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Esmolol and Lidocaine in Blunting Hemodynamic Response to Extubation

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ABSTRACT

Background

Tracheal extubation is one of the routinely performed procedures in the practice of anaesthesiology. It has been reported that it increases heart rate and blood pressure by 15-30% lasting for 5 to 15 minutes. Incidence of increase in HR and BP is four times more during extubation in comparison to intubation. Various pharmacological interventions have been tried to attenuate these hemodynamic responses as Lidocaine, Propofol, Esmolol or opioids. Objective of this study is to evaluate and compare the efficacy of Esmolol and Lidocaine in blunting hemodynamic response to extubation.

Methods

This was a comparatove study performed in 70 patients of ASA grade I and II. Patients were divided into Esmolol group and Lidocaine group. Data were collected and Statistical analysis was done using independent t-test to compare means for continuous variable like age, weight, heart rate, SBP, DBP and MBP. Chi-square test was used to test difference in proportion of categorical outcomes in different drug groups.

Results

There was significant increase in heart rate at the time of extubation and at 1 min after extubation in-group L compared to group E (p-value<0.005). SBP was also found significantly increased at the time of extubation and 3 min after extubation in group L as compared to group E (p-value<0.05).

Conclusions

Esmolol 1.5mg/kg given 3 minutes before extubation is better than Lidocaine 1.5mg/kg in attenuating hemodynamic responses to extubation.

Keywords: esmolol; lidocaine; extubation.

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INTRODUCTION

Endotracheal extubation is one of the widely performed procedures as a part of general anaesthesia.[1] Extubation causes 10% to 30% increase in blood pressure and heart rate by the release of catecholamines from stimulation of respiratory tract at supraglottic and subglottic levels.[2-4] This hemodynamic change lasts for around 5-15mins.[5-7] Tracheal extubation is also associated with complications as coughing, agitation, bronchospasm, increased bleeding and raised intracranial and intraocular pressure.[8-9] These responses have been found to have an add on effect to above-mentioned cardiovascular responses. Therefore, hemodynamic responses during extubation should be blunted. Various methods have been used to blunt these hemodynamic response like Esmolol,[10] Labetalol,[11] Lidocaine spray,[12] intravenous Lidocaine,[10] calcium channel blockers,[13] opioids,[5] prostaglandins,[14] Dexmedetomidine,[4] and blocking of glossopharyngeal nerve and superior laryngeal nerve.[10] This study has been done to compare the efficacy of intravenous Esmolol and Lidocaine in blunting such cardiovascular reflex responses during extubation.

METHODS

This study was a comparative interventional study conducted in Department of Anaesthesiology, College of Medical Sciences-Teaching Hospital, Bharatpur, Chitwan from Jan 2017 to August 2018. The sample size of the study was 70 patients dividing 35 patients in each group (Esmolol Group and Lidocaine Group). The sample size is calculated by using the following formula [15]

$$n = \frac{2(Z\alpha + Z\beta)^2 \times SD^2}{d^2}$$

Where, n = Number of sample, Zα = 1.96 (at 95% confidence interval and power of 90%), Zβ = 1.282 (at 95% confidence interval and power of 90%), SD = Sum of standard deviation of the two groups which is 11.02 from previous study[10], d = Difference of mean between two groups which is 9.[10] Following calculation based on previous published data, the sample size in each group came to be 31.2. Based on this result a decision to take 35 patients in each group was made. The patients of Esmolol group (Group E) received 1.5 mg/kg of Esmolol and patients of lidocaine group (Group L) received 1.5 mg/kg of 2% Lidocaine. Randomization was done using closed envelope method. Seventy envelopes were prepared before start of study. Half of the envelopes was written group E and the other half receipt was written group L. These envelopes were mixed and arranged randomly. The accompanying anaesthesia assistant was trained regarding protocol of the study and was allowed to open the envelope before induction of anaesthesia and prepare the drug as per the protocol and code the patient and give the drug to attending anaesthesiologist of the OT to administer the drug at the time of extubation. Attending anaesthesiologist did not know the drug being given and decoding was done during analysis of the collected data. Patients from ASA I and II undergoing elective surgery under general anesthesia without having history of cardiovascular or respiratory diseases and significant renal, hepatic or any metabolic disorders or allergy to study drugs were taken. After selection of the sample informed written consent were taken. All patients were premedicated with diazepam (5 mg orally if the patient's weight was <50 kg and 10 mg if the patient's weight was > 50 kg) on the night before surgery. All patients were preoxygenated with 100% Oxygen for at least 3 minutes before induction. Patient then received Inj. Midazolam 0.04mg/kg intravenous followed by Inj.Fentanyl 1.5mcg/kg iv. Induction of anaesthesia was done with Inj. Propofol iv in titrated dose sufficient to obtund the verbal contact. Inj. Vecuronium 0.1mg/kg was used for neuromuscular relaxation and manual bag and mask ventilation was done for 3 minutes before orotracheal intubation. Maintenance of anaesthesia was done using a mixture of 50% oxygen and 50% air with isoflurane 1- 1.5%. Intermittent bolus doses of Vecuronium was given to facilitate muscle relaxation. Isoflurane was discontinued at the end of surgery and patients were ventilated with 100 % oxygen. Residual muscle paralysis was reversed with Neostigmine (0.05mg/kg) and Glycopyrrolate (0.01mg/kg) following signs of

