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# ATTENUATION OF HEMODYNAMIC RESPONSE DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION WITH TWO DIFFERENT DOSES OF LABETALOL IN CONTROLLED HYPERTENSIVE PATIENTS

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**Keywords:**

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**ABSTRACT: Aim:** The aim of the present study was to compare the efficacy of two different doses of labetalol for controlling these hemodynamic responses to laryngoscopy and tracheal intubation in controlled hypertensive patients. **Material and Methods:** Present study conducted on 90 patients posted for various elective surgeries under general anesthesia at our institute. Study populations were randomly divided into 3 groups. Group L1: Received Inj Labetalol 0.15 mg/kg iv 5 min before intubation, Group L2: Received Inj. Labetalol 0.3 mg/kg iv 5 min before intubation and Group C: Received 0.9% normal saline iv 5 min before intubation. Heart rate (HR), systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded prior to induction, at time of intubation and various intervals after intubation. **Results:** The control in HR, SBP, DBP, MAP and RPP was statistically significant between Group L1 and Group L2 at 3 min, 5 min post intubation. There was significant difference in HR throughout study time between L1 C and L2 C. **Conclusion:** Labetalol in both doses is effective in reducing the hemodynamic stress response in controlled hypertensive patients in dose dependent manner during laryngoscopy and tracheal intubation and was attenuated more with Labetalol 0.3mg/kg iv compared to 0.15mg/kg iv dosage.

**INTRODUCTION:** Despite the emergence of new airway devices in the recent years, rigid laryngoscopy and tracheal intubation still remain the gold standard in airway management.

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Laryngoscopy, endotracheal intubation, and other airway manipulations (e.g., placement of a nasopharyngeal or oropharyngeal supralaryngeal airway) are noxious stimuli that may induce profound changes in cardiovascular physiology, primarily through reflex responses. These changes are even more profound in hypertensive patients. Direct Laryngoscopy and endotracheal intubation are invariably associated with certain cardiovascular changes such as hypertension, tachycardia and wide variety of cardiac arrhythmias [1, 2, 3].

Although these responses may be of short duration, variable, unpredictable and of little consequence in healthy individuals. However, these changes can facilitate and accelerate the development of myocardial ischemia, arrhythmia, infarction and cerebral haemorrhage in patients with coronary artery disease, hypertension or cerebrovascular disease.

Regardless of the preoperative blood pressure control in hypertensive patients there was an excessive rise in BP following endotracheal intubation.

A variety of drugs and methods have been tried to attenuate this stress response considering their ability to block the intense sympathetic discharge during airway stimulation [5-10].

1. Premedicating patient with antihypertensive drugs.
2. Vasodilators (eg. hydralazine).
3. Beta blockers (eg. Esmolol, labetalol).
4. Calcium channel blockers (eg. nifedipine).
5. α-2 agonists (clonidine, dexmedetomidine).
6. Nitroglycerine (intravenous, intranasal spray or sublingual).
7. ACE inhibitors (eg. captopril, enalapril).
8. Opioids (fentanyl, remifentanil, alfentanyl, sufentanyl).
9. Lignocaine (intravenous, spray or gargles).

Deepen plane of anaesthesia by intravenous induction agent or increasing concentration of volatile anaesthetic during mask ventilation. Decreasing laryngoscopy time to less than 15 seconds.

Labetalol is an unique oral and parenteral antihypertensive drug that is alpha- 1and nonselective b1- and b2-adrenergic antagonist. It reaches its peak effect at 5–15 min after intravenous (IV) injection and rapidly redistributes (5.9 min redistribution half-life). It lowers BP by decreasing systemic vascular resistance (α1-blockade), whereas reflex tachycardia triggered by vasodilatation is attenuated by simultaneous β-blockade. Cardiac output remains unchanged [11].

The aim of the present study was to compare the efficacy of two different doses of labetalol for controlling these hemodynamic responses to laryngoscopy and tracheal intubation under the same anaesthetic techniques in controlled hypertensive patients.

**MATERIAL AND METHODS:** Present randomized Placebo controlled study conducted on 90 patients aged between 30 years to 60 years of either sex belonging to ASA class II (controlled hypertensives) posted for various elective surgeries under general anesthesia at our institute.

