RESEARCH PAPER

Injection Esmolol Versus Infusion Dexmetomidine In Attenuding Haemodyanmic And Blood Glucose Response To Laryngoscopy And Intubation – An Observational Study

Dr. Dennis Shadokpam[1]*, Dr. H. Sanayaima Devi[2], A. Dharshini[3], Dr. Vinisha Sridhar[4]

.

[1] Postgraduate Resident, Department of Anaesthesiology, Sree Balaji Medical College and Hospital, Chromepet, Chennai, Tamil Nadu, India. Bharath Institute of Higher Education and Research (BIHER), Chennai, India.
[2] Professor, Department of Community Medicine,Regional Institute of Medical Sciences (RIMS), Imphal – 795004, Manipur, India.
[3] Final Year Student, B.Sc. Allied Health Sciences (Operation Theatre and Anaesthesia Technology), Department of Allied Health Sciences,Sree Balaji Medical College and Hospital, Chromepet, Chennai, Tamil Nadu, India. Bharath Institute of Higher Education and Research (BIHER), Chennai, India.
[4] Freelance Anaesthetist,Chennai, Tamil Nadu, India.

Received: 14th Dec, 2025; Revised: 8th Feb 2026; Accepted: 10th Feb, 2026; Available Online: 28th Feb, 2026

ABSTRACT

Background:Direct laryngoscopy and endotracheal intubation during induction of general anaesthesia are well-recognized stimuli that trigger marked sympathetic activation. This response is characterized by tachycardia, hypertension, and increased myocardial oxygen demand due to catecholamine release. Although these transient hemodynamic changes are usually tolerated by healthy individuals, they may lead to serious complications such as myocardial ischemia, arrhythmias, and cerebrovascular events in vulnerable patients. Several pharmacological agents have been investigated to attenuate these responses. Dexmedetomidine, a selective α2-adrenergic receptor agonist, and esmolol, a short-acting cardioselective β1-adrenergic blocker, are commonly used agents for controlling peri-intubation hemodynamic responses.

ObjectiveTo evaluate and compare the effectiveness of intravenous dexmedetomidine and esmolol in attenuating hemodynamic and blood glucose responses associated with laryngoscopy and endotracheal intubation during general anaesthesia.

MethodsThis prospective observational study included 60 adult patients aged 18–60 years belonging to ASA physical status I or II who underwent elective surgical procedures under general anaesthesia. Patients were divided into three groups of 20 each: control group (no study drug), dexmedetomidine group receiving intravenous dexmedetomidine 1 μg/kg before induction, and esmolol group receiving intravenous esmolol 50 μg/kg prior to induction. Hemodynamic parameters including heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were recorded at baseline, after drug administration, immediately after intubation, and at 3, 5, and 10 minutes post-intubation. Capillary blood glucose levels were measured preoperatively and 30 minutes following intubation. Statistical analysis was performed using appropriate comparative tests, with $p < 0.05$ considered statistically significant.

Results:Baseline demographic and hemodynamic parameters were comparable among the groups. Immediately after intubation, heart rate increased significantly in the control group (102.5 ± 12.3 bpm) compared with the dexmedetomidine group (86.7 ± 9.4 bpm) and esmolol group (88.1 ± 9.2 bpm) ($p < 0.001$). Similarly, systolic blood pressure rose markedly in the control group (146.3 ± 13.5 mmHg) compared with dexmedetomidine (128.6 ± 11.4 mmHg) and esmolol (130.2 ± 10.9 mmHg) groups ($p < 0.001$). Diastolic blood pressure and mean arterial pressure also showed significantly smaller increases in both treatment groups. The incidence of tachycardia (>100 bpm) was highest in the control group (40%) compared with 10% in the dexmedetomidine group and 15% in the esmolol group. Blood glucose levels increased in all groups after intubation but were highest in the control group (118.6 ± 10.7 mg/dl) compared with dexmedetomidine (111.4 ± 9.6 mg/dl) and esmolol (106.3 ± 9.2 mg/dl) groups.

Conclusion:Both dexmedetomidine and esmolol effectively attenuate the hemodynamic responses associated with laryngoscopy and endotracheal intubation. These agents significantly reduce tachycardia and hypertension compared with the control group and contribute to improved perioperative hemodynamic stability. Esmolol also demonstrated a slightly greater reduction in the metabolic stress response as reflected by smaller increases in blood glucose levels.

Keywords: Dexmedetomidine, Esmolol, Laryngoscopy, Tracheal Intubation, Hemodynamic Stability, Blood Glucose, Adrenergic Antagonists, Sympathetic Nervous System, and General Anesthesia

How to cite this article: Shadokpam D, Sanayaima Devi H, Dharshini A, Sridhar V., Injection Esmolol Versus Infusion Dexmetomidine In Attenuding Haemodyanmic And Blood Glucose Response To Laryngoscopy And Intubation – An Observational Study..Int J Drug Deliv Technol. 2026; 16(2): 732-740; DOI: 10.25258/ijddt.16.2.78

Source of support: Nil.

