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Assessing the Hemodynamic Effects of Labetalol Compared to Lignocaine During Intubation in Normotensive Patients at Tertiary Hospital at Barisal

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Abstract **Original Research Article**

Background: Laryngoscopy and endotracheal intubation provoke transient sympathetic stimulation leading to tachycardia and hypertension, which may be harmful even in normotensive patients with occult cardiovascular risk, particularly in resource-limited tertiary settings. Various pharmacological strategies have been used to attenuate this response; however, the comparative efficacy of labetalol versus lignocaine in normotensive adults in Bangladeshi tertiary hospitals has not been well defined. ***Objective:*** To assess and compare the hemodynamic effects of intravenous labetalol versus lignocaine in attenuating the cardiovascular responses to laryngoscopy and endotracheal intubation among normotensive adult patients undergoing general anesthesia at a tertiary hospital in Barisal. ***Methods:*** This prospective, randomized, double-blind clinical trial included 80 ASA I II normotensive adults (18 - 60 years) scheduled for elective non-cardiac surgery under general anesthesia with orotracheal intubation, allocated to labetalol (LB, n=40) or lignocaine (LG, n=40). Patients in group LB received labetalol 0.25 mg/kg IV and those in group LG received lignocaine 1 mg/kg IV, each diluted to 10 ml and administered over 60 seconds, 5 minutes before laryngoscopy. Standardized anesthesia was used in both groups; heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at baseline and at predefined time points, with primary analysis focused on MAP and HR at 1 minute after intubation. Data were analyzed using appropriate parametric and non-parametric tests with $p < 0.05$ considered statistically significant. ***Results:*** Demographic variables (age, sex, weight, ASA status) and baseline hemodynamic parameters were comparable between groups. At 1 minute after intubation, patients in the labetalol group exhibited significantly lower HR (88.9 ± 8.5 vs. 96.7 ± 9.4 beats/min), SBP (132.9 ± 11.2 vs. 144.8 ± 13.6 mmHg), DBP (83.1 ± 8.6 vs. 92.4 ± 9.2 mmHg), and MAP (99.7 ± 8.4 vs. 109.9 ± 9.1 mmHg) compared with the lignocaine group (all $p < 0.001$). The mean increase in MAP from baseline was significantly smaller in the labetalol group (+9.2 ± 4.6 vs. +19.0 ± 6.3 mmHg) and the rise in HR was also attenuated (+8.3 ± 4.2 vs. +15.4 ± 5.1 beats/min; both $p < 0.001$). Episodes of peri-intubation hypertension and tachycardia were less frequent with labetalol, while the incidence of hypotension and bradycardia remained low and comparable between groups, with no serious drug-related adverse events. These findings are in line with contemporary trials reporting superior attenuation of intubation-induced pressor responses by labetalol compared with lignocaine. ***Conclusion:*** Intravenous labetalol 0.25 mg/kg was more effective than lignocaine 1 mg/kg in attenuating the hemodynamic responses to laryngoscopy and endotracheal intubation in normotensive adult patients undergoing elective surgery at a tertiary hospital in Barisal, without an increase in clinically relevant adverse events. Labetalol can therefore be recommended as a preferred agent over lignocaine for hemodynamic control during intubation in similar settings, with potential benefit in reducing peri-intubation cardiovascular risk.

Keywords: Labetalol, Lignocaine, Laryngoscopy, Endotracheal intubation, Hemodynamic response, Normotensive adults.

INTRODUCTION

Endotracheal intubation with direct laryngoscopy is an essential component of general anesthesia for securing the airway, but it is also a potent noxious stimulus that provokes significant sympathetic activation and hemodynamic perturbations. The resulting transient surges in heart rate (HR) and blood pressure (BP) are usually well tolerated in healthy individuals but they can precipitate myocardial ischemia,

arrhythmias, cerebral hemorrhage, or heart failure in vulnerable patients with limited cardiovascular reserve. Even among normotensive surgical candidates, repeated or prolonged laryngoscopy, suboptimal anesthetic depth or coexisting risk factors may accentuate these responses and increase perioperative morbidity. Consequently, attenuation of the pressor response to laryngoscopy and intubation remains a central objective of modern anesthetic practice, particularly in resource-limited tertiary hospitals where baseline cardiovascular disease burden is high and intensive care resources are constrained. In Bangladesh, tertiary hospitals such as those in Barisal serve a large catchment population and manage a wide spectrum of elective and emergency surgeries, underscoring the need for simple, effective and safe pharmacologic strategies to blunt intubation-induced hemodynamic stress in normotensive patients. [1]

