Journal of Advanced Medical and Dental Sciences Research

@Society of Scientific Research and Studies

Journal home page:www.jamdsr.com doi:10.21276/jamdsr Index Copernicus value [ICV] =82.06

(e) ISSN Online: 2321-9599; (p) ISSN Print: 2348-6805

Original Research

IV versus topical lignocaine for attenuation of hemodynamic response during ocular surgery: A comparative study

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ABSTRACT:
Background: Laryngoscopy and tracheal intubation during general anesthesia evoke a sympathetic stress response characterized by tachycardia and hypertension. In ocular surgery, such transient hemodynamic surges are undesirable because they may compromise cardiovascular stability and adversely affect operative conditions. Lignocaine is commonly used to blunt these responses, but the comparative effectiveness of intravenous versus topical administration in ocular surgery remains clinically relevant. **Aim:** To compare intravenous (IV) lignocaine with topical lignocaine for attenuation of hemodynamic responses during laryngoscopy and tracheal intubation in patients undergoing ocular surgery under general anesthesia. **Material and Methods:** This comparative, parallel-group study was conducted at a tertiary care hospital and included 84 adult patients (ASA I–II) scheduled for elective ocular surgery under general anesthesia. Patients were allocated into two equal groups: Group IV received IV lignocaine 1.5 mg/kg administered 90 seconds before laryngoscopy, while Group TOP received topical lignocaine spray to the oropharynx and supraglottic area prior to laryngoscopy. Heart rate (HR) and mean arterial pressure (MAP) were recorded at baseline, post-induction, at laryngoscopy, immediately after intubation, and at 1, 3, 5, and 10 minutes post-intubation. Clinically significant responses were defined as a ≥20% rise in HR and/or MAP from baseline. Adverse events including bradycardia, hypotension, coughing, and laryngospasm were documented. **Results:** Baseline demographic variables were comparable between groups. HR and MAP responses were significantly lower in Group IV at laryngoscopy, immediately after intubation, and during the early post-intubation period, indicating superior attenuation of sympathetic response. Clinically significant HR and MAP rises occurred less frequently in Group IV than Group TOP. Coughing during intubation was also reduced in the IV group, while other adverse events were infrequent and comparable between groups. **Conclusion:** Intravenous lignocaine provided better attenuation of hemodynamic responses to laryngoscopy and intubation than topical lignocaine in ocular surgery, with a favorable safety profile and improved airway tolerance.

Keywords: intravenous lignocaine; topical lignocaine; hemodynamic response; laryngoscopy; ocular surgery

Received: 14 April, 2018 Accepted: 19 May, 2018 Published: 25 May, 2018

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This article may be cited as: Choudhary R, Kumar V. IV versus topical lignocaine for attenuation of hemodynamic response during ocular surgery: A comparative study. J Adv Med Dent Scie Res 2018;6(5):212-218.

INTRODUCTION

Ocular surgery is frequently performed in patients at the extremes of age and in those with co-existing systemic disease, where even brief cardiovascular instability may be undesirable. A major peri-operative challenge during general anesthesia for eye surgery is the sympathetic “stress response” triggered by airway manipulation—particularly direct laryngoscopy and endotracheal intubation—which may produce abrupt tachycardia and hypertension. These short-lived surges can increase myocardial oxygen demand, precipitate dysrhythmias in susceptible patients, and may be especially relevant in ophthalmic procedures where a calm surgical field, stable perfusion, and avoidance of sudden pressure fluctuations are preferred. In addition, the anesthetic plan for ocular surgery must anticipate not only sympathetic stimulation from intubation but also reflex responses related to ocular handling, such as the oculocardiac reflex (OCR), which can cause clinically important

