International Journal of Research in Medical Sciences
Gurudatta KN et al. Int J Res Med Sci. 2014 Aug;2(3):866-871
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: 10.5455/2320-6012.ijrms20140814

Research Article

Attenuation of hemodynamic responses to laryngoscopy and endotracheal intubation by intravenous esmolol

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Received: 18 April 2014
Accepted: 4 May 2014

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ABSTRACT

Background: Sympathetic response associated with laryngoscopy and endotracheal intubation is a potential cause for a number of complications especially in patients with cardio-vascular compromise. The aim of our study was to evaluate and study the efficiency of intravenous esmolol in the attenuation of hemodynamic response to laryngoscopy and intubation in normotensive individuals.
Methods: 100 surgical patients of either sex of physical status ASA I/II were randomly divided into 2 groups. Group C (10 ml of 0.9% normal saline) and group E (Esmolol 100 mg IV) given 2 minutes before induction. Baseline parameters - heart rate, systolic blood pressure, diastolic blood pressure and rate pressure product were noted at baseline level, just before induction, 1 min., 3 min., 5 min and 10 minutes after tracheal intubation.
Results: Intravenous esmolol showed statistically significant attenuation of hemodynamic response to laryngoscopy and intubation when compared with the control.
Conclusion: We conclude that esmolol in a dose of 100 mg given 2 minute before induction is highly effective in attenuation hemodynamic response to laryngoscopy and intubation.

Keywords: Hemodynamic response, Laryngoscopy, Intubation, Esmolol

INTRODUCTION

The frequent occurrence of hemodynamic responses to laryngoscopy and endotracheal intubation has attracted the attention of anesthesiologists since 1940s. Ensuing tachycardia, rise in blood pressure, sometimes dysrhythmia that occur during intubation, are potentially harmful especially in cardiac patients. In hypertensive patients, the cardiovascular response to laryngoscopy and intubation is exaggerated due to the narrow arterial lumen, blunted baro reflex response and increased sympathetic activity.[1]

Many studies have been conducted to attenuate this response. The search for effective attenuation of these responses include IV or topical lignocaine[2] vasodialtors[3] like NTG, adrenergic blockers,[4] narcotics[5] and inhaled anesthetics[6] by using deeper plane of anesthesia, administration of alpha & beta blockers, calcium channel blockers, opioids etc., esmolol is a short acting cardio selective drug, whose half-life is 9 min.

The topic of study was chosen because it has been noted by many workers that increase in blood pressure and heart rate resulting from sympathetic discharge in response to laryngyo-tracheal stimulation may get further enhanced and proves dangerous to hypertensive and ischemic heart disease patients and esmolol is fast-acting, has a short duration of action, and has few to no side effects.

METHODS

After obtaining approval from the hospital ethical committee and patients written informed consent, 100 patients in the age group of 20 to 50 years and physical status ASA I/II, posted for various surgical procedures were included in the study. Exclusion criteria were patients with the anticipated difficult airway, emergency cases, and patients with bronchial asthma, uncontrolled hypertension, and diabetes mellitus. Patients were randomized into two groups. Group C (control) - who received 10 ml of 0.9% normal saline and group E - who received 100 mg esmolol intravenously slowly, 2 minutes before induction. The patients were monitored throughout the procedure by pulse oxymeter, NIBP, ECG. All the patients received tab. diazepam 5 mg previous night at bed time. Patients were premedicated with pentazocine 0.5 mg/kg. Induction was done with intravenous, thiopentone sodium (2.5%) dose being 5 mg/kg with abolition of eye lash reflex as end point and followed by succinylcholine 1.5 mg/kg. Laryngoscopy was performed with a Macintosh laryngoscope and intubation was performed with a cuffed oral endotracheal tube of appropriate size, with a strict and vigil monitoring of hemodynamic parameters - heart Rate, systolic blood pressure, Diastolic Blood Pressure (DBP) Rate Pressure Product (RPP) at regular interval: pre-induction, just prior to induction and at 0 min (laryngoscopy and intubation), 1 min, 5 min and 10 minutes post intubation. Surgical stimulus was avoided during the study period. Anesthesia was maintained with oxygen, nitrous oxide, halothane and vecuronium bromide (0.08 mg/kg) incremental dose of which was given every 20 minutes. At the end of surgery, neuromuscular blockade was reversed with inj. neostigmine 0.05 mg/kg and inj. glycopyrrolate 0.01 mg/kg. Extubation done and patients were shifted to recovery ward for further observation.