The pathophysiology of the hemodynamic response to laryngoscopy and intubation is primarily mediated by reflex sympathetic stimulation resulting from mechanical stimulation of the supraglottic and infraglottic structures. Direct laryngoscopy and passage of the endotracheal tube activate afferent fibers of the glossopharyngeal and vagus nerves, leading to increased catecholamine release, vasoconstriction, tachycardia, and elevations in systolic, diastolic, and mean arterial pressures. These changes typically begin within seconds of laryngoscopy, peak within the first 1 2 minutes after intubation, and may persist for several minutes if not pharmacologically modulated. Intraoperative surges in rate pressure product, a surrogate of myocardial oxygen demand, have been associated with ischemic electrocardiographic changes, particularly in patients with coronary artery disease, long-standing hypertension, or diabetes. Although normotensive patients are considered lower risk, undiagnosed cardiovascular comorbidities are common in South Asian populations, making routine attenuation of this reflex response clinically relevant even in apparently healthy adults. [1-4]

Multiple pharmacologic approaches have been explored to blunt the hemodynamic response to airway instrumentation, including deepening the plane of anesthesia and the use of opioids, volatile agents, local anesthetics, vasodilators, and beta-adrenergic blockers. Intravenous lignocaine (lidocaine) has long been used because of its local anesthetic and antiarrhythmic properties with early reports suggesting some efficacy in attenuating HR and BP increases when administered before laryngoscopy or extubation. More recently, beta-adrenergic antagonists such as esmolol and labetalol have gained prominence due to their ability to directly blunt sympathetic responses, reduce rate pressure product and improve peri-intubation hemodynamic stability. However, each class of drug has distinct pharmacodynamic profiles, onset times, durations of action and side-effect spectra which influence their suitability for routine use in normotensive patients undergoing a variety of surgical procedures. [2,5]

Lignocaine is an amide local anesthetic that exerts its action by blocking voltage-gated sodium channels, thereby stabilizing neuronal membranes and attenuating conduction in sensory and autonomic fibers. When administered intravenously in doses of around 1 1.5 mg/kg shortly before intubation, it has been reported to reduce cough, limit airway reflexes and partially blunt increases in HR and BP associated with airway manipulation. Clinical studies evaluating peri-operative intravenous lignocaine infusions have also demonstrated benefits on pain scores and postoperative recovery, though these regimens are more complex to implement in routine practice. Nevertheless, evidence regarding the magnitude and consistency of lignocaine's effect on the pressor response to laryngoscopy is mixed; some trials report modest attenuation of hemodynamic changes, while others show limited benefit compared with placebo or alternative agents. Moreover, lignocaine lacks direct alpha- or beta-adrenergic blocking activity and its hypotensive or bradycardic effects are usually mild, which may be advantageous in normotensive patients but may also limit its ability to fully control the sympathetic surge during difficult or repeated intubation attempts. [5]

Labetalol is a combined nonselective beta-adrenergic (β1 and β2) and selective alpha1-adrenergic antagonist that produces a balanced decrease in heart rate, myocardial contractility, and peripheral vascular resistance. This dual mechanism makes labetalol particularly attractive for blunting the hemodynamic responses to acute sympathoadrenergic surges, such as those occurring during laryngoscopy, by simultaneously limiting tachycardia and hypertension without causing significant reflex tachycardia. Studies in the last decade have shown that low doses of intravenous labetalol administered before induction can significantly attenuate increases in HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and rate pressure product during laryngoscopy and intubation when compared with placebo or other agents such as esmolol. In one trial, labetalol at 0.25 mg/kg provided better protection than esmolol 0.5 mg/kg against rises in HR and SBP, with comparable safety profiles. Other clinical data suggest that labetalol, in appropriate doses, maintains hemodynamic stability without inducing clinically significant hypotension or bradycardia in ASA I II patients. This profile is especially relevant for normotensive adults, where excessive BP reduction is undesirable and may compromise organ perfusion. [4, 6]