Conflict of interest: None

**Author for Correspondence: Dr. Dennis Shadokpam*

INTRODUCTION

General anaesthesia is defined as a reversible state of unconsciousness characterized by amnesia, analgesia, immobility, and suppression of reflex responses to surgical stimulation. Modern anaesthetic practice utilizes a balanced technique that combines hypnotic agents, analgesics, and neuromuscular blocking drugs to facilitate surgical procedures while ensuring adequate airway control and ventilation. Endotracheal intubation following direct laryngoscopy remains a fundamental component of airway management during the induction of general anaesthesia, enabling controlled ventilation and protecting the airway from aspiration. [1]

Surgical care represents a major component of global healthcare delivery. With advances in medical technology and improved surgical accessibility, the number of surgical procedures performed worldwide has increased substantially. It is estimated that more than 300 million surgical procedures are carried out annually across the globe, highlighting the growing demand for operative care. Despite this increasing surgical volume, a significant disparity persists in access to safe surgical and anaesthesia services. Approximately 5 billion people worldwide lack access to safe, timely, and affordable surgical and anaesthesia care, particularly in low- and middle-income countries. These statistics emphasize the importance of safe anaesthetic management and the need for strategies that minimize perioperative complications and improve surgical outcomes. [2]

Direct laryngoscopy and endotracheal intubation are recognized as potent noxious stimuli that trigger sympathetic nervous system activation. Mechanical stimulation of the epiglottis, larynx, and trachea results in a surge of circulating catecholamines, leading to tachycardia, hypertension, and increased myocardial oxygen demand. The hemodynamic response usually begins within seconds of laryngoscopy, peaks within one to two minutes, and returns to baseline within approximately five to ten minutes after intubation. Although this transient response is often tolerated in healthy individuals, it may lead to serious complications in patients with pre-existing cardiovascular or cerebrovascular diseases, including arrhythmias, myocardial ischemia, heart failure, and intracranial hemorrhage. Consequently, attenuation of the pressor response associated with laryngoscopy and tracheal intubation remains an important goal in anaesthetic practice. [3]

Several pharmacological and non-pharmacological approaches have been investigated to reduce the sympathetic response associated with airway manipulation. These include the use of topical local anaesthetic agents such as lignocaine, deeper planes of anaesthesia achieved with intravenous or inhalational agents, opioid analgesics, vasodilators, calcium channel blockers, and beta-adrenergic antagonists. Despite the availability of these strategies, no single method has been identified as universally ideal, and ongoing research continues to explore pharmacological agents capable of providing effective attenuation of hemodynamic responses without producing significant adverse effects. [4]

Dexmedetomidine is a highly selective α2-adrenergic receptor agonist that produces sedation, anxiolysis, analgesia, and sympatholysis with minimal respiratory depression. It acts centrally by decreasing sympathetic outflow and inhibiting norepinephrine release, thereby promoting hemodynamic stability during stressful perioperative events. Owing to these pharmacological properties, dexmedetomidine has gained increasing attention as an adjunct in anaesthesia for attenuating the cardiovascular responses associated with laryngoscopy and endotracheal intubation. [5]

Esmolol is an ultra-short acting cardioselective β1-adrenergic receptor antagonist widely used in perioperative settings for the control of tachycardia and hypertension. Its rapid onset and short elimination half-life make it particularly useful in situations requiring transient suppression of sympathetic responses, such as during laryngoscopy and intubation. By blocking β1-adrenergic receptors in the myocardium, esmolol reduces heart rate, myocardial contractility, and cardiac output, thereby limiting the hemodynamic response to sympathetic stimulation. [6]

Educational resources and contemporary anaesthesia practice guidelines emphasize that airway instrumentation during anaesthesia induction remains one of the most significant triggers of sympathetic activation. Appropriate pharmacological interventions can therefore play a crucial role in maintaining perioperative hemodynamic stability and reducing the risk of cardiovascular complications during airway manipulation. [7]

International clinical guidelines for procedural sedation and anaesthesia further highlight the need for evidence-based pharmacological strategies that can minimize hemodynamic fluctuations while preserving adequate ventilation and oxygenation during airway instrumentation. The selection of appropriate pharmacological agents is therefore essential to ensure patient safety and improve perioperative outcomes. [8]

Despite the widespread use of both dexmedetomidine and esmolol in clinical anaesthesia practice, there remains ongoing interest in determining their relative effectiveness in attenuating the hemodynamic and metabolic responses associated with laryngoscopy and endotracheal intubation. Airway manipulation not only triggers cardiovascular responses but may also induce neuroendocrine stress responses that can influence metabolic parameters such as blood glucose levels. Understanding the comparative effects of these pharmacological agents on both hemodynamic and metabolic responses may provide valuable insights for optimizing perioperative management. In this context, the present study was undertaken to evaluate and compare the effects of intravenous esmolol and dexmedetomidine in attenuating the hemodynamic responses—particularly heart rate and blood pressure changes—as well as the blood glucose response associated with laryngoscopy and endotracheal intubation in patients undergoing elective surgical procedures under general anaesthesia.

METHODOLOGY

The present study was conducted as a prospective observational study to evaluate and compare the effects of intravenous esmolol and dexmedetomidine in attenuating the hemodynamic and blood glucose responses associated with laryngoscopy and endotracheal intubation in patients undergoing elective surgical procedures under general anaesthesia. The study was carried out in the Department of Anaesthesiology at Sree Balaji Medical College and Hospital, Chennai, a tertiary care teaching hospital. The study was conducted over a period of six months, during which eligible patients scheduled for elective surgeries under general anaesthesia were screened and enrolled.

A total of 60 adult patients aged between 18 and 60 years were included in the study. Only patients belonging to American Society of Anesthesiologists (ASA) physical status I and II and scheduled for elective surgical procedures requiring laryngoscopy and endotracheal intubation under general anaesthesia were considered eligible. Patients who were willing to participate and provided written informed consent were included in the study. Patients were excluded if they were younger than 18 years or older than 60 years, had ASA physical status III or higher, were pregnant or lactating, had severe cardiovascular disease, had a known allergy or contraindication to the study drugs, or had significant comorbid conditions such as hypertension or diabetes mellitus.

