Original Article

Efficacy of esmolol in attenuation of cardiovascular response to laryngoscopy and endotracheal intubation

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Abstract

Introduction: Direct laryngoscopy and endotracheal intubation frequently induces a cardiovascular stress response characterized by hypertension, tachycardia due to reflex sympathetic stimulation which in turn leads to increased plasma catecholamine concentration. Increase in heart rate and blood pressure are well documented sequelae of direct laryngoscopy and endotracheal intubation in normotensive individuals. **Aims and Objectives:** To study Efficacy of Esmolol in attenuation of cardiovascular response to Laryngoscopy and Endotracheal intubation **Materials and Methods:** The study was approved by the Ethics Committee of Kidwai Memorial Institute of Oncology, Bangalore and all patients gave valid written informed consent. One Sixty inpatients, 20 – 60 years of age, of either sex undergoing elective surgical procedures at Kidwai Memorial Institute of Oncology, Bangalore requiring general anaesthesia with endotracheal intubation were selected randomly. The study was conducted in the Department Of Anaesthesia and Pain Relief, Kidwai Memorial Institute of Oncology, Bangalore for a period of one year 01-01-2013 to 01-01-2014.Descriptive and inferential statistical analysishas been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance. **Result:** In Esmolol group The decrease in mean HR at 1,3 and 5 minutes after intubation compared to basal HR was statistically significant ($p<0.001$). The decrease in mean SBP observed at 1, 3, 5 and 10 minutes after intubation when compared with basal SBP was statistically significant ($p<0.05$).The decrease in mean DBP observed at 3 and 5 minutes after intubation when compared with basal DBP was statistically significant ($p<0.05$). The decrease in MAP observed at 3, 5 and 10 minutes after intubation when compared with basal MAP was statistically significant ($p<0.05$). **Conclusion:** Esmolol significantly attenuates the cardiovascular response to laryngoscopy and intubation

Keywords: Esmolol, cardiovascular response to Laryngoscopy and Endotracheal intubation.

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INTRODUCTION

Direct laryngoscopy and endotracheal intubation frequently induces a cardiovascular stress response characterized by hypertension, tachycardia due to reflex sympathetic stimulation which in turn leads to increased plasma catecholamine concentration. Increase in heart rate and blood pressure are well documented sequelae of direct laryngoscopy and endotracheal intubation in normotensive individuals.[1,2,3,4] This transient, self-limiting hypertension and tachycardia are innocuous in healthy individuals but either or both may be hazardous to patients with hypertension, coronary insufficiency or with cerebro-vascular disease.[5] Pressor response to intubation is exaggerated in hypertensive patients even though rendered normotensive preoperatively by antihypertensive medications.[6] Pressor response may result in intra-operative myocardial infarction,[7] acute

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L.V.F,[7] dysarrhythmias[8] and intracerebral bleed[7] in individuals with end organ decompensation. Intravenous anesthetic induction agents do not adequately or predictably suppress the circulatory responses evolved by endotracheal intubation.[5] So prior to initiating laryngoscopy additional pharmacological measures like use of volatile anaesthetics,[2] topical and intravenous lidocaine,[9] opioids[10,11,12], vasodilators – SNP[13], NTG[14], calcium channel blockers[15, 16, 17] and β-blockers[18, 19] have been tried by various authors. These measures attenuate but do not completely abolish the pressor response. Each technique has its own disadvantage which suggests lack of an ideal measure. Minimum alveolar concentration of Halothane, Isofluraneand Enflurane required to attenuate the pressorresponse is very difficult to attain during the short period available for anaestheticinduction.[5] If at all attained, volatile anaesthetic may cause unacceptable mycocardialdepression[2,21] hazardous to hypertensive patient with ischaemic heart disease. Intravenous lidocaine in varying doses has been shown to attenuate the stress response to laryngoscopy and intubation.[20, 22] Sodium Nitroprusside requires special administration technique and invasive arterial BP monitoring and undue hypotension can still occur. In year 1982,[23] a new intravenous cardio-selective β blocker Esmolol Hydrochloride became available for clinical use. Many authors used esmolol to blunt the short lived haemodynamicsequelae associated with laryngoscopy and intubation because of its unique properties such as rapid onset of action, peak effect within minutes and short elimination half-life after intravenous administration. Intravenous esmolol has been extensively tried in patients[24,25,26,27,28,29] by many authors to blunt the pressor response to tracheal intubation and found to be effective and safe.

