International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2025: 7.089

A Comparative Study of the Efficacy of Intravenous Dexmedetomidine versus Intravenous Esmolol for Attenuation of Cardiovascular Response to Laryngoscopy and Endotracheal Intubation: A Randomized Double-Blind

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Abstract: ***Background: Laryngoscopy and endotracheal intubation trigger a reflex sympathetic discharge leading to tachycardia, hypertension, and arrhythmias. In patients with compromised cardiovascular reserve, these responses can precipitate myocardial ischemia, pulmonary edema, or cerebrovascular events. Pharmacological attenuation of this stress response is therefore essential. Objectives: To compare the efficacy of intravenous (IV) dexmedetomidine (0.6 µg/kg) versus IV esmolol (1 mg/kg) in attenuating hemodynamic responses to laryngoscopy and endotracheal intubation, and to document associated adverse effects. Methods: A randomized double-blind study was conducted on 60 ASA I–II adult patients (18–60 years) scheduled for elective surgery under general anaesthesia. Patients were randomly allocated into two groups of 30 each. Group D received dexmedetomidine 0.6 µg/kg and Group E received esmolol 1 mg/kg, each infused over 30 seconds beginning at the 7th minute of a 10-minute saline drip. Anaesthesia was induced with thiopentone and succinylcholine and intubation was performed at 90 seconds. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and oxygen saturation (SpO_2) were recorded at baseline, induction, and 1, 3, 5, and 10 minutes post-intubation. Adverse effects were monitored throughout the surgery. Results: Both groups were comparable at baseline for age, sex, ASA grade, and hemodynamic parameters. Group D demonstrated significantly lower HR from induction onwards through all post-intubation time points ($P \leq 0.002$ at all intervals). SBP was significantly lower in Group D at 1 minute (113.87 ± 11.92 vs. 127.83 ± 10.73 mmHg; $P < 0.0001$), 3 minutes ($P = 0.002$), and 10 minutes (117.37 ± 9.21 vs. 135.37 ± 12.36 mmHg; $P < 0.0001$) post-intubation. DBP and MAP were also significantly lower in Group D at 1 minute ($P = 0.013$; $P = 0.0005$) and 10 minutes ($P = 0.001$; $P < 0.0001$). SpO_2 was comparable between groups at all time points. No adverse effects were recorded in either group. Conclusion: Intravenous dexmedetomidine (0.6 µg/kg) provides superior attenuation of cardiovascular responses to laryngoscopy and endotracheal intubation compared to IV esmolol (1 mg/kg), maintaining significantly lower HR, SBP, DBP, and MAP across multiple post-intubation time points, without adverse effects.***

Keywords: cardiovascular stress response; dexmedetomidine; esmolol; endotracheal intubation; laryngoscopy; haemodynamic attenuation

1. Introduction

Laryngoscopy and endotracheal intubation are fundamental to the practice of general anaesthesia, yet they reliably provoke a transient but potentially dangerous haemodynamic stress response.[1] Mechanical stimulation of the epipharyngeal and laryngopharyngeal regions triggers reflex sympathetic discharge, causing an acute rise in circulating catecholamines.[2,3] The resulting tachycardia, hypertension, and occasional arrhythmias typically peak within 30 seconds of intubation and generally resolve within 5–10 minutes.[4]

While these haemodynamic changes are usually well-tolerated in healthy individuals, they can be hazardous in patients with coronary artery disease, hypertensive heart disease, valvular disorders, cerebrovascular disease, intracranial vascular abnormalities, or conditions such as pheochromocytoma and pre-eclampsia. In such patients, the surge in myocardial oxygen demand may upset the supply-demand balance, precipitating myocardial ischaemia, acute pulmonary oedema, or cerebrovascular accidents.[5]

A variety of pharmacological strategies have been employed to modify this response. Beta-adrenergic blockers, opioids, calcium channel blockers, lidocaine, magnesium sulphate, and α2-adrenergic agonists have all been studied, each with distinct haemodynamic profiles and side-effect burdens. Among these, esmolol attenuates heart rate by peripheral adrenergic blockade without influencing central sympathetic tone.[6] Dexmedetomidine, a highly selective α2-adrenergic agonist with a selectivity ratio of 1600:1 for the α2 over α1 receptor, acts centrally by decreasing sympathetic outflow from the locus coeruleus, providing anxiolysis, sedation, and sympatholysis with minimal respiratory depression.[7,8]

Several studies have compared these two agents, with most reporting superior haemodynamic control with dexmedetomidine.[9,10] However, dosage protocols have varied widely, and data comparing a lower, potentially safer dose of dexmedetomidine (0.6 µg/kg) against the standard esmolol dose (1 mg/kg) remain limited. The present study was therefore designed to compare IV dexmedetomidine 0.6 µg/kg with IV esmolol 1 mg/kg for attenuation of the cardiovascular response to laryngoscopy and endotracheal intubation in ASA I–II patients who underwent elective surgery.

