Section: Anaesthesiology

Original Research Article

COMPARATIVE EVALUATION OF INTRAVENOUS ESMOLOL AND DILTIAZEM FOR ATTENUATION OF HEMODYNAMIC STRESS RESPONSE DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

D. Suvankar[1], Anjutgi Reshma Parmeshwar[1], Priyambad Sahu[2]

[1]Assistant Professor, Department of Critical Care Medicine, IMS & SUM Hospital Campus II, Bhubaneswar, Odisha, India.
[2]Assistant Professor, Department of General Surgery, PGIMER & Capital Hospital, Bhubaneswar, Odisha, India.

Received *: 25/05/2026*
Received in revised form *: 29/06/2026*
Accepted *: 14/07/2026*

Corresponding Author:
Dr. Anjutgi Reshma Parmeshwar,
Assistant Professor, Department of Critical Care Medicine, IMS & SUM Hospital Campus II, Bhubaneswar, Odisha, India.
Email: reshmaanjutgi@gmail.com

DOI: 10.70034/ijmedph.2026.3.252

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 1570-1576

ABSTRACT

Background: Laryngoscopy and endotracheal intubation are associated with significant hemodynamic responses due to sympathetic stimulation, resulting in tachycardia and hypertension. Although these changes are transient in healthy individuals, they may lead to adverse cardiovascular outcomes in susceptible patients. Various pharmacological agents have been used to attenuate this pressor response, among which esmolol and diltiazem have shown promising results. The Objective is to compare the efficacy of intravenous esmolol and intravenous diltiazem in attenuating the hemodynamic response to laryngoscopy and tracheal intubation.

Materials and Methods: A prospective, double-blind, randomized controlled study was conducted in the Department of Anaesthesiology over a period of two years after obtaining Institutional Ethics Committee approval. A total of 120 ASA physical status I patients, aged 18–65 years, undergoing elective surgery under general anaesthesia were randomly allocated into three groups (n=40 each). Group I received normal saline (20 mL IV), Group II received diltiazem 0.2 mg/kg IV, and Group III received esmolol 0.5 mg/kg IV, administered 5 minutes before intubation. Hemodynamic parameters including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at baseline, after induction, before and after intubation, and at predefined intervals up to 20 minutes post-intubation. Data were analyzed using appropriate statistical methods, and $p < 0.05$ was considered statistically significant.

Results: Demographic variables were comparable among the three groups. Both diltiazem and esmolol significantly attenuated the hemodynamic response to laryngoscopy and tracheal intubation compared with placebo ($p < 0.001$). Esmolol demonstrated superior control of heart rate, with significantly lower post-intubation tachycardia than diltiazem and placebo. In contrast, diltiazem showed comparatively better attenuation of systolic, diastolic, and mean arterial pressures, particularly during the early post-intubation period. No major adverse effects such as severe hypotension, bradycardia, or arrhythmias were observed in any group.

Conclusion: Both intravenous esmolol and diltiazem effectively attenuated the hemodynamic response to laryngoscopy and tracheal intubation compared to placebo. Esmolol was more effective in controlling tachycardia, whereas diltiazem provided better control of blood pressure responses. The choice of agent may therefore be individualized according to the predominant hemodynamic concern and patient profile.

Keywords: Esmolol, Diltiazem, Laryngoscopy, Tracheal intubation, Hemodynamic response, General anaesthesia.

INTRODUCTION

Laryngoscopy and endotracheal intubation are essential components of general anaesthesia and airway management. However, these procedures are frequently associated with significant sympathetic stimulation resulting in transient but marked hemodynamic alterations, including tachycardia, hypertension, and increased myocardial oxygen demand. These cardiovascular responses are primarily mediated through reflex sympathetic activation caused by stimulation of the epipharyngeal and laryngotracheal structures during laryngoscopy and tracheal intubation. Such changes are usually transient in healthy individuals but may be potentially hazardous in patients with cardiovascular disease, systemic hypertension, raised intracranial pressure, cerebrovascular disease, or ischemic heart disease.[1–3]

The hemodynamic stress response to laryngoscopy and tracheal intubation has been attributed to increased catecholamine release, particularly plasma norepinephrine, resulting in elevated heart rate and arterial blood pressure.[4] Although these responses generally subside within a few minutes, exaggerated pressor responses may precipitate myocardial ischemia, arrhythmias, pulmonary edema, or cerebrovascular complications in vulnerable patients. Therefore, attenuation of this response remains an important objective in anaesthetic practice.[5,6]

Various pharmacological interventions have been investigated to blunt the cardiovascular response associated with laryngoscopy and tracheal intubation. These include opioids, local anaesthetics such as intravenous lignocaine, vasodilators, beta-adrenergic blockers, calcium channel blockers, and alpha-2 adrenergic agonists.[7–10] Despite numerous available options, no single agent has proven ideal, and the search for an effective, safe, and predictable method for attenuating hemodynamic responses continues.