bradycardia or rhythm disturbances during extra-ocular muscle traction or orbital stimulation. The OCR is a well-recognized trigemino-vagal reflex and remains a relevant concern in strabismus and other orbital surgeries, reinforcing the overall need for vigilant hemodynamic control during ophthalmic anesthesia.[1]Hemodynamic changes at induction are clinically important in eye surgery because airway instrumentation can influence intraocular physiology as well as systemic cardiovascular variables. Although small intraocular pressure (IOP) changes may be tolerated in healthy eyes, acute elevations can be harmful in patients with fragile ocular structures, compromised optic nerve perfusion, glaucoma, penetrating eye injury, or during procedures where vitreous or choroidal congestion is undesirable. Intubation is known to cause a greater ocular stress response than less stimulating airway techniques. In adults undergoing general anesthesia, endotracheal tube (ETT) placement has been associated with a statistically significant increase in IOP compared with laryngeal mask airway (LMA), supporting the clinical observation that airway stimulation contributes to ocular pressure fluctuations.[2] Because many ocular operations still require a secured airway with controlled ventilation, strategies that blunt the intubation response without prolonging recovery or increasing adverse events remain clinically relevant.Among the pharmacologic options used to attenuate the intubation response, lignocaine (lidocaine) is attractive because it is inexpensive, familiar, and has multiple potentially beneficial mechanisms: suppression of airway reflex afferents, reduction of cough, mild sympatholytic effects, and antiarrhythmic membrane-stabilizing properties. Lidocaine can be delivered systemically (intravenous bolus with or without infusion) or applied locally to the airway (topical spray, atomization, or laryngotracheal application). Intravenous lidocaine has been widely adopted in anesthesia practice because of its rapid onset and predictable dosing. Beyond elective operating room use, safety concerns about IV lidocaine pretreatment have also been examined in acute care intubations; in a cohort of severe traumatic brain injury patients undergoing rapid sequence intubation, IV lidocaine pretreatment was not associated with significant adverse post-intubation hemodynamic changes, lending support to its cardiovascular tolerability when appropriately dosed and monitored.[3] Although this emergency-care population differs from elective ophthalmic surgery, the findings add confidence that IV lidocaine does not inherently destabilize hemodynamics in many clinical settings.Topical (local) airway anesthesia with lidocaine offers an alternative approach aimed at blocking afferent stimulation at the mucosal level. Topicalization can be performed in different ways, and effectiveness may depend on distribution to the laryngeal inlet and trachea, timing relative to laryngoscopy, and the anesthetic regimen used for induction. In hypertensive surgical patients, local airway anesthesia using topical lidocaine has been shown to attenuate hemodynamic responses around intubation and extubation compared with saline control, supporting its role in reducing airway-related sympathetic surges in higher-risk populations.[4] Such findings are relevant to ophthalmic practice because a substantial proportion of eye-surgery patients are elderly and may have hypertension, diabetes, or other systemic comorbidities. However, topical techniques can be variably applied in routine practice and may not uniformly anesthetize the critical regions responsible for the greatest reflex stimulation.More focused peri-intubation evidence also supports topical lidocaine when applied strategically to key airway structures. In a prospective, randomized study where 10% lidocaine was sprayed on the laryngoscope blade and/or directly to the trachea during propofol–remifentanil induction, post-intubation MAP and HR changes were significantly reduced versus no-lidocaine control, suggesting that targeted topical delivery can meaningfully blunt the cardiovascular response.[5] These results highlight an important practical point: “topical lidocaine” is not a single, uniform intervention, and the site of application (oropharynx vs laryngeal inlet vs trachea) likely influences clinical effectiveness. In many tertiary care settings, however, topical spray to the oropharynx and supraglottic area is a commonly used approach because it is quick and requires no specialized devices. Whether this pragmatic topical strategy performs as well as a standard IV bolus regimen in attenuating the peri-intubation response during ocular surgery remains clinically important.Systemic administration of lidocaine as a bolus with continued infusion has also been investigated for broader peri-operative advantages, including stable hemodynamics and improved postoperative comfort. In an Indian Journal of Anaesthesia clinical investigation, patients receiving an IV lignocaine bolus followed by infusion showed a significantly smaller rise in pulse rate and mean arterial pressure during intubation and extubation compared with saline, suggesting that systemic lidocaine can blunt airway-related hemodynamic fluctuations across multiple peri-operative phases.[6]

MATERIAL AND METHODS

This comparative, parallel-group clinical study was conducted in a tertiary care hospital to evaluate and compare the efficacy of intravenous (IV) lignocaine versus topical lignocaine in attenuating the hemodynamic response associated with ocular surgery performed under general anesthesia. The study was planned to ensure uniform anesthetic technique, standardized stimulation points, and objective measurement of cardiovascular responses at predefined time intervals.A total of 84 adult patients scheduled for elective ocular surgery and requiring general anesthesia were enrolled. Patients were

allocated into two equal groups (n = 42 each): Group IV (intravenous lignocaine) and Group TOP (topical lignocaine). All participants underwent a pre-anesthetic evaluation, and baseline demographic and clinical characteristics were recorded to assess group comparability.

