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Comparison of iv lignocane and iv labetalol for the attenuation of hemodynamic response to laryngoscopy and endotracheal intubation

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Abstract

Introduction: Laryngoscopy and endotracheal intubation result in pressor and sympathoadrenal responses. Certain agents such as Lignocaine and Labetalol can be given to prevent these hemodynamic changes prior to intubation.

Materials and Methods: 60 patients of ASA grade I, scheduled for various surgeries under general anaesthesia were divided into 2 Groups. Patients in Group A were given 2% Inj. Lignocaine hydrochloride (preservative free) intravenously at 1.5mg/kg over 10 seconds, 90 seconds before laryngoscopy. Patients in Group B were administered 0.5 mg/kg Inj. Labetalol IV, 10 minutes before intubation. Laryngoscopy and endotracheal intubation were performed after induction. Heart rate, systolic, diastolic and the mean arterial blood pressure were recorded before induction, after induction, during intubation and at 1, 3 and 5 minutes post intubation.

Results: Heart rate, systolic and diastolic blood pressures and the mean arterial pressures were significantly lower in the Labetalol group when compared to the Lignocaine group, with the mean arterial pressure and the heart rate attaining the basal value in the Labetalol group after 5 minutes of intubation, while it was still higher in the Lignocaine group.

Conclusion: Labetalol is a better drug for the attenuation of the hemodynamic changes that take place during intubation compared to Lignocaine.

Keywords: Labetalol, lignocaine, hemodynamic changes, laryngoscopy, endotracheal intubation

Introduction

Laryngoscopy and endotracheal intubation cause stimulation of oropharynx and laryngopharynx and result in pressor and sympathoadrenal responses [1]. Even without intubation, similar pressor response is observed with laryngoscopy. In healthy adults, it is seen to start within 5-30 seconds, reach peak at 1-2 minutes and return back to baseline in 5-10 minutes [2, 3].

The placement of the endotracheal tube into the trachea is called the endotracheal intubation. This may be done through the nasal or oral route. Factors that precipitate pressor response to laryngoscopy and intubation are light planes of anesthesia, greater force to displace tongue, prolonged duration of laryngoscopy and repeated attempts at laryngoscopy/ intubation. Since 1950, some of the hemodynamic changes that have been recognized are increase in serum catecholamine's, hypertension and tachycardia. These responses are transient and normally seen in all patients during laryngoscopy and manipulation of the epiglottis [4, 5].

Increase in cardiac output results in the increase in arterial blood pressure and is further

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associated with a rise in the central venous pressure [6]. In healthy individuals, these hemodynamic changes may be negligible and well tolerated. But in with patients with comorbidities like hypertension, coronary artery disease, myocardial insufficiency etc., these changes may be exaggerated and even life threatening in certain cases [6, 7]. In patients with decreased intracranial compliance like those with a head injury, cerebral neoplasms or cerebral aneurysms, this sudden rise in blood pressure may have catastrophic results. In pre-eclampsia patients, there may be a precipitation of convulsions [8].

Attenuation of stress response can be achieved by limiting the duration of laryngoscopy to 15-20 seconds, use of various pharmacological agents like Lignocaine, Magnesium sulphate, α-2 blockers like Dexmedetomidine, β adrenergic blockers like Labetolol, Esmolol, Opioids such as Fentanyl etc, but none has yet been considered ideal.

Labetalol is a combined α-1 and nonselective β adrenergic blocker, oral and a parenteral antihypertensive drug. After IV injection, it reaches its peak in 5-15 mins and immediately gets redistributed, causing a reduction in the blood pressure by decreasing systemic vascular resistance (α-1 blockade) and the reflex tachycardia triggered by vasodilation is attenuated by β blockade and stabilizing the cardiac output [5]. Lignocaine is an amide local anaesthetic and also an anti-arrythmic drug. It can be administered in the form of a spray or gargle or used intravenously (preservative free) prior to the laryngoscopy [9]. It is very cost effective and a has been used extensively for attenuation of pressor response during intubation [9, 10]; however its exact mechanism of action is still unclear. After IV injection, its action starts within 45 seconds. Its half-life is around 8 minutes after which it is metabolized in the liver and excreted.

This study was conducted to compare the attenuation of hemodynamic response during laryngoscopy and endotracheal intubation of Lignocaine with Labetalol.