Esmolol, an ultra-short-acting selective β1-adrenergic receptor antagonist, has a rapid onset and short duration of action, making it particularly suitable for controlling transient tachycardia and hypertension associated with airway instrumentation. It has been shown to effectively attenuate increases in heart rate and blood pressure during laryngoscopy and intubation with minimal prolonged cardiovascular effects.[11,12]

Diltiazem, a calcium channel blocker, exerts its effect by inhibiting calcium influx across cardiac and vascular smooth muscle cells, thereby reducing myocardial contractility, slowing atrioventricular conduction, and producing peripheral vasodilatation. Previous studies have demonstrated its efficacy in controlling pressor responses during endotracheal intubation, particularly by attenuating blood pressure elevation.[13,14]

Although both esmolol and diltiazem have demonstrated beneficial effects in controlling hemodynamic responses during laryngoscopy and tracheal intubation, evidence comparing their relative efficacy remains inconsistent. Therefore, the present study was undertaken to compare the effectiveness of intravenous esmolol and intravenous diltiazem in attenuating hemodynamic responses to laryngoscopy and tracheal intubation and to evaluate their associated adverse effects.

Aims and Objectives

To compare the efficacy of intravenous esmolol and intravenous diltiazem in attenuating the hemodynamic response to laryngoscopy and tracheal intubation.

MATERIALS AND METHODS

A prospective, double-blind, randomized controlled study was conducted in the Department of Anaesthesiology over a period of two years after obtaining approval from the Institutional Ethics Committee.

A total of 120 patients belonging to American Society of Anesthesiologists (ASA) Physical Status I undergoing elective surgical procedures under general anaesthesia were included in the study.

Patients were randomly allocated into one of the three study groups using a computer-generated random number sequence (OPEN API statistical package).

- Group I (Control Group): Received 20 mL of normal saline intravenously over 2 minutes
- Group II (Diltiazem Group): Received intravenous diltiazem 0.2 mg/kg diluted to 20 mL and administered over 2 minutes
- Group III (Esmolol Group): Received intravenous esmolol 0.5 mg/kg diluted to 20 mL and administered over 2 minutes

The study was conducted in a double-blind manner, wherein both the patient and the anaesthesiologist responsible for data collection were blinded to the study drug administered.

Eligibility Criteria

Inclusion Criteria

- Patients aged 18–65 years
- ASA Physical Status I
- Patients scheduled for elective surgical procedures under general anaesthesia
- Patients willing to provide written informed consent

Exclusion Criteria

- Patients unwilling or unable to provide informed consent
- ASA Physical Status II–IV
- Age <18 years or >65 years
- Anticipated difficult airway/intubation
- Obesity
- Patients receiving sedatives, hypnotics, beta blockers, calcium channel blockers, or antihypertensive medications

- Known allergy or hypersensitivity to any study or anaesthetic drug

Preoperative Evaluation

All patients underwent a detailed pre-anaesthetic assessment on the day prior to surgery, including history taking, systemic examination, and routine investigations. Demographic and clinical details including name, age, sex, body weight, registration number, and diagnosis were recorded.

Patients were informed regarding the study objectives, procedure, drugs used, and their potential effects and adverse reactions. Written informed consent was obtained prior to enrolment. Patients were kept nil per oral (NPO/NBM) according to ASA fasting guidelines.

Anaesthetic Procedure

On the day of surgery, patient investigations and fasting status were reconfirmed. After shifting to the operation theatre, standard monitoring including electrocardiography (ECG), pulse oximetry (SpO_2), non-invasive blood pressure (NIBP), and heart rate monitoring was established.

The allocated study drug was administered 5 minutes prior to laryngoscopy and tracheal intubation.