AIMS AND OBJECTIVES

To study Efficacy of Esmolol in attenuation of cardiovascular response to Laryngoscopy and Endotracheal intubation

MATERIALS AND METHODS

The study was approved by the Ethics Committee of Kidwai Memorial Institute of Oncology, Bangalore and all patients gave valid written informed consent. One Sixty inpatients, 20 – 60 years of age, of either sexundergoing elective surgical procedures at Kidwai Memorial Institute of Oncology, Bangalore requiring general anaesthesia with endotracheal intubation were selected randomly. The study was conducted in the Department Of Anaesthesia and Pain Relief, Kidwai Memorial Institute of Oncology, Bangalore for a period of one year 01-01-2013 to 01-01-2014. Patients aged between 20 - 60 years of age posted for elective non cardiac surgical procedures under general anaesthesia, ASA grade I and II patients, Patients with Mallampatti grade I and II were Included into study. Whereas Patients with any known medical comorbidities like hypertension, ischemic heart diseases, diabetes, asthma etc, Basal Heart Rate < 60 and> 100, Basal Systolic Blood Pressure < 100mmHg, Predicted difficult intubation If patient is allergic to any of the study drugs, More than 1 attempt at intubation and attempt lasting more than 40 seconds, Cormack – Lehane Laryngoscopic Grading > 2, On any medications with cardiovascular effects, If patient is allergic to any of the study drugs, More than 1 attempt at intubation and attempt lasting more than 40 seconds Cormack – Lehane Laryngoscopic Grading > 2.On any medications with cardiovascular effects were excluded from the study. The patients were connected to multichannel monitor which records Heart Rate, non-invasive measurements of SBP, DBP, MAP, EtCO2 and continuous ECG monitoring and oxygen saturation. The baseline systolic, diastolic blood pressure, mean arterial pressure and heart rate were recorded. The cardiac rate and rhythm were also monitored from a continuous visual display of electrocardiogram from lead II. Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance.

RESULT

Table 1: Heart rate (bpm) changes – intragroup comparison - Esmolol

HR	Mean	StdDev	SE of Mean	Mean Difference	t	P-Value
Baseline	85.28	8.76	1.38	---	---	---
1 Min after drug administration	80.38	8.97	1.44	-4.890	6.993	<0.001*
Immediately after intubation	85.03	8.82	1.41	-0.249	0.702	0.487
1 Min post intubation	82.95	8.74	1.40	-2.326	2.354	0.024*
3 Min post intubation	80.03	8.57	1.37	-5.249	4.195	<0.001*
5 Min post intubation	77.33	8.16	1.31	-7.942	6.350	<0.001*
10 Min post intubation	83.49	6.71	1.07	-1.788	1.241	0.222

p <0.05 is significant; p<0.01 highly significant

In the pre-induction mean HR was 85.2±8.76 bpm. The mean heart rate after 1min of study drug administration was 80.4±8.97 bpm, immediately after intubation was 85.03±8.82 bpm, one minute after intubation it was 82.95±11.4 bpm representing a fall of 2.25 bpm from the pre-induction heart rate. By 3, 5 and 10 minutes mean HR were 80.03±8.57 bpm, 77.33±8.16 bpm and 83.49±6.71 bpm respectively. The decrease in mean HR at 1,3 and 5 minutes after intubation compared to basal HR was statistically significant ($p<0.001$).

