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Evaluation Of Two Different Doses of Injection Lignocaine in Attenuating Hemodynamic and Airway Responses to Extubation –A Clinical Study

Dr. Ashwini Bagade[1], Dr. Akshita Mohan[2], Dr. Anshu Priyanka Lakra*[3], Dr. Ankita[4], Dr. Jitendra Kushwaha[5], Dr. Rahul Choudhary[6]

[1]Assistant Professor, [2,6]Resident, RD Gardi Medical College & Hospital, Surasa, Ujjain, MP, India
[3,4,5]Assistant Professor, Trauma and Emergency Medicine, AIIMS Bhopal, India

***Corresponding Author:**
Dr. Anshu Priyanka Lakra,
Assistant Professor, Trauma and Emergency Medicine, AIIMS Bhopal, India

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ABSTRACT

Background and Aim: Tracheal extubation at the end of anaesthesia induces exaggerated haemodynamic and airway reflexes. Tracheal extubation accompanied by marked sympatho-adrenal response leading to tachycardia, hypertension,coughing,bucking,laryngospasm and branchospasm. Lignocaine is being used for many years to suppress the reflex responses to laryngoscopy and intubation but only a few studies have been carried out to suppress reflex response to extubation.

The objective of our study is to evaluate and compare the effectiveness of two different intravenous lignocaine doses (1 mg/kg and 1.5 mg/kg) in attenuating haemodynamic responses and airway reflexes during extubation.

Material and Method: This prospective, comparative study was carried out at GMC Bhopal following approval from the Institutional Ethics Committee. A total of 75 patients, aged between 20 and 50 years and classified as ASA physical status I and II, undergoing various surgeries under general anaesthesia were enrolled. The patients were randomly allocated into three groups of 25 each: Group I (control group) received normal saline, Group II received lignocaine 1 mg/kg, and Group III received lignocaine 1.5 mg/kg. Data analysis was performed using IBM SPSS Statistics software, applying the unpaired t-test. A p-value of less than 0.05 was considered statistically significant.

Result: In the control group (Group I), a significant increase in heart rate (HR), systolic blood pressure (SBP), and mean arterial pressure (MAP) was observed throughout the study period, with moderate and severe cough occurring in 44% and 32% of patients, respectively. The attenuation of diastolic blood pressure and mean arterial pressure was significantly better with lignocaine 1.5 mg/kg (Group III) compared to the other groups ($P<0.001$). No significant difference in systolic blood pressure was noted between patients who received 1 mg/kg and 1.5 mg/kg of lignocaine. A significant reduction in heart rate was observed in Group III compared to Group II. Complete suppression of post-extubation cough was achieved in patients administered 1.5 mg/kg of lignocaine.

Conclusion: Based on our study findings, we conclude that lignocaine at a dose of 1.5 mg/kg is significantly more effective than 1 mg/kg in attenuating Hemodynamic responses and Cough reflex during and following extubation.

Keywords: *Lignocaine, haemodynamic and airway reflexes, tracheal extubation*

1. INTRODUCTION

Tracheal extubation following anaesthesia is often linked to undesirable circulatory and airway reflexes, including tachycardia, hypertension, coughing, bucking, laryngospasm, and bronchospasm.[1,2,3] These reflexes can be potentially life-threatening, particularly in patients with cardiac or respiratory conditions, as they may result in increased myocardial oxygen demand, myocardial ischemia, infarction, or pulmonary edema.[4,5,6]

Various drugs such as beta-blockers, calcium channel blockers, alpha-2 agonists, and lignocaine have been employed to attenuate these reflexes, though each has its own limitations and potential side effects. Lignocaine, in particular, suppresses autonomic reflexes, enhances analgesia, induces central sedation, and may offer protection to ischemic myocardium against

damage caused by elevated circulating catecholamine levels.[7]
Lignocaine has been used for many years to suppress reflex responses associated with laryngoscopy and intubation; however, only a limited number of studies have focused on its role in controlling reflex responses during extubation.[6,8,9] Therefore, we designed this study to determine the optimal dose of lignocaine for attenuating airway and hemodynamic responses during laryngoscopy and extubation.