All patients received premedication with:

- Midazolam 0.03 mg/kg intravenously
- Fentanyl 2 µg/kg intravenously

Anaesthesia was induced using:

- Propofol 2 mg/kg intravenously
- Vecuronium 0.1 mg/kg intravenously to facilitate endotracheal intubation.

Laryngoscopy and endotracheal intubation were performed by an experienced anaesthesiologist.

Anaesthesia was maintained with:

- Isoflurane (1 MAC)
- Fresh gas flow of 3 L/min, consisting of 50% oxygen and 50% nitrous oxide

Hemodynamic Monitoring

Hemodynamic parameters including Heart Rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), Electrocardiography (ECG), Peripheral Oxygen Saturation (SpO_2), End-tidal Carbon Dioxide ($EtCO_2$)

For intraoperative analgesia, patients received intravenous paracetamol 15 mg/kg.

At the end of surgery, residual neuromuscular blockade was reversed using:

- Glycopyrrolate 8 µg/kg intravenously
- Neostigmine 0.05 mg/kg intravenously

Following extubation, patients were shifted to the Post-Anaesthesia Care Unit (PACU) and observed for 12 hours after administration of the study drug for any adverse events or complications.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequency and percentage. Comparison between groups was performed using appropriate statistical tests, and a p-value <0.05 was considered statistically significant.

RESULTS

A total of 120 patients scheduled for elective surgery under general anaesthesia were enrolled and randomly allocated into three equal groups (n=40 each). Group I received normal saline (control), Group II received intravenous diltiazem (0.2 mg/kg), and Group III received intravenous esmolol (0.5 mg/kg) prior to laryngoscopy and tracheal intubation.

The three study groups were comparable with respect to demographic characteristics. The mean age of patients in Group I, Group II, and Group III was 43.35 ± 7.76 years, 43.57 ± 8.45 years, and 44.22 ± 9.35 years, respectively, with no statistically significant difference between groups (p = 0.893). Female participants predominated across all groups, accounting for 67.5% of the total study population. No statistically significant difference in gender distribution was observed (p = 0.634), indicating baseline comparability among the study groups.

Baseline heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were comparable among all groups before induction (p > 0.05).

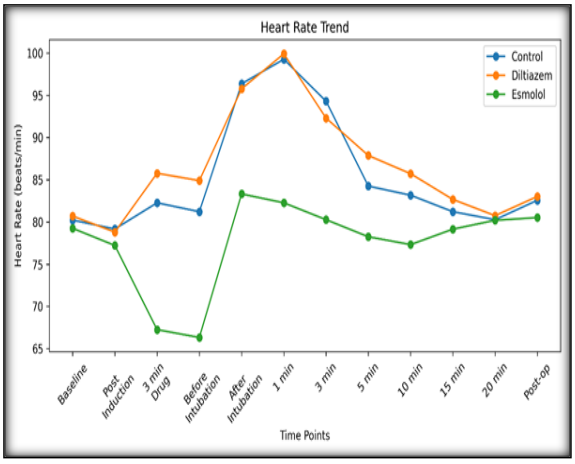

Figure 1: Trend of heart rate changes among the control, diltiazem, and esmolol groups at different peri-intubation time points

Following study drug administration, patients in the esmolol group demonstrated a significant reduction in heart rate compared to both the control and diltiazem groups. Immediately after intubation and at 1 minute post-intubation, heart rate increased markedly in the control and diltiazem groups, whereas the rise was significantly attenuated in the esmolol group (p < 0.001). Hemodynamic values gradually returned toward baseline in all groups by 15–20 minutes post-intubation.

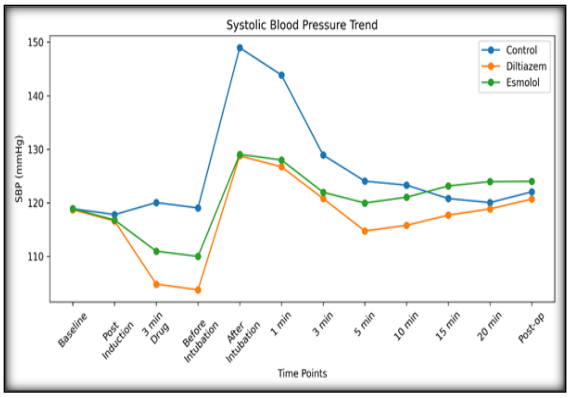

Figure 2: Trend of systolic blood pressure changes among the control, diltiazem, and esmolol groups during laryngoscopy and tracheal intubation

A significant rise in systolic blood pressure occurred immediately after intubation in the control group (148.95 ± 11.65 mmHg) compared to the diltiazem (128.77 ± 7.58 mmHg) and esmolol (129.05 ± 9.26 mmHg) groups ($p < 0.001$). Both diltiazem and esmolol effectively attenuated the pressor response, with diltiazem demonstrating slightly greater reduction in systolic blood pressure during the pre-intubation and early post-intubation period.