The data were statistically analyzed using student's T test and chi-square test. A value of P <0.05 was considered significant and <0.001 was considered highly significant.

RESULTS

The Patients in the two groups were comparable with respect to age, weight and sex (Table 1).

Table 1: Demographic profile.

	C	E	
Age (Y)	39.04 ± 12.07	40.26 ± 12.50	P >0.05 NS
Sex (M/F)	14/16	16/14	X^2 = 0.0815 P >0.05 NS
Weight (Kg)	52.08 ± 7.48	53.68 ± 6.44	P >0.05NS

Heart rate (Table 2)

The effect of esmolol on heart rate in the groups is shown in Table 2. In the control group C, heart rate increased significantly from the time of laryngoscopy and intubation up to 5 minutes post intubation, (P <0/001) and at 10 minutes it was comparable with the basal level. The increase in mean HR at laryngoscopy in the control group was 42.16% from the basal value. In the E group the heart rate decreased below the basal value by 4.8% just before laryngoscopy and, HR increased by 5.13% at laryngoscopy. Thereafter HR was comparable with the base value.

Table 2: Within the group and between the group compressions of changes in the mean heart rate presented as HR ± SD.

Time of assessment	Control 'C'	Esmolol 'E'	Within the group changes from basal value (%)		Between the group difference P value
			C	E	
Basal	83.5 ± 6.42	83.38 ± 6.45			P >0.05
Just prior to induction (after the drug)	85.34 ± 6.17	79.31 ± 6.81	+2.15	-4.8	P >0.05
0 min	118.49 ± 10.72	87.23 ± 8.3	+42.16	+5.13	P <0.001
1 min	114.44 ± 7.12	83.6 ± 6.57	+37.34	-0.3	P <0.001
3 min	101.98 ± 8.85	79.98 ± 5.38	+21.6	-4.8	P <0.001
5 min	92.48 ± 6.41	82.58 ± 4.97	+9.1	-1.41	P <0.001
10 min	84.3 ± 5.5	83.64 ± 4.8			P >0.05

-ve sign indicates decrease; +ve indicates increase

P <0.05 - Significant, P <0.001 - Highly significant

Systolic blood pressure (SBP) (Table 3)

In group 'C' there was no significant change in SBP just before laryngoscopy. At laryngoscopy and intubation (0 min) there was an increase in SBP by 27.4%. SBP increased by 23.84% at 1 min, by 10.76% at 3 min and by 3.84% at 5 min. The increases were highly significant. At 10 min post intubation SBP was comparable with the basal value. In group E there was a decrease in SBP by just before laryngoscopy by 3.18%. After laryngoscopy and intubation the changes in the SBP were not significant.

Table 3: Comparison of changes in mean systolic blood pressure in mmHg.

Time of assessment	Control 'C'	Esmolol 'E'	Within the group changes from basal value (%)		Between the group difference P value
			C	E	
Basal	130.54 ± 10.9	128.74 ± 11.8			P >0.05
Just prior to induction (after the drug)	132.8 ± 8.6	124.64 ± 82	+1.3%	-3.18%	P <0.05
0 min	166 ± 11.41	128.33 ± 11.18	+27.4	+1.17	P <0.001
1 min	160.44 ± 7.72	126.61 ± 6.57	+23.84	-1.56	P <0.001
3 min	144.44 ± 6.8	126.31 ± 5.7	+10.76	-4.6	P <0.001
5 min	135.4 ± 4.6	124.56 ± 4.2	+3.84	-3.12	P <0.05
10 min	131.64 ± 5.2	125.7 ± 5.3	+0.76	-2.34	P <0.05

Diastolic blood pressure changes (DBP) (Table 4)

Rise in the DBP in the control group at laryngoscopy and intubation was significant. DBP at '0' and 1 min showed an increase by 31.5% and 28.9% respectively in the control group. While in the group E the corresponding increases in DBP were 5.2% and 3.8% respectively.

Table 4: Comparison of mean diastolic blood pressure in mmHg.