Table 2: Systolic blood pressure changes – intragroup comparison –Esmolol

SBP	Mean	Std. Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	127.78	10.98	1.74	---	---	---
1 Min after drug administration	122.36	11.35	1.82	-5.416	6.935	<0.001*
Immediately after intubation	130.77	11.16	1.79	2.994	-3.176	0.003*
1 Min post intubation	124.74	9.75	1.56	-3.031	2.552	0.015*
3 Min post intubation	116.51	10.15	1.63	-11.262	9.197	<0.001*
5 Min post intubation	110.33	8.35	1.34	-17.442	14.289	<0.001*
10 Min post intubation	116.95	10.70	1.71	-10.826	6.984	<0.001*

p<0.01–highly significant, p<0.05–significant, p>0.05–Not Significant

In the group E (Esmolol) the baseline value of mean SBP was 127.78 ± 10.98 mmHg and at immediately after intubation was 130.77 ± 11.16 mmHg. The mean systolic blood pressure one minute after intubation was 124.74 ± 9.75 mmHg, representing a fall of 3.04 mmHg. By 3, 5 and 10 minutes mean SBP values were 116.51 ± 10.15 mmHg, 110.33 ± 8.35 mmHg, 116.95 ± 10.70 mmHg and with a fall of 11.27, 17.45 and 10.83 mmHg respectively compared to baseline values. The decrease in mean SBP observed at 1, 3, 5 and 10 minutes after intubation when compared with basal SBP was statistically significant ($p<0.05$).

Table 3: Diastolic blood pressure changes – intragroup comparison –Esmolol

DBP	Mean	Std. Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	77.43	6.93	1.10	---	---	---
1 Min after drug administration	74.77	7.61	1.22	-2.656	3.264	0.002*
Immediately after intubation	82.44	7.03	1.13	5.011	-5.014	<0.001*
1 Min post intubation	76.62	6.35	1.02	-0.810	0.749	0.458
3 Min post intubation	72.74	7.60	1.22	-4.681	3.153	0.003*
5 Min post intubation	70.67	6.62	1.06	-6.758	4.385	<0.001*
10 Min post intubation	77.62	8.05	1.29	0.190	-0.078	0.938

p<0.01–highly significant, p<0.05–significant, p>0.05–Not Significant

In the group E (Esmolol) the baseline value of mean DBP was 77.43 ± 6.93 mmHg and at immediately after intubation was 82.44 ± 7.03 mmHg. The mean diastolic blood pressure one minute after intubation was 76.62 ± 6.35 mmHg, representing a fall of 0.81 mmHg which is not statistically significant. By 3 and 5 minutes mean DBP values were 72.74 ± 7.60 mmHg and 70.67 ± 6.62 mmHg with a fall of 4.69 and 6.76 mmHg respectively compared to baseline values. At 10 min post intubation, the mean DBP returned to the basal values. The decrease in mean DBP observed at 3 and 5 minutes after intubation when compared with basal DBP was statistically significant ($p<0.05$).

Table 4: Mean Arterial blood pressure changes – intragroup comparison - Esmolol

MAP	Mean	Std. Dev	SE of Mean	Mean Difference	t	P-Value
Baseline	94.23	7.26	1.16	---	---	---
1 Min after drug administration	90.36	8.26	1.32	-3.872	5.485	<0.001*
Immediately after intubation	98.64	7.33	1.17	4.410	-5.226	<0.001*
1 Min post intubation	92.74	6.50	1.04	-1.487	1.440	0.158
3 Min post intubation	87.33	7.36	1.18	-6.897	5.528	<0.001*
5 Min post intubation	83.92	6.53	1.05	-10.308	7.997	<0.001*
10 Min post intubation	90.72	8.32	1.33	-3.513	2.467	0.018*

p<0.01–highly significant, p<0.05–significant, p>0.05–Not Significant

In the group E (Esmolol) the baseline value of MAP was 94.23 ± 7.26 mmHg and at immediately after intubation was 98.64 ± 7.33 mmHg. The MAP at one minute after intubation was 92.74 ± 6.50 mmHg, representing a fall of 1.49 mmHg which is not statistically significant. By 3, 5 and 10 minutes MAP values were 87.33 ± 7.36 mmHg, 83.92 ± 6.53 mmHg, 90.72 ± 8.32 mmHg and with a fall of 6.9, 10.31 and 3.51 mmHg respectively compared to baseline values. The decrease in MAP observed at 3, 5 and 10 minutes after intubation when compared with basal MAP was statistically significant ($p<0.05$).