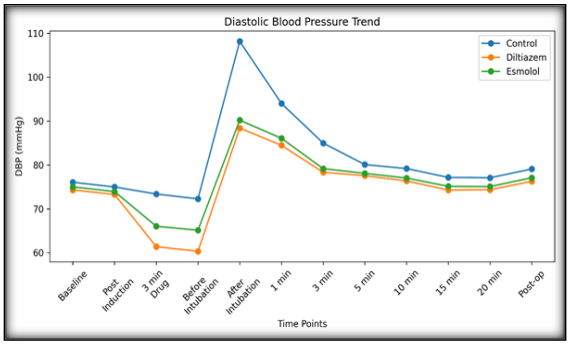

Figure 3: Trend of diastolic blood pressure changes among the control, diltiazem, and esmolol groups across study time points

Diastolic blood pressure increased significantly after intubation in all groups; however, the increase was substantially lower in the diltiazem and esmolol groups compared to the control group. Immediately after intubation, DBP in the control group reached 108.15 ± 4.82 mmHg, whereas lower values were observed in the diltiazem (88.40 ± 4.40 mmHg) and esmolol (90.20 ± 4.10 mmHg) groups ($p < 0.001$).

Mean arterial pressure increased significantly after intubation in the control group (121.74 ± 6.47 mmHg) compared with the diltiazem (101.85 ± 4.81 mmHg) and esmolol (103.14 ± 5.35 mmHg) groups ($p < 0.001$). Both drugs significantly attenuated the pressor response, although diltiazem showed a comparatively greater reduction in blood pressure parameters.

No major adverse effects, severe bradycardia, hypotension, arrhythmia, or hypersensitivity reactions requiring discontinuation of the study drug were observed in any of the groups.

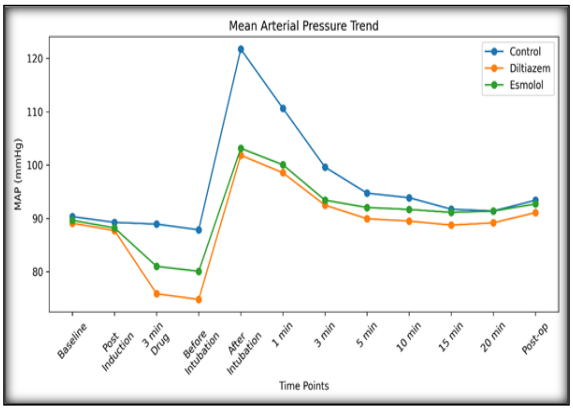

Figure 4: Trend of mean arterial pressure changes among the control, diltiazem, and esmolol groups during the peri-intubation period

Table 1: Baseline Demographic Characteristics of Study Participants

Variable	Group I (Control) n=40	Group II (Diltiazem) n=40	Group III (Esmolol) n=40	p-value
Age (years), Mean ± SD	43.35 ± 7.76	43.57 ± 8.45	44.22 ± 9.35	0.893
Female, n (%)	29 (72.5)	25 (62.5)	27 (67.5)	0.634
Male, n (%)	11 (27.5)	15 (37.5)	13 (32.5)	

Table 2: Key Hemodynamic Parameters at Critical Time Points (Mean ± SD)

Parameter	Time Point	Control	Diltiazem	Esmolol	Significance
Heart Rate (beats/min)	Baseline	80.22 ± 7.70	80.70 ± 6.49	79.25 ± 6.22	NS
	Immediately after intubation	96.37 ± 7.28	95.77 ± 6.35	83.32 ± 5.87	p<0.001
	1 min after intubation	99.27 ± 7.58	99.92 ± 6.17	82.27 ± 6.32	p<0.001
SBP (mmHg)	Baseline	118.9 ± 11.7	118.7 ± 8.10	118.9 ± 9.48	NS
	Immediately after intubation	148.95 ± 11.65	128.77 ± 7.58	129.05 ± 9.26	p<0.001
	1 min after intubation	143.87 ± 11.52	126.72 ± 8.06	128.00 ± 9.33	p<0.001
DBP (mmHg)	Baseline	76.05 ± 5.04	74.30 ± 4.56	75.00 ± 3.90	NS
	Immediately after intubation	108.15 ± 4.82	88.40 ± 4.40	90.20 ± 4.10	p<0.001
MAP (mmHg)	Baseline	90.33 ± 6.67	89.10 ± 4.87	89.64 ± 5.00	NS
	Immediately after intubation	121.74 ± 6.47	101.85 ± 4.81	103.14 ± 5.35	p<0.001