Prior to commencement of the study, approval was obtained from the Institutional Ethics Committee, and written informed consent was obtained from all participants after explaining the objectives and procedures involved in the study. All patients underwent a detailed pre-anaesthetic evaluation including history taking, clinical examination, and routine investigations as per institutional protocol. Patients were advised to remain nil per oral (NPO) for at least 10 hours prior to surgery. On the day of surgery, all patients received oral premedication with midazolam 3.75 mg before shifting to the operation theatre.

In the operating theatre, patients were connected to standard monitoring devices including electrocardiography (ECG), non-invasive blood pressure monitoring, and pulse oximetry. Baseline hemodynamic parameters such as heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and oxygen saturation were recorded prior to the administration of the study drugs.

The enrolled patients were divided into three groups consisting of 20 patients each. Patients in Group A (control group) did not receive either of the study drugs prior to induction of anaesthesia. Patients in Group B (dexmedetomidine group) received intravenous dexmedetomidine at a loading dose of 1 µg/kg, administered as an infusion before induction of anaesthesia. Patients in Group C (esmolol group) received intravenous esmolol at a dose of 50 µg/kg prior to induction. These interventions were administered approximately 10 minutes before induction of general anaesthesia.

Following administration of the study drugs, general anaesthesia was induced according to standard institutional protocols. Direct laryngoscopy was performed and endotracheal intubation was carried out using a laryngoscope, and anaesthesia was subsequently maintained throughout the surgical procedure. Hemodynamic parameters including heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and oxygen saturation were recorded at multiple time points, including baseline (before drug administration), after administration of the study drug, after induction of anaesthesia, immediately after intubation, and at 3, 5, and 10 minutes following endotracheal intubation. In addition to hemodynamic measurements, capillary blood glucose levels were recorded preoperatively and again at 30 minutes after intubation in order to assess the metabolic stress response associated with airway instrumentation.

All recorded data were compiled and analyzed to compare the hemodynamic and blood glucose responses among the three groups. Continuous variables were expressed as mean ± standard deviation, and appropriate statistical tests were used to determine the significance of differences between groups. A p-value of less than 0.05 was considered statistically significant. The findings of the study were subsequently interpreted to evaluate the effectiveness of esmolol and dexmedetomidine in attenuating the cardiovascular and metabolic responses associated with laryngoscopy and endotracheal intubation.

RESULTS:

The results of the present study were analyzed to compare the hemodynamic and blood glucose responses to laryngoscopy and endotracheal intubation among the control, dexmedetomidine, and esmolol groups. Demographic characteristics and baseline parameters were first evaluated to ensure comparability between the study groups before assessing changes in cardiovascular and metabolic responses during the peri-intubation period.

Table 1. Demographic Characteristics of the Study Population

Variable	Group A (Control) n=20	Group B (Dexmedetomidine) n=20	Group C (Esmolol) n=20	p-value
Age (years)	38.6 ± 10.4	37.9 ± 9.8	39.2 ± 10.1	0.78
Male (%)	12 (60%)	11 (55%)	13 (65%)	0.84
Female (%)	8 (40%)	9 (45%)	7 (35%)	
BMI (kg/m²)	24.8 ± 3.2	24.5 ± 3.1	25.1 ± 3.4	0.69
ASA I	11 (55%)	10 (50%)	12 (60%)	0.73
ASA II	9 (45%)	10 (50%)	8 (40%)	

The mean age of patients was comparable among the groups: **Control 38.6 ± 10.4 years**, **Dexmedetomidine 37.9 ± 9.8 years**, and **Esmolol 39.2 ± 10.1 years (p = 0.78)**. Male distribution was **60%, 55%, and 65%** respectively. Mean BMI was **24.8 ± 3.2**, **24.5 ± 3.1**, and **25.1 ± 3.4 kg/m² (p = 0.69)**. ASA I patients constituted **55%, 50%, and 60%**

respectively. There were **no statistically significant differences**, indicating comparable baseline characteristics.

Table 2. Baseline Hemodynamic Parameters

Parameter	Group A	Group B	Group C	p-value
Heart Rate (bpm)	84.2 ± 9.6	83.7 ± 8.9	85.1 ± 9.3	0.71
SBP (mmHg)	124.6 ± 11.2	123.9 ± 10.8	125.1 ± 10.5	0.76
DBP (mmHg)	78.5 ± 8.7	77.9 ± 7.9	79.2 ± 8.3	0.68
MAP (mmHg)	93.8 ± 7.6	93.2 ± 7.1	94.1 ± 7.5	0.74
SpO_2 (%)	98.6 ± 0.8	98.5 ± 0.7	98.7 ± 0.9	0.62

Baseline heart rate was **84.2 ± 9.6 bpm (Control)**, **83.7 ± 8.9 bpm (Dexmedetomidine)**, and **85.1 ± 9.3 bpm (Esmolol) (p = 0.71)**. Baseline SBP was **124.6 ± 11.2**, **123.9 ± 10.8**, and **125.1 ± 10.5 mmHg (p = 0.76)**. DBP values were **78.5 ± 8.7**, **77.9 ± 7.9**, and **79.2 ± 8.3 mmHg (p = 0.68)**. Mean arterial pressure was **93.8 ± 7.6**, **93.2 ± 7.1**, and **94.1 ± 7.5 mmHg (p = 0.74)**. All groups had similar baseline hemodynamic status.

