preintubation values have been reported after placement of an endotracheal tube.A rise in mean heart rate of 29.9 beats/min has also been noted[19]. Many factors influence the cardiovascular changes associated with laryngoscopy and intubation. Age, drugs, type and duration of procedures, depth of anaesthesia, hypoxia, hypercarbia influence the pressor response[20,21,22]. Marked fluctuations in haemodynamic responses are often seen in geriatric patients[22]. In our study we selected the patients above 18 years. Patients on antihypertensive drugs may exhibit a decrease in pressor response. We excluded the patients on beta –blockers from our study. A variable combination of drugs used for premedication, induction, relaxation and maintenance of anaesthesia can influence the sympathetic response to laryngoscopy and intubation. Propofol was selected for induction since it still continues to be the most popular agent for induction. In normovolemic patients propofol 2 mg kg^{-1} IV can transiently decrease 10-20mm Hg of blood pressure and decrease the heart rate by 15- 20 beats min^{-1}. Succinylcholine has negative inotropic and chronotrpic effect. It acts on the muscarinic receptors of SA node. A marked noradrenergic response was noted when intubation was performed under succinylcholine.[24] Nitrous oxide may increase the tone of sympathetic nervous system. The direct action of nitrous oxide is negative inotropism which is offset by increased sympathetic tone.[25] Halothane has a potency to decrease the heart rate but at concentration used for maintenance it does not appreciably change the heart rate.[26] Nasotracheal intubation comprises of three distinct phases a) nasopharyngeal intubation b) direct laryngoscopy to identify the vocal cords and c) Passage of tracheal tube into the trachea. Nasopharyngeal intubation causes significant pressor response. This response in heightened by the passage of tracheal tube in the larynx and trachea. In a study conducted, direct laryngoscopy did not increase the response significantly.[27] In our study, we included only direct laryngoscopy and orotracheal intubation. Laryngoscopy alone may produce most of the cardiovascular responses reported after laryngoscopy and tracheal intubation during anaesthesia. The most significant laryngoscopic factor influencing cardiovascular responses is found to be the duration of laryngoscopy. A linear increase in heart rate and mean arterial pressure during the first 45 seconds has been observed. Further prolongation has little effect. The force applied during laryngoscopy has only minor effect.[16] In our study the duration of laryngoscopy and intubation was limited to 20 seconds. Adequate care was taken to achieve the required depth of anaesthesia avoiding hypoxia and hypercarbia which can influence the hemodynamic variations. Other contributory causes of hypertension and tachycardia could be continued manifestation of anxiety concerning anaesthesia and surgery, glycopyrrolate premedication and possible effect of suxamethonium. But they seem to be less important than laryngotracheal stimulation during laryngoscopy and intubation. Attenuation of sympathetic responses during laryngoscopy and intubation is of prime concern to the anaesthetist more so in high risk subjects as mentioned earlier. Many strategies have been recommended which include minimising the duration of laryngoscopy to less than 20 seconds[16], iv β-blockers,[8,9,10,11] calcium channel blockers[28], nifedepine[29], clonidine.[30] sodium nitroprusside[12], lignocaine[2,8,9,10,31]. No single drug or technique is satisfactory. Each technique has its own advantages and disadvantages. Optimal time for administration is 2 minutes before laryngoscopy and intubation.[2,8] Esmolol is a betablocking agent with several desirable properties. It is relatively cardioselective, ultrashort acting, with rapid onset of action. It has no significant drug interaction. Metabolism independent of vital organs and it is metabolized by RBC esterase. Frequent side effect is hypotension which doesn't require any other treatment other than discontinuation of theraphy.[4,5,6] Previous studies have shown that the unique pharmacokinetic behavior of esmolol makes it well suited for controlling the cardiovascular responses to tracheal intubation when used as a continuous infusion technique.[18,32,33,34] However the dosing schedule and the time required for preparation of infusion may add a degree of complexity. An alternative approach is to use a bolus dose of esmolol and many studies have investigated this and concluded it to be efficacious. Various bolus doses ranging from 100, 150 and 200mg have been investigated. In our study we have used 50mg, 100mg, 150mg of esmolol.