Eligibility criteria

Patients of either sex, belonging to ASA physical status I–II, and planned for elective ocular surgery under general anesthesia were included. Patients were excluded if they had known hypersensitivity to lignocaine or amide local anesthetics, significant cardiovascular disease (e.g., uncontrolled hypertension, ischemic heart disease, arrhythmias), conduction abnormalities, severe hepatic or renal impairment, pregnancy or lactation, anticipated difficult airway, use of drugs affecting autonomic responses (e.g., beta-blockers, clonidine, antiarrhythmics) when not clinically avoidable, or if intraoperative events necessitated deviation from the standardized protocol.

Methodology

Patients were assigned to one of two intervention arms. In Group IV, lignocaine was administered intravenously as a bolus dose of 1.5 mg/kg given 90 seconds before laryngoscopy and tracheal intubation. In Group TOP, topical lignocaine was used to anesthetize the upper airway: 10% lignocaine spray was applied to the oropharynx and supraglottic area as per institutional practice, targeting adequate mucosal anesthesia prior to laryngoscopy while ensuring safe maximum dose limits. All lignocaine dosing (total administered dose from all sources) was kept within recommended safety margins.

Standardization of anesthesia technique: All patients were kept fasting as per institutional protocol and received standard preoperative preparation. In the operating room, routine monitoring was applied, and baseline hemodynamic values were documented before induction. General anesthesia was induced using a standardized regimen (hypnotic induction agent and opioid as per protocol), followed by a neuromuscular blocker to facilitate intubation. Laryngoscopy and endotracheal intubation were performed by an experienced anesthesiologist to minimize variability; cases requiring prolonged or multiple attempts at intubation were excluded from analysis to avoid confounding hemodynamic surges. Anesthesia was maintained with inhalational agent/oxygen–air mixture with controlled ventilation, and intraoperative analgesia and muscle relaxation were provided according to a uniform plan. Fluid therapy and ventilatory parameters were standardized, and surgical stimulation points were noted.

Hemodynamic monitoring and time points of assessment: Hemodynamic variables were recorded at predefined intervals to capture responses to airway manipulation and early surgical stimulation. Measurements were taken at baseline (pre-induction), post-induction (before laryngoscopy), at laryngoscopy, immediately after intubation (0 min), and at 1, 3, 5, and 10 minutes after intubation. Additional recordings were obtained at placement of ocular speculum and at initial surgical incision where applicable, as these steps may provoke sympathetic stimulation. Hemodynamic monitoring included heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP), recorded using a non-invasive blood pressure monitor at consistent intervals; continuous ECG was used for rhythm surveillance.

Outcome measures: The primary outcome was attenuation of the pressor and tachycardic response to airway manipulation, assessed by changes in HR and MAP from baseline at the specified post-intubation time points. Secondary outcomes included changes in SBP and DBP, the rate-pressure product (RPP = HR × SBP) as an index of myocardial oxygen demand, and the proportion of patients exhibiting clinically significant hemodynamic responses, defined operationally as ≥20% increase in HR or MAP from baseline. Safety outcomes included the incidence of bradycardia, hypotension, arrhythmias, airway-related adverse events (coughing, laryngospasm), and any suspected lignocaine toxicity symptoms.

Statistical analysis

Data were entered and analyzed using standard statistical software. Continuous variables were expressed as mean ± standard deviation (or median with interquartile range when non-normally distributed), and categorical variables as frequencies and percentages. Baseline comparability between groups was assessed using independent t-test/Mann–Whitney U test for continuous variables and Chi-square/Fisher's exact test for categorical variables. Hemodynamic changes over time were compared between groups using repeated-measures analysis (repeated-measures ANOVA or mixed-effects model as appropriate), with post-hoc testing for inter-group differences at specific time points. A p-value < 0.05 was considered statistically significant.

RESULTS

Table 1: Demographic characteristics and baseline clinical variables

The demographic profile and baseline clinical parameters of patients in both groups were comparable. The mean age of patients in Group IV was 46.82 ± 9.34 years, while that in Group TOP was 47.26 ± 8.91 years, with no statistically significant difference (p = 0.82). Gender distribution was also similar between the two groups, with males constituting 57.14% in Group IV and 52.38% in Group TOP (p = 0.66). Mean body weight did not

differ significantly between the groups (64.35 ± 7.88 kg vs. 63.92 ± 8.11 kg; p = 0.79). The distribution of ASA physical status I and II patients was comparable, indicating similar baseline health status. Additionally, the duration of laryngoscopy was nearly identical between groups (p = 0.64), suggesting uniform airway manipulation.