**Inclusion Criteria:**

- Age 30 to 60 years.
- ASA II.
- Undergoing elective surgery of longer than one hour duration.

**Exclusion Criteria:**

- ASA grade I, III, IV and V.
- Known case of diabetes, bronchial asthma, COPD, IHD.
- Patients with atrial/ventricular arrhythmias, second/third degree A-V conduction block.
- Patients with heart disease, congestive Heart Failure and terminal valvular insufficiency.
- Patients with severe hemodynamic in stability like severe anaemia, hypotension.
- Patients on beta adrenergic antagonist therapy
- Patients with anticipated difficult airway.
- Patients requiring more than 20s or requiring one attempt at intubation.
- Patients known to have allergy to anaesthetic drugs used in study.

After obtaining institutional ethical committee approval, written informed consent was obtained from the patients. Data was collected in study proforma meeting the aims and objectives of the study. Study population (90 patients) were randomly divided by computer generated numbers into 3 groups with 30 patients in each group.

**Group L1:** Received Inj Labetalol 0.15 mg/kg iv 5 min before intubation.

**Group L2:** Received Inj. Labetalol 0.3 mg/kg iv 5 min before intubation.

**Group C:** Received 0.9% normal saline (5ml) iv 5 min before intubation.

All the patients underwent a detailed pre anaesthetic check-up on the day before surgery and all the routine and specific investigations were advised and evaluated in the morning on the day of surgery. History pertaining to hypertension like duration, medications (ACE inhibitors, calcium channel blockers, beta blockers, diuretics. Whenever necessary special tests were carried out.

The patients were electively kept nil by mouth for 6 hours before surgery and prior to surgery patients were explained about the procedure and informed consent were taken. Morning dose of antihypertensive drugs was given at 6 am on the day of the surgery with sips of water. After the patient was shifted to the operation theatre, standard monitors like ECG, NIBP, and pulse oximetry, $ETCO_2$ applied and baseline parameters [$SpO_2$, Heart rate (HR), Systolic blood pressure (SBP), Diastolic blood pressure (DBP), mean arterial pressure (MAP), Rate pressure product (RPP)] were recorded. Two intravenous lines with 18/20-gauge cannula were secured and intravenous fluid was started.

Patients were premedicated with:

- Inj. Ondansetron 0.15 mg/kg i.v. 10 min prior to induction
- Inj. Glycopyrrolate 4µg/kg i.v. 10 min prior to induction
- Inj. Fentanyl 2 µg/kg iv 10 min prior to induction

The patients were pre oxygenated for 5 min using 100% oxygen with Bain's circuit with $O_2$ flow at 8-10L/min. Induction of anaesthesia was carried out using Inj. Propofol 2mg/kg iv, Inj. Vecuronium bromide 0.1 mg/kg iv to facilitate intubation and Intermittent positive pressure ventilation given for 3 min. Anaesthesia was maintained using 50% oxygen, 50% nitrous oxide, 1% sevoflurane and Inj. Vecuronium Bromide 0.08mg/kg IV.

**Monitoring:**

- Heart rate (HR)
- Systolic blood pressure (SBP)
- Diastolic blood pressure (DBP)
- Mean arterial blood preesure (MAP)
- Rate Pressure Product (RPP)
- Pulse oximetry (SpO2)
- End tidal carbon dioxide (ETCO2)

All parameters were recorded at following stages:

- ✓ Baseline
- ✓ After pre-medication.
- ✓ After intubation.
- ✓ At 1,2,3,5 and 10 min after intubation.

**Reversal of Anesthesia:** Neuromuscular blockade was reversed after onset of spontaneous respiration by using Inj. Glycopyrrolate 8µg/kg iv and Inj. Neostigmine 0.05 mg/kg i.v.

**Extubation:** After satisfied criteria for extubation, thorough oral and endotracheal suction was done, and trachea was extubated. Any intraoperative complications like bradycardia, hypotension, arrhythmias, laryngospasm were recorded and managed according to standard protocols.