TIMING OF DRUG ADMINISTRATION

In our study we administer the drug 2 minute before laryngoscopy and intubation. It correlates with study conducted by Helfman SM *et al*[8] who concluded that, esmolol controls the mean rise in systolic blood pressure when it is given 2 minutes before intubation.

COMPARISION OF HEART RATE

In our study the maximum heart rate in group A was 45% after 3 minutes. It correlated with study conducted by Schroff PP *et al*[36] which showed 31.2% increase, Rathore A *et al*[37] which showed 33.4% increase, Sharma J *et al* [38] which showed 48.1% increase, Korpinen R *et al*[39] which

showed 41% increase, Helfman MS *et al*[8] which showed 44% and Oxorn D *et al*[40] which showed 44.1% increase in maximal heart rate. In our study the maximum heart rate in group B was 35.6% after 3 minutes. It correlated with conducted by Atlee JL *et al*[41] which showed 21.8% increase and Rathore A *et al*[37] which showed 19.8% increase in heart rate In our study the maximum heart rate in group C was 13.8% after 3 minutes. It correlated with study conducted by Rathore A *et al*[37] which had 15.2% increase, Sheppard S *et al*[43] which had 18.1 increase, Chung KS *et al*[11] which had 18.2% increase, Korpinen R *et al* [39]which had 18.2% increase and Oxorn D *et al*[40] which had 19.7% increase in heart rate. Our study did not correlate with study of Sharma S *et al*[44] who had 3.4 % decrease in heart rate, Sharma J *et al* [50]who had 6.4% decrease in heart rate. It correlated with study conducted by Schroff PP *et al* [36]which had 10% increase, Rathore A *et al*[37] which had 10% increase, and Sheppard S *et al* [43]which had 8.8% increase in heart rate.

SYSTOLIC BLOOD PRESSURE

In our study, the maximum increase in mean systolic blood pressure in group A was 23.6% after 3 minutes. It correlated with study conducted by Schroff PP *et al*[36] which had 16.7% increase, Rathore A *et al*[37] which had 31.6% increase, Kumar S *et al*[45] which had 23.8% increase , Atlee JL *et al*[41] which had 21.8% increase, Sheppard S *et al* [43]which had 27.8 % increase in maximum mean systolic blood pressure. In our study the maximum increase in mean systolic blood pressure in group B was 19.9% after 3 minutes. It correlated with the study conducted by Rathore A *et al*[37] which had 25.8% and Atlee JL *et al*[41] which had 13.1% increase in maximum mean systolic blood pressure. In our study, the maximum increase in mean systolic blood pressure in group C was 6.2% after 2 minutes. After administration of drug the mean systolic blood pressure decreased below the baseline mean systolic blood pressure. It correlated with the study conducted by Kumar S *et al*[45] which had 7.4% increase, Sheppard S *et al*[43] which had 13.6% increase, Korpinen R *et al*[39] which had 13.6% increase and Venkatesha SL *et al*[46] which had 8% increase in maximum mean systolic blood pressure.