A growing body of research has directly compared labetalol with lignocaine for attenuation of hemodynamic responses to laryngoscopy and intubation. A randomized study comparing low-dose labetalol and lignocaine observed that labetalol produced more stable HR and BP values during the peri-intubation period, with

significantly lower increases in SBP, DBP, MAP, and rate pressure product in the labetalol group. Another trial demonstrated that intravenous labetalol 1 mg/kg, given prior to induction, attenuated the pressor response more effectively than lignocaine, with only minimal variations in BP after intubation and no serious adverse events. Recent randomized controlled work from 2023 2024 also reported that patients receiving labetalol had significantly lower SBP and MAP after laryngoscopy compared with those given lignocaine, reinforcing the superiority of labetalol in controlling pressor responses during endotracheal intubation. These studies collectively indicate that although both drugs can blunt the hemodynamic response, labetalol offers more robust and consistent attenuation of sympathetic surges associated with airway instrumentation. [7]

Despite these advances, evidence specific to normotensive patients in the context of South Asian tertiary hospitals remains limited. Most published trials have been conducted in different geographic and health-system settings, often with varying anesthetic techniques, intubation times and patient profiles, which may limit direct applicability to routine practice in Bangladeshi institutions. Factors such as higher background rates of subclinical hypertension, diabetes, and ischemic heart disease, variability in anesthetic drug availability and differences in perioperative monitoring can influence both the baseline hemodynamic status and the clinical impact of pharmacologic interventions. Moreover, data from Bangladesh and neighboring countries suggest that perioperative cardiovascular complications remain a significant contributor to morbidity, even among patients classified as ASA I II, emphasizing the importance of context-specific evidence to inform practice. In Barisal, a major tertiary care hub in southern Bangladesh, there is a particular need to identify simple, cost-effective regimens that can be integrated into standard anesthetic protocols to improve hemodynamic control during intubation in normotensive adult patients.

Normotensive patients represent the majority of individuals presenting for elective surgery in tertiary hospitals, yet their hemodynamic responses to laryngoscopy and intubation can be unpredictable. Standard anesthetic induction techniques alone may not suffice to prevent significant transient increases in HR and BP, especially in patients with heightened anxiety, difficult airways or prolonged laryngoscopy times. While much of the literature focuses on high-risk or hypertensive populations, understanding how labetalol and lignocaine perform in normotensive adults is essential for developing rational, broadly applicable protocols that enhance safety without introducing unnecessary drug-related risks. Furthermore, the balance between efficacy and safety is critical in this group: agents that produce profound hypotension or bradycardia may be inappropriate, whereas drugs that offer stable attenuation with minimal adverse events are particularly valuable. Labetalol, by virtue of its mixed adrenergic blockade and lignocaine, with its local anesthetic and modest systemic effects, are both promising candidates but comparative data in normotensive patients in the Bangladeshi tertiary care context are scarce.

Given these considerations, there is a clear rationale to systematically evaluate and compare the hemodynamic effects of labetalol and lignocaine during laryngoscopy and endotracheal intubation in normotensive patients undergoing surgery at a tertiary hospital in Barisal. Such an evaluation will help clarify which agent offers superior control of HR, SBP, DBP, and MAP during the critical peri-intubation period, while also documenting any adverse hemodynamic events such as hypotension, bradycardia, or arrhythmias. By generating locally relevant evidence, this research can support evidence-based modifications of institutional anesthetic protocols, optimize drug selection for routine practice, and potentially reduce the risk of peri-intubation cardiovascular complications in a large cohort of normotensive surgical patients in southern Bangladesh.

Objective: To assess and compare the hemodynamic effects of intravenous labetalol versus lignocaine in attenuating the cardiovascular responses to laryngoscopy and endotracheal intubation among normotensive adult patients undergoing general anesthesia at a tertiary hospital in Barisal.

METHODOLOGY AND MATERIALS

Study design and setting

This study was designed as a prospective, randomized, double-blind, controlled clinical trial conducted in the Department of Anesthesiology of a tertiary care hospital in Barisal, Bangladesh. The study was carried out over a defined period after approval from the institutional ethics committee and after obtaining written informed consent from all participants.

Study population

The study population comprised adult patients scheduled for elective surgical procedures under general anesthesia requiring orotracheal intubation. Only normotensive patients were included to evaluate hemodynamic effects in this commonly encountered but relatively lower-risk group.

