RESEARCH PAPER

Effect of Intravenous Lignocaine on Hemodynamic Response to Intubation: A Randomized Controlled Study

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ABSTRACT

Background:

Laryngoscopy and endotracheal intubation frequently provoke transient tachycardia and hypertension, which may harm patients with limited cardiovascular reserve. Intravenous lignocaine has been investigated for blunting this response, but evidence remains inconsistent due to methodological limitations.

Aim:

To evaluate whether intravenous lignocaine (1.5 mg/kg) administered 90 seconds before laryngoscopy significantly attenuates the hemodynamic response to endotracheal intubation compared to placebo.

Methods:

This double-blind, randomized study enrolled 74 adults (ASA I–II, aged 18–60 years) undergoing elective non-cardiac surgery. Patients received either lignocaine 1.5 mg/kg or normal saline 90 seconds before laryngoscopy under a standardized anaesthetic protocol (fentanyl 2 µg/kg, propofol 2 mg/kg, succinylcholine 1.5 mg/kg). Heart rate, systolic, diastolic, and mean arterial pressure were recorded at baseline, after study drug, immediately post-intubation, and at 1, 3, and 5 minutes.

Results:

Seventy-four patients completed the study (37 per group) with comparable baseline characteristics. Following intubation, the lignocaine group demonstrated significantly lower heart rate, systolic, diastolic, and mean arterial pressure than placebo at all post-intubation time points ($p < 0.001$ for immediate to 3 minutes; $p \leq 0.007$ for 5 minutes). At peak response (immediately post-intubation), lignocaine reduced systolic blood pressure by 13.4 mmHg and heart rate by 10.8 bpm. Hemodynamic values returned closer to baseline more rapidly in the lignocaine group. Oxygen saturation remained stable in both groups, and no adverse events were observed.

Conclusion:

Intravenous lignocaine (1.5 mg/kg) given 90 seconds before laryngoscopy safely and significantly attenuates the hemodynamic response to intubation, providing enhanced cardiovascular stability during general anaesthesia.

Key words: Lignocaine; lidocaine; hemodynamic response; laryngoscopy; endotracheal intubation; randomized controlled trial

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INTRODUCTION

Laryngoscopy and endotracheal intubation are essential manoeuvres in general anaesthesia, but they frequently provoke a transient sympathetic stress response characterised by tachycardia and hypertension [1]. This response results from direct mechanical stimulation of the supraglottic and laryngeal tissues, leading to catecholamine release [2].. While often well tolerated in healthy individuals, such hemodynamic surges can precipitate myocardial ischemia, acute heart failure, or intracranial haemorrhage in patients with limited cardiovascular or cerebrovascular reserve [3,4].

Numerous pharmacological interventions have been investigated to blunt this response, including opioids, beta blockers, calcium channel blockers, and local anaesthetics [5]. Among these, intravenous (IV) lignocaine (lidocaine) has attracted considerable interest due to its low cost, wide availability, and favourable safety profile [6]. However, the evidence base is not without controversy. A systematic review concluded that IV lignocaine reduces cardiovascular responses to intubation but explicitly called for further studies on optimal dosage and timing.

A meta-analysis by Qin et al. [8] reported mean reductions in MAP of 3.85 mmHg and HR of 4.72 bpm, yet noted that lignocaine's effectiveness may vary by dosage and patient ethnicity. Additional comparative studies have yielded mixed results and found that low-dose ketamine and lidocaine provided comparable hemodynamic attenuation [9]. A trial reported that the intravenous route was superior to nebulised lidocaine [10]. The Ghanaian trial compared esmolol and lidocaine, reporting different side effect profiles [11]. In hypertensive patients, combining lignocaine with magnesium sulphate offered superior blunting compared to lignocaine alone [12]. A dose-finding study in elderly females suggested that higher doses (2 mg/kg) provided better attenuation but raised toxicity concerns [13].

Despite decades of studies, a significant gap persists: most prior studies suffer from methodological limitations such as lack of blinding, variable co-induction agents, inconsistent timing of lignocaine administration, and inadequate sample sizes [7,8,14]. Few double-blind, placebo-controlled trials have tested a standardised dose (e.g., 1.5 mg/kg) given at a precise interval (e.g., 90 seconds before laryngoscopy) under a fully uniform anaesthetic protocol. Therefore, we conducted this double blind, randomised, placebo-controlled trial to evaluate whether IV lignocaine (1.5 mg/kg) administered 90 seconds before laryngoscopy significantly attenuates the hemodynamic response to endotracheal intubation while maintaining safety.

MATERIALS AND METHODS

This prospective, randomized, double-blind controlled study was conducted in the Department of Operation Theatre, NIMS Hospital, NIMS University, Jaipur, after obtaining written informed consent.

A total 74 ASA I–II patients between the ages of 18 and 60 who were having elective non-cardiac surgery were included. Individuals with pregnancy, hepatic/renal dysfunction, uncontrolled hypertension, cardiovascular illness, anticipated difficult airway, or drug allergies were not included.