NS = Not Significant

DISCUSSION

Laryngoscopy and endotracheal intubation are associated with transient but significant hemodynamic alterations due to reflex sympathetic stimulation of the upper airway and laryngeal structures. This sympathetic surge leads to tachycardia, hypertension, and increased myocardial oxygen demand secondary to catecholamine release, particularly norepinephrine. Although healthy individuals generally tolerate these changes, they may be detrimental in patients with cardiovascular disease, hypertension, cerebrovascular disorders, or raised intracranial pressure. Consequently, attenuation of this pressor response remains an important objective in anaesthetic practice.[6,15–17]

Several pharmacological interventions have been investigated to blunt the hemodynamic response to airway instrumentation, including opioids, lignocaine, vasodilators, β-adrenergic blockers, calcium channel blockers, and α2-adrenergic agonists.[6,18,19] However, an ideal agent capable of effectively attenuating both tachycardia and hypertension with minimal adverse effects remains elusive. The present study evaluated the comparative efficacy of intravenous esmolol and diltiazem in attenuating the hemodynamic response to laryngoscopy and tracheal intubation.

In the present study, the demographic characteristics were comparable across all three groups with respect to age and sex distribution, suggesting homogeneity of the study population and minimizing the possibility of confounding due to baseline differences. Similar demographic comparability has been reported in previous studies comparing esmolol and diltiazem for

The findings of the present study demonstrated that esmolol provided superior control of heart rate compared with diltiazem and placebo. Although all groups exhibited an increase in heart rate following laryngoscopy and tracheal intubation, the magnitude of increase was significantly lower in the esmolol group, particularly during the immediate post-intubation period and up to 10 minutes thereafter. Peak heart rate was observed at 1 minute after intubation in all groups, after which values gradually returned toward baseline.

The superior efficacy of esmolol in controlling tachycardia can be attributed to its pharmacological profile as an ultra-short-acting selective β1-adrenergic receptor antagonist. By blocking sympathetic stimulation of cardiac β1 receptors, esmolol effectively suppresses reflex tachycardia associated with airway manipulation without producing prolonged cardiovascular depression.[22]

Our findings are consistent with the observations of Rathore et al., who reported effective attenuation of tachycardia with esmolol administration during laryngoscopy and intubation, particularly at higher doses.[23] Similarly, Kumar et al. observed significantly lower heart rates in patients receiving esmolol compared with diltiazem and magnesium sulphate, concluding that esmolol was more effective in controlling intubation-induced tachycardia.[24] Comparable findings were also reported by Agrawal et al. and Parvez Gazi et al, who demonstrated superior heart rate attenuation with esmolol when compared with diltiazem and placebo.[21,25]

In contrast, diltiazem showed only modest efficacy in controlling heart rate. Although some reduction in tachycardia was observed compared to the control group, the effect was less pronounced than esmolol. This finding is biologically plausible, as diltiazem primarily acts through calcium channel blockade and atrioventricular nodal suppression, resulting in comparatively weaker control of acute sympathetic tachycardia.

In the present study, both diltiazem and esmolol significantly attenuated the rise in systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) compared to placebo. However, diltiazem demonstrated comparatively better control of blood pressure parameters, particularly during the early post-intubation period.

The control group exhibited a marked rise in blood pressure immediately after intubation, reflecting the sympathoadrenal response associated with airway instrumentation. In contrast, both intervention groups experienced significantly attenuated pressor responses, with peak blood pressure elevations occurring immediately after intubation followed by gradual normalization.

Diltiazem appeared to offer superior attenuation of systolic, diastolic, and mean arterial pressures, particularly at 5 and 10 minutes after intubation. This observation may be explained by its vasodilatory effect mediated through inhibition of calcium influx into vascular smooth muscle, leading to reduced peripheral vascular resistance and arterial pressure.