**Statistical Analysis:** All patient's data were recorded in proforma of study. Data was expressed as mean values ± standard deviation (SD). Quantitative data was analyzed using t-test and qualitative by chi square test. Statistical calculations were carried out using Microsoft Office Excel 2010 and Graph Pad Prism 6.05 (quickcalc) Software (Graph pad software inc. La Jalla CA USA). Changes in hemodynamic variables from baseline and a comparison of means were analyzed by paired t- test for each time interval. A p-value $<0.05$ was considered statistically significant and p-value $>0.05$ was considered non-significant.

**RESULTS:** 90 patients aged between 30 years to 60 years of either sex belonging to ASA class II posted for various elective surgeries under general anesthesia at our institute were randomly selected

and divided by computer generated numbers into 3 groups with 30 patients in each group.

Group L1 received Inj. Labetalol 0.15mg/kg iv 5 min before intubation.

Group L2 received Inj. Labetalol 0.3mg/kg iv 5 min before intubation.

Group C received 0.9%. normal saline (5ml) iv 5 min before intubation.

**TABLE 1: DEMOGRAPHIC DATA**

| Variables | Group L1(n=30) Mean±SD | Group L2(n=30) Mean±SD | Group C(n=30) Mean±SD |
|---|---|---|---|
| Age (yrs) | 46.87±6.56 | 48.6 ± 6.85 | 46.73±6.17 |
| Sex (female/male) | 18/12 | 17/13 | 13/17 |
| Weight (kg) | 55.63± 6.71 | 58.4 ± 4.36 | 58.667±5.48 |
| **Antihypertensive medications** | | | |
| Calcium channel blockers (no of patients) | 18 | 16 | 19 |
| Renin angiotens ininhibitors (no of patients) | 12 | 14 | 11 |

As per **Table 1**, all the three groups were comparable with respect to age, sex, weight and type of antihypertensive drug.

**TABLE 2: COMPARATIVE CHANGES IN MEAN HEART RATE**

| Time | Mean heart rate (bpm) | | | | | |
|---|---|---|---|---|---|---|
| | GroupL1 | Group L2 | Group C | Pvalue | | |
| | Mean± SD | Mean± SD | Mean± SD | L1L2 | L2C | L1C |
| Baseline | 89.43±12.52 | 87 ±13.98 | 88.43±14.29 | 0.480 | 0.696 | 0.774 |
| After premedication | 85.11±12.96 | 81.73±12.16 | 86.56±13.53 | 0.303 | 0.151 | 0.669 |
| After intubation | 101.21±10.19 | 97.81 ±9.35 | 105.53±12.82 | 0.183 | 0.009 | 0.152 |
| 1 min after intubation | 102.76±10.50 | 99.71±10.63 | 110.6±11.73 | 0.265 | 0.000 | 0.008 |
| 2 min after intubation | 100.76±9.49 | 96.43±10.29 | 112.76±10.839 | 0.095 | 0.0000 | 0.000 |
| 3 min after intubation | 99.96±9.29 | 94.13±10.28 | 108.83±10.645 | 0.024 | 0.0000 | 0.001 |
| 5 min after intubation | 91.43 ±9.01 | 84.63±10.86 | 101.73±10.32 | 0.010 | 0.0000 | 0.000 |
| 10min after intubation | 86.033±9.66 | 81.23±10.59 | 94.80 ±8.70 | 0.071 | 0.0000 | 0.000 |

**Table 2** shows the comparison of changes in mean heart rate at various predetermined time interval and P value of group L1L2, group L2 C and group L1C to determine the significance of the changes in heart rate between three groups. The values of HR prior to intubation were statistically not significant between all three groups ($p>0.05$). At 3 min and 5 min post intubation, there was statistically significant difference in between L1 and L2 groups ($p<0.05$). There was significant difference in HR throughout study time between L1 and control group ($p<0.05$), and L2 and control group ($p<0.05$).

