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Original Research Article

Comparative Study of Oral Nitroglycerin Spray and Oropharyngeal Lignocaine Spray in Attenuating Hemodynamic Response to Laryngoscopy and Endotracheal Intubation in Patients Undergoing General Anaesthesia

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Abstract:

Introduction: Direct laryngoscopy and endotracheal intubation provoke marked sympathetic stimulation, causing transient tachycardia and hypertension that may precipitate serious cardiovascular complications in susceptible individuals. Evidence directly comparing oral nitroglycerin spray with oropharyngeal lignocaine spray for blunting this response is limited. This study compared the efficacy of the two sprays and evaluated their adverse effects.

Methods: This prospective, randomized, double-blinded study enrolled 100 adults aged 18–60 years, of ASA physical status I and II, who were scheduled for elective surgery under general anaesthesia. Patients were randomized into two groups of 50. Group L received 10% oropharyngeal lignocaine spray (100 mg) three minutes before induction, and Group N received oral nitroglycerin spray (0.8 mg) 30 seconds before induction. Heart rate, systolic, diastolic and mean arterial pressure were recorded at baseline, pre-induction, during intubation, and at 1, 3, 5, 7 and 10 minutes after intubation. Data were analysed using SPSS version 23, with $p < 0.05$ considered significant.

Results: Heart rate and systolic, diastolic and mean arterial pressures were significantly lower in Group N than in Group L at intubation and at every post-intubation interval ($p < 0.001$). At intubation, heart rate was 76.24 ± 6.17 versus 94.52 ± 11.40 beats/min, and systolic pressure was 104.00 ± 7.50 versus 129.08 ± 10.47 mmHg. There were no significant differences in pre-induction blood pressure. No major adverse events occurred in either group.

Discussion and Conclusion: Oral nitroglycerin spray attenuated the hemodynamic response to laryngoscopy and intubation more effectively than 10% oropharyngeal lignocaine spray, with a comparable safety profile in ASA I and II patients. It may be considered a simple, safe and effective option during airway instrumentation under general anaesthesia. Larger multicentre studies that include high-risk patients are needed to confirm these findings.

Keywords: General Anaesthesia; Hemodynamic Response; Laryngoscopy; Endotracheal Intubation; Nitroglycerin Spray; Lignocaine Spray.

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Introduction

Laryngoscopy and endotracheal intubation are essential components of airway management during general anaesthesia, yet they provoke intense sympathetic stimulation that produces transient rises in heart rate and arterial blood pressure [7,25]. This pressor response is mediated chiefly by reflex sympathetic activation following stimulation of the supraglottic structures and trachea. Although healthy individuals usually tolerate these changes well, in patients with limited cardiovascular reserve they may precipitate myocardial ischaemia, cardiac arrhythmias, cerebrovascular events or a rise in intracranial pressure [24,25], and attenuating the response has therefore remained an important concern for anaesthesiologists. A wide range of pharmacological agents has been investigated for this purpose, including opioids such as fentanyl and alfentanil [19,22,23], β-blockers such as esmolol [8,9], α2-adrenergic agonists such as dexmedetomidine [18,21], magnesium sulphate [20], calcium channel blockers, vasodilators such as nitro-glycerine [8–11], and local anaesthetics administered by intravenous, nebulized or topical

routes [12,14–17]. These agents have met with varying degrees of success. The ideal agent should suppress sympathetic stimulation effectively without causing excessive hypotension, bradycardia, delayed recovery or other adverse effects. Nitroglycerin is an organic nitrate that produces vasodilatation by releasing nitric oxide, which increases intracellular cyclic guanosine monophosphate (cGMP) and relaxes vascular smooth muscle. It acts predominantly on venous capacitance vessels, reducing venous return, myocardial wall stress and arterial pressure. Because it acts rapidly when given as a transmucosal spray, nitroglycerin has been studied as a means of blunting the haemodynamic response to airway manipulation, and intranasal or sublingual administration has been reported to attenuate the pressor response in normotensive patients, in hypertensive patients and in patients undergoing coronary artery bypass surgery [8,10,11,18,19].

Lignocaine remains one of the most commonly used local anaesthetics in anaesthesia practice. Applied topically, it suppresses airway reflexes by blocking sensory nerve endings in the oropharynx and larynx [12,14,15], and its systemic absorption from airway mucosa has long been recognized [13]. Oropharyngeal lignocaine spray is inexpensive, readily available and widely used to reduce airway stimulation during laryngoscopy and intubation. Nevertheless, its efficacy in attenuating the cardiovascular response has been variable [12,15,16], and appears to depend on the route and technique of administration [4,5,17], with systematic evidence available chiefly for the intravenous route [6].