Time of assessment	Control 'C'	Esmolol 'E'	Within the group changes from basal value (%)		Between the group difference P value
			C	E	
Basal	76.3 ± 6.1	76.2 ± 6.5			P >0.05 NS
Just prior to induction (after the drug)	77.78 ± 5.03	74.91 ± 4.6	+1.97	-1.7	P <0.05 S
0 min	100.90 ± 9.4	80.4 ± 4.6	+31.5	+5.2	P <0.001 HS
1 min	98.48 ± 6.89	78.96 ± 5.42	+28.9	+3.8	P <0.001 HS
3 min	94.56 ± 7.2	76.41 ± 4.3	+23.6	+0.26	P <0.001 HS
5 min	90.42 ± 6.54	75.56 ± 5.2	+18.5%	-1.7	P <0.001 HS
10 min	89.42 ± 6.12	75.6 ± 2.1	+12.98	-1.18	P <0.001 HS

+ Indicates increase, - Indicates decrease

Rate pressure product (RPP) (Table 5)

It is a derived parameter obtained by multiplying HR with SBP. RPP is said to correlate with myocardial oxygen consumption.[7] It is a simple and useful means of clinically assessing the work load of the heart. In our study RPP in the control group increased by 68% and 58% from the basal value at 0 and 1 min respectively. The increase was highly significant (P <0.001). At 3rd minute after intubation the increase was by 33.4% and

RPP reached comparable value by 10 m post intubation. In the esmolol group, there was a significant fall in RPP, when measured just before laryngoscopy. At laryngoscopy (0 min) RPP increased by 4.8% and thereafter the values during the study period were slightly below the basal value.

No ECG changes were present.

Table 5: Comparison of mean rate pressure product between the groups.

Time of assessment	Control 'C'	Esmolol 'E'	Within the group changes from basal value (%)		Between the group difference P value
			C	E	
Basal	10896 ± 1461	10731 ± 1496			P >0.05 NS
Just prior to induction (after the drug)	11328 ± 1120	9796 ± 1216	+14	-8.7	P <0.05 S
0 min	18408 ± 2421	11223 ± 1554	+68	+4.8	P <0.001 HS
1 min	16872 ± 2375	10458 ± 1493	+54.8	-2.5	P <0.001 HS
3 min	14544 ± 1608	9760 ± 1028	+34.4	-9.1	P <0.001 HS
5 min	12421 ± 1490	10230 ± 1104	+13.9	-4.6	P <0.001 HS
10 min	11008 ± 1300	10375 ± 1223	+1.02	-4.66	P <0.05 S

+ Indicates increase, - Indicates decrease

DISCUSSION

It is well documented that laryngoscopy and endotracheal intubation following induction of anesthesia is commonly associated with hemodynamic changes due to reflex sympathetic discharge caused by epipharyngeal and laryngopharyngeal stimulation.[8] The more common response to airway manipulation is hypertension and tachycardia mediated by cardioaccelerator nerves and sympathetic chain ganglion.[9] This response includes wide spread release of norepinephrine from the adrenergic nerve terminals and epinephrine from adrenal medulla.[10] Hypertension response to the endotracheal intubation partly results from activation of renin angiotensin system which is innervated by beta adrenergic nerve terminals. This increased sympatho-adrenal activity may frequently result in hypertension, tachycardia and arrhythmias.[11,12] The average increase in blood pressure by 40-50% and 20% increase in heart rate[13] has been observed. This increase in blood pressure and heart rate are usually transitory, variable and unpredictable. Transitory hypertension and tachycardia are probably of no consequence in healthy individuals,[14] but either or both may be hazardous to those with history of diabetes, pre-eclampsia, myocardial insufficiency or cerebrovascular diseases.[15,16] This sympathoadrenal response to laryngoscopy results in an increased cardiac work load which in turn may culminate in perioperative myocardial ischemia and acute heart failure in susceptible individuals. This response is undesirable in any patient with heart disease undergoing surgery, irrespective of the nature of surgery. Various agents have been used to attenuate hypertensive response including: topical lignocaine - sprays, deeper plane of anesthesia - by inhalational agents, narcotics like fentanyl, alfentanil, sufentanil, remifentanyl, magnesium sulphate, calcium channel blockers, vasodilators like SNP and NTG. Deaths attributable to anesthesia could be reduced by controlling the hemodynamic changes that occur during endotracheal intubation. There is increasing evidence that control of the heart rate and blood pressure response to endotracheal intubation is essential to prevent adverse cardiovascular outcomes.[17,18] Heart rate is a major determinant of myocardial oxygen consumption and tachycardia is poorly tolerated in patients with coronary heart disease. Studies show that incidence of myocardial ischemia is high when intra operative heart rate exceeds 110/min.[19]

