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ORIGINAL ARTICLE

ATTENUATION OF HEMODYNAMIC RESPONSE TO EXTUBATION WITH I.V. LIGNOCAINE: A RANDOMIZED CLINICAL TRIAL

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ABSTRACT: BACKGROUND AND OBJECTIVES: Hemodynamic and cough response to extubation can result in raised heart rate, blood pressures and intracavitary pressures which could be detrimental in high-risk patients. The aim of our study was to estimate the difference in hemodynamic and cough response to orotracheal tube extubation with saline (control group), I.V lignocaine 0.5mg/kg and I.V lignocaine 1mg/kg and to evaluate the comparative efficacy between the groups. **METHODS:** In our clinical prospective descriptive double blind study 90 patients of either sex scheduled for elective surgical procedures requiring orotracheal intubation, who met inclusion criteria, were considered. They were randomly divided into three groups of 30 each, Group-1 (control-saline), group-2 (lignocaine 0.5mg/kg) and group-3 (lignocaine 1mg/kg). They were administered study drug 2 minutes prior to extubation, following a standard peri operative anesthetic course. Hemodynamic parameters like heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure (HR, SBP, DBP and MAP) prior to administration of the study drug and at 1min, 3min, 5min and 10min post extubation were considered for statistical analysis. Post extubation cough graded as per Eshak's grading (Grade 0, 1, 2 and 3). Data obtained were analyzed using Analysis of variance (ANOVA), Post-hoc Tukey test and Chi-square/Fisher Exact test. Results on continuous measurement were, presented on Mean±SD. Significance was assessed at 5% level of significance. **RESULTS:** In control group, there was significant rise in HR, SBP and MAP throughout the study period and the incidence of moderate and severs cough was 43.3% and 30% respectively. Diastolic blood pressure and mean arterial pressures attenuation with lignocaine 1mg/kg found to be superior ($P<0.001$). There was no significant difference in heart rate and systolic blood pressure attenuation between patients who received 0.5mg/kg lignocaine and 1mg/kg lignocaine at 1min (P - 0.101 and P - 0.938 respectively). Post extubation cough suppression was 100% in patients who received lignocaine 1mg/kg. **CONCLUSION:** Study concludes that lignocaine 1 mg/kg is superior to 0.5 mg/kg in attenuating the hemodynamic responses to tracheal extubation. For post extubation cough suppression (100%) lignocaine 1mg/kg is ideal.

KEYWORDS: Extubation, hemodynamic response, attenuation, lignocaine.

INTRODUCTION: Physiologic responses to emergence from anesthesia and tracheal extubation include unwanted circulatory and airway reflexes. Is due to lighter planes of anesthesia with inadequate analgesia, which result in tachycardia, hypertension, coughing, bucking, laryngospasm and bronchospasm.[1- 3]

In the clinical practice respiratory complications after tracheal extubation are three times more common than during tracheal intubation and induction of anesthesia (12.6% vs. 4.6%).[4]

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Significant decrease in ejection fractions (from 55% ± 7% to 45% ± 7%) after extubation without electrocardiographic signs of myocardial ischemia was demonstrated with coronary artery disease patients.[2, 5]

Sympathetic stimulation during extubation may be detrimental, due to increased myocardial oxygen demand, subjecting the patient to have arrhythmia, myocardial ischemia, and infarction, pulmonary edema, cerebral hemorrhage, etc. These responses are marked in hypertensive patients, coronary artery disease patients and cerebrovascular disease patients.[6, 7, 8]

Bucking and coughing frequently occurs during extubation. Bucking physiologically mimics Valsalva maneuver. It could cause negative pressure pulmonary edema if lung volumes are less than vital capacity. They also cause abrupt increases in intracavitary pressures (intraocular, intrathoracic, intra-abdominal, and intracranial) which could put patient at high risk.[9, 10]

To blunt above mentioned hemodynamic and cough responses to extubation, several pharmacological strategies and extubation in deeper planes of anesthesia have been studied.[11, 12] Each one has its own merits and demerits.

In our study we had considered lignocaine in view of its action and to find the clinical effective dose to practice. It produces central sedation, suppresses autonomic reflexes, potentiates analgesia and may protect the ischemic myocardium from ultra-structural damage associated with high circulating catecholamine levels.[13]

The aim of our study was to estimate the difference in hemodynamic and cough response to orotracheal tube extubation with saline (control group), I.V lignocaine 0.5mg/kg and I.V lignocaine 1mg/kg and to evaluate the comparative efficacy between the groups.