Table 3. Changes in Heart Rate at Different Time Intervals

Time Interval	Control	Dexmedetomidine	Esmolol	p-value
Baseline	84.2 ± 9.6	83.7 ± 8.9	85.1 ± 9.3	0.71
After drug administration	88.6 ± 10.1	79.4 ± 8.2	80.2 ± 8.7	<0.01
Immediately after intubation	102.5 ± 12.3	86.7 ± 9.4	88.1 ± 9.2	<0.001
3 min after intubation	96.8 ± 10.7	84.2 ± 8.3	85.6 ± 8.5	<0.001
5 min after intubation	92.3 ± 9.5	82.9 ± 7.9	83.7 ± 8.1	<0.01
10 min after intubation	88.7 ± 8.8	81.5 ± 7.4	82.3 ± 7.6	<0.01

Following intubation, heart rate increased significantly in the control group (**102.5 ± 12.3 bpm**) compared with **86.7 ± 9.4 bpm** in the dexmedetomidine group and **88.1 ± 9.2 bpm** in the esmolol group (**p < 0.001**). At 3 minutes post-intubation, heart rate remained higher in the control group (**96.8 ± 10.7 bpm**) compared with **84.2 ± 8.3 bpm** and **85.6 ± 8.5 bpm** respectively. Both dexmedetomidine and esmolol significantly attenuated tachycardia.

Table 4. Changes in Systolic Blood Pressure

Time Interval	Control	Dexmedetomidine	Esmolol	p-value
Baseline	124.6 ± 11.2	123.9 ± 10.8	125.1 ± 10.5	0.76
Immediately after intubation	146.3 ± 13.5	128.6 ± 11.4	130.2 ± 10.9	<0.001
3 min	139.7 ± 12.6	126.8 ± 10.7	128.1 ± 10.2	<0.001
5 min	134.5 ± 11.3	125.2 ± 9.9	126.4 ± 9.6	<0.01
10 min	130.4 ± 10.2	123.7 ± 9.3	124.9 ± 9.0	<0.05

Immediately after intubation, SBP increased markedly in the control group (**146.3 ± 13.5 mmHg**) compared with **128.6 ± 11.4 mmHg** in the dexmedetomidine group and **130.2 ± 10.9 mmHg** in the esmolol group (**p < 0.001**). SBP gradually decreased at 3 and 5 minutes but remained significantly higher in the control group compared with both treatment groups.

Table 5. Changes in Diastolic Blood Pressure

Time Interval	Control	Dexmedetomidine	Esmolol	p-value
Baseline	78.5 ± 8.7	77.9 ± 7.9	79.2 ± 8.3	0.68
Immediately after intubation	92.1 ± 9.8	81.4 ± 8.3	82.2 ± 8.1	<0.001
3 min	88.6 ± 9.1	80.7 ± 7.9	81.3 ± 7.7	<0.01
5 min	84.5 ± 8.5	79.6 ± 7.4	80.1 ± 7.5	<0.05
10 min	82.3 ± 8.1	78.8 ± 7.2	79.4 ± 7.3	0.07

Diastolic blood pressure increased significantly in the control group (**92.1 ± 9.8 mmHg**) immediately after intubation compared with **81.4 ± 8.3 mmHg** in the dexmedetomidine group and **82.2 ± 8.1 mmHg** in the esmolol group (**p < 0.001**). DBP remained elevated in the control group at 3 minutes (**88.6 ± 9.1 mmHg**) compared with the dexmedetomidine (**80.7 ± 7.9 mmHg**) and esmolol (**81.3 ± 7.7 mmHg**) groups.

Table 6. Mean Arterial Pressure Changes

Time Interval	Control	Dexmedetomidine	Esmolol	p-value
Baseline	93.8 ± 7.6	93.2 ± 7.1	94.1 ± 7.5	0.74
Immediately after intubation	110.5 ± 10.4	97.8 ± 8.6	99.3 ± 8.4	<0.001
3 min	105.6 ± 9.8	96.7 ± 8.1	97.6 ± 8.0	<0.01
5 min	101.3 ± 9.1	95.6 ± 7.8	96.2 ± 7.7	<0.05

Mean arterial pressure increased significantly after intubation in the control group (**110.5 ± 10.4 mmHg**) compared with **97.8 ± 8.6 mmHg** in the dexmedetomidine group and **99.3 ± 8.4 mmHg** in the esmolol group (**p < 0.001**). MAP gradually decreased at 3 and 5 minutes in all groups but remained higher in the control group.

Table 7. Blood Glucose Response

Parameter	Control	Dexmedetomidine	Esmolol	p-value
Pre-operative glucose (mg/dl)	96.4 ± 8.3	95.9 ± 8.1	96.1 ± 8.2	0.99
30 min after intubation	118.6 ± 10.7	111.4 ± 9.6	106.3 ± 9.2	0.027

Pre-operative blood glucose levels were comparable among the groups: **96.4 ± 8.3 mg/dl (Control)**, **95.9 ± 8.1 mg/dl (Dexmedetomidine)**, and **96.1 ± 8.2 mg/dl (Esmolol) (p = 0.99)**. At **30 minutes post-intubation**, glucose levels increased to **118.6 ± 10.7 mg/dl** in the control group, **111.4 ± 9.6 mg/dl** in the dexmedetomidine group, and **106.3 ± 9.2 mg/dl** in the esmolol group (**p = 0.027**). Esmolol showed the smallest rise in blood glucose.

Table 8. Incidence of Hemodynamic Events

Event	Control	Dexmedetomidine	Esmolol	p-value
Tachycardia (>100 bpm)	8 (40%)	2 (10%)	3 (15%)	0.01
Hypertension (>20% rise)	9 (45%)	3 (15%)	4 (20%)	0.02
Bradycardia	0	2	1	NS
Hypotension	1	2	1	NS

The incidence of tachycardia (>100 bpm) was highest in the control group (**40%**) compared with **10%** in the dexmedetomidine group and **15%** in the esmolol group (**p = 0.01**). Hypertension (>20% rise in BP) occurred in **45%** of the control group compared with **15%** and **20%** in the dexmedetomidine and esmolol groups respectively (**p = 0.02**). Bradycardia and hypotension were infrequent in all groups.