DIASTOLIC BLOOD PRESSURE

In group A, there was a decrease in mean diastolic blood pressure values after induction and after administration of drug. In our study, the maximum increase in mean diastolic blood pressure was 19.1% after 3 minutes. It correlated with the study conducted by Sharma S *et al*[44] which had 20.4% increase, Atlee JL *et al*[52] which had 16.6% increase , Venkatesha SL *et al*[46] which had 23.4% increase in maximum mean diastolic blood pressure values. In our study, the maximum increase in mean diastolic blood pressure in group B was 18.2% after 3 minutes. It correlated with the study conducted by Atlee JL *et al*[41] which had 29.3% increase in mean diastolic blood pressure values. In our study, the maximum increase in mean diastolic blood pressure in group C was 3.8 % after 3 minutes. There was decrease in mean diastolic blood pressure values after induction and after administration of drug. It correlated with the study conducted by Sharma J *et al*[38] which had 2.2% increase, Korpinen R *et al* [39]which had 11.7% increase and Venkatesha SL *et al* [46]which had 18.3 % increase in maximum mean diastolic blood pressure values. In our study, the maximum increase in mean arterial blood pressure in group A was 21.3% after 3 minutes. There was decrease in mean arterial blood pressure values after induction and after administration of drug. It correlated with the study conducted by Sharma S *et al* [44]which had 18.4% increase , Sharma J *et al*[38] which had 11.3 % increase and Atlee JL *et al*[41] which had 22.5 % increase in mean arterial blood pressure values. In our study, the maximum increase in mean arterial blood pressure in group B was 19.1% after 3 minutes .There was decrease in mean arterial blood pressure values after induction and after administration of drug. It correlated with the study conducted by Atlee JL *et al*[41] which had 20.5 % increase and Menigaux C *et al*[42] which had 23.8% increase in mean arterial blood pressure values. In our study the maximum increase in mean arterial blood pressure in group C there was 4.6 % after 3 minutes. There was decrease in mean arterial blood pressure values after induction and after administration of drug. It correlated with the study conducted by Sharma J *et al* [38]which had 0.1% increase, and Kumar S *et al* [45] which had 14.4 % increase in mean arterial blood pressure values.

CONCLUSION

In this study, all the groups in which esmolol was used showed a decrease in mean value of systolic blood pressure, diastolic blood pressure and mean arterial pressure after administration of drug. The reduction is less in esmolol 50 mg group. Esmolol 50 mg group adequately attenuated the heart rate, but the systolic blood pressure, diastolic blood pressure and mean arterial pressure were not satisfactorily attenuated compared to esmolol 100mg group. In esmolol 100 mg group, heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were adequately attenuated during laryngoscopy and tracheal intubation. From this study, it is concluded that esmolol 100 mg is near an ideal drug for attenuation of pressor response to laryngoscopy and endotracheal intubation.