**TABLE 3: COMPARATIVE CHANGES IN MEAN SYSTOLIC BLOOD PRESSURE**

| | Mean systolic blood pressure (mmHg) | | | | | |
|---|---|---|---|---|---|---|
| | Group L1 | Group L2 | Group C | P value | | |
| | MEAN ± SD | MEAN ± SD | MEAN ± SD | L1L2 | L2C | L1C |
| Baseline | 125.81 ± 9.87 | 126.13 ± 10.72 | 127.23 ± 9.27 | 0.9007 | 0.6724 | 0.5645 |
| After premedication | 123.16 ± 8.94 | 124.16 ± 9.02 | 126.31 ± 8.03 | 0.6679 | 0.3371 | 0.1587 |
| After intubation | 135.03 ± 6.66 | 134.43 ± 9.83 | 143.43 ± 7.91 | 0.7830 | 0.0002 | 0.0000 |
| 1 min after intubation | 136.33 ± 6.80 | 132.43 ± 9.40 | 147.3 ± 7.53 | 0.0708 | 0.0000 | 0.0000 |
| 2 min after intubation | 133.36 ± 6.76 | 128.7 ± 9.15 | 147.96 ± 6.40 | 0.0285 | 0.0000 | 0.0000 |
| 3 min after intubation | 129.13 ± 6.22 | 121.03 ± 8.79 | 144.86 ± 6.12 | 0.0001 | 0.0000 | 0.0000 |
| 5 min after intubation | 124.76 ± 6.63 | 118.46 ± 9.09 | 139.36 ± 6.02 | 0.0033 | 0.0000 | 0.0000 |
| 10 min after intubation | 121.56 ± 6.80 | 111.86 ± 9.73 | 132.96 ± 5.92 | 0.0000 | 0.0000 | 0.0000 |

**Table 3** shows the comparison of changes in SBP at various predetermined time interval and P value of group $L_1L_2$, group $L_2C$ and group $L_1C$ to determine the significance of the changes in SBP between three groups. The values of SBP between L1 and L2 at intubation and 1 min post intubation were statistically not significant ($p>0.05$). However, there was statistically significant difference between L1 and L2 group at 2min,3min,5min and 10 min post intubation ($p<0.05$). These may be because of higher dose of labetalol used in L2 (0.3mg/kg iv) group as compared to L1 (0.15mg/kg iv) group. In placebo group C, values of SBP from baseline to the time of

intubation were high compared to the labetalol group L1 and L2. There was significant difference in SBP throughout study time, between L1 and control group C ($p<0.05$), and L2 and control group C ($p<0.05$).

**TABLE 4: COMPARATIVE CHANGES IN MEAN DBP**

| Time | Mean diastolic blood pressure (mmHg) | | | | | |
|---|---|---|---|---|---|---|
| | Group L1 | Group L2 | Group C | P value | | |
| | MEAN ± SD | MEAN ± SD | MEAN ± SD | L1L2 | L2C | L1C |
| Baseline | 80.26 ± 9.38 | 82.06 ± 8.44 | 81.26 ± 6.90 | 0.4372 | 0.6893 | 0.5998 |
| After premedication | 78.23 ± 7.45 | 80.46 ± 7.58 | 81.2 ± 5.02 | 0.2547 | 0.6604 | 0.0761 |
| After intubation | 89.53 ± 6.40 | 88.63 ± 6.19 | 89.8 ± 3.79 | 0.5822 | 0.3826 | 0.0027 |
| 1 min after intubation | 86.2 ± 6.75 | 86.36 ± 5.79 | 91.63 ± 3.61 | 0.9189 | 0.0001 | 0.0024 |
| 2 min after intubation | 85.86 ± 6.83 | 82.53 ± 5.91 | 94.06 ± 2.80 | 0.0480 | 0.0000 | 0.0001 |
| 3 min after intubation | 83.6 ± 6.64 | 77.76 ± 5.58 | 91.33 ± 2.83 | 0.0005 | 0.0000 | 0.0001 |
| 5 min after intubation | 78.2 ± 6.86 | 71.36 ± 5.14 | 88.66 ± 2.46 | 0.0001 | 0.0000 | 0.0001 |
| 10 min after intubation | 76.6 ± 6.83 | 67.53 ± 4.62 | 84.8 ± 2.86 | 0.0000 | 0.0000 | 0.0001 |

**Table 4** shows the comparison of changes in mean DBP at various predetermined time intervals and P value of group $L_1$ $L_2$, group $L_2$C and group $L_1$ C to determine the significance of the changes in DBP between three groups. The values of DBP between L1 and L2 at intubation and 1 min post intubation were statistically not significant ($p>0.05$). However, there was statistically significant difference between L1 and L2 group at 2 min, 3 min, 5 min and 10 min post intubation ($p<0.05$). These may be because of higher dose of labetalol used in L2 (0.3mg/kgiv) group as compared to L1 (0.15mg/kgiv) group. In placebo group C, values of DBP from base line to the time of intubation were high compared to the labetalol group L1 and L2. There was significant difference in DBP throughout study time, between L1 and control group C($p<0.05$), and L2 and control group C($p<0.05$).