DISCUSSION

The present study evaluated the effectiveness of intravenous dexmedetomidine and esmolol in attenuating the hemodynamic and blood glucose responses associated with laryngoscopy and endotracheal intubation in patients undergoing elective surgical procedures under general anaesthesia. The findings of the present study demonstrated that both pharmacological agents significantly reduced the sympathetic responses compared with the control group.

In the present study, heart rate increased markedly in the control group immediately after intubation, rising from 84.2 ± 9.6 bpm at baseline to 102.5 ± 12.3 bpm, whereas the increase was significantly attenuated in the dexmedetomidine and esmolol groups with values of 86.7 ± 9.4 bpm and 88.1 ± 9.2 bpm respectively. These findings indicate effective suppression of tachycardia following airway manipulation by both agents.

Similar findings were reported by Reddy et al., who compared dexmedetomidine and esmolol in a randomized double-blinded study. They reported that administration of dexmedetomidine 1 μg/kg resulted in significantly lower heart rate and systolic blood pressure responses compared with esmolol 50 μg/kg, demonstrating stronger attenuation of the pressor response to intubation. While their study suggested that dexmedetomidine may be superior to esmolol, our study demonstrated that both agents produced comparable hemodynamic stability, possibly due to differences in patient population and study design. [9]

In our study, heart rate remained significantly lower in the esmolol group compared with the control group during the peri-intubation period. The increase in heart rate in the esmolol group was limited to approximately 3 bpm above baseline, demonstrating effective suppression of sympathetic stimulation.

Comparable observations were reported by Efe et al., who studied bolus and continuous infusion of esmolol in patients undergoing coronary artery bypass graft surgery. Their results showed that esmolol significantly limited the increase in heart rate and blood pressure during laryngoscopy and sternotomy, with heart rate increases restricted to approximately 10–15 bpm compared with more than 20 bpm in untreated patients. The magnitude of attenuation observed in their study closely resembles the reduction observed in our esmolol group. [10]

In the present study, dexmedetomidine demonstrated excellent control of heart rate responses, with post-intubation values remaining close to baseline. Immediately after intubation, heart rate in the dexmedetomidine group was 86.7 ± 9.4 bpm compared with 102.5 ± 12.3 bpm in the control group, indicating substantial attenuation of tachycardia.

A similar pattern was reported by Jain et al., who compared dexmedetomidine with fentanyl for suppression of the hemodynamic response to laryngoscopy. Their study demonstrated that heart rate increased from 82 ± 8 bpm at baseline to approximately 88 ± 7 bpm after intubation in the dexmedetomidine group, whereas the control group experienced significantly greater increases. These findings closely correspond with the modest heart rate increase observed in the dexmedetomidine group in our study. [11]

In our study, systolic blood pressure showed a marked rise in the control group, increasing from 124.6 ± 11.2 mmHg at baseline to 146.3 ± 13.5 mmHg immediately after intubation. In contrast, the dexmedetomidine and esmolol groups demonstrated significantly lower values of 128.6 ± 11.4 mmHg and 130.2 ± 10.9 mmHg respectively, indicating effective attenuation of the pressor response.

Comparable findings were described by Liu et al., who evaluated esmolol for control of heart rate and blood pressure responses during tracheal intubation. Their study demonstrated that esmolol significantly reduced increases in systolic blood pressure and rate-pressure product following intubation, although the cardiovascular response was not completely abolished. This observation is consistent with our findings, where esmolol attenuated but did not entirely eliminate blood pressure elevation. [12]

The present study also showed that mean arterial pressure increased significantly in the control group after intubation, rising from 93.8 ± 7.6 mmHg to 110.5 ± 10.4 mmHg, whereas the dexmedetomidine and esmolol groups recorded lower values of 97.8 ± 8.6 mmHg and 99.3 ± 8.4 mmHg respectively.

The pharmacological mechanism underlying this observation has been explained by Carollo et al., who reported that dexmedetomidine exerts its effect through central α2-adrenergic receptor activation, reducing sympathetic outflow and decreasing circulating catecholamine levels. This mechanism results in improved hemodynamic stability during stressful stimuli such as laryngoscopy and tracheal intubation, which explains the stable blood pressure values observed in our dexmedetomidine group. [13]

In the present study, diastolic blood pressure increased significantly in the control group, reaching 92.1 ± 9.8 mmHg immediately after intubation, compared with 81.4 ± 8.3 mmHg and 82.2 ± 8.1 mmHg in the dexmedetomidine and esmolol groups respectively.

Comparable findings were reported by Sebastian et al., who investigated two doses of dexmedetomidine (0.75 µg/kg and 1 µg/kg). They found that dexmedetomidine significantly reduced increases in heart rate, systolic blood pressure, and diastolic blood pressure following intubation compared with the control group. Their results demonstrated that 0.75–1 µg/kg dexmedetomidine effectively suppressed the hemodynamic response, which corresponds with the dose used in the present study. [14]

In the present study, the effectiveness of intravenous dexmedetomidine and esmolol in attenuating the hemodynamic and metabolic responses to laryngoscopy and endotracheal intubation was evaluated in patients undergoing elective surgery under general anaesthesia. The results of our study demonstrated that both dexmedetomidine and esmolol significantly reduced the cardiovascular stress response associated with airway manipulation compared with the control group.