2. MATERIAL AND METHOD

The study will be conducted following approval from the Institutional Ethics Committee. Patients who refuse to participate, have a known hypersensitivity to lignocaine, are on beta-blockers or calcium channel blockers, or those with bronchial asthma, chronic obstructive pulmonary disease (COPD), cardiovascular disease, active upper respiratory tract infections, or anticipated difficult airway will be excluded. All patients will undergo a pre-anaesthetic evaluation. After obtaining informed written consent, standard anaesthetic management will be administered as follows: Premedication will include intravenous glycopyrrolate (0.1 mg/kg), midazolam (0.05 mg/kg), fentanyl (2 mcg/kg), and ondansetron (0.1 mg/kg). After pre-oxygenation with 100% oxygen for three minutes, anaesthesia induction will be carried out using intravenous propofol (2 mg/kg), followed by muscle relaxation with vecuronium bromide (0.1 mg/kg) to facilitate tracheal intubation. Anaesthesia maintenance will involve a mixture of oxygen and nitrous oxide in a 40:60 ratio along with a step-down infusion of propofol. Non-invasive monitoring, including pulse oximetry, electrocardiography, non-invasive blood pressure measurement, and end-tidal CO_2 monitoring, will be used throughout the procedure. At the end of surgery, both the propofol infusion and nitrous oxide will be discontinued. The study drug will be prepared and administered two minutes prior to extubation by an independent third party, who will not be involved in further phases of the study.

Parameters to be monitored and recorded

1. **Hemodynamic parameters:** Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) will be recorded before administration of the study drug and subsequently at 1, 3, 5, and 10 minutes after extubation.
2. **Post-extubation cough incidence:** Coughing after extubation will be assessed using a 4-point scale proposed by Eshak, where Grade 0 indicates no coughing or straining, Grade 1 represents moderate coughing, Grade 2 signifies marked coughing or straining, and Grade 3 denotes poor extubation associated with laryngospasm.

Statistical Analysis

Results for continuous variables will be expressed as Mean ± Standard Deviation (SD). Data analysis will be performed using IBM SPSS Statistics software. The unpaired t-test will be applied for statistical evaluation. The level of significance will be interpreted as follows: suggestive significance for P-values between 0.05 and 0.10, moderate significance for P-values between 0.01 and 0.05, and strong significance for P-values less than or equal to 0.01.

3. RESULTS

Table1: Patient data

	Group I	Group II	Group III
Age (Years)	39.50±10.46	41.70±11.23	40.59±10.33
Male/Female	12/13	11/14	13/12
Weight (kg)	61.33±7.24	62.23±6.72	61.87±6.83
ASA I/II	14/111	12/13	13/12

All three groups were comparable in terms of age, sex, weight, and ASA physical status. Baseline measurements of heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were similar across the groups ($p>0.05$). However, the control group (Group I) showed a significantly greater increase in HR, SBP, and MAP compared to Group II and Group III throughout the study duration.
On comparison between group I and group II ,there was significant fall in heart rate systolic bp mean bp throughout study period. Fall in dbp Between group Igroup II was statistically signicant upto 5 min.at 10 min fall in dbp was was comparable between group. Therewas significant attenuation of heart rate,systolic bp , diastolic bp,mean aeterial bp in group III as compare to group I throught study period.

Table2: Showing mean heart rate(beats/min)changes

HR (bpm)	Group-I	Group-II	Group-III	Group wise significance

				Group-I vs. group-II	**Group-I vs. group-III**	**Group-II vs. group-III**
Baseline	90.32±5.79	89.07±4.26	89.78±5.26	0.389	0.731	0.602
At 1 minute	111.42±10.27	98.21±7.20	92.62±4.12	<0.0001*	<0.0001*	0.0015*
At 3 minutes	108.11±11.11	96.70±6.59	90.70±3.38	<0.0001*	<0.0001*	<0.0002*
At 5 minutes	106.42±10.76	92.86±6.12	89.72±2.41	<0.0001*	<0.0001*	0.0210*
At 10 minutes	92.22±7.34	88.26±5.32	85.12±2.12	0.0334*	0.0016*	0.0086*

Figure1:showing mean heart rate(beats/min)changes

A significant rise in heart rate was observed postoperatively at one, three, five, and ten minutes after extubation in Group I when compared to Group II and Group III. Throughout the study period, heart rate attenuation was significantly greater in Group III compared to Group II ($p<0.05$).

Table3: Showing mean systolic blood pressure changes

Systolic Blood Pressure (mmHg)	**Group-I**	**Group-II**	**Group-III**	**Group Wise Significance**		
				Group-I Vs Group-II	**Group-I Vs Group-III**	**Group-II Vs Group-III**
Baseline	122.42±3.14	123.12±3.91	124.21±7.14	< 0.32	< 0.21	< 0.54
At 1 minute	148.24±2.89	120.39±6.13	119.48±4.17	< 0.0001	< 0.0001	0.2211
At 3 minute	146.34±3.78	119.17±5.74	119.48±2.12	< 0.0001	< 0.0001	0.6130
At 5 minute	135.70±5.72	118.13±5.66	117.28±6.28	< 0.0001	< 0.0001	0.3159
At 10 minute	133.11±2.10	117.79±4.79	116.89±8.37	< 0.0001	< 0.0001	0.6429

A significant postoperative rise in systolic blood pressure was noted in Group I compared to Group II and Group III at one, three, five, and ten minutes after extubation. However, no significant difference in systolic blood pressure was observed between patients who received 1 mg/kg and 1.5 mg/kg of lignocaine ($p>0.05$) throughout the study period.