Esmolol, (methyl 3-[4- [2 – hydroxyl – 3) isopropylamino) propoxyl] phenyl] proprionate HCl) is a cardio selective water soluble ultra-short acting β_1 adrenergic receptor antagonist that can be administered only intravenously.[20] Esmolol is rapidly hydrolyzed by cytoplasmic esterases in red blood cells, therefore has short elimination of approximately 9 min., with distribution half-life of 2 min and peak hemodynamic effect at 6 to 10 min of administration. Its metabolism is not influenced by renal or hepatic function and less than 1% excreted in urine as unchanged drug. Esmolol is a cardio-selective β-blocker with no action on bronchial smooth muscles and hence safe for use in smokers.[21]

In our study, the increase in mean HR at laryngoscopy in the control group C was 42.16% from the basal value. In the study group E, the heart rate decreased below the

basal value by 4.8% just before laryngoscopy and at laryngoscopy, HR increased by 5.13%. Thereafter HR was comparable with the base value. Thus increases in the heart rate at laryngoscopy and intubation were significantly attenuated by the use of esmolol in the study period. Similar results were found in the studies done by Shorff PP[19] et al., Suresh Lakshmanappa[22] et al., Sheppard S[23] et al., and Kumar.[24]

In the control group C, during laryngoscopy and intubation (0 min) SBP increased by 27.4%, at 1 min. by 23.84%, at by 10.76% at 3 min and by 3.84% at 5 min. At 10 min post intubation SBP was comparable with the basal value. In group E there was a decrease in SBP by 3.18% just before laryngoscopy. After Laryngoscopy and intubation, SBP did not change significantly. Esmolol attenuated the rise in SBP. These findings are similar to those of Mikawa[25] et al. and P Agarwal[20] et al.

Rise in the DBP in the group C at '0' and 1 min showed an increase by 31.5% and 28.9% respectively. While in the group E the corresponding increases in DBP were 5.2% and 3.8% respectively. We found that DBP showed a significant increase in the value during laryngoscopy and intubation in group C and persisted up to 10 min. as compared to the group E where the significant rise in DBP was attenuated.

With respect to RPP, group C showed a significant rise from the baseline, peaking at laryngoscopy and intubation (18408 ± 2421 from 10896 ± 1481, Increase by 68%) and at 5 min. post intubation it was 12421 ± 1490. In the E group, RPP values increased by 4.8% at laryngoscopy and thereafter remained below the basal level throughout the study period. Thus esmolol successfully attenuated the increase in RPP following laryngoscopy and intubation. These findings are consistent with the findings of P. Agarwal[20] et al., Mikawa[25] et al. and Kumar Santhosh[26] et al.

M. Begum[27] et al. found in their study that esmolol at the dose of 1.5 mg/kg is superior to lignocaine for attenuation of hemodynamic response to laryngoscopy and endotracheal intubation. S. Sharma[28] et al. observed esmolol appears quite suitable for use during a short lived stress such as tracheal intubation, organ manipulation like handling adrenal and thyroid gland and extubation. Harbhej Singh[29] et al. concluded that in comparison with lignocaine 1.5 mg/ kg and NTG 2 mcg/kg IV, esmolol 1.4 mg/kg IV was significantly more effective in controlling heart rate and minimizing the increase in MAP following tracheal intubation. Miller[30] et al. demonstrated 1.5 mg/kg of esmolol is optimal for blunting hemodynamic responses to intubation. Gazi Parvez[31] et al. concluded in a study that attenuating effect of presser responses to laryngoscopy and tracheal intubation of esmolol when compared to diltiazem was significantly more.

CONCLUSION

We conclude that intravenous administration of 100 mg esmolol 2 minutes before induction of general anesthesia is very effective in attenuating the hemodynamic response to laryngoscopy and tracheal intubation.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the hospital ethics committee

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DOI: 10.5455/2320-6012.ijrms20140814
Cite this article as: Gurudatta KN, Kiran M, Ravindra GL. Attenuation of hemodynamic responses to laryngoscopy and endotracheal intubation by intravenous esmolol. Int J Res Med Sci 2014;2:866-71.