spontaneous breath efforts. Three minutes prior to extubation study drug was given by the attending anaesthesiologist. Group E received a bolus dose of Esmolol 1.5mg/kg and Group L received Lidocaine 1.5mg/kg, diluted to a 10ml solution of saline, given over a period of one minute. Endotracheal extubation was done after patient met the extubation criteria. After extubation, patients were shifted to Post Anaesthesia Care Unit and monitored for hemodynamic instability in the form of hypotension/hypertension or bradycardia/tachycardia. Heart Rate, Systolic Blood Pressure, Diastolic Blood Pressure, Mean Blood Pressure were monitored and recorded at the end of surgery, at the time of study drug administration, at extubation and at 1, 3, 5 and 10 minutes after extubation. Any side effects observed were recorded. Statistical analysis was done using statistical software SPSS version 16. Independent t-test was used to compare means for continuous variable data like age, weight, heart rate, SBP, DBP and MBP in two drug categories. Chi-square test for proportions was used to test difference in proportion of categorical outcomes in different drug groups.

RESULTS

The mean duration of anaesthesia in Group E was 63.86 ± 24.25 and in Group L was 89.86 ± 27.53 which is statistically insignificant (p >0.05) (Table 1).

Table 1. Demographic data of the patients.

Parameters	Groups		p-value
	Group L	Group E	
Age (years)	32.71 ± 7.99	33.94 ± 10.51	0.584
Body weight (kgs)	59.94 ± 9.4	58.46 ± 7.35	0.52
Male/ Female	33/2	30/5	0.42

Table 2. Preoperative hemodynamic variables: comparison between two groups.

Parameters	Group Esmolol		Group Lidocaine		p-value
	Mean	SD	Mean	SD	
Heart rate (bpm)	80.17	8.86	80.54	9.47	0.86
Systolic BP (mmHg)	117.69	11.12	122.57	13.03	0.09
Diastolic BP(mmHg)	76.11	8.94	73.77	7.84	0.24
MAP (mmHg)	90.03	8.58	90.03	7.33	1
SpO2	99.89	0.4	99.91	0.37	0.76

Heart rate was significantly increased in Group L at the time of extubation (p<0.001). SBP at the end of surgery and at extubation in Group E was 115.97 ± 13.667 and 118.26 ± 24.21 with p value of 0.616 and that in group L was 119.60 ± 11.107 and 133.91 ± 16.67 with p-value<0.001 respectively. DBP at the end of surgery and at extubation in group E and Group L were comparable. MAP at the time of surgery and at extubation in-group L was significantly raised (p-value <0.05) whereas the change was not statistically significant in Group E (p-value >0.05) (Table 1).There were insignificant changes in HR, SBP, DBP or MAP in group E at the time of extubation (p >0.05), however significant increase in HR, SBP and MAP was observed in group L at the time of extubation and up to 3 mins post extubation (p-value<0.05) (Table 3).

Table 3. Comparison of various parameters at the end of surgery with the value at the time of tracheal extubation, comparison within the groups.