Materials and Methods

60 patients scheduled for various surgeries under general anaesthesia, were included in this study done by the Department of Anaesthesiology, Mediciti Institute of Medical Sciences. After obtaining clearance from the Institute's ethical committee, this study was done over a period of one year from March 2021 to February 2022. Patients of either gender, between the age of 18 to 65 years, with ASA Grade I status were included in this study and their demographic details were noted. The nature of the study was explained to all the patients and their relatives in detail and informed consent was obtained from them.

All the patients underwent a thorough pre-anesthetic clinical and laboratory evaluation prior to surgery. Airway assessment was done in detail and any patient with features suggestive of an anticipated difficult airway was excluded from the study. Patients suffering from respiratory, cardiac, hepatic and renal disorders were also excluded from the study.

The patients who were finally included in the study were divided into 2 groups of 30 each. Patients in Group A received 1.5 ml/kg of Lignocaine hydrochloride (preservative free) intravenous over 10 seconds. This was given 90 seconds prior to laryngoscopy and intubation. Patients in Group B were given 0.5 mg/kg Labetalol intravenous over 5 minutes. This was given 10 minutes before intubation.

In the pre-operative ward, intravenous access was secured with an 18G cannula and infusion of Ringer Lactate was started. Patients were then shifted to OT and Non-invasive monitors such as blood pressure, pulse oximeter and ECG were connected. All patients were pre-medicated with Inj. Ondansetron 4 mg, Inj. Glycopyrrolate 0.2 mg and Inj. Tramadol 1 mg/kg. Patients were pre-oxygenated with 100% oxygen for 3 minutes and then induced with 5mg/kg Inj. Thiopentone sodium. Inj Vecuronium bromide 0.1mg/kg loading dose was given for muscle relaxation.

Intubation was done using Macintosh laryngoscope blade and Portex cuffed endotracheal tube. The total time taken for intubation in each patient was <20 seconds. If intubation time exceeded 20 seconds, then these patients were excluded from the study. Heart rate, systolic and diastolic blood pressure and the mean arterial pressure were recorded before induction, after induction, during intubation and at 1, 3 and 5 minutes post intubation. Anesthesia was

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maintained with intermittent positive pressure ventilation with nitrous oxide and oxygen in the ratio of 66%: 33% with Isoflurane 0.8-1%. Analgesia and muscle relaxation were supplemented with aliquots of Inj. Tramadol 0.1 mg/kg and 0.02 mg/kg Vecuronium bromide respectively. After the completion of surgery, Inj. Glycopyrolate 0.2 mg and Inj. Neostigmine 0.05 mg/kg were administered for reversal of the neuromuscular blockade and extubated patients were. Follow-up was done for all the patients.

Results

There were 30 patients each in Group A and Group B. In both the groups, number of male and female patients were comparable (Fig: 1).The number of males in Group A were 18 (60%) and females were 12 (40%), while in Group B, the number of males were 16 (53.33%) and the females were 14 (46.67%) There was no statistically significant difference between the groups (p>0.05) (Fig: 1).

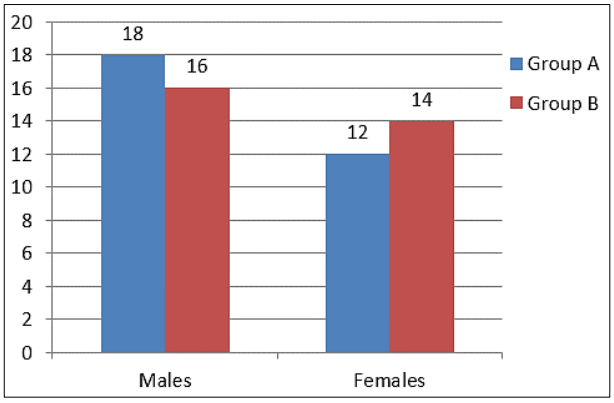

Fig 1: Gender wise distribution of patients

The mean age of the patients in Group A was 33.8 years and in Group B, it was 35.9 years. The mean weight of the patients in Group A was 58.9 kgs and in Group B it was 57.3 kgs. No statistical difference was seen among both the groups. (p>0.05) (Table: 1).