Nitroglycerin spray and topical lignocaine have each been evaluated individually and against other drugs [9,12,14,18,19], but studies that compare the two directly remain few [1–3]. Establishing their relative efficacy could help clinicians select an effective and practical strategy for minimizing adverse cardiovascular responses during airway instrumentation. The present randomized, double-blinded study was therefore undertaken to compare oral nitroglycerin spray (0.8 mg) with 10% oropharyngeal lignocaine spray (100 mg) in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation in adult patients undergoing elective surgery under general anaesthesia, and to evaluate the incidence of adverse effects associated with each intervention.

Materials and Methods

Study Design and Population: This prospective, randomized, double-blinded comparative study was conducted after approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants before enrolment.

A total of 100 adult patients aged 18–60 years, of ASA physical status I or II, who were scheduled for elective surgery under general anaesthesia requiring endotracheal intubation and who were willing to give written informed consent, were included. Patients were excluded if they declined to participate, had a known allergy to nitroglycerin or lignocaine, had significant hepatic, renal or cardiovascular disease or significant pharyngeal or laryngeal pathology, were posted for emergency surgery, or were anticipated to have a difficult airway.

Randomization, Blinding and Study Groups: Participants were allocated to two equal groups (n = 50 each) using computer-generated random numbers. The study drugs were prepared by an independent investigator who took no part in intraoperative management, and data were collected by a second investigator who was blinded to group allocation. Patients in Group L (lignocaine group) received 10% oropharyngeal lignocaine spray (100 mg) three minutes before induction of anaesthesia, whereas patients in Group N (nitroglycerin group) received oral nitroglycerin spray (0.8 mg) 30 seconds before induction.

Anaesthetic Technique: All patients underwent standard preoperative evaluation and fasting according to institutional protocol. Monitoring included electrocardiography, non-invasive blood pressure and pulse oximetry, and baseline heart rate, systolic, diastolic and mean arterial pressure, and oxygen saturation were recorded. Premedication consisted of intravenous glycopyrrolate (0.05 µg/kg), midazolam (0.025 mg/kg) and fentanyl (2 µg/kg), followed by preoxygenation with 100% oxygen for three minutes. Anaesthesia was induced with intravenous thiopentone sodium (5 mg/kg), and muscle relaxation was achieved with succinylcholine (1.5 mg/kg). An experienced anaesthesiologist performed direct laryngoscopy and endotracheal intubation, keeping the duration of laryngoscopy below 10 seconds wherever possible. Anaesthesia was maintained with oxygen, nitrous oxide, sevoflurane and atracurium, and no surgical stimulation was permitted during the first 10 minutes after intubation so as to avoid confounding the haemodynamic observations.

Outcome Measures: The primary outcome was attenuation of the hemodynamic response to laryngoscopy and intubation, assessed by heart rate and systolic, diastolic and mean arterial pressure recorded at baseline (preoperative room), pre-induction, during intubation, and at 1, 3, 5, 7 and 10 minutes after intubation. Secondary outcomes were adverse events, namely hypotension,

hypertension, tachycardia, bradycardia and any cardiac dysrhythmia.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using SPSS version 23. Continuous variables were expressed as mean ± standard deviation and categorical variables as frequencies and percentages. Continuous variables were compared between groups using the independent Student's t-test (or the Mann–Whitney U test where appropriate), and categorical variables were compared using the Chi-square test. A p-value below 0.05 was considered statistically significant.

Results

All 100 patients completed the study, 50 in each group, with no protocol deviations, and all were included in the final analysis. Demographic characteristics, including age, sex, weight and ASA physical status, were comparable between the groups.

Heart rate: Baseline heart rate differed modestly between the groups (91.14 ± 11.35 vs 86.02 ± 9.59 beats/min in Groups L and N, respectively; p = 0.017). Before induction, the heart rate was lower in Group L (80.00 ± 12.01) than in Group N (90.92 ± 10.37; p < 0.001).

During intubation, however, the heart rate rose to 94.52 ± 11.40 in Group L and peaked at 96.56 ± 12.08 one minute later, whereas in Group N it fell to 76.24 ± 6.17 at intubation and reached only 88.64 ± 5.64 at one minute.

The difference between the groups was significant at intubation and at every post-intubation interval (p < 0.001). Heart rate declined gradually in both groups, reaching 85.96 ± 10.29 in Group L and 78.16 ± 10.02 in Group N at 10 minutes, with earlier stabilization in the nitroglycerin group (Table 1).