Table 2: Heart rate changes at different time intervals

Baseline heart rate and post-induction heart rate were comparable between Group IV and Group TOP, with no statistically significant differences (p > 0.05). However, a significant divergence was observed at the time of laryngoscopy and tracheal intubation. Group TOP exhibited a marked increase in heart rate at laryngoscopy (89.64 ± 8.21 beats/min) compared to Group IV (81.36 ± 7.42 beats/min), which was statistically highly significant (p < 0.001). This trend persisted immediately after intubation and at 1, 3, and 5 minutes post-intubation, with Group TOP consistently demonstrating higher heart rates than Group IV (p < 0.01 at all these time points). By 10 minutes after intubation, heart rates in both groups returned close to baseline levels, and the difference was no longer statistically significant (p = 0.24).

Table 3: Mean arterial pressure changes at various time intervals

Mean arterial pressure values at baseline and post-induction were comparable between the two groups (p > 0.05). At laryngoscopy, Group TOP showed a significantly higher rise in MAP (106.26 ± 8.11 mmHg) compared to Group IV (96.84 ± 7.42 mmHg), with a highly significant p value (<0.001). This exaggerated pressor response in Group TOP persisted immediately after intubation and at 1 and 3 minutes post-intubation, with statistically significant differences favoring Group IV (p < 0.001). Although MAP values remained higher in Group TOP at 5 minutes post-intubation, the magnitude of difference was smaller but still statistically significant (p = 0.01). At 10 minutes, MAP values in both groups were comparable (p = 0.38).

Table 4: Incidence of clinically significant hemodynamic response (≥20% rise from baseline)

A significantly greater proportion of patients in Group TOP experienced clinically significant hemodynamic responses. An increase in heart rate of ≥20% from baseline was observed in 42.86% of patients in Group TOP compared to only 14.29% in Group IV (p = 0.004). Similarly, a ≥20% rise in MAP occurred in 38.10% of patients in Group TOP versus 11.90% in Group IV (p = 0.006). The combined occurrence of both heart rate and MAP increases ≥20% was also significantly higher in Group TOP (33.33%) compared to Group IV (9.52%), with a p value of 0.01.

Table 5: Adverse events and safety profile

The incidence of adverse events was low in both groups. Bradycardia occurred in 7.14% of patients in Group IV and 2.38% in Group TOP, while hypotension was observed in 9.52% and 4.76% of patients, respectively; however, these differences were not statistically significant (p > 0.05). Coughing during intubation was significantly more frequent in Group TOP (21.43%) compared to Group IV (4.76%), with a statistically significant difference (p = 0.03). Laryngospasm was observed only in Group TOP (4.76%), though this difference did not reach statistical significance (p = 0.15). No serious arrhythmias or signs of lignocaine toxicity were noted in either group, indicating that both techniques were generally safe, with intravenous lignocaine offering better airway tolerance.

Table 1. Demographic characteristics and baseline clinical variables

Variable	Group IV (n = 42)	Group TOP (n = 42)	p value
Age (years), mean ± SD	46.82 ± 9.34	47.26 ± 8.91	0.82
Sex (Male/Female), n (%)	24 (57.14%) / 18 (42.86%)	22 (52.38%) / 20 (47.62%)	0.66
Weight (kg), mean ± SD	64.35 ± 7.88	63.92 ± 8.11	0.79
ASA I, n (%)	26 (61.90%)	25 (59.52%)	0.82
ASA II, n (%)	16 (38.10%)	17 (40.48%)	0.82
Duration of laryngoscopy (sec), mean ± SD	14.26 ± 2.31	14.51 ± 2.48	0.64