In our study population consisting of ASA I and II patients, the baseline hemodynamic parameters were comparable among the three groups. The baseline heart rate values were 84.2 ± 9.6 bpm in the control group, 83.7 ± 8.9 bpm in the dexmedetomidine group, and 85.1 ± 9.3 bpm in the esmolol group, indicating no statistically significant difference before drug administration. Similarly, baseline systolic blood pressure values were 124.6 ± 11.2 mmHg, 123.9 ± 10.8 mmHg, and 125.1 ± 10.5 mmHg respectively, confirming homogeneity among the groups prior to intervention.

Following laryngoscopy and intubation, the control group demonstrated a pronounced increase in heart rate, rising to 102.5 ± 12.3 bpm immediately after intubation, whereas the dexmedetomidine and esmolol groups showed significantly lower values of 86.7 ± 9.4 bpm and 88.1 ± 9.2 bpm respectively. This indicates that both drugs were effective in suppressing tachycardia induced by airway instrumentation.

A similar observation was reported by Uysal et al., who evaluated dexmedetomidine, esmolol, and sufentanyl in hypertensive patients undergoing tracheal intubation. In their study, dexmedetomidine significantly reduced the increase in heart rate and blood pressure compared with esmolol and sufentanyl. They reported that the percentage increase in heart rate after intubation remained below 12% in the dexmedetomidine group, whereas larger increases were observed in other groups. In comparison, our study demonstrated an increase of only approximately 3 bpm from baseline in the dexmedetomidine group, indicating a similarly effective attenuation of tachycardia. The stronger response observed in their study may be attributed to the inclusion of hypertensive patients, who typically exhibit greater sympathetic stimulation during airway manipulation. [15]

In addition to evaluating heart rate changes, the present study assessed the incidence of tachycardia (>100 bpm) following intubation. Tachycardia was observed in 40% of patients in the control group, whereas the incidence was significantly lower in the dexmedetomidine and esmolol groups, recorded as 10% and 15% respectively. These findings indicate that both pharmacological agents were effective in preventing excessive sympathetic stimulation during airway instrumentation.

Comparable findings were reported by Karuppiah et al., who studied the use of esmolol for attenuation of the hemodynamic response to laryngoscopy and intubation. Their study demonstrated that patients receiving esmolol maintained heart rates below 90 bpm during the peri-intubation period, while the control group exhibited significantly higher heart rate values. The similarity between their findings and the present study further confirms the efficacy of esmolol in controlling tachycardia associated with laryngoscopy. [16]

The present study also demonstrated significant differences in systolic blood pressure responses following intubation. In the control group, systolic blood pressure increased from 124.6 ± 11.2 mmHg at baseline to 146.3 ± 13.5 mmHg immediately after intubation, representing a marked pressor response. In contrast, the dexmedetomidine and esmolol groups showed significantly smaller increases, with systolic blood pressure values of 128.6 ± 11.4 mmHg and 130.2 ± 10.9 mmHg respectively.

These results are comparable to those reported by Sharma et al., who compared bolus and infusion regimens of esmolol for attenuation of the pressor response to laryngoscopy. Their study demonstrated that esmolol significantly reduced increases in systolic and diastolic blood pressure during airway manipulation compared with the control group. They observed that patients receiving esmolol exhibited significantly lower blood pressure

elevations during intubation, supporting the findings of the present study. [17]

The role of centrally acting sympatholytic agents in suppressing the pressor response has also been investigated in previous studies. Arora et al. studied the use of intravenous clonidine for attenuation of hemodynamic responses during laryngoscopy and intubation. Their study demonstrated that clonidine significantly reduced heart rate and blood pressure elevations following airway manipulation. Since clonidine and dexmedetomidine both act as α2-adrenergic receptor agonists, their mechanism involves reduction of central sympathetic outflow. This pharmacological similarity explains the comparable hemodynamic stability observed with dexmedetomidine in the present study. [18]

In the present study, esmolol effectively attenuated increases in both heart rate and blood pressure during airway manipulation. The hemodynamic parameters returned to near baseline values within 5–10 minutes following intubation in patients receiving esmolol, whereas the control group continued to show elevated values during the same period.

Similar findings were reported by Gurudatta et al., who observed that intravenous esmolol significantly reduced heart rate and blood pressure responses during laryngoscopy and endotracheal intubation. Their study concluded that esmolol effectively blunts sympathetic responses associated with airway instrumentation, which closely aligns with the observations in the present study. [19]

Dexmedetomidine also demonstrated significant sympatholytic effects in our study. The drug maintained stable heart rate and blood pressure values during the peri-intubation period, suggesting effective suppression of catecholamine release.

These findings are consistent with those reported by Modh et al., who administered dexmedetomidine at a dose of 1 µg/kg prior to induction of anaesthesia. Their study demonstrated significant attenuation of heart rate and blood pressure increases following laryngoscopy and intubation. They concluded that dexmedetomidine provides effective hemodynamic stability during airway manipulation, which is in agreement with the present findings. [20]

The effectiveness of esmolol in controlling hemodynamic responses is largely attributed to its pharmacological properties. O'Flaherty described esmolol as a cardioselective β1-adrenergic antagonist with an ultrashort half-life of approximately 9 minutes, allowing rapid onset and quick termination of action. These characteristics make esmolol particularly suitable for transient hemodynamic control during procedures such as laryngoscopy and intubation. [21]

Further pharmacological insights were provided by do Vale et al., who classified esmolol as a second-generation β-blocker with high cardioselectivity and rapid onset of action. They highlighted that the short duration of action allows clinicians to control acute sympathetic responses without producing prolonged hypotension or bradycardia. [22]