Table4: showing mean diastolic blood pressure changes

Diastolic Blood Pressure (mmHg)	**Group-I**	**Group-II**	**Group-III**	**Group Wise Significance**		
				Group-I Vs Group-II	**Group-I Vs Group-III**	**Group-II Vs Group-III**
Baseline	76.70±3.83	76.25±4.77	76.42±5.11	0.7126	0.8274	0.9037
At 1 minute	87.12±10.16	82.33±6.49	78.91±4.11	< 0.0001	< 0.0001	< 0.0001
At 3 minute	89.59±3.89	80.34±5.19	77.41±5.51	< 0.0001	< 0.0001	< 0.0001
At 5 minute	86.82±7.69	79.17±6.14	76.50±6.10	< 0.0001	< 0.0001	0.0023
At 10 minute	76.11±4.03	76.28±4.65	74.35±3.11	< 0.7826	< 0.0010	0.0014

A significant postoperative rise in diastolic blood pressure was observed in Group I compared to Group II and Group III at one, three, five, and ten minutes after extubation. The attenuation of diastolic blood pressure was found to be superior with lignocaine 1.5 mg/kg (Group III) compared to the other groups throughout the study period ($P<0.05$).

Table5: showing mean blood pressure changes

Mean Systolic Blood Pressure (mmHg)	**Group-I**	**Group-II**	**Group-III**	**Group Wise Significance**		
				Group-I Vs Group-II	**Group-I Vs Group-III**	**Group-II Vs Group-III**
Baseline	91.47±6.32	92.82±7.09	92.66±6.29	0.1567	0.1835	0.8661
At 1 minute	114.20±6.20	90.80±8.00	81.57±5.65	< 0.0001	< 0.0001	< 0.0001
At 3 minute	114.53±7.04	89.62±7.96	80.72±7.02	< 0.0001	< 0.0001	< 0.0001
At 5 minute	162.49±5.61	89.16±5.60	80.59±4.93	< 0.0001	< 0.0001	< 0.0001

At 10 minute	91.72±5.47	87.03±5.71	75.71±4.93	< 0.0001	< 0.0001	< 0.0001

A significant postoperative rise in mean arterial pressure was noted in Group I compared to Group II and Group III at one, three, five, and ten minutes after extubation. Attenuation of mean arterial pressure was significantly better with lignocaine 1.5 mg/kg (Group III) than in the other groups throughout the study period (P<0.001).
Table 6 presents the comparison of cough incidence among the three groups. In Group I, the incidence of moderate and severe coughing was 44% and 32%, respectively. Complete suppression of post-extubation cough was achieved in patients administered lignocaine 1.5 mg/kg.

4. DISCUSSION

- A significant increase in hemodynamic variables (20% or more) has been demonstrated following extubation in normotensive patients (Bidwai et al).[6]
- Lignocaine attenuates these hemodynamic responses by its direct myocardial depressant and peripheral vasodilatory effects and a suppressant effect on synaptic transmission transmission responsible for cough reflex. [10]
- Earlier studies were unable to determine an effective clinical dose for suppressing these reflexes, with 0.5 mg/kg found to be ineffective and 1 mg/kg shown to be only moderately effective in controlling cough reflexes.[11]
- Hence, we carried out this study to evaluate lignocaine in doses of 1mg/kg and 1.5mg/kg.As Intravenous lignocaine attains its peak effect in 1-2 minutes, hence we gave it 2 minutes prior to extubation. [12]
- Lowrie et al [13] evaluated significant increase in plasma ephinephrine level over the period of 5 min.after extubation
- In our study heart rate had increased at 1 min after extubation. .This could be due to increased ephinephrine level.
- The findings of our study are consistent with those reported by Sanikop CS. [8]

who administered lignocaine 2 mg/kg to prevent post-extubation laryngospasm, and Chandra K. Pandey et al., who utilized lignocaine 1.5 mg/kg to suppress fentanyl-induced cough.[14]

5. LIMITATION OF STUDY

• We did not measure plasma norepinephrine levels or assess the effect of the drug on the requirement for other anaesthetic agents.
• A limitation of the present study is its small sample size, indicating the need for replication in studies with a larger population.

6. CONCLUSION

A dose of 1.5 mg/kg significantly attenuates hemodynamic and cough responses compared to the control group (Group I) and is also clinically more effective than 1 mg/kg of lignocaine.

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