The values of MAP between L1 and L2 at intubation and 1 min post intubation were statistically not significant ($p>0.05$). However, there was statistically significant difference between L1 and L2 group at 2 min, 3 min, 5 min and 10 min post intubation ($p<0.05$). These may be because of higher dose of labetalol used in L2 (0.3mg/kg iv) group as compared to L1 (0.15mg/kg iv) group. In placebo group C, values of MAP from baseline to the time of intubation were high compared to the labetalol group L1 and L2. There was significant difference in MAP throughout study time, between L1 and control group C ($p<0.05$), and L2 and control group C ($p<0.05$). The values of RPP between L1 and L2 at intubation and 1 min post intubation were statistically not significant ($p>0.05$). However, there was statistically significant difference between L1 and L2 group at 2min,3min,5min and 10 min post intubation ($p<0.05$). These may be because of higher dose of labetalol used in L2 (0.3mg/kg iv) group as compared to L1(0.15mg/kg iv )group. In placebo group C, values of RPP from baseline to the time of intubation were high compared to the labetalol group L1 and L2. There was significant difference in RPP throughout study time, between L1 and control group C ($p<0.05$), and L2 and control group C ($p<0.05$). The values of $SpO_2$ were comparable between the three study groups. There were no adverse effects or complications observed in the study groups.

**DISCUSSION:** In normal patients, hemodynamic stress response to laryngoscopy and endotracheal intubation has always become a challenge for anaesthetists. It manifests in the form of hypertension, tachycardia, and arrhythmias and may prove disastrous. C. Prys-Roberts *et al* (1971) [5] reported high incidence of cardiac arrhythmias, myocardial ischemia-infarction, acute LVF and cerebrovascular accidents following intubation in patients of hypertension, myocardial insufficiency, pre-eclampsia, eclampsia and in raised intracranial tension. It would seem prudent to adopt preventive measures to attenuate the hemodynamic stress response which otherwise may lead to dangerous complications or even sudden death. Hence it becomes the moral obligation of anesthesiologist towards any patient to ensure attenuation of this cardiovascular to laryngoscopy an intubation. The adverse cardiovascular changes and catecholamine discharge seen during laryngoscopy and tracheal intubation appear in two phases. The effects of

laryngoscopy should be distinguished from effects seen while the endotracheal tube is placed through trachea. Laryngoscopy alone without intubation causes a supraglottic stimulus in which both SBP and DBP increased when compared to Heart rate. Increase in BP is due norepinephrine, while increase in heart rate is due to epinephrine. Endotracheal intubation creates an extra cardiovascular response and catecholamine discharge due to infraglottic stimulus. Stress response increases at this stage and both SBP and DBP increase by 36-40% in contrast to control levels. HR levels increase more than 20% with tracheal intubation in contrast to laryngoscopy [12]. As discussed earlier, various drugs and techniques have been tried to attenuate this hemodynamic response to laryngoscopy and tracheal intubation [1, 3, 4, 13, 14, 15].

Labetalol is an antihypertensive drug that decreases the pressure response of intubation by alpha-1($\alpha_1$) and nonselective beta (β) adrenergic receptor blockade. It reaches its peak effect at 5-15 min after iv injection and rapidly distributes. It lowers BP by decreasing systemic vascular resistance (α1-blockade), whereas reflex tachycardia triggered by vasodilatation is attenutated by simultaneous β-blockade. Cardiac output remains unchanged. The onset of action of labetalol 2-3 min and peak effect reaches at 5-15min. The hemodynamic response to laryngoscopy and intubation is believed to lasts for a period of 10 mins. Roelofse *et al* [16] found that labetalol of dosage 1mg/kg given as an iv bolus 1 min before laryngoscopy was not effective in attenuation of HR. This failure of the study drug can be explained by the time of administration of the study drug because labetalol has peak effect after 5-10 min.