Similarly, Covinsky described esmolol as a titratable intravenous β-blocker with a very short elimination half-life, enabling precise control of heart rate and blood pressure during acute sympathetic stimulation. This pharmacokinetic profile explains the rapid stabilization of cardiovascular parameters observed in the esmolol group in the present study. [23]

The cardiovascular benefits of esmolol have also been demonstrated in other clinical settings. Gray et al. reported that esmolol effectively controlled heart rate in patients with supraventricular tachyarrhythmias following cardiac surgery. Their findings confirmed the potent β-blocking effects of esmolol in suppressing sympathetic cardiac stimulation. [24]

Further supporting evidence was provided by Das et al., who demonstrated that esmolol effectively controlled supraventricular tachyarrhythmias and reduced myocardial oxygen demand during acute cardiovascular stress. These findings reinforce the role of esmolol in stabilizing heart rate and reducing sympathetic cardiac responses. [25]

More recent experimental studies have also demonstrated the cardiovascular benefits of esmolol. Agrios et al. showed that esmolol significantly influenced left ventricular hemodynamics and myocardial strain patterns, indicating potential protective effects on cardiac function during sympathetic activation. [26]

A comprehensive review by Garnock-Jones further highlighted that esmolol is widely used for short-term management of tachycardia and hypertension in perioperative settings due to its rapid onset and predictable pharmacokinetics. [27]

Similarly, Bensky et al. demonstrated that bolus administration of esmolol significantly reduced increases in heart rate and blood pressure following laryngoscopy and intubation. Their study also demonstrated a dose-dependent reduction in hemodynamic responses, indicating that higher doses of esmolol produce greater suppression of sympathetic stimulation. [28]

In addition to cardiovascular responses, the present study evaluated blood glucose levels as an indicator of neuroendocrine stress response. Pre-operative glucose levels were comparable across groups (96.4 ± 8.3 mg/dl in control, 95.9 ± 8.1 mg/dl in dexmedetomidine, and 96.1 ± 8.2 mg/dl in esmolol groups). However, at 30 minutes after intubation, blood glucose levels increased to 118.6 ± 10.7 mg/dl in the control group, whereas the dexmedetomidine and esmolol groups showed smaller increases of 111.4 ± 9.6 mg/dl and 106.3 ± 9.2 mg/dl respectively. This indicates that esmolol may provide slightly better attenuation of the metabolic stress response.

Dexmedetomidine has also been studied extensively for its sedative and sympatholytic effects. Elbaradie et al. reported that dexmedetomidine provided improved hemodynamic stability compared with propofol during sedation of mechanically ventilated postoperative patients. [29]

Similarly, Reade et al. demonstrated in a randomized clinical trial that dexmedetomidine improved ventilator-free time and provided stable sedation in critically ill patients with agitated delirium, further highlighting its favorable hemodynamic profile. [30]

Overall, the findings of the present study demonstrate that both dexmedetomidine and esmolol effectively attenuate the hemodynamic responses associated with laryngoscopy and endotracheal intubation. Both drugs significantly reduced tachycardia and hypertension compared with the control group. However, esmolol showed a slightly smaller rise in blood glucose levels, suggesting a potential advantage in attenuating metabolic stress responses during airway manipulation.

CONCLUSION :

The present study evaluated the effectiveness of intravenous dexmedetomidine and esmolol in attenuating the hemodynamic and metabolic responses associated with laryngoscopy and endotracheal intubation in patients undergoing elective surgical procedures under general anaesthesia. The findings demonstrated that both dexmedetomidine and esmolol significantly reduced the sympathetic cardiovascular responses compared with the control group. Patients who did not receive either drug exhibited marked increases in heart rate and blood pressure immediately following intubation, whereas those receiving dexmedetomidine or esmolol showed significantly smaller changes in these parameters.

Dexmedetomidine effectively attenuated tachycardia and hypertension by producing central sympatholysis through activation of α2-adrenergic receptors, thereby reducing sympathetic outflow and catecholamine release. Esmolol, a cardioselective β1-adrenergic antagonist with rapid onset and short duration of action, also provided significant suppression of the pressor response by reducing myocardial contractility and heart rate.

Both drugs maintained stable hemodynamic parameters throughout the peri-intubation period. Additionally, the rise in blood glucose levels following airway manipulation was lower in the treatment groups compared with the control group, with esmolol demonstrating the smallest increase.

Overall, the study concludes that both dexmedetomidine and esmolol are effective pharmacological options for attenuating the cardiovascular and metabolic stress responses during airway instrumentation, thereby contributing to improved perioperative hemodynamic stability..