Table 2. Heart rate (beats/min) changes at different time intervals

Time point	Group IV (mean ± SD)	Group TOP (mean ± SD)	p value
Baseline	76.42 ± 6.85	75.96 ± 7.12	0.74
Post-induction	72.18 ± 6.34	73.05 ± 6.81	0.52
At laryngoscopy	81.36 ± 7.42	89.64 ± 8.21	**<0.001**
Immediately after intubation	84.12 ± 7.96	94.38 ± 8.64	**<0.001**
1 min	82.04 ± 7.18	91.52 ± 8.06	**<0.001**
3 min	79.26 ± 6.94	87.18 ± 7.62	**<0.001**
5 min	77.14 ± 6.52	82.36 ± 7.08	**0.002**

10 min	75.98 ± 6.21	77.62 ± 6.48	0.24

Table 3. Mean arterial pressure (MAP, mmHg) at various time intervals

Time point	Group IV (mean ± SD)	Group TOP (mean ± SD)	p value
Baseline	92.36 ± 6.74	91.88 ± 7.02	0.76
Post-induction	86.42 ± 6.18	87.04 ± 6.36	0.64
At laryngoscopy	96.84 ± 7.42	106.26 ± 8.11	**<0.001**
Immediately after intubation	99.12 ± 7.96	109.48 ± 8.52	**<0.001**
1 min	97.04 ± 7.18	106.36 ± 7.94	**<0.001**
3 min	94.18 ± 6.82	101.92 ± 7.36	**<0.001**
5 min	92.44 ± 6.36	96.18 ± 6.88	**0.01**
10 min	90.96 ± 6.12	92.14 ± 6.28	0.38

Table 4. Incidence of clinically significant hemodynamic response (≥20% rise from baseline)

Parameter	Group IV (n = 42)	Group TOP (n = 42)	p value
HR increase ≥20%, n (%)	6 (14.29%)	18 (42.86%)	**0.004**
MAP increase ≥20%, n (%)	5 (11.90%)	16 (38.10%)	**0.006**
Both HR and MAP ≥20%, n (%)	4 (9.52%)	14 (33.33%)	**0.01**

Table 5. Adverse events and safety profile

Adverse event	Group IV (n = 42)	Group TOP (n = 42)	p value
Bradycardia, n (%)	3 (7.14%)	1 (2.38%)	0.30
Hypotension, n (%)	4 (9.52%)	2 (4.76%)	0.40
Coughing during intubation, n (%)	2 (4.76%)	9 (21.43%)	**0.03**
Laryngospasm, n (%)	0 (0.00%)	2 (4.76%)	0.15

DISCUSSION

The two study groups in the present work were well matched, which strengthens the validity of inter-group comparisons. Age (46.82 ± 9.34 vs 47.26 ± 8.91 years), sex distribution (57.14% vs 52.38% males), weight (64.35 ± 7.88 vs 63.92 ± 8.11 kg), ASA grading (ASA I: 61.90% vs 59.52%), and laryngoscopy duration (14.26 ± 2.31 vs 14.51 ± 2.48 s) did not differ significantly (all $p>0.05$). Similar baseline comparability has been emphasized in controlled intubation-response trials to ensure that observed hemodynamic differences reflect the intervention rather than demographic imbalance, as demonstrated by Valeshabad et al. (2014), where age, sex, weight and ASA status were comparable between study arms before evaluating post-laryngoscopy cardiovascular responses.[7]A key finding of this study was superior attenuation of tachycardic response with IV lignocaine at the major stimulation points. Although baseline HR and post-induction HR were similar, Group IV showed significantly lower HR at laryngoscopy (81.36 ± 7.42 vs 89.64 ± 8.21 beats/min) and immediately after intubation (84.12 ± 7.96 vs 94.38 ± 8.64 beats/min), with persistence at 1, 3 and 5 minutes (all $p\leq0.002$), before convergence by 10 minutes ($p=0.24$). This pattern—largest separation at airway manipulation with gradual normalization—mirrors the physiology reported by Sklar et al. (1992), where placebo inhalation produced a larger HR rise (≈15.6 bpm), while adequate airway lidocaine exposure reduced the HR increase markedly (e.g., ≈3.1 bpm with higher-dose inhaled lidocaine), supporting the concept that better suppression of airway reflex input translates into smaller sympathetic surges.[8]The pressor response followed the same direction as HR in the present study, with IV lignocaine producing a clinically and statistically meaningful reduction in MAP rise. Group TOP had higher MAP at laryngoscopy (106.26 ± 8.11 vs 96.84 ± 7.42 mmHg) and immediately after intubation (109.48 ± 8.52 vs 99.12 ± 7.96 mmHg), and the difference remained significant through 5 minutes ($p=0.01$) before becoming non-significant at 10 minutes ($p=0.38$). Timing of systemic lignocaine is known to be critical for suppressing the neurohumoral component of the response; Wilson et al. (1991) showed that varying the interval between IV lignocaine and intubation influenced sympathoadrenal responses, reinforcing why, in the present protocol, giving IV lignocaine shortly before laryngoscopy plausibly contributed to the lower early MAP and HR peaks observed.[9]Beyond mean values, the present study demonstrated that IV lignocaine reduced the *proportion* of patients developing clinically significant surges (≥20% rise from baseline). HR ≥20% occurred in 14.29% in Group IV versus 42.86% in Group TOP ($p=0.004$), MAP ≥20% occurred in 11.90% versus 38.10% ($p=0.006$), and both HR+MAP ≥20% occurred in 9.52% versus 33.33% ($p=0.01$). These categorical outcomes align with the way earlier trials have reported responder rates; for instance, Kindler et al. (1996) highlighted clinically relevant thresholds (e.g., proportion with maximum HR exceeding 90/min), showing higher "excess response" rates in controls than in active-drug arms, supporting