Variables		Group E			Group L		
		Mean	SD	p-value	Mean	SD	p-value
HR	At the end of surgery	82.83	12.93	0.084	85.09	16.68	<0.001*
	At extubation	87.43	10.6		100.2	20.81	
SBP	At the end of surgery	116	13.67	0.616	119.6	11.11	<0.001*
	At extubation	118.3	24.21		133.9	16.67	
DBP	At the end of surgery	75.31	15.09	0.317	78.89	11.54	0.081
	At extubation	78.54	18.4		84.69	15.99	
MAP	At the end of surgery	87.4	14.08	0.271	91.29	19.86	0.004*
	At extubation	91.29	19.86		102.3	14.86	

**Statistically significant*

DISCUSSION

In our study, significant rise in heart rate at the time of extubation was observed in the Lidocaine group only (p-value <0.001). This significant increase in heart rate persisted up to 1 min after extubation, which returned to baseline, three mins post extubation. Therefore, compared to Lidocaine group, Esmolol group had better attenuation of heart rate at the

time of extubation. The greater efficacy of Esmolol in attenuating heart rate response could be due to its cardio selective beta 1 property.[14] The SBP also increased significantly in the Lidocaine group during the study time and up to 3 mins post extubation (p-value <0.05). After 3 minutes of extubation, the SBP returned to baseline values and persisted same in the recovery unit. In the Esmolol group insignificant rise in SBP was seen at the time of extubation and was comparable with the baseline values throughout the study period (p-value >0.05). From these results Esmolol was found out to be superior in attenuating SBP response at extubation.

Similarly, mean DBP rose at the study drug time, at extubation, 1 min, 3 min and 5 min post extubation in both the groups which was statistically insignificant (p-value >0.05). The changes in mean MAP in Esmolol group, before and after extubation were statistically insignificant as compared to baseline values (p >0.05). Similarly, increase in mean MAP at extubation, at 1 min and 3 min post extubation were also statistically insignificant (p-value >0.05). However, the mean MAP at the time of extubation from the basal value was highly significant in Lidocaine group (p-value <0.001). So, compared to Lidocaine group, Esmolol group had statistically significant attenuation of mean MAP at extubation and at 1min and 3 min after extubation. The lesser efficacy of iv bolus Lidocaine in attenuating stress response could be due to single iv bolus administration. Lee JH et al.,[23] advocated that serum concentration of Lidocaine should be 1-5 mcg/ml for suppressing stress response which may be difficult to achieve in a timely manner by single bolus administration.

Dyson et al., in 1990[16] did a comparative study between different doses of Esmolol for blunting hemodynamic responses during extubation with placebo group. Esmolol was given at a dose of 1mg/kg, 1.5mg/kg and 2mg/kg respectively, 2 mins after giving reversal agents. They concluded that Esmolol at a dose of 1.5mg/kg attenuates both increase in heart rate and blood pressure. Dose of 2mg/kg produced significant bradycardia and hypotension whereas dose of 1mg/kg was sufficient only to prevent increase in heart rate and not blood pressure. Nagrale MH et al.,[10] studied the effect of intravenous Esmolol, Lidocaine and Propofol on attenuation of stress response to extubation in 90 patients and found that HR, MAP and SBP were markedly decreased by Esmolol upto 10mins after extubation (p-value<0.05) at a dose of 1.5 mg/kg. These findings agree to our study results where HR, mean SBP and mean MAP were attenuated. Sagedi M et al. in 2008[17] conducted a study to compare iv Lidocaine 1.5mg/kg and iv Alfentanil 1.5mcg/kg in blunting hemodynamic response to extubation. They concluded that Lidocaine could not attenuate airway and circulatory reflexes during emergence from anaesthesia (p-value <0.001).

In contrast to our study Singh A et al.[18] in their study concluded that Esmolol 1mg/kg was not able to control the SBP response to extubation when compared with Diltiazem 0.1mg/kg. They however, have used lesser dose of Esmolol (1 mg/kg) than what we used in this study. This difference could be due to lesser drug dosage of Esmolol in comparison to our study. We did not observe any of the significant side effects or complications related to drugs or extubation process. We however found that two patients in Esmolol group developed bradycardia 2 minutes following drug administration (HR<60) which responded to bolus dose of 0.3 mg atropine.

CONCLUSIONS

From the observation and results of this study, it is concluded that Esmolol 1.5 mg/kg given 3 minutes before extubation effectively attenuates hemodynamic response (Hypertension and tachycardia) to extubation immediately and remains effective until 10 minutes post extubation without any side effects. IV Esmolol 1.5mg/kg is preferred for attenuation when compared with IV Lidocaine 1.5mg/kg given 3 minutes prior to extubation. IV Lidocaine 1.5mg/kg when given 3 minutes prior to extubation is not effective immediately and attenuation of hemodynamic response to extubation occurred only after 3 minutes of extubation.

Conflict of interest: None

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