By using a random sample method, patients were split into two groups of 37 each. Group A received intravenous lignocaine (1.5 mg/kg diluted to 10 mL)., while Group B received 10 mL of regular saline. The investigator and the patient were blinded.

Baseline values were noted upon IV access and routine monitoring. Fentanyl (2 µg/kg) and propofol (2 mg/kg) were used to induce Anesthesia before the study medication was administered. Using a Macintosh blade, intubation was completed in 15 seconds with the help of succinylcholine (1.5 mg/kg).

At baseline, following medication administration, right after intubation, and one, three, and five minutes after intubation, hemodynamic parameters (HR, SBP, DBP, MAP, and SpO_2) were measured.

Change in MAP was the main endpoint; HR, SBP, DBP, incidence of SBP rise >30%, and adverse events were the secondary outcomes.

Data was analyzed using SPSS version 26.0. Independent t-test, chi-square test, and repeated-measures ANOVA were applied. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 74 patients (37 in Group A [lignocaine], 37 in Group B [control]) completed the study without any dropouts or protocol violations. Baseline demographic and physiological characteristics were similar between groups, confirming successful randomisation.

Table 1: Comparison of changes in Heart rate at different time-intervals

Time	Group A (Lignocaine)	Group B (Control)	P-value *

Time	Group A (Lignocaine)	Group B (Control)	P-value *
T_0 (baseline)	77.6 ± 6.1	78.2 ± 6.4	0.67
T_1 (after study drug)	75.9 ± 5.8	77.5 ± 6.1	0.21
T_2 (immediate post-intubation)	90.8 ± 7.0	101.6 ± 8.3	< 0.001
T_3 (1 min post-intubation)	86.5 ± 6.4	96.2 ± 7.5	< 0.001
T_4 (3 min post-intubation)	82.3 ± 5.9	89.8 ± 6.7	< 0.001
T_5 (5 min post-intubation)	79.4 ± 5.6	84.6 ± 6.2	0.002

Table 1: Laryngoscopy and intubation caused a sharp rise in heart rate in both groups, peaking at T_2. However, the lignocaine group had consistently lower heart rates at all post-intubation time points, with the difference reaching statistical significance from T_2 to T_5 ($p \leq 0.002$). The attenuation (≈11 bpm lower at T_2) is clinically meaningful and confirms lignocaine's moderating effect on tachycardia**.**

Table 2: Comparison of changes in Systolic Blood Pressure at different time-intervals

Time	Group A (Lignocaine)	Group B (Control)	p-value*
T_0	121.8 ± 7.5	122.6 ± 7.9	0.64
T_1	119.6 ± 7.2	121.9 ± 7.6	0.18
T_2	134.2 ± 8.8	147.6 ± 10.5	< 0.001
T_3	130.5 ± 8.3	142.3 ± 9.4	< 0.001
T_4	126.4 ± 7.7	134.8 ± 8.6	< 0.001
T_5	123.3 ± 7.2	128.7 ± 7.8	0.005

Table 2: Both groups experienced a rise in systolic blood pressure following intubation, but the lignocaine group had significantly lower values at T_2 through T_5. The peak difference of 13.4 mmHg at T_2 (147.6 vs 134.2) is both statistically and clinically significant, indicating that lignocaine blunts the hypertensive response to intubation**.**

Table 3: Comparison of changes in Diastolic Blood Pressure at different time-intervals

Time	Group A (Lignocaine)	Group B (Control)	p-value*
T_0	78.2 ± 5.2	78.9 ± 5.4	0.59
T_1	76.7 ± 5.0	78.3 ± 5.2	0.16
T_2	86.1 ± 6.1	94.7 ± 6.9	< 0.001
T_3	83.6 ± 5.7	91.2 ± 6.3	< 0.001
T_4	80.9 ± 5.3	87.4 ± 5.9	< 0.001
T_5	78.8 ± 5.0	82.9 ± 5.6	0.007

Table 3: Diastolic blood pressure followed a similar pattern to systolic pressure. The lignocaine group had an 8.6 mmHg lower DBP at T_2 compared to the

control ($p < 0.001$). The attenuation persisted at T_5 ($p = 0.007$). This reinforces that lignocaine mitigates the overall pressor response, not just systolic hypertension.

Table 4: Comparison of changes in Oxygen Saturation (SpO_2, %) Oxygen Saturation (SpO_2, %) at different time-intervals

Time	Group A (Lignocaine)	Group B (Control)	p-value*
T_0	99.1 ± 0.6	99.0 ± 0.5	0.48
T_1	99.2 ± 0.5	99.1 ± 0.6	0.52
T_2	98.9 ± 0.7	98.8 ± 0.8	0.61
T_3	99.0 ± 0.6	98.9 ± 0.7	0.57
T_4	99.1 ± 0.5	99.0 ± 0.6	0.49
T_5	99.2 ± 0.5	99.1 ± 0.5	0.55

Table 4: There were no significant differences in SpO_2 between groups at any time point, and all values remained within the normal range (≥98.8%). This confirms that lignocaine administration did not compromise respiratory function or oxygenation, establishing its safety profile in this setting.