METHODOLOGY: The randomized prospective double blind single centre study, was undertaken at tertiary care hospital after obtaining hospital ethical committee approval and informed written consent from the patients. The study included 90 patients of either sex of ASA (American Society of Anesthesiologist) grade I and II, with airway assessment of Mallampatti grade 1 and 2, between the age group of 18-60 years, scheduled for elective surgeries under general anesthesia posted for General Surgery, ENT (ear, nose and throat), Orthopedic, Gynecological, Plastic and Neurosurgical procedures requiring oral intubation. Patients were excluded if they were unwilling to participate, history of allergy to any drug, emergency surgeries, patients on beta blockers or calcium channel blockers, patients with bronchial asthma and cardiovascular disease, patients with documented intra operative hemodynamic compromise, patients with active upper respiratory tract infection, sore throat and history of laryngeal/tracheal surgery or pathology.

Randomization was done using random number table and were divided into three groups (n=30) Group-1 (control-saline), group-2 (lignocaine 0.5mg/kg) and group-3 (lignocaine 1mg/kg) to administer study drug 2 minutes prior to extubation. Two senior residents who were not involved in patient care generated random sequence and they were sequentially numbered. For blinding purpose, calculated dose of study drug was diluted to 5ml with saline in a 5cc syringe and labeled with sequential number. Anesthesiologist blinded to the study administered the study drug, recorded parameters and rating of post extubation cough. Patients were also blinded to the study drug. Samples were decoded for statistical analysis after the completion of the study.

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The incidence of post extubation cough was evaluated using a 4-point rating scale suggested by Eshak.[14] Grade 0 = no coughing or straining, Grade 1= moderate coughing, Grade 2 = marked coughing, straining and Grade 3 = poor extubation with laryngospasm.

On the day of surgery, confirming the pre-anesthetic check-up, patients were mobilized to the operation theatre. Securing the I.V line all patients were started on maintenance fluid (ringer's lactate). All patients were monitored with pulse oximetry, non-invasive blood pressure, electrocardiography and end tidal carbon dioxide throughout the surgery.

Premedication, induction, maintenance and perioperative analgesia was standardized in all three groups. At the conclusion of the surgery residual neuromuscular blockade was reversed with inj Glycopyrrolate 6μg/kg and inj neostigmine 0.05mg/kg when swallowing reflex was present, followed by study drug (either normal saline or inj Lignocaine 0.5mg/kg or inj Lignocaine 1mg/kg). Patients were extubated 2min after the study drug administration, establishing adequate tidal volume and oropharyngeal suctioning. Patients were assessed clinically for eye opening, and handgrip before extubation. At the time of extubation patients were assessed for the incidence of post extubation cough using 4-point scale suggested by Eshak. Following extubation patients were oxygenated with 100% oxygen through facemask for 5min. Later patients were shifted to recovery room. HR, SBP, DBP and MAP recorded just before reversal (base line value) and 1min, 3min, 5min and 10min following extubation was considered for statistical analysis.

Descriptive statistical analysis was carried out in our study. Results on continuous measurements were presented on Mean±SD (min-max). Significance was assessed at 5% level of significance. Analysis of variance (ANOVA) was used to find the significance of study parameters between the three groups of patients. Post-hoc Tukey test was employed to find the pair wise significance. Chi-square/Fisher Exact test was used to find the significance of study parameters on categorical scale between two or more groups. For analysis + Suggestive significance (P value: $0.05<P<0.10$), * moderately significant (P value: $0.01<P \leq 0.05$) and ** strongly significant (P value: $P \leq 0.01$).

ANALYSIS: The demographic characteristics age, sex, weight, ASA grade and duration of surgery are detailed in table I and samples are matching each other statistically.

In all three groups rise in HR, DBP and MAP were statistically significant throughout the study period in comparison with base line values, but SBP was comparable with base line values at 10min. DBP and MAP attenuation was superior with lignocaine 1mg/kg (Table 2, 3, 4 and 5) ($p < 0.001$). Post extubation cough suppression was 100% in patients who received lignocaine 1mg/kg (Table 6).