the usefulness of responder analyses in addition to comparing mean hemodynamic values.[10]

Airway tolerance outcomes in this study also favored IV lignocaine. Coughing during intubation was significantly lower in Group IV (4.76%) compared with Group TOP (21.43%; p=0.03), while laryngospasm occurred only in Group TOP (4.76%). This agrees with the known antitussive effect of systemic lignocaine: Yukioka et al. (1985) demonstrated a clear dose-response reduction in coughing during intubation, with significant suppression at doses ≥1 mg/kg and near-complete suppression at higher doses, providing mechanistic support for why IV lignocaine in the present study likely improved intubation tolerance compared with topical oropharyngeal spray alone.[11]Hemodynamic safety outcomes in the current work were acceptable and broadly comparable between groups, with no serious arrhythmias or toxicity. Bradycardia (7.14% vs 2.38%; p=0.30) and hypotension (9.52% vs 4.76%; p=0.40) were numerically higher in Group IV but not statistically significant, suggesting that at the administered dose, IV lignocaine's benefit occurred without a major penalty in clinically important adverse cardiovascular events. Lev et al. (1994) reviewed prophylactic pre-intubation lignocaine use and noted that while efficacy varies across endpoints and techniques, harmful effects were not consistently documented when conventional dosing is used, supporting the overall safety signal seen in the present cohort.[12]The relatively weaker attenuation seen with topical lignocaine spray in this study (higher HR and MAP peaks and more ≥20% responders) can be interpreted in light of variability in topical delivery, distribution, and onset time. "Topical lignocaine" encompasses multiple methods (spray, jelly, transtracheal, nebulized, ETT-delivered), and effectiveness is influenced by where and when the drug contacts airway receptors. Soltani et al. (2002) demonstrated that different lignocaine application methods yield different airway outcomes (notably cough/sore throat), underscoring that not all topical approaches are equivalent; therefore, the present finding that simple oropharyngeal/supraglottic spray did not blunt pressor/tachycardic responses as effectively as IV lignocaine is biologically plausible and consistent with the broader methodological heterogeneity reported in topical lignocaine literature.[13]Finally, it is important to contextualize these findings against populations and techniques where IV lignocaine has shown limited benefit. While our adult ocular-surgery cohort displayed clear reduction in HR/MAP surges with IV lignocaine, Splinter et al. (1990) reported that IV lignocaine did *not* attenuate intubation-related increases in HR and arterial pressure in children, highlighting that age-related physiology, baseline heart rates, anesthetic technique, and reflex intensity can modify observed efficacy. This helps explain why IV lignocaine's apparent benefit in the present adult ASA I–II population should not be generalized without considering patient factors and procedural context.[14]

CONCLUSION

Intravenous lignocaine was more effective than topical lignocaine in attenuating the hemodynamic response to laryngoscopy and tracheal intubation during ocular surgery under general anesthesia. The IV route provided better control of heart rate and blood pressure changes at critical stimulation points and reduced the occurrence of clinically significant sympathetic surges. Both techniques were generally safe, with no serious adverse effects observed. Overall, IV lignocaine can be considered a preferable option for achieving greater peri-intubation hemodynamic stability in ocular surgical patients.

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