Inclusion criteria

Patients were included if they met the following criteria:

- Age between 18 and 60 years.
- American Society of Anesthesiologists (ASA) physical status I or II.
- Normotensive on preoperative assessment (systolic BP 100 139 mmHg and diastolic BP 60 89 mmHg).

- Scheduled for elective non-cardiac surgery under general anesthesia with endotracheal intubation.
- Mallampati airway class I II with anticipated easy intubation.

Exclusion criteria

Patients were excluded if they had:

- History of hypertension, ischemic heart disease, arrhythmias, heart failure or cerebrovascular disease.
- Current use of beta-blockers, calcium channel blockers, antiarrhythmics or other drugs affecting autonomic function.
- Bronchial asthma or chronic obstructive pulmonary disease.
- Known allergy or contraindication to labetalol, lignocaine or any study medication.
- Pregnancy or lactation.
- Anticipated difficult airway (Mallampati III IV, restricted mouth opening, cervical spine pathology).
- Baseline bradycardia (HR <60 beats/min) or hypotension (SBP <100 mmHg).

Sample size

The required sample size for this study was 40 patients in each group (total 80 patients), after adjustment for potential dropouts.

Using data from previous trials that compared labetalol and lignocaine, the sample size was calculated to detect a clinically significant difference of about 10 mmHg in mean arterial pressure (MAP) at 1 minute after intubation between the two groups, assuming a common standard deviation of approximately 15 mmHg, a two-sided alpha of 0.05 and a power of 80%. The initial calculation yielded around 36 participants per group, and this number was rounded up to 40 per group to compensate for an anticipated 10% dropout or protocol violation rate.

Randomization and blinding

Eligible patients were randomly allocated into two equal groups:

- Group LB (Labetalol group): received intravenous labetalol.
- Group LG (Lignocaine group): received intravenous lignocaine.

Randomization was performed using a computer-generated random number table and allocation concealment was ensured by sequentially numbered, opaque, sealed envelopes opened immediately before drug preparation. A trained anesthesia assistant not involved in patient management or data collection prepared the study drugs in identical 10-ml syringes labeled only with the study code, so that patients, the attending anesthesiologist, and the investigator recording data remained blinded to group allocation.

Study drugs and dosing

Patients in the two groups received study medications as follows:

- **Group LB:** Injection labetalol hydrochloride 0.25 mg/kg diluted with 0.9% saline to a total volume of 10 ml, administered intravenously over 60 seconds, 5 minutes before laryngoscopy.
- **Group LG:** Injection lignocaine (lidocaine) hydrochloride 1 mg/kg diluted with 0.9% saline to a total volume of 10 ml, administered intravenously over 60 seconds, 5 minutes before laryngoscopy.

These doses and timing were selected in accordance with contemporary randomized trials comparing labetalol and lignocaine for attenuation of intubation-induced hemodynamic responses.

Preoperative assessment and preparation

All patients underwent standard pre-anesthetic evaluation including medical history, physical examination, airway assessment and review of routine laboratory investigations. Baseline HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), and MAP were recorded in the preoperative area after at least 5 minutes of rest in the supine position. Patients were kept fasting as per institutional guidelines and premedication (such as oral anxiolytics or antacids) was administered uniformly, avoiding drugs known to significantly alter cardiovascular parameters.

Anesthetic technique

On arrival in the operating room, patients were connected to standard monitors: non-invasive BP, continuous ECG, and pulse oximetry. Baseline intraoperative HR, SBP, DBP, and MAP were recorded before any intravenous drugs were given. Intravenous access was secured with an appropriate-gauge cannula. All patients received a standardized premedication regimen, for example intravenous midazolam 0.02 mg/kg and fentanyl 1 2 µg/kg, according to departmental protocol.

General anesthesia was induced with intravenous propofol 2 2.5 mg/kg until loss of response to verbal commands and eyelash reflex. Neuromuscular blockade was achieved with succinylcholine 1.5 2 mg/kg or rocuronium 0.6 0.9 mg/kg, depending on institutional practice. After adequate jaw relaxation and muscle paralysis, direct laryngoscopy was performed using a standard Macintosh laryngoscope by an experienced anesthesiologist with a minimum of two years of post-qualification experience to minimize operator-related variability. Orotracheal intubation was attempted within 30 seconds; patients in whom intubation required more than two attempts or lasted

longer than 30 seconds were excluded from final analysis due to the additional hemodynamic impact of prolonged airway manipulation.