Fall in HR, SBP, DBP and MAP between group-1 and group-3 was statistically significant up to 5min. At 10min fall in SBP was comparable between the groups. Comparison between group-1 and group-2, in group-2 there was significant fall in HR and SBP up to 5min ($p <0.001$), whereas DBP and MAP were inconsistent (Table 2, 3, 4 and 5). There was no statistically significant difference in HR between group-2 and group-3 except for recordings at 1min (Figure 1). Infers that lignocaine 0.5mg/kg and 1mg/kg both are effective in suppressing the HR. However, comparison between group-1 and group-3 there was statistically significant difference in the HR ($p < 0.001$) (Table 2). Fall in SBP, DBP and MAP between group-2 and group-3 are not statistically significant throughout the study period (Table 3 and 4). The mean arterial pressures were blunted best in group-3 at 1min, 3min and 5 min post extubation in comparison with group-1{($p<0.001$) (Table 5) (Figure 2)}. In

conclusion, lignocaine 1mg/kg significantly attenuates the presser response compared to lignocaine 0.5mg/kg and control group.

Post extubation cough suppression was 100% in group-3 where as it was 56.7% in group-2 and 26.7% in group-1. Severe cough documented was 30% in group-1 and 13.3% in group-2 (Table 6). By the clinical observation lignocaine 1mg/kg was ideal in attenuating cough responses to tracheal extubation.

In conclusion, lignocaine 1mg/kg significantly attenuates the presser and cough response compared to control group. It is also clinically more effective than 0.5mg/kg lignocaine.

DISCUSSION: Significant increase in HR, MAP, cardiac index, systemic vascular resistance, pulmonary artery pressure and unwanted airway reflexes have been observed in response to tracheal extubation, which persist into the recovery period.[3] Bidwai et al[8] and Dyson et al[15] demonstrated increases of 20% or more in these hemodynamic variables following extubation in normotensive patients.

Lignocaine attenuates the hemodynamic response to tracheal extubation by its direct myocardial depressant effect, central stimulant effect, and peripheral vasodilatory effect and finally it suppresses the cough reflex, an effect on synaptic transmission.[16] So we had considered lignocaine in our study.

Mahmood Saghaei et al[17] has done 2 phases study on 200 adult patients, randomly divided into two groups to receive either IV lignocaine 1mg/kg or normal saline as placebo. Post extubation cough were compared between the two groups. In the study population patients who had cough were randomly divided into 2 groups to receive IV lignocaine 0.5mg/kg or saline to abort the cough. Portion of patients, with successful response to lignocaine 0.5mg/kg were, compared with 1mg/kg lignocaine group. Outcome result of the study was that prophylactic administration of lignocaine prior to extubation could be ineffective in preventing post extubation cough. Recommendation was, to treat cough upon occurrence, instead of routine prophylactic administration of lignocaine. In this study, the comparison was between 1gm/kg and 0.5mg/kg I.V lignocaine. Outcome does not suggest, which dose is ideal to suppress cough reflex. Keeping it in view, we had considered 3groups in our study. Group I control (saline), Group II (lignocaine 0.5mg/kg) and Group III (lignocaine 1mg/kg).

Hamaya et al[18] reported that the maximal plasma lignocaine concentration was 4.3 - 2.5µg/mL 5 minutes after IV injection of lignocaine 1mg/ kg. It is more than the plasma concentration required to suppress cough reflex (2.3µg/mL). Cough suppression was 100% with lignocaine 1mg/kg in our study. This explains lignocaine 1mg/kg is the clinical dose to suppress cough.

Bidwai AV, Stanley TH, and BidwaiVA[8] did a study on heart rate and blood pressure response to endotracheal extubation with I.V lignocaine 1mg/kg and control group (saline). Patients in lignocaine group did not sustain significant elevation in HR or SBP at or after extubation compared with saline group.

Considering the above two references, we chose 1mg/kg lignocaine as one of our study drug dose.

Lidocaine when administered I.V has an onset of action within 45-30 seconds with peak effect at 1-2 min. Mikawa et al[19] reported that IV lignocaine two minutes prior to tracheal extubation attenuates increases in HR, SBP, DBP and the cough reflex. Considering it, in our study we administered the study drug 2min prior to extubation.

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Lowrie et al[20] evaluated the impact of tracheal extubation on changes in plasma concentrations of epinephrine and Norepinephrine in 12 patients undergoing major elective surgery. They found significant increase in epinephrine levels from 0.9 to 1.4 pmol/mL 5min, after extubation. In our study in all three groups HR had increased at 1min following extubation. Explanation could be due to increase in plasma epinephrine levels.