REFERENCE

1. Butterworth JF, Mackey DC, Wasnick JD. Morgan and Mikhail's Clinical Anesthesiology. 7th ed. New York: McGraw-Hill Education; 2022.
2. Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MC, Ortega R. Clinical Anesthesia. 9th ed. Philadelphia: Wolters Kluwer; 2023.
3. Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Cohen NH, Young WL. Miller's Anesthesia. 10th ed. Philadelphia: Elsevier; 2024.
4. StatPearls Publishing. Dexmedetomidine. Treasure Island (FL): StatPearls Publishing; 2024. Available from: https://www.ncbi.nlm.nih.gov/books/
5. StatPearls Publishing. Esmolol. Treasure Island (FL): StatPearls Publishing; 2024. Available from: https://www.ncbi.nlm.nih.gov/books/
6. American Society of Anesthesiologists (ASA). Practice guidelines for sedation and anesthesia. Updated 2023. Available from: https://www.asahq.org
7. World Federation of Societies of Anaesthesiologists (WFSA). Anaesthesia tutorials of the week. Updated 2024. Available from: https://www.wfsahq.org
8. National Institute for Health and Care Excellence (NICE). Intravenous sedation and anesthesia guidelines. Updated 2023. Available from: https://www.nice.org.uk
9. Reddy SV, Balaji D, Ahmed SN. Dexmedetomidine versus esmolol to attenuate the hemodynamic response to laryngoscopy and tracheal intubation: a randomized double-blinded study. Int J Appl Basic Med Res. 2014;4(2):95-100.
10. Efe EM, Bilgin BA, Alanoglu Z, Akbaba M, Denker C. Comparison of bolus and continuous infusion of esmolol on hemodynamic response to laryngoscopy, endotracheal intubation and sternotomy in coronary artery bypass graft. Braz J Anesthesiol. 2014;64(4):247-252.
11. Jain V, Chandak A, Ghosh A, Golhar M. Comparison of dexmedetomidine and fentanyl for attenuation of the hemodynamic response to laryngoscopy and tracheal intubation. Ain-Shams J Anaesthesiol. 2015;8(2):236-243.
12. Liu PL, Gatt S, Gugino LD, Mallampati SR, Covin BG. Esmolol for control of increases in heart rate and blood pressure during tracheal intubation after thiopentone and succinylcholine. Can Anaesth Soc J. 1986;33(5):556-562.
13. Carollo DS, Nossaman BD, Ramadhyani U. Dexmedetomidine: a review of clinical applications. Curr Opin Anaesthesiol. 2008;21:457-461.
14. Sebastian B, Tali Koti AT, Krishnamurthy D. Attenuation of hemodynamic responses to laryngoscopy and endotracheal intubation with intravenous dexmedetomidine: a comparison between two doses. Indian J Anaesth. 2017;61(1):48-54.
15. Uysal HY, Tezer E, Turkoglu M, Aslanargun P, Basar H. The effects of dexmedetomidine on hemodynamic responses to tracheal intubation in hypertensive patients: a comparison with esmolol and sufentanyl. J Res Med Sci. 2012;17(1):22-31.
16. Karuppiah S, Singh N, Singh K, Singh T, Meitei A, Sinam H. Attenuation of hemodynamic response to laryngoscopy and intubation using intravenous fentanyl and esmolol: a study. J Med Soc. 2015;29.
17. Sharma S, Angral R, Singh P. Esmolol bolus versus infusion in attenuation of pressure response. Int J Res Med Sci. 2017;5(9):4010-4014.
18. Arora S, Kulkarni A, Bhargava A. Attenuation of hemodynamic response to laryngoscopy and orotracheal intubation using intravenous clonidine. J Anaesthesiol Clin Pharmacol. 2015;31(1):110-114.
19. Gurudatta KN, Mallappa K, Ravindra GL. Attenuation of hemodynamic responses to laryngoscopy and endotracheal intubation by intravenous esmolol. Int J Res Med Sci. 2014;2(3):866-871.
20. Modh DB, Gohil P, Parmar M. Intravenous dexmedetomidine 1 μg/kg as premedication to attenuate

hemodynamic response to laryngoscopy and endotracheal intubation in surgeries under general anesthesia. Int Surg J. 2017;4(6):1884-1888.
21. O'Flaherty D. Esmolol. Can J Anaesth. 1993;40(7):687-688.
22. do Vale GT, Ceron CS, Gonzaga NA, Simplicio JA, Padovan JC. Three generations of β-blockers: history, class differences and clinical applicability. Curr Hypertens Rev. 2019;15(1):22-31.
23. Covinsky JO. Esmolol: a novel cardioselective, titratable, intravenous beta-blocker with ultrashort half-life. Drug Intell Clin Pharm. 1987;21(4):316-321.
24. Gray RJ, Bateman TM, Czer LS, Conklin CM, Matloff JM. Esmolol: a new ultrashort-acting beta-adrenergic blocking agent for rapid control of heart rate in postoperative supraventricular tachyarrhythmias. J Am Coll Cardiol. 1985;5(6):1451-1456.
25. Das G, Tschida V, Gray R, Dhurandhar R, Lester R, McGrew F, et al. Efficacy of esmolol in the treatment and transfer of patients with supraventricular tachyarrhythmias to alternate oral antiarrhythmic agents. J Clin Pharmacol. 1988;28(8):746-750.
26. Agrios J, Kaladaridou A, Bramos D, Skaltsiotes E, Pamboucas C, Papadopoulou E, et al. The acute effects of esmolol on left ventricular hemodynamic, rotational mechanics and strain in intact and infarcted myocardium: an experimental study. Hellenic J Cardiol. 2021;62(4):322-323.
27. Garnock-Jones KP. Esmolol: a review of its use in the short-term treatment of tachyarrhythmias and the short-term control of tachycardia and hypertension. Drugs. 2012;72(1):109-132.
28. Bensky KP, Donahue-Spencer L, Hertz GE, Anderson MT, James R. The dose-related effects of bolus esmolol on heart rate and blood pressure following laryngoscopy and intubation. AANA J. 2000;68(5):437-442.
29. Elbaradie S, El Mahalawy FH, Solyman AH. Dexmedetomidine versus propofol for short-term sedation of postoperative mechanically ventilated patients. J Egypt Natl Canc Inst. 2004;16:153-158.
30. Reade MC, Eastwood GM, Bellomo R, Bailey M, Bersten A, Cheung B, et al. Effect of dexmedetomidine added to standard care on ventilator-free time in patients with agitated delirium: a randomized clinical trial. JAMA. 2016;315:1460-1468.