REFERENCES

1. King BD, Harris LC, Greifenstein FE, Dripps RD. Reflex circulatory responses to direct laryngoscopy and tracheal intubation performed during general anaesthesia.Anaesthesiology .1951; 12: 556-561.
2. Bachofen M. Suppression of blood pressure increases during intubation : Lidocaine or fentanyl ? Anesthesist 1988; 37(3) : 156-61.
3. Prys - Roberts C, Greene LT, Melocher, Foex P. Studies of anaesthesia in relation to hypertension II haemodynamic consequences of induction and endotracheal intubation. Br J Anaesth 1971; 43 : 531-46.
4. Richard Gorezynski J. Basic pharmacology of esmolol. Am J Cardiol.1985; 56:3F-13F.
5. Greenspan AM, Scott R, Spielman, Leonard NH, Sheila S, Steck J, Senior CRM, *et al*. Electrophysiology of esmolol. Am J Cardiol 1985;56: 19F-26F.
6. Wiest DB. Esmolol a review of it's therapeutic efficacy and pharmacokinetic characteristics. Clin Pharmacokinetics. 1995; 28(3): 190-202.
7. Anthony LS, James H, Edward H, Edward LC.Pharmacokinetics and pharmacokinetics of esmolol administered as an intravenous bolus. Clin Pharmacol Ther.1987;41:216-19.
8. Helfman SM, Gold MI, Delisser EA, Herrington CA. Which drug prevents tachycardia and hypertension associated with tracheal intubation : lidocaine, fentanyl, or esmolol? Anesth.Analg. 1991; 73(4): 502-04.
9. Kindler CH, Schumacher DG, Schneider MC, Urwyler A. Effects of intravenous lidocaine and/or esmolol on hemodynamic responses to laryngoscopy and intubation: a double blind – controlled clinical trial. J Clin Anaesth 1996; 8(6): 491-6.
10. Feng CK, Chan KH, Liu KN, Lee TY. A comparision of lidocaine,
11. fentanyl and esmolol for attenuation of cardiovascular response to laryngoscopy and tracheal intubation. Acta Anaesthesiol Sin 1996 ; 34(3): 172.
12. Chung KS, Sintara RS, Halvey JD, Paige D, Silvermen DG. Comparison of fentanyl, esmolol and their combination for blunting the haemodynamic response during rapid sequence induction. Can J Anesth 1992; 39: 774-79.
13. Stoelting RK. Attenuation of blood pressure response to laryngoscopy and tracheal intubation with sodium nitroprusside. Anesth.Analg.
14. 1979; 58: 116-19.
15. Williams, Warwick; Gray's anatomy. 36th edn., Edinburgh : Churchill Livingstone, 2000.
16. Black TE, Kay B and Healy TEJ. Reducing the hemodynamic responses to laryngoscopy and intubation. Anaesthesia 1984; 39: 883-887.
17. Shapiro HM, Wyte SR, Harris AB, Galindo H. Acute intraoperative intracranial hypertension in neurosurgical patients: Mechanical and pharmacologic factors. Anaesthesiology. 1972;37: 399-405.
18. Stoelting RK. Blood pressure and heart rate changes during short duration laryngoscopy for tracheal intubation. Anaesth Analg 1978; 57: 197-199.
19. Fox EJ,Sklar GS,Hill CH , Villanueva R, King BD. Complications related to the pressor response to endotracheal intubation. Anesthesiology.1977;47:524- 5.
20. Figueredo E, Garcia EM,Fuenter. Assessment of the efficancy of esmolol on the haemodynamic changes induced by laryngoscopy and tracheal intubation : a meta analysis . Acta Anaesthesiol Scand.2001;45(8):1011-22.
21. Derbyshire DR, Smith G. Sympathoadrenal response to anaesthesia and surgery.Br J Anaesth 1984; 56(7) : 725-737.
22. Bucx MJL, Van Geel RTM, Scheck PAE, Stijnen T. Cardiovascular effects of forces applied during laryngoscopy. Anaesthesia .1992; 47: 1029-33.
23. Khan FA, Mehaboobi SK. Effect of laryngoscopy and tracheal intubation on pulse pressure and influence of age on this response. Anaesth Intensive Care 2004; 32(4): 535-41.
24. Splinter WM, Cervenko F. Haemodynamic responses to laryngoscopy and tracheal intubation in geriatric patients : effects of fentanyl, lidocaine and thiopentone. Can J Anaesth 1989; 36(4): 370-6.
25. Lindgren L, Yli-Hankala A, Rnandell T. Haemodynamic and catecholamine responses to induction of anaesthesia and tracheal intubation.
26. Comparision between propofol and thiopentone. Br J Anesth 1993; 70: 306- 310.
27. Stoelting RK, Peterson C. Heart rate slowing and junctional rhythm following intravenous succinyl choline with or without intramuscular atropine premedication. Anesth.Analg. 1974; 54: 705.
28. Smith NT, Eger EI, Stoelting RK. The cardiovascular and sympathomimetic responeses to the addition of nitrous oxide to halothane in man. Anesthesiology 1970; 32: 410.
29. Karagoz AH, Basgul E, Celiker V, Aypar U. The effect of inhalational anaesthetics on QTc interval. Eur J Anaesthesiol 2005; 22(3): 171-4.
30. Singh S, Smith JE. Cardiovascular changes after the 3 stages of nasotracheal intubation. Br J Anaesth 2003; 91(5): 667-71. Onkar Singh, P.Kumar, Swarn Kaur. Attenuation of the pressure response to
31. laryngoscopy and tracheal intubation : Comparision of beta blockers and calcium channel blockers. Ind J Anaesth. 1993; 41: 320-24.
32. .Khan RM, TZ Khan, Eqbal Ahmed. Nifedipine and attenuation of blood pressure and pulse rate changes in response to laryngoscopy and tracheal intubation. Ind J Anaesth 1987; 35(5): 346-49.
33. Roy S, Rudra A, Gupta K, Mondal T.. Attenuation of cardiovascular response to laryngoscopy and tracheal intubation with oral clonidine (Arkamine). Ind J Anaesth 1993; 41: 62-65.
34. Ugur B ,Ogurlu M, Gezer E, Aydin NO ,GursoyF. Effects of esmolol , lidocaine and fentanyl on haemodynamic responses to endotracheal intubation: a comparative study Clin Drug Investig.2007;27(4):269-77.
35. Vucevic M, Purdy GM, Ellis FR. Esmolol hydrochloride for management of the cardiovascular stress responses to laryngoscopy and tracheal intubation.
36. Br J Anaesth 1992; 68: 529-530.
37. Saif Ghaus M, Vinita Singh, Kumar A, Wahal R, Bhatia VK, Agarwal J. A study of cardiovascular response during laryngoscopy and intubation and their attenuation by ultra short acting β-blockers - Esmolol. Indian J Anaesth 2002; 46(2):104-106.
38. Liu PL, Gatt S, Gugino LD, Mallampati SR, Covino BG. Esmolol for control of increase in heart rate and blood pressure during tracheal intubation after thiopentone and succinycholine.Can Anaesth Soc J. 1986;33(5):556-62.