Table 1: Demographic details of the patients

Details	Group A (Lignocaine)	Group B (Labetalol)
Mean Age (in years)	33.8	35.9
Mean weight (in kgs)	58.9	57.3

Before induction, the hemodynamic parameters in the two groups were comparable. The heart rate (beats/min) pre induction was 89.5 ± 10.4 in Group A, while it was 88.9 ± 10.4 beats/min in Group B. The systolic and diastolic pressure in Group A before induction was 123.4 ± 4.82 mmHg and 78.2 ± 7.1 mmHg respectively, while in Group B, it was 119.5 ± 6.2 mmHg and 76.1 ± 5.2 mmHg respectively. Mean arterial pressure before induction was 94.2 ± 4.7 in Group A and 92.2 ± 8.5 in Group B. There was no statistical difference among the groups before induction. (p > 0.05).

After induction, the heart rate in Group A was 114.45 ± 9.46 beats/min and in Group B was 91.46 ± 6.75 beats/min. There was an increase in heart rate in both the groups after induction, but the rise in heart rate in Labetolol group was less when compared to Lignocaine group. The systolic and diastolic pressure after induction were 111.7 ± 8.1mmHg and 72.82 ± 3.67 mmHg respectively in Group A and 107.3 ± 6.2 and 69.86 ± 3.29 mmHg respectively in Group B. Mean arterial pressure after induction was 84.24 ± 5.2 in Group A and 83.24 ± 6.74 in Group B. It is seen that there was a slight reduction in the systolic, diastolic and mean blood pressure in both the groups. There was no statistical significance between both the groups. This slight decrease in blood pressure and increase in Heart rate in both the groups after induction is probably due to vasodilatory action of Thiopentone sodium. (Table: 2).

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Table 2: Blood pressure and heart rate before induction and after induction

Blood pressure	Group A (Lignocaine)		Group B (Labetalol)	
	Before induction	After induction	Before induction	After induction
Heart Rate (Rate/min)	89.5 ± 10.4	114.45 ± 9.46	88.9 ± 10.4	91.46 ± 6.75
Systolic Blood Pressure (mm/Hg)	123.4 ± 4.82	111.7 ± 8.1	119.5 ± 6.2	107.3 ± 6.2
Diastolic Blood Pressure (mm/Hg)	78.2 ± 7.1	72.82 ± 3.67	76.1 ± 5.2	69.86 ± 3.29
Mean arterial Pressure (mm/Hg)	94.2 ± 4.7	84.24 ±5.2	92.2 ± 8.5	83.24 ± 6.74

The Systolic and Diastolic Blood pressure in Group A during intubation were 147.34 ± 12.45 mmHg and 98.33 ± 8.3 mmHg and respectively. The Systolic, Diastolic Blood pressure and Mean Arterial Pressure in Group B, at the time of intubation were 116.35 ± 5.8 mmHg and 81.56 ± 9.15 mmHg respectively. The mean arterial pressure in Group A and Group B was 119.42 ± 12.4 mmHg and 95.28 ± 7.2 mmHg respectively. The mean Heart rate (beats/per minute) in Group A during intubation was 123.71 ± 9.22 and in Group B was 92.85 ± 8.82. There was a rise in all the parameters in both the groups during intubation; however, the rise in Group A (Lignocaine) was significantly higher compared to Group B (Labetolol), showing that Labetolol has attenuated the rise in heart rate and blood pressure during intubation better than Lignocaine ($p < 0.05$).

One minute after intubation, heart rate in Group A and Group B was 116.04 ± 7.99 & 90.29 ± 9.16 beats/min respectively. One minute after intubation, systolic and diastolic blood pressures in Group A were 144.56 ± 9.39, 96.35 ± 7.2 mm Hg respectively, whereas in Group B were 117.34 ± 5.34, 79.33 ± 8.4 mm Hg respectively. Mean arterial pressures, one minute post intubation in Group A and B were 109.94 ± 11.45 mm Hg 88.47 ± 8.3 mm Hg respectively. Heart rate and Blood pressures were significantly lower in Labetolol group ($p< 0.05$).