Fujii et al[21] did a randomized double blind study on hypertensive (ASA 2) patients undergoing elective orthopedic surgeries with 0.2mg/kg diltiazem or 1mg/kg lignocaine IV or both together before tracheal extubation. Hemodynamic changes were less in patients receiving diltiazem plus lignocaine than in those receiving diltiazem or lignocaine as sole agent. In our study too, lignocaine 1mg/kg was not effective in suppressing hemodynamic response to extubation.

C.S. Sanikop[22] in his study on pre extubation intravenous lignocaine 2mg/kg, to prevent post extubation laryngospasm in children operated for cleft lip and cleft palate surgery found HR, BP and oxygen saturation being well maintained at 1min, 2min, 3min, 5min and 10min following administration of I.V lignocaine. In our study, observations are comparable with lignocaine 1mg/kg.

Chandra K. Pandey et al[23] studied the effects of lignocaine 1.5mg/kg to suppress fentanyl (3μg/kg) induced cough in a randomized double blind pattern. Lignocaine administered 1min prior to fentanyl, had significantly reduced fentanyl induced cough in comparison with placebo. In our study, we had administered lignocaine 1mg/kg 2min prior to extubation to suppress post extubation cough. Cough suppression was 100% in our study group.

CONCLUSION Based on the present clinical comparative study the following conclusions are drawn.

With lignocaine 1mg/kg rise in HR, SBP, DBP and MAP were 11.02%, 3.64%, 8.13%, and 5.72% at 3 minutes post extubation compared to pre extubation values. Where as in control group it was 45.29%, 21.09%, 20.31% and 21.57% respectively, which is statistical strongly significant ($p < 0.001$)

Post extubation cough suppression was 100% with lignocaine 1mg/kg, which was 56.7% with 0.5mg/kg lignocaine and 26.7% with saline (control group).

Inference from our study is that lignocaine 1mg/kg is superior to 0.5 mg/kg in attenuating the hemodynamic responses to tracheal extubation, which is statistically highly significant ($p < 0.001$). For post extubation cough suppression (100%) lignocaine 1mg/kg is ideal.

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	Group I	Group II	Group III
Age (Years)	38.60±11.64	42.50±12.03	40.30±10.21
Male/Female	18/12	11/19	14/16
Weight (kg)	61.10±8.27	62.17±7.06	58.20±7.83
ASA I/II	20/10	16/14	17/13
Duration of surgery (hrs.)	3.50±1.36	2.68±1.05	2.98±1.39

Table 1: Patients data

HR (bpm)	Group I	Group II	Group III	P value	Pair wise significance		
					Group I vs Group II	Group I vs Group III	Group II vs Group III
Baseline	80.27±6.49	81.60±8.29	80.10±5.22	0.645	0.728	0.995	0.669
1 minute	117.10±10.92	94.00±7.28	89.27±7.86	<0.001**	<0.001**	<0.001**	0.101
3 minutes	116.63±13.83	98.03±6.12	88.93±7.39	<0.001**	<0.001**	<0.001**	0.001**
5 minutes	106.17±13.50	95.10±7.73	86.63±7.99	<0.001**	<0.001**	<0.001**	0.005**
10 minutes	84.77±6.35	86.23±6.69	78.03±4.69	<0.001**	0.610	<0.001**	<0.001**

Table 2: Comparison of heart rate in three groups

SBP (mm hg)	Group I	Group II	Group III	P value	Pair wise significance		
					Group I vs Group II	Group I vs Group III	Group II vs Group III
Baseline	127.33±9.54	126.53±6.26	129.10±11.86	0.566	0.943	0.752	0.550
1 minute	155.37±9.75	134.67±8.80	133.8±10.92	<0.001**	<0.001**	<0.001**	0.938
3 minutes	154.1±11.38	137.6±8.58	133.8±10.42	<0.001**	<0.001**	<0.001**	0.323
5 minutes	143.73±12.68	136.77±9.15	133.17±9.44	0.001**	0.032*	0.001**	0.387
10 minutes	131.50±9.52	133.27±8.48	128.27±9.76	0.112	0.741	0.371	0.098+