39. Derbyshire DR, Smith G. Sympathoadrenal response to anaesthesia and surgery.Br J Anaesth 1984; 56(7) : 725-737.
40. Shroff PP, Mohite SM, Panchal ID. Bolus administration of esmolol in controlling the haemodynamic response to tracheal intubation. J Anaesth Clin Pharmacol 2004; 20(1): 69-72.
41. Rathore A, Gupta HK, Tanwar GL, Rehman H. Attenuation of the pressure response to laryngoscopy and endotracheal intubation with different doses of esmolol. Indian J Anaesthesia.2002; 46(6):449-452.
42. Sharma J, Sharma V, Ranbhushan, Gupta S. Comparative study of Magnesium sulphate and Esmolol in attenuating the pressor response to endotracheal intubation in controlled hypertensive patients. J Anaesth Clin Pharmacol.2006;22(3):255-259.
43. Korpinen R, Simola M, Saarnivaara L. Effect of esmolol on the heart rate, arterial pressure and electrocardiographic changes during laryngoscopy. Acta Anaesthesiol Scand 1997 Mar;41(3):371-5.
44. Oxorn D, Knox JWD, Jeremy Hill. A bolus dose of esmolol for the prevention of perioperative hypertension and tachycardia. Can J Anaesth .1990; 37 (2): 206-209.
45. Atlee JL, Dhamee MS, Olund TL, George V. The use of Esmolol , Nicardipine
46. ,or their combination to blunt hemodynamic changes after laryngoscopy and tracheal intubation. Anesth Analg .2000; 90:280-5.
47. Menigaux C, Guignard B, Adam F, Sessler DI, Joly V, and Chauvin M. Esmolol prevents movement and attenuates the BIS response to ortotracheal intubation. Br J Anesth 2002; 89(6): 857-62.
48. Sheppard S, Eagle CJ, Strunin L. A bolus dose of esmolol attenuates tachycardia and hypertension after tracheal intubation. Can J Anesth 1990; 37: 202-205.
49. Sharma S, Mitra S, Kumar V, Kalra R. Esmolol blunts the haemodynamic response to tracheal intubation in treated hypertensive patients. Can J Anaesth 1996;43(8):778-82.
50. Kumar S, Mishra MN, Mishra LS, Bathla S. Comparative study of the efficacy of I.V .Esmolol , Diltiazem and Magnesium sulphate in attenuating haemodynamic response to laryngoscopy and tracheal intubation. Indian J Anaesth. 2003; (1):41-44.
51. Venkatesha SL, Grover GD, Pandav SS. Esmolol partially attenuates intraocular pressure response to suxamethonium and tracheal intubation. J Anaesth Clin Pharmacol.2002;18(1):63-67.

Source of Support: None Declared
Conflict of Interest: None Declared