Three minutes after intubation, heart rate was 111.73 ± 8.37 & 88.76 ± 7.89 beats/min in Group A and Group B respectively. Systolic and diastolic blood pressures were 135.48 ± 8.24, 87.49 ± 8.1 mm Hg respectively in Group A and 116.25 ± 7.2 and 77.4 ± 6.1mm Hg respectively in Group B. Mean arterial pressures, three minutes post intubation in Group A and Group B were 101.4 ± 5.3 and 87.43 ± 7.28 mm Hg respectively. Heart rate and blood pressure were still significantly higher in Lignocaine group compared to Labetolol group; three minutes after intubation ($p< 0.05$)

Five minutes post intubation, heart rate was 109.47 ± 6.91 & 86.27 ± 8.66 beats/min in Group A and group B respectively. Systolic and diastolic blood pressures were 123.64 ± 6.35 and 79.49 ± 6 mm Hg respectively in Group A and 117.57 ± 8.12 and 76.44 ± 5.1 mm Hg respectively in Group B. Five minutes post intubation; mean arterial pressures in Group A and B were 99.27 ± 8.33 and 85.02 ± 5.99 mmHg respectively. Blood pressure and Heart rate were significantly lower in Labetolol group compared to Lignocaine group ($p< 0.01$). Hemodynamic parameters in Labetolol group almost reached baseline values, five minutes after intubation (Table: 3).

Table 3: Blood pressure post intubation

Blood pressure	During intubation		1 min after intubation		3 mins after intubation		5 mins after intubation	
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
Heart Rate (Rate/min)	123.71± 9.22	92.85 ± 8.82	116.04 ± 7.99	90.29 ± 9.16	111.73 ± 8.37	88.76 ± 7.89	109.47 ± 6.91	86.27 ± 8.66
Systolic	147.34 ±	116.35 ±	144.56 ±	117.34 ±	135.48 ±	116.25 ±	123.64	117.57 ±

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Blood Pressure (mm/Hg)	12.45	5.8	9.39	5.3	8.24	7.2	± 6.35	8.12
Diastolic Blood Pressure (mm/Hg)	98.33 ± 8.3	81.56 ± 9.15	96.35 ± 7.2	79.33 ± 8.4	87.49 ± 8.1	77.4 ± 6.1	79.49 ± 6.9	76.44 ± 5.1
Mean arterial Pressure (mm/Hg)	119.42 ± 12.4	95.28 ± 7.2	109.94 ± 11.45	88.47 ± 8.3	101.4 ± 5.3	87.43 ± 7.28	99.27 ± 8.33	85.02 ± 5.99

Discussion

Endotracheal intubation is an essential procedure in all patients undergoing major surgery under general anesthesia. Laryngoscopy, endotracheal intubation, and other airway manipulations are noxious stimuli that may induce profound changes in cardiovascular physiology, initiated by proprioceptors responding to tissue irritation in the supraglottic region and in the trachea. The glossopharyngeal and vagal afferent nerves transmit these impulses to the brainstem, which, in turn, causes widespread activation of autonomic nervous system [11]. The result of laryngoscopy and endotracheal intubation is transient but marked pressor and sympathoadrenal response, causing tachycardia and hypertension [12, 13]. Some anesthesiologists consider the period of intubation to be of the greatest risk in surgeries of patients with hypertension, coronary artery disease and increased intracranial pressure [14]. In patients with limited cardiovascular reserve, Heart rate is an important factor affecting myocardial oxygen demand; therefore tachycardia may be detrimental as it may precipitate perioperative myocardial ischemia and myocardial infarction [15]. Certain conditions such as pulmonary edema, ventricular arrhythmias and rupture of cerebral aneurysms have been documented as complications of exaggerated response to laryngoscopy. The cardiovascular responses to laryngoscopy and tracheal intubation are well known and linked with increase in catecholamine blood levels [16]. These effects/responses can be reduced to a certain extent by deeper planes of anesthesia, but even this may be poorly tolerated in patients with reduced cardiovascular reserve. Various methods and drugs have been tried for blunting hemodynamic responses, but no single technique or drug has proven to be very effective. Therefore anesthesiologists are in constant search for an ideal agent to counteract the catecholamine surge during laryngoscopy and endotracheal intubation.

Lignocaine is a local anesthetic and an anti-arrythmic drug, which is used topically as well as intravenously. It is thought to blunt hemodynamic and cerebrovascular response to intubation when given in dose of 1.5 mg/kg IV by adding approximately 0.3 MAC of anesthetic potency [11] it is commonly used for attenuation of pressor response as it is readily available and very cost effective. Labetalol is a unique antihypertensive drug with selective α-1 blocking properties and nonselective β adrenergic blocking effects. It causes reduction in the blood pressure by decreasing systemic vascular resistance and attenuates reflex tachycardia triggered by vasodilation thereby stabilizing the cardiac output [5].