Anesthesia was maintained with a mixture of oxygen and nitrous oxide (if used) along with a volatile anesthetic such as isoflurane or sevoflurane, titrated to maintain an adequate depth of anesthesia and stable hemodynamics in both groups. Controlled ventilation was adjusted to maintain normocapnia according to end-tidal carbon dioxide monitoring.

Hemodynamic measurements

Hemodynamic variables recorded were HR, SBP, DBP, and MAP. Measurements were documented at predefined time points:

- T0: Baseline value in the operating room before premedication.
- T1: Just before administration of the study drug.
- T2: Immediately before laryngoscopy (after study drug and induction).
- T3: At intubation.
- T4: 1 minute after intubation.
- T5: 3 minutes after intubation.
- T6: 5 minutes after intubation.

Rate pressure product (RPP = HR × SBP) was calculated at the same time points as an indirect index of myocardial oxygen demand.

Outcome measures

Primary outcome

The primary outcome was the change in MAP from baseline (T0) to 1 minute after intubation (T4) between the labetalol and lignocaine groups.

Secondary outcomes

Secondary outcomes included:

- Changes in HR, SBP, DBP, and MAP at all time points (T2 T6) compared with baseline within and between groups.
- Changes in RPP at specified time points.
- Incidence of adverse hemodynamic events:
- Hypotension (MAP <60 mmHg or >20% decrease from baseline).
- Hypertension (>20% increase in MAP from baseline).
- Bradycardia (HR <50 beats/min).
- Tachycardia (HR >100 beats/min or >20% increase from baseline).
- Requirement for rescue medications such as ephedrine for hypotension and atropine for bradycardia.

Safety monitoring

Patients were continuously monitored for potential adverse effects related to the study drugs including severe hypotension, severe bradycardia, arrhythmias, bronchospasm, or allergic reactions. Any serious adverse event was managed promptly according to standard resuscitation protocols and was documented in detail. Patients with major protocol deviations or serious intraoperative complications not attributable to the study drugs were withdrawn from per-protocol analysis.

Data collection and management

All hemodynamic readings and perioperative events were recorded using a predesigned case-record form by an investigator who remained blinded to the group assignment. Data were subsequently entered into a password-protected electronic database, with periodic cross-checking against source documents to minimize transcription errors. Continuous variables were summarized as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages.

Statistical analysis

Statistical analysis was performed using a standard statistical software package. Normality of continuous variables was assessed with the Shapiro Wilk test. Between-group comparisons of normally distributed continuous variables (such as MAP at T4) were carried out using the independent-samples t-test, while non-normally distributed data were analyzed with the Mann Whitney U test. Within-group changes over time were evaluated using repeated-measures analysis of variance (ANOVA) or the Friedman test as appropriate. Categorical variables, including incidence of hypotension or bradycardia, were compared using the chi-square test or Fisher's exact test. A p-value of less than 0.05 was considered statistically significant.

Ethical considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee of the tertiary hospital in Barisal. All participants received a clear explanation of the study aims, procedures, possible risks and benefits in a language they understood, and written informed consent was obtained prior to enrollment. Confidentiality was ensured by de-identifying patient data and restricting access to study records. The study adhered to the principles of the Declaration of Helsinki and Good Clinical Practice guidelines, similar to other contemporary randomized clinical trials in anesthesiology.

RESULTS

Demographic and baseline characteristics

A total of 80 normotensive patients were analyzed with 40 patients in the labetalol group (LB) and 40 in the lignocaine group (LG). Demographic characteristics and baseline hemodynamic variables were comparable between the two groups, with no statistically significant differences.

Table 1: Demographic characteristics of the study population

Variable	Labetalol group (n=40)	Lignocaine group (n=40)	p-value
Age (years), mean ± SD	39.8 ± 10.2	40.6 ± 9.8	0.72
Weight (kg), mean ± SD	63.4 ± 8.7	64.1 ± 9.1	0.76
Sex, n (%) Male	24 (60.0)	23 (57.5)	0.82
Sex, n (%) Female	16 (40.0)	17 (42.5)	
ASA I, n (%)	26 (65.0)	25 (62.5)	0.81
ASA II, n (%)	14 (35.0)	15 (37.5)	

Baseline HR, SBP, DBP, and MAP before premedication were also similar between groups, indicating that randomization achieved well-balanced groups.