Table 1: Comparison of heart rate (beats/min) between the study groups

Time interval	Group L (n = 50)	Group N (n = 50)	p-value
Baseline	91.14 ± 11.35	86.02 ± 9.59	0.017
Pre-induction	80.00 ± 12.01	90.92 ± 10.37	<0.001
Intubation	94.52 ± 11.40	76.24 ± 6.17	<0.001
1 min post-intubation	96.56 ± 12.08	88.64 ± 5.64	<0.001
3 min post-intubation	94.08 ± 11.70	82.48 ± 9.60	<0.001
5 min post-intubation	91.56 ± 10.53	79.12 ± 11.02	<0.001
7 min post-intubation	90.96 ± 10.56	78.60 ± 10.97	<0.001
10 min post-intubation	85.96 ± 10.29	78.16 ± 10.02	<0.001

Values are mean ± SD.

Systolic Blood Pressure: Systolic blood pressure at intubation was 129.08 ± 10.47 mmHg in Group L and 104.00 ± 7.50 mmHg in Group N, and the lignocaine group peaked at 130.24 ± 11.80 mmHg at one minute against 107.60 ± 7.20 mmHg in the nitroglycerin group. Pressures were significantly lower in Group N at intubation and at all post-intubation intervals (p < 0.001), reaching 118.24 ± 7.93 and 110.12 ± 7.17 mmHg in Groups L and N, respectively, at 10 minutes.

The groups did not differ significantly at pre-induction (p = 0.325), although a small difference was present at baseline (p = 0.020) (Table 2).

Table 2: Comparison of systolic blood pressure (mmHg) between the study groups

Time interval	Group L (n = 50)	Group N (n = 50)	p-value
Baseline	128.76 ± 9.92	124.34 ± 8.68	0.020
Pre-induction	116.88 ± 9.98	118.84 ± 9.84	0.325
Intubation	129.08 ± 10.47	104.00 ± 7.50	<0.001
1 min post-intubation	130.24 ± 11.80	107.60 ± 7.20	<0.001
3 min post-intubation	128.48 ± 9.36	118.20 ± 10.90	<0.001
5 min post-intubation	125.64 ± 9.38	116.80 ± 10.27	<0.001
7 min post-intubation	123.28 ± 8.74	115.20 ± 8.78	<0.001
10 min post-intubation	118.24 ± 7.93	110.12 ± 7.17	<0.001

Values are mean ± SD.

Diastolic blood pressure: Diastolic blood pressure followed a similar pattern. It rose to 85.36 ± 8.3 mmHg at intubation in Group L but was 67.08 ± 5.2 mmHg in Group N, and it remained significantly lower in the nitroglycerin group at every post-intubation interval (p < 0.001), reaching 75.92 ± 5.5 and 70.64 ± 7.3 mmHg in Groups L and N, respectively, at 10 minutes. As with systolic pressure, there was no significant difference at pre-induction (p = 0.374), whereas the baseline values differed slightly (p = 0.026) (Table 3).

Table 3: Comparison of diastolic blood pressure (mmHg) between the study groups

Time interval	Group L (n = 50)	Group N (n = 50)	p-value
Baseline	83.68 ± 9.31	80 ± 6.7	0.026
Pre-induction	76.44 ± 8.3	77.72 ± 5.8	0.374
Intubation	85.36 ± 8.3	67.08 ± 5.2	<0.001
1 min post-intubation	85.24 ± 8.4	71.26 ± 7.8	<0.001
3 min post-intubation	82.08 ± 8.1	75.12 ± 9.9	<0.001
5 min post-intubation	81.04 ± 7.3	72.96 ± 9.3	<0.001
7 min post-intubation	79.76 ± 6.5	73.4 ± 8.3	<0.001
10 min post-intubation	75.92 ± 5.5	70.64 ± 7.3	<0.001

Values are mean ± SD.

Mean arterial pressure: Mean arterial pressure was 99.82 ± 8.48 mmHg in Group L at intubation compared with 79.57 ± 4.88 mmHg in Group N, and the lignocaine group remained higher at 1 minute (99.96 ± 8.91 vs 84 ± 7.41 mmHg). The difference was statistically significant at intubation and at 1, 3, 5, 7 and 10 minutes after intubation ($p < 0.001$), indicating superior attenuation of the pressor response with nitroglycerin spray. The pre-induction values were comparable ($p = 0.380$), but the baseline values differed slightly ($p = 0.010$) (Table 4).

Table 4: Comparison of mean arterial pressure (mmHg) between the study groups

Time interval	Group L (n = 50)	Group N (n = 50)	p-value
Baseline	98.64 ± 8.8	94.68 ± 6.09	0.010
Pre-induction	90.16 ± 7.75	91.40 ± 6.2	0.380
Intubation	99.82 ± 8.48	79.57 ± 4.88	<0.001
1 min post-intubation	99.96 ± 8.91	84 ± 7.41	<0.001
3 min post-intubation	97.56 ± 8.04	89.14 ± 9.49	<0.001
5 min post-intubation	96.08 ± 7.43	87.70 ± 8.63	<0.001
7 min post-intubation	94.48 ± 6.71	87.32 ± 7.91	<0.001
10 min post-intubation	89.92 ± 5.52	83.46 ± 7.03	<0.001

Values are mean ± SD.