In the present study we have chosen to evaluate the efficacy of 0.5 mg/kg Labetalol and 1.5 mg/kg Lignocaine in attenuating stress response to laryngoscopy and intubation. Findings of each group were discussed in comparison with their pre-operative values and values at different time intervals with regard to heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure.

The number of males in Group A (Lignocaine) in our study was 60% and in Group B (Labetalol) it was 53.33%. The mean age of the patients in Group A was 33.8 years and Group B was 35.9 years. The mean weight was 58.9 kgs in Group A and 57.3 kgs in Group B. There was no significant difference in the age, gender and weight distribution of patients in both the groups. ($P > 0.05$).

Before induction, hemodynamic parameters in both the groups were comparable. After induction, there was an increase in heart rate in both the groups, but the rise in heart rate in

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Labetolol group was less compared to Lignocaine group. There was a slight reduction in the systolic, diastolic and mean blood pressure in both the groups but no statistical significance was noted. This slight decrease in blood pressure and increase in Heart rate in both the groups after induction is probably due to vasodilatory action of Thiopentone sodium.

During intubation, there was a rise in all the hemodynamic parameters in both the groups; however, the rise in Group A (Lignocaine) was significantly higher compared to Group B (Labetolol), showing that Labetolol has attenuated the rise in heart rate and blood pressure during intubation better than Lignocaine ($p< 0.05$).

One minute after intubation, heart rate and Blood pressures were significantly high in Lignocaine group compared to Labetolol group. ($p< 0.05$). Heart rate and blood pressure were still significantly higher in Lignocaine group compared to Labetolol group; three minutes after intubation ($p< 0.05$).

Five minutes post intubation, Blood pressure and Heart rate were significantly lower in Labetolol group compared to Lignocaine group ($p< 0.01$). Hemodynamic parameters in Labetolol group almost reached baseline values, five minutes after intubation.

In our study, Labetalol was found to be a better agent for effective attenuation of the heart rate during intubation when compared to Lignocaine and this was also observed by Jaiswal *et al*. [17] Jaiswal *et al*. also reported that the Lignocaine did not prevent a rise in the heart rate post induction, but blunted the rise seen with laryngoscopy and endotracheal intubation to certain degree, although it could not totally attenuate it. A peak rise of the heart rate post induction was probably due to vasodilation caused by Thiopentone sodium and subsequently during intubation was due to catecholamine release. Another study by Ramanathan *et al*. reported that 1mg/kg Labetalol was more effective than Lidocaine in attenuating the pressor response to intubation [18].

Rise in heart rate during intubation was successfully attenuated by Labetalol in a study by Maharaja RJ *et al*. [19]. A study done by Ratnani. E *et al.*, showed that Labetalol found to be better for the attenuation of HR, SBP, DBP, mean arterial pressure and RPP during and after laryngoscopy and endotracheal intubation [20]. The hemodynamic parameters were relatively more stable in labetalol group intraoperatively as compared to esmolol and lignocaine. This coroborates with our study. A study by Kiran Kumar *et al*, also showed that Labetalol was more effective in attenuating the heart rate and the blood pressure compared to Esmolol and Lignocaine [21].

A study by Singh *et al*, where Labetalol was compared with Esmolol in low doses, it was found that Esmolol had no significant effect on the pressor response while Labetalol showed attenuation of the hemodynamic response to intubation [8]. In our study both Labetalol and Lignocaine did not cause any side effects in intraoperative and postoperative period.

Newer aids have been developed to facilitate laryngoscopy and intubation and lessen the hemodynamic stress response. Video laryngoscopes like Airtraq, C Mac, Mc Grath laryngoscope, McCoy laryngoscope, Fibreoptic bronchoscope etc have been found to lower the sympathoadrenal response among patients, thereby reducing the rise in blood pressure and heart rate during intubation. Concurrent use of these devices with drugs like Labetolol may provide better results for attenuation of stress response to laryngoscopy**.**

Conclusion

Labetalol is a more effective drug in attenuation of hemodynamic response to laryngoscopy and endotracheal intubation in comparison to Lignocaine. Since its half-life is 5.5 hours, it prevents adverse reactions even during extubation. Labetalol is readily available, cost effective and easily administrable with minimal side effects and a better choice than lignocaine for use during general anesthesia.

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