DISCUSSION

Laryngoscopy and endotracheal intubation elicit a reflex cardiovascular response in the form of hypertension and tachycardia in adults. Though well tolerated in healthy adult patients it can have catastrophic consequences in patients with coronary artery disease and cerebrovascular diseases.[30] It is very much essential to minimize the hemodynamic response to laryngoscopy and intubation in high risk patients such as patients with history of coronary artery disease, hypertension and cerebrovascular diseases. To achieve this, it is important to understand the dynamic interactions between the drugs used and onset of drug effects. One should avoid over treating these responses which are usually short lived and well tolerated by most patients-one ounce of prevention is worth a pound of cure.[31] In our study the pre-induction mean HR was 85.2±8.76 bpm. The mean heart rate after 1min of study drug administration was 80.4±8.97 bpm, immediately after intubation was 85.03±8.82 bpm, one minute after intubation it was 82.95±11.4 bpm representing a fall of 2.25 bpm from the pre-induction heart rate. By 3, 5 and 10 minutes mean HR were 80.03±8.57 bpm, 77.33±8.16 bpm and 83.49±6.71 bpm respectively. The decrease in mean HR at 1,3 and 5 minutes after intubation compared to basal HR was statistically significant ($p<0.001$). These findings resemble that of Menkhaus *et al*[24] who found that esmolol given by continuous infusion attenuated heart rate response at 1, 3 and 4 minutes after laryngoscopy and intubation. In the group E (Esmolol) the baseline value of mean SBP was 127.78 ± 10.98 mmHg and at immediately after intubation was 130.77 ± 11.16 mmHg. The mean systolic blood pressure one minute after intubation was 124.74 ± 9.75 mmHg, representing a fall of 3.04 mmHg. By 3, 5 and 10 minutes mean SBP values were 116.51 ± 10.15 mmHg, 110.33 ± 8.35 mmHg, 116.95 ± 10.70 mmHg and with a fall of 11.27, 17.45 and 10.83 mmHg respectively compared to baseline values. The decrease in mean SBP observed at 1, 3, 5 and 10 minutes after intubation when compared with basal SBP was statistically significant ($p<0.05$). In the group E (Esmolol) the baseline value of mean DBP was 77.43 ± 6.93 mmHg and at immediately after intubation was 82.44 ± 7.03 mmHg. The mean diastolic blood pressure one minute after intubation was 76.62 ± 6.35 mmHg, representing a fall of 0.81 mmHg which is not statistically significant. By 3 and 5 minutes mean DBP values were 72.74 ± 7.60 mmHg and 70.67 ± 6.62 mmHg with a fall of 4.69 and 6.76 mmHg respectively compared to baseline values. At 10 min post intubation, the mean DBP returned to the basal values. The decrease in mean DBP observed at 3 and 5 minutes after intubation when compared with basal DBP was statistically significant ($p<0.05$). In the group E (Esmolol) the baseline value of MAP was 94.23 ± 7.26 mmHg and at immediately after intubation was 98.64 ± 7.33 mmHg. The MAP at one minute after intubation was 92.74 ± 6.50 mmHg, representing a fall of 1.49 mmHg which is not statistically significant. By 3, 5 and 10 minutes MAP values were 87.33 ± 7.36 mmHg, 83.92 ± 6.53 mmHg, 90.72 ± 8.32 mmHg and with a fall of 6.9, 10.31 and 3.51 mmHg respectively compared to baseline values. The decrease in MAP observed at 3, 5 and 10 minutes after intubation when compared with basal MAP was statistically significant ($p<0.05$). These findings are in agreement with study of Menkhaus *et al* [24] and Vucevic *et al* [32].

CONCLUSION

In patients with no interventions to attenuate the cardiovascular responses to laryngoscopy and intubation, the maximum rises in heart rate, systolic, diastolic and mean arterial pressures were statistically and clinically very high and can be detrimental in high risk patients. Esmolol significantly attenuates the cardiovascular response to laryngoscopy and intubation.

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