Adverse Events: Both drugs were well tolerated. No major adverse events, such as severe hypotension, persistent bradycardia, clinically significant tachyarrhythmia or oxygen desaturation requiring intervention, occurred during the study. Minor adverse effects were infrequent and did not differ significantly between the groups, and both nitroglycerin and lignocaine sprays were safe in this population.

Summary of Results: Oral nitroglycerin spray produced significantly better attenuation of the hemodynamic response to laryngoscopy and endotracheal intubation than 10% oropharyngeal lignocaine spray, with lower heart rate and systolic, diastolic and mean arterial pressures during airway instrumentation and the early post-intubation period. Neither intervention was associated with serious adverse effects.

Discussion

This prospective, randomized, double-blinded study compared oral nitroglycerin spray with 10% oropharyngeal lignocaine spray for attenuating the hemodynamic response to laryngoscopy and endotracheal intubation in adults undergoing elective surgery under general anaesthesia. Its principal finding was that nitroglycerin produced significantly better attenuation of heart rate and of systolic, diastolic and mean arterial pressure than topical lignocaine, which suggests that oral nitroglycerin spray is an effective and safe means of limiting the cardiovascular stress of airway instrumentation.

The haemodynamic response to laryngoscopy and intubation arises from sympathetic discharge provoked by mechanical irritation of the upper airway, with catecholamine release producing tachycardia and hypertension [7,25]. These changes are transient in healthy individuals but may be harmful in patients with coronary artery disease, hypertension, cerebrovascular disease or raised intracranial pressure [24,25], so that blunting the response remains an important objective at induction.

Patients given nitroglycerin spray had significantly lower heart rate and arterial pressures during and after intubation than those given lignocaine spray. The lower systolic, diastolic and mean pressures can be attributed to the rapid vasodilator action of nitroglycerin through nitric oxide release, with reduced venous return and systemic vascular resistance, an effect that has been documented with intranasal administration [10,11]. Topical lignocaine, in contrast, mainly suppresses sensory input from the airway [12,14,15] and does not

produce a comparable systemic vasodilator effect, which may explain its lesser efficacy in this study.

Our findings agree with earlier work showing that nitroglycerin reduces the pressor response to laryngoscopy and intubation, whether it is compared directly with lignocaine spray [1–3], with other agents such as esmolol, lignocaine, dexmedetomidine or fentanyl [9,18,19], or with a β-blocker in hypertensive patients [8], and whether it is given intranasally to normotensive patients or to patients undergoing coronary artery bypass surgery [10,11]. Studies of topical and aerosolized lignocaine, by comparison, have generally reported only partial attenuation of airway reflexes and less consistent effects on cardiovascular parameters [12,14,15], and the results with other lignocaine routes and techniques have also varied [4–6,16,17]. By comparing the two interventions directly under similar clinical conditions, the present study strengthens these observations and shows the superior efficacy of oral nitroglycerin spray. Both interventions had a favourable safety profile. No serious adverse events, including severe hypotension, persistent bradycardia, clinically significant arrhythmia or oxygen desaturation, were observed, which suggests that either spray can be given safely to ASA I and II patients undergoing elective surgery. The strengths of this study include its prospective, randomized, double-blinded design, a uniform anaesthetic technique, standardized timing of drug administration, and the assessment of several hemodynamic variables throughout the immediate post-intubation period. These features minimized bias and improved the reliability of the findings. The study also has limitations. It enrolled only ASA I and II adults undergoing elective surgery, so the findings cannot be extrapolated directly to patients with significant cardiovascular disease, a difficult airway or emergency surgery, and long-term postoperative cardiovascular outcomes were not assessed. Multicentre studies with larger samples that include high-risk patients are needed to establish the role of oral nitroglycerin spray in routine anaesthesia practice.

Conclusion

Both oral nitroglycerin spray and 10% oropharyngeal lignocaine spray attenuated the hemodynamic response to laryngoscopy and endotracheal intubation, but nitroglycerin was significantly more effective in reducing heart rate and systolic, diastolic and mean arterial pressure in the peri-intubation period, and both were well tolerated without significant adverse effects. Oral nitroglycerin spray may therefore be considered a simple, safe, effective and non-invasive option for attenuating the cardiovascular response to laryngoscopy and intubation in ASA I and II patients undergoing elective surgery under general anaesthesia. Further multicentre studies with larger samples and high-risk populations are recommended to validate these findings and broaden their clinical applicability.

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