DISCUSSION

This double-blind, randomized, placebo-controlled trial showed that intravenous lignocaine (1.5 mg/kg) given 90 seconds before laryngoscopy significantly attenuates the hemodynamic response to endotracheal intubation. The effect was greatest immediately post-intubation (T_2) and persisted for 5 minutes without compromising oxygenation. These findings support lignocaine as a safe, effective adjunct for blunting the pressor response.

The temporal pattern of hemodynamic changes in our study followed a predictable course. Both groups exhibited a sharp rise in HR, SBP, DBP, and MAP at T_2, but the lignocaine group had consistently lower values across all post-intubation time points (Tables 1–3). At the peak response (T_2), lignocaine reduced SBP by 13.4 mmHg ($p<0.001$) and HR by 10.8 bpm ($p<0.001$). By T_5, significant differences remained (SBP: 123.3 vs. 128.7 mmHg, $p=0.005$; HR: 79.4 vs. 84.6 bpm, $p=0.002$), indicating sustained attenuation. Oxygen saturation remained stable (Table 5), confirming safety.

Our results align with existing literature. The meta-analysis by Qi et al. (2013) reported that IV lignocaine reduces SBP by ~4–5 mmHg and HR by ~4–5 bpm [9]. The greater attenuation in our study may reflect precise timing (90 seconds before laryngoscopy) and a standardised anaesthetic protocol. A more recent meta-analysis by Qin et al. (2025) confirmed reductions in MAP (MD −3.85 mmHg) and HR (MD −4.72 bpm) [10]. Comparative studies show lignocaine is comparable to low-dose ketamine (Nazemroaya et al., 2023) [8] and superior to nebulised lidocaine (Laurito et al., 1991) [4], while esmolol has a different side-effect profile (Saroa et al., 2015) [7]. In hypertensive patients, combining lignocaine with magnesium sulphate provided superior attenuation (West African Journal of Medicine, 2023) [14], suggesting monotherapy may be insufficient in some subgroups. A 2025 dose-finding study in elderly females recommended 2 mg/kg, with caution for toxicity [2]. Gulabani et al. (2015) found dexmedetomidine more effective than lignocaine at 1 μg/kg [6].

Mechanisms underlying lignocaine's attenuation are multifactorial. First, it exerts a central depressant effect on the brainstem, reducing sensitivity of medullary cardiovascular and cough centres to afferent stimuli from supraglottic and laryngeal regions [11]. Second, it blocks voltage-gated sodium channels, decreasing action potential propagation along glossopharyngeal and superior laryngeal nerves; this membrane-stabilising effect is well established [9,13]. Third, it may reduce reflex catecholamine release from the adrenal medulla and sympathetic nerve endings, although evidence is limited [13]. Fourth, a direct myocardial depressant effect—reducing contractility and heart rate—may contribute [9]. Timing is critical: the 90-second pre-intubation interval corresponds to peak plasma concentration of IV lignocaine (~1–2 minutes), ensuring optimal effect during laryngoscopy [9,10]. Collectively, these actions explain why lignocaine attenuates—without abolishing—the tachycardia and hypertension induced by airway manipulation.

Clinical significance extends beyond statistics. A 13-mmHg reduction in SBP and a 10 bpm reduction in HR correspond to a rate-pressure product (RPP) decrease from ~15,000 to 12,180. The ischemic threshold in many patients with coronary artery disease is ~12,000–13,000 (Singh et al., 1994) [12], suggesting potential protection against myocardial

ischemia. In patients with intracranial pathology, blunting the hypertensive surge may reduce the risk of aneurysm rupture or worsening cerebral oedema (Shribman et al., 1990) [11]. The absence of desaturation or adverse events (Table 5) supports safety. Given its low cost and wide availability, lignocaine is an attractive first-line option, particularly in resource-limited settings (Qin et al., 2025) [10].

Limitations include: (1) enrolment of only ASA I–II patients, limiting generalizability to high-risk populations; (2) lack of plasma catecholamine measurement; (3) short follow-up (5 minutes); (4) use of succinylcholine (results may differ with non-depolarizing agents); and (5) co-administration of fentanyl, which itself attenuates the stress response, potentially masking some of lignocaine's independent effect.

CONCLUSION

In this double-blind, randomized, placebo-controlled trial, IV lignocaine (1.5 mg/kg) given 90 seconds before laryngoscopy significantly attenuated the hemodynamic response to endotracheal intubation without compromising oxygenation. The effect was sustained for at least 5 minutes. Lignocaine is a safe, cost-effective, and evidence-based strategy to enhance hemodynamic stability during general anaesthesia, particularly in patients at risk for complications from acute hypertensive or tachycardic responses.

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