All patients in our study were demographically comparable with respect to age, sex, weight and type of antihypertensive drug in between three groups.

There was significant difference in HR throughout study time post intubation, between L1 and control group ($p<0.05$), and L2 and control group ($p<0.05$). Heart rate was found to be more controlled in L2 group than L1 group which demonstrates the dose dependent property of labetalol. D. Amar, H. Shamoon, W.H. Frishman *et al* [7] who administered 0.15- 0.3mg/kg of labetalol, showed in placebo group C an increase in HR up to 33% compared to labetalol group. We found similar results in our study. Kim SS, Kim JY, Lee JR *et al* [17] reported that a single dose of labetalol 0.25mg/kg iv given 5 min before intubation decreases HR significantly after intubation up to 10 min.

In our study the values between L1 and L2 at intubation and 1 min post intubation were statistically not significant ($p>0.05$). However, there was statistically significant difference between L1 and L2 group at 2 min, 3 min, 5 min and 10 min post intubation($p<0.05$). These may be because of higher dose of labetalol used in L2 (0.3mg/kg iv) group as compared to L1 (0.15mg/kg iv) group. Leslie JB *et al.*[18] demonstrated that a dose dependent blocking effect of labetalol on the hemodynamic stress response at various doses 0.25, 0.5,0.75, 1mg/kg produce significant reductions in SBP. Babita *et al* [1] reported that single dose of labetalol 0.25mg/kg iv given 5 min before intubation showed statistically significant decrease in SBP ($p<0.05$) when compared to fentanyl 2mcg/kg iv. In our study, group L2 (0.3mg/kg iv) SBP values were more controlled when compared to group L1 (0.15mg/kg iv).

In our study, the values between L1 and L2 at intubation and 1 min post intubation were statistically significant ($p>0.05$). However, there was statistically significant difference between L1 and L2 group at 2 min, 3 min, 5 min and 10 min post intubation ($p<0.05$). Leslie JB *et al* [18] demonstrated that a dose dependent blocking effect of labetalol on the hemodynamic stress response at various doses 0.25,0.5,0.75,1mg/kg produce significant reductions in DBP.

There was statistically significant difference between L1 and L2 group at 2 min, 3 min, 5 min and 10 min post intubation ($p<0.05$).In placebo group C, values of MAP from baseline to the time of intubation were high compared to the labetalol group L1 and L2.D. Amar *et al* [7] who administered 0.15-0.3mg/kg of labetalol, showed in placebo group C increase in MAP upto 52% compared to labetalol group($P<0.001$). In our study similar results were found, labetalol group L1 and L2 reduced MAP by 15% (L2>L1) over the hemodynamic stress response than control group C.

RPP in various study groups were recorded after the administration of the study drug. In our study the values between L1 and L2 at intubation and 1 min post intubation were statistically not significant ($p>0.05$). However, there was statistically significant difference between L1 and L2 group at 2 min, 3 min, 5 min and 10 min post intubation ($p<0.05$). In the present study, the RPP changes from baseline values after intubation in Group L1 and L2 were significantly less than Group C. Singh SP *et al* [19] showed that there was statistically significant difference in RPP values of labetalol group at all times ($p<0.001$)), at intubation, 1min and 10 min post intubation in the labetalol group($p=0.042$). The RPP in the labetalol group never crossed the critical 1500 mark. During anesthesia, it is desirable to avoid the combination of tachycardia and hypertension both of which increase myocardial oxygen consumption ($mVO_2$). $mVO_2$ correlates well with the product of systolic pressure and heart rate. RPP is an index of myocardial oxygen demand. All three groups were observed for complications like bradycardia, bronchospasm, hypotension, arrhythmia and allergy to drug. In our study none of the patients developed the above-mentioned complications.

**CONCLUSION:** Labetalol in both doses is effective in reducing the hemodynamic stress response in controlled hypertensive patients in dose dependent manner during laryngoscopy and tracheal intubation and was attenuated more with Labetalol 0.3mg/kg iv compared to 0.15mg/kg iv dosage.

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