Table 3: Comparison of SBP (mm Hg) in three groups

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DBP (mm hg)	Group I	Group II	Group III	P value	Pair wise significance		
					Group I vs Group II	Group I vs Group III	Group II vs Group III
Baseline	74.47±6.69	75.70±3.77	76.17±6.15	0.491	0.679	0.481	0.946
1 minute	88.87±7.07	91.17±7.05	81.83±5.63	<0.001**	0.374	<0.001**	<0.001**
3 minutes	89.60±4.95	93.53±7.00	82.37±4.43	<0.001**	0.021*	<0.001**	<0.001**
5 minutes	85.33±6.16	91.23±7.65	81.67±6.07	<0.001**	0.003**	0.089÷	<0.001**
10 minutes	78.33±5.59	85.90±8.13	77.80±5.89	<0.001**	<0.001**	0.948	<0.001**

Table 4: Comparison of DBP (mm Hg) in three groups

MAP (mm hg)	Group I	Group II	Group III	P value	Pair wise significance		
					Group I vs Group II	Group I vs Group III	Group II vs Group III
Baseline	91.33±6.44	92.63±10.16	93.70±6.23	0.505	0.796	0.473	0.858
1 minute	111.70±5.60	105.20±7.04	99.07±6.26	<0.001**	<0.001**	<0.001**	0.001**
3 minutes	111.03±6.19	108.33±7.60	99.37±5.18	<0.001**	0.237	<0.001**	<0.001**
5 minutes	104.63±7.02	106.03±8.00	98.50±5.93	<0.001**	0.722	0.003**	<0.001**
10 minutes	95.47±5.72	103.27±8.30	94.53±6.04	<0.001**	<0.001**	0.856	<0.001**

Table 5: Comparison of MAP (mm Hg) in three groups

Cough	Group-I	Group-II	Group-III
No cough	8(26.7%)	17(56.7%)	30(100%)
Moderate cough	13(43.3%)	9(30.0%)	0
Severe cough	9(30.0%)	4(13.3%)	0
Poor extubation with laryngospasm	0	0	0
Total	30(100%)	30(100%)	30(100%)

Table 6: Comparison of Cough in three groups

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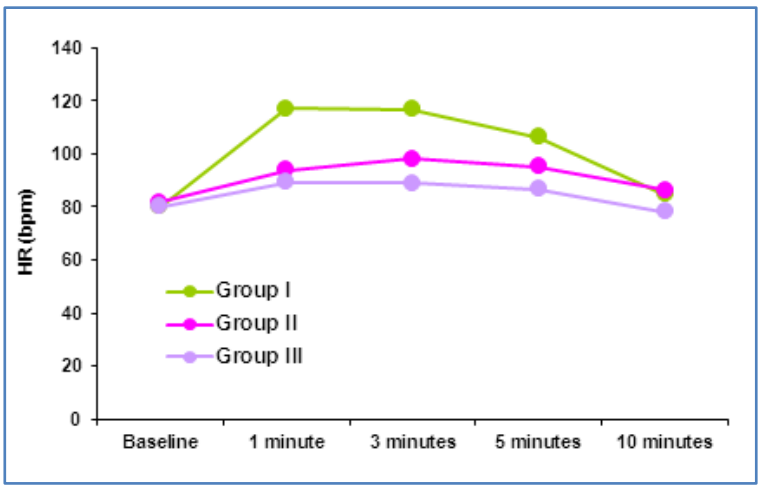

Fig. 1: Graphs depicting mean heart rate changes

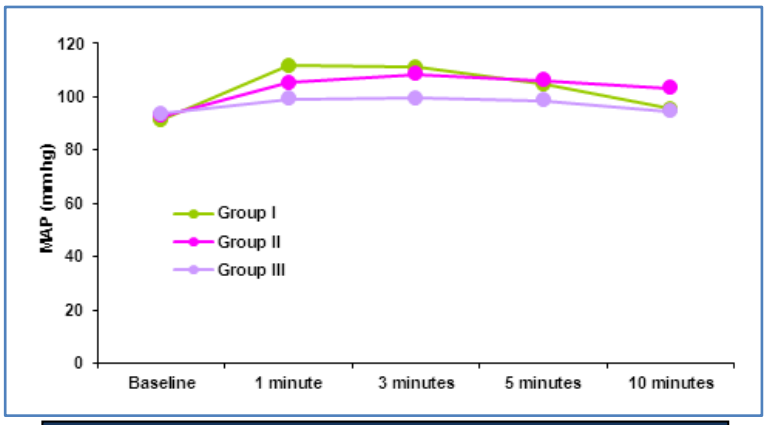

Fig. 2: Graphs depicting mean arterial changes

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