Table 2: Baseline hemodynamic variables (T0)

Parameter	Labetalol group (n=40) mean ± SD	Lignocaine group (n=40) mean ± SD	p-value
HR_T0 (beats/min)	80.6 ± 7.9	81.3 ± 8.1	0.68
SBP_T0 (mmHg)	120.8 ± 9.6	121.5 ± 10.1	0.78
DBP_T0 (mmHg)	75.4 ± 7.7	76.0 ± 8.0	0.74
MAP_T0 (mmHg)	90.5 ± 7.3	91.0 ± 7.5	0.79

Hemodynamic changes during intubation

After administration of the study drugs and following laryngoscopy and intubation, both groups showed an increase in HR and BP compared with baseline, but the magnitude of rise was significantly lower in the labetalol group at 1 minute after intubation (T4). This difference was most evident in MAP, which was the primary outcome.

Table 3: Hemodynamic variables 1 minute after intubation (T4)

Parameter	Labetalol group (n=40) mean ± SD	Lignocaine group (n=40) mean ± SD	p-value
HR_T4 (beats/min)	88.9 ± 8.5	96.7 ± 9.4	<0.001
SBP_T4 (mmHg)	132.9 ± 11.2	144.8 ± 13.6	<0.001
DBP_T4 (mmHg)	83.1 ± 8.6	92.4 ± 9.2	<0.001
MAP_T4 (mmHg)	99.7 ± 8.4	109.9 ± 9.1	<0.001

The mean increase in MAP from baseline (ΔMAP = MAP_T4 − MAP_T0) was markedly lower in the labetalol group compared with the lignocaine group, demonstrating superior attenuation of the pressor response.

Table 4: Change in mean arterial pressure and heart rate from baseline to 1 minute after intubation

Variable	Labetalol group (n=40) mean ± SD	Lignocaine group (n=40) mean ± SD	p-value
ΔMAP (T4 − T0) (mmHg)	+9.2 ± 4.6	+19.0 ± 6.3	<0.001
ΔHR (T4 − T0) (beats/min)	+8.3 ± 4.2	+15.4 ± 5.1	<0.001

The rate pressure product (RPP) at 1 minute after intubation was also significantly lower in the labetalol group, suggesting a lower myocardial oxygen demand compared with the lignocaine group, consistent with findings from similar comparative trials.

Adverse hemodynamic events

The incidence of adverse hemodynamic events was lower in the labetalol group compared with the lignocaine group. No patient in either group experienced severe hypotension, bronchospasm, or drug-related allergic reactions.

Table 5: Adverse hemodynamic events

Event	Labetalol group (n=40) n (%)	Lignocaine group (n=40) n (%)	p-value
Hypertension (>20% MAP rise)	4 (10.0)	14 (35.0)	0.008
Tachycardia (>20% HR rise)	5 (12.5)	16 (40.0)	0.006
Hypotension (<20% MAP fall)	2 (5.0)	1 (2.5)	0.55
Bradycardia (HR <50/min)	1 (2.5)	0 (0.0)	0.31
Need for rescue drugs	3 (7.5)	7 (17.5)	0.18

labetalol provided better control of the hemodynamic response to laryngoscopy and intubation, with significantly smaller increases in HR, SBP, DBP, MAP, and a lower frequency of hypertensive and tachycardic episodes compared with lignocaine, while

maintaining an acceptable safety profile, in agreement with contemporary clinical evidence.

DISCUSSIONS

The present study demonstrated that intravenous labetalol 0.25 mg/kg was more effective than lignocaine 1 mg/kg in attenuating the hemodynamic responses to laryngoscopy and endotracheal intubation in normotensive adult patients undergoing elective surgery at a tertiary hospital in Barisal. Labetalol significantly limited the rise in mean arterial pressure and heart rate at 1 minute after intubation, with smaller increases in SBP, DBP, and rate pressure product, while maintaining an acceptable safety profile without clinically important hypotension or bradycardia. These findings support the preferential use of labetalol over lignocaine when the primary goal is to blunt the transient but potentially harmful sympathetic surge associated with airway instrumentation in otherwise normotensive patients.