The present findings are comparable to those reported by Kumar et al., who observed that diltiazem was particularly effective in attenuating blood pressure changes, although it was less effective than esmolol in controlling heart rate.[24] Similarly, Sangawar et al. demonstrated that diltiazem significantly reduced systolic blood pressure following laryngoscopy and endotracheal intubation and concluded that diltiazem effectively blunted the pressor response.

Our findings are also supported by the study conducted by Agrawal et al., who reported that both esmolol and diltiazem attenuated blood pressure responses, although esmolol showed superior heart rate control.[21] Likewise, Parvez Gazi et al. demonstrated that both agents effectively attenuated pressor responses in hypertensive surgical patients, while esmolol remained more effective in controlling pulse rate.[25]

Interestingly, although esmolol attenuated blood pressure significantly compared to placebo, its effect appeared less sustained than diltiazem beyond the

immediate post-intubation period. This finding may be attributable to esmolol's very short elimination half-life, which contributes to rapid onset but comparatively shorter duration of action.[22]

No major adverse effects such as severe bradycardia, hypotension, arrhythmias, or hypersensitivity reactions requiring intervention were observed in the present study. Both drugs were well tolerated and demonstrated acceptable safety profiles when administered at the selected doses.

The findings of this study suggest that drug selection may be individualized according to the predominant hemodynamic concern. In patients where tachycardia and increased myocardial oxygen demand are major concerns, such as ischemic heart disease, esmolol may be preferred due to its superior heart rate control. Conversely, in patients with hypertension or conditions where blood pressure elevation may be particularly harmful, diltiazem may provide better attenuation of pressor responses.

Strengths and Limitations

The strengths of the present study include its prospective, randomized, double-blind design, standardized anaesthetic protocol, and uniform timing of hemodynamic measurements. However, certain limitations should be acknowledged. The study included only ASA physical status I patients, thereby limiting generalizability to high-risk populations. Additionally, the study was conducted at a single centre with a relatively modest sample size, and biochemical markers such as plasma catecholamine levels were not measured. Future multicentric studies involving high-risk surgical populations and varying drug dosages may further clarify the relative efficacy of these agents in attenuating hemodynamic responses during airway instrumentation.

Overall, the present study demonstrated that both intravenous esmolol and diltiazem effectively attenuated the hemodynamic response to laryngoscopy and tracheal intubation compared with placebo. However, esmolol was superior in controlling tachycardia, whereas diltiazem provided comparatively better control of blood pressure parameters, suggesting complementary clinical roles for both agents in perioperative haemodynamic management.

CONCLUSION

Laryngoscopy and endotracheal intubation are associated with significant hemodynamic responses characterized by transient tachycardia and hypertension due to sympathetic stimulation. The present study demonstrated that both intravenous esmolol and intravenous diltiazem effectively attenuated the hemodynamic response to laryngoscopy and tracheal intubation when compared with placebo.

Esmolol was found to be more effective in controlling tachycardia and heart rate changes, whereas diltiazem provided comparatively better attenuation of blood pressure parameters, including systolic, diastolic, and mean arterial pressures. Both drugs exhibited acceptable safety profiles without significant adverse effects.

Therefore, the choice between esmolol and diltiazem may be individualized based on the predominant clinical concern and patient profile. Esmolol may be preferred in situations where tachycardia and increased myocardial oxygen demand are major concerns, while diltiazem may be advantageous in patients requiring greater control of blood pressure response during laryngoscopy and tracheal intubation.