The observed pressor and tachycardic responses in the lignocaine group are consistent with the well-described pathophysiology of laryngoscopy and intubation, where mechanical stimulation of the upper airway provokes reflex sympathetic activation and catecholamine release, leading to transient elevations in HR and BP. In our study, both drugs reduced the magnitude of this response compared with the expected unattenuated rise reported in historical controls, but labetalol produced a significantly smaller increase in MAP and HR than lignocaine. This aligns with the pharmacologic profile of labetalol as a combined α1- and β-adrenergic blocker, which can simultaneously blunt tachycardia and hypertension, whereas lignocaine, lacking direct adrenergic blockade, primarily acts by reducing airway reflexes and has a more modest effect on systemic sympathetic outflow. [8-10]

Our results are in agreement with recent randomized trials directly comparing labetalol and lignocaine for attenuation of intubation-induced hemodynamic responses. A 2024 randomized controlled trial from Jamshoro found that mean SBP and MAP after laryngoscopy were significantly lower in the labetalol group than in the lignocaine group (SBP 122.13 ± 2.67 vs. 127.48 ± 2.54 mmHg; MAP 92.29 ± 4.42 vs. 104.74 ± 3.51 mmHg; both $p < 0.001$), concluding that labetalol provided superior attenuation of the pressor response. Similarly, a study comparing low-dose labetalol with low-dose lignocaine reported that the mean increases in HR and MAP after intubation were significantly greater in the lignocaine group, reinforcing the greater efficacy of labetalol in controlling these variables. Earlier clinical trials that evaluated labetalol, lignocaine and other agents such as esmolol also showed that labetalol produced more pronounced reductions in HR and BP compared with lignocaine, with better attenuation of diastolic pressure and MAP. The magnitude and direction of the differences in MAP and HR between groups in our study are therefore consistent with the existing body of evidence. [9]

In contrast, some studies have suggested that lignocaine is reasonably effective in blunting hemodynamic fluctuations during airway manipulation, particularly at higher doses or when combined with other agents. A randomized trial in 2016 comparing lidocaine, labetalol and a control group in elective non-cardiac surgery reported that both lidocaine and labetalol significantly attenuated increases in SBP, DBP and MAP compared with the control group and concluded that both drugs could provide protection against hemodynamic changes, though HR decreased significantly only in the labetalol group. Another trial in preeclampsia patients showed that adding labetalol to lignocaine produced further attenuation of the hemodynamic response compared with lignocaine alone, suggesting an additive or synergistic effect when adrenergic blockade is combined with local anesthetic mechanisms. These data support the concept that lignocaine does have a role in hemodynamic control, but that its efficacy is often inferior to, or enhanced by, concomitant use of β-blockade.[11]

Our findings differ in part from studies that reported similar hemodynamic profiles for labetalol and lidocaine in settings of controlled hypotension. A randomized clinical trial in tympanoplasty patients found that both labetalol and lidocaine infusions were similarly effective at achieving targeted hypotension, with no significant difference in MAP trends or time to reach target blood pressure, although bleeding tended to be less with labetalol. However, that study focused on sustained intraoperative hypotension rather than the acute response to laryngoscopy and intubation, used different dosing regimens (continuous infusions rather than single pre-intubation bolus), and involved a different surgical population. The discrepancy highlights that the relative performance of these agents can depend on clinical context, dosing strategy, and outcome measures. In the acute, short-lived stress of intubation, the rapid, combined α- and β-blockade of labetalol appears particularly advantageous. [8]

The lower incidence of hypertensive and tachycardic episodes in the labetalol group in our study has important clinical implications. Although our population consisted of normotensive ASA I II patients, a proportion of such patients in South Asian settings may harbor undiagnosed hypertension, coronary artery disease or other cardiovascular risk factors. Even transient surges in BP and HR during laryngoscopy can precipitate myocardial ischemia, arrhythmias, or cerebrovascular events in susceptible individuals. Comparative studies have shown that labetalol more effectively limits rises in diastolic BP and MAP than lignocaine or esmolol, and reduces the risk of exaggerated pressor responses. Our results, showing significantly smaller increases in MAP and HR and

fewer hypertensive and tachycardic events with labetalol, suggest that its routine use in normotensive patients may provide a margin of safety against unrecognized cardiovascular vulnerability, particularly in resource-limited tertiary hospitals where postoperative monitoring and intensive care capacity may be constrained. [12]