REFERENCES

1. Derbyshire DR, Chmielewski A, Fell D, Vater M, Achola K, Smith G. Plasma catecholamine responses to tracheal intubation. *Br J Anaesth.* 1983;55(9):855–860.
2. Shribman AJ, Smith G, Achola KJ. Cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. Br J Anaesth. 1987;59(3):295–299.
3. Fox EJ, Sklar GS, Hill CH, Villanueva R, King BD. Complications related to the pressor response to endotracheal intubation. Anesthesiology. 1977;47(6):524–525.
4. Stoelting RK. Blood pressure and heart rate changes during short-duration laryngoscopy for tracheal intubation: influence of intravenous lidocaine. Anesth Analg. 1978;57(2):197–199.
5. Kovac AL. Controlling the hemodynamic response to laryngoscopy and endotracheal intubation. J Clin Anesth. 1996;8(1):63–79.
6. Prys-Roberts C, Greene LT, Meloche R, Foex P. Studies of anaesthesia in relation to hypertension: haemodynamic consequences of induction and endotracheal intubation. Br J Anaesth. 1971;43(6):531–547.
7. Helfman SM, Gold MI, Delisser EA, Herrington CA. Which drug prevents tachycardia and hypertension associated with tracheal intubation: lidocaine, fentanyl, or esmolol? Anesth Analg. 1991;72(4):482–486.
8. Kindler CH, Schumacher PG, Schneider MC, Urwyler A. Effects of intravenous lidocaine and/or esmolol on hemodynamic responses to laryngoscopy and intubation: a double-blind controlled clinical trial. J Clin Anesth. 1996;8(6):491–496.
9. Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young WL. Miller's Anesthesia. 8th ed. Philadelphia: Elsevier; 2015.
10. Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 5th ed. New York: McGraw-Hill Education; 2013.
11. Ebert TJ, Pearson JD, Gelman S, Harris C, Bradley EL. Circulatory responses to laryngoscopy: the comparative effects of placebo, fentanyl and esmolol. Can J Anaesth. 1989;36(3):301–306.
12. Figueredo E, Garcia-Fuentes EM. Assessment of the efficacy of esmolol on the haemodynamic changes induced by laryngoscopy and tracheal intubation: a meta-analysis. Acta Anaesthesiol Scand. 2001;45(8):1011–1022.
13. Mikawa K, Nishina K, Maekawa N, Obara H. Comparison of nicardipine, diltiazem, and verapamil for controlling hemodynamic responses to tracheal intubation. Br J Anaesth. 1996;76(2):221–226.
14. Nishikawa T, Namiki A. Attenuation of the pressor response to laryngoscopy and tracheal intubation with intravenous diltiazem. Can J Anaesth. 1989;36(1):1–5.
15. Rathore A, Gupta HK, Tanwar GL, Rehman H. Attenuation of pressure response to laryngoscopy and endotracheal intubation with different doses of esmolol. Indian J Anaesth. 2002;46(6):449–452.
16. Kumar S, Mishra MN, Mishra LS, Bathla S. Comparative study of the efficacy of intravenous esmolol, diltiazem and

magnesium sulphate in attenuating hemodynamic response to laryngoscopy and tracheal intubation. Indian J Anaesth. 2003;47(1):41–44.
17. Agrawal P, Harode D, Saxena KN. Comparative study of intravenous esmolol, lignocaine and diltiazem in attenuating haemodynamic response to laryngoscopy and intubation. Indian J Anaesth. 2010;54(5):426–430.
18. Sarkar A, Khurana P, Bhattacharya D. Attenuation of pressor response: a comparison of diltiazem and esmolol. J Anaesthesiol Clin Pharmacol. 2013;29(2):172–176.
19. Sangawar MA, Medha S. Attenuation of cardiovascular response to endotracheal intubation with diltiazem. Int J Med Res Rev. 2016;4(8):1321–1326.
20. Parvez Gazi M, Islam MM, Hossain MH, Rahman MH. Comparative efficacy of esmolol and diltiazem in attenuation of haemodynamic response to laryngoscopy and intubation. Bangladesh J Med Sci. 2017;16(4):572–578.
21. Bruder N, Ortega D, Granthil C. Consequences and prevention methods of hemodynamic changes during laryngoscopy and intratracheal intubation. Ann Fr Anesth Reanim. 1992;11(1):57–71.
22. Singh H, Vichitvejpaisal P, Gaines GY, White PF. Comparative effects of lidocaine, esmolol, and nitroglycerin in modifying the hemodynamic response to laryngoscopy and intubation. J Clin Anesth. 1995;7(1):5–8.
23. Gupta S, Tank P. A comparative study of efficacy of esmolol and diltiazem in attenuating pressor response to laryngoscopy and endotracheal intubation. J Evol Med Dent Sci. 2014;3(52):12145–12153.
24. Khan RM, Khan TZ, Ahmed A. Comparative evaluation of esmolol and diltiazem in attenuation of cardiovascular response during laryngoscopy and tracheal intubation. Pak J Med Sci. 2015;31(2):321–326.
25. Miller KA, Harkin CP, Bailey PL. Postoperative tracheal extubation. Anesth Analg. 1995;80(1):149–172.