Safety is a key concern when administering β-blockers to normotensive patients, as excessive dosing or overly strong adrenergic blockade can lead to hypotension, bradycardia, and reduced organ perfusion. In our series, labetalol at 0.25 mg/kg did not produce clinically significant hypotension or bradycardia, and the incidence of these events was low and comparable to that in the lignocaine group. This observation is consistent with other trials that used similar or slightly higher labetalol doses and reported good hemodynamic tolerance in ASA I II patients. Moreover, studies in neurosurgical and craniotomy patients have suggested that labetalol provides stable intraoperative and postoperative BP control without frequent bradycardia or hypotension, and may reduce coughing and other emergence reactions. Thus, when appropriately dosed and monitored, labetalol appears safe for use even in normotensive adults. [10]

The present study adds to the existing literature by focusing specifically on normotensive adults in a Bangladeshi tertiary care setting and by using a relatively robust sample size with balanced demographic and baseline hemodynamic characteristics. Prior work from South Asian and Middle Eastern centers has demonstrated similar trends favoring labetalol over lignocaine but often included mixed hypertensive and normotensive populations or added other agents such as esmolol or opioids, making it difficult to isolate the comparative effect of these two drugs alone. Our findings, showing a clear and statistically significant advantage of labetalol for controlling MAP and HR with a single pre-intubation dose, provide context-specific evidence that can be directly translated into practice guidelines in similar tertiary hospitals.

Several limitations should be acknowledged. First, the study evaluated hemodynamic responses only up to 5 minutes after intubation and did not assess longer-term postoperative outcomes such as myocardial ischemia, arrhythmias, or neurological events. While transient hemodynamic control is important, demonstration of reduced clinical endpoints would provide stronger evidence for routine labetalol use. Second, the study included only ASA I II normotensive patients undergoing elective non-cardiac surgery; results may not be generalizable to high-risk populations, emergency surgeries or patients with significant cardiac comorbidities. Third, only a single dose of each drug was tested; dose-response relationships and the potential benefits of combining labetalol with lignocaine or other agents were not explored. Some studies suggest that higher labetalol doses or combination regimens may offer even better control but at the cost of more frequent hypotension or bradycardia, and this balance warrants further investigation. [11]

Despite these limitations, the overall pattern of our results is consistent with contemporary evidence: labetalol provides superior attenuation of the pressor and tachycardic responses to laryngoscopy and intubation compared with lignocaine, with an acceptable safety margin in appropriately selected patients. For anesthesiologists practicing in tertiary hospitals similar to Barisal, incorporating low-dose labetalol into pre-intubation protocols for normotensive adults could enhance hemodynamic stability during one of the most stressful phases of anesthesia, potentially reducing peri-intubation cardiovascular risk. Future research should examine the impact of this strategy in higher-risk cohorts, compare different dosing regimens, and evaluate combinations of labetalol with other attenuating agents as well as explore cost-effectiveness and implementation feasibility in resource-limited environments.

CONCLUSIONS

In this randomized, double-blind clinical trial, intravenous labetalol 0.25 mg/kg proved more effective than lignocaine 1 mg/kg in attenuating the hemodynamic responses to laryngoscopy and endotracheal intubation in normotensive adult patients undergoing elective surgery at a tertiary hospital in Barisal. Labetalol significantly reduced the rise in mean arterial pressure, systolic and diastolic blood pressure, and heart rate at 1 minute after intubation, with fewer episodes of peri-intubation hypertension and tachycardia, while maintaining hemodynamic stability without a clinically important increase in hypotension or bradycardia. These findings are concordant with contemporary studies that have shown superior attenuation of the pressor response with labetalol compared with lignocaine during airway instrumentation.

Given its combined α1- and β-adrenergic blocking properties, labetalol appears to offer a more comprehensive blunt of the sympathoadrenal surge than lignocaine, which primarily acts by raising the threshold for airway stimulation. In the context of a high-volume tertiary hospital in Bangladesh, where undiagnosed cardiovascular risk is common and resources for managing peri-intubation complications may be limited, routine use of low-dose labetalol as a pre-intubation agent in normotensive ASA I II patients may improve hemodynamic control and provide an additional safety margin. Future studies should evaluate different dosing regimens, higher-risk populations, and combinations with other attenuating agents but based on current evidence, labetalol can be recommended as a preferred option over lignocaine for mitigating hemodynamic responses to laryngoscopy and intubation in similar clinical settings.

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