2. Materials and Methods

Study design and setting

This was a prospective, randomized, double-blind study conducted over 18 months. Institutional ethics committee approval was obtained prior to enrolment, and written informed consent was obtained from all participants.

Participants

Sixty patients of either sex, aged 18–60 years, with American Society of Anesthesiologists (ASA) physical status I or II, who were admitted for elective surgical procedures who required general anaesthesia and orotracheal intubation were enrolled. Patients with Mallampati score I or II were included.[11]

Patients were excluded if they had: known allergy to dexmedetomidine or esmolol; anticipated difficult airway (Mallampati score III–IV); atrioventricular heart block; renal dysfunction (blood urea nitrogen > 25 mg/dl or serum creatinine > 1.5 mg/dl); hepatic dysfunction; or if intubation required more than 60 seconds.

Sample size

Sample size was calculated using the formula $N = (Z_{1-}\alpha/2 + Z_{1-}\beta)^2 \times 4\sigma^2 / (\mu_1 - \mu_2)^2$, with a two-tailed type I error (α) of 0.05 and a power of 80%. Based on an expected difference in mean diastolic blood pressure of 8 mmHg at 5 minutes post-intubation, a minimum of 30 patients per group was required, giving a total of 60 patients.

Randomization and blinding

Patients were allocated to two groups of 30 (Group D and Group E) using a computer-generated random number sequence. One envelope was prepared with 30 labelled Group D and 30 labelled Group E. An anaesthesiologist who was not involved in patient care opened an envelope and prepared the study drug. The primary investigator who performed laryngoscopy and recorded haemodynamic data was blinded to group allocation.

All patients received premedication with oral diazepam 10 mg the night before surgery and 10 mg orally two hours before induction, with a sip of water. In the operating theatre, intravenous access was established and 0.9% normal saline was infused at 10–15 ml/kg/hour. Patients were monitored with ECG, pulse oximetry, and non-invasive blood pressure. A blinded drip of 0.9% normal saline was started at 50 ml/hour. At the 7th minute of the infusion:

- Group D: dexmedetomidine 0.6 µg/kg diluted to 10 ml in 0.9% saline, administered IV over 30 seconds.
- Group E: esmolol 1 mg/kg diluted to 10 ml in 0.9% saline, administered IV over 30 seconds.

The saline drip was continued for 10 minutes and then discontinued. Patients were pre-oxygenated with 100% oxygen via face mask for 3 minutes. General anaesthesia was induced with IV thiopentone 5 mg/kg (to loss of eyelash reflex), and neuromuscular blockade was achieved with succinylcholine 1–2 mg/kg. Ninety seconds after succinylcholine administration, intubation was performed with an appropriate sized cuffed endotracheal tube using a Macintosh laryngoscope by the same experienced anaesthesiologist throughout the study. Anaesthesia was maintained with oxygen, nitrous oxide, isoflurane, and intermittent atracurium.

Outcome measures

The primary outcomes were changes in HR, SBP, DBP, and MAP at baseline, induction, and 1, 3, 5, and 10 minutes post-intubation. The secondary outcome was the incidence of adverse effects (bradycardia defined as HR < 50 beats/min; hypotension defined as a fall in MAP > 30% from baseline; nausea; vomiting). Bradycardia was managed with IV atropine 0.6 mg and hypotension with IV mephentermine 6 mg.

Statistical analysis

Data were entered into Microsoft Excel and analysed using SPSS version 28.0 (IBM Corp., Armonk, NY, USA). Categorical variables were compared using the Chi-square test or Fisher's exact test as appropriate. Continuous variables were expressed as mean ± standard deviation (SD) and compared using the independent samples t-test. A P value < 0.05 was considered statistically significant.

3. Results

Demographic and baseline characteristics

Sixty patients were enrolled and completed the study—30 in each group. Baseline demographic characteristics (figure 1 and 2), ASA status (figure 3), and duration of surgery were comparable between the two groups (Table 1).

Table 1: Demographic and baseline characteristics of the D and E groups

Variable	Group D (Dexmedetomidine) n = 30	Group E (Esmolol) n = 30	P value
Age (years), Mean ± SD	36.23 ± 11.44	35.07 ± 13.66	0.7
Sex			
Male	19 (63.3)	13 (43.3)	0.1
Female	11 (36.7)	17 (56.7)	
ASA Grade			
I	6 (20)	10 (33.3)	0.2
II	24 (80)	20 (66.7)	
Duration of surgery (hours), Mean ± SD	2.3 ± 0.71	2.17 ± 0.75	0.5

Values expressed as mean ± SD or n (%). P values from independent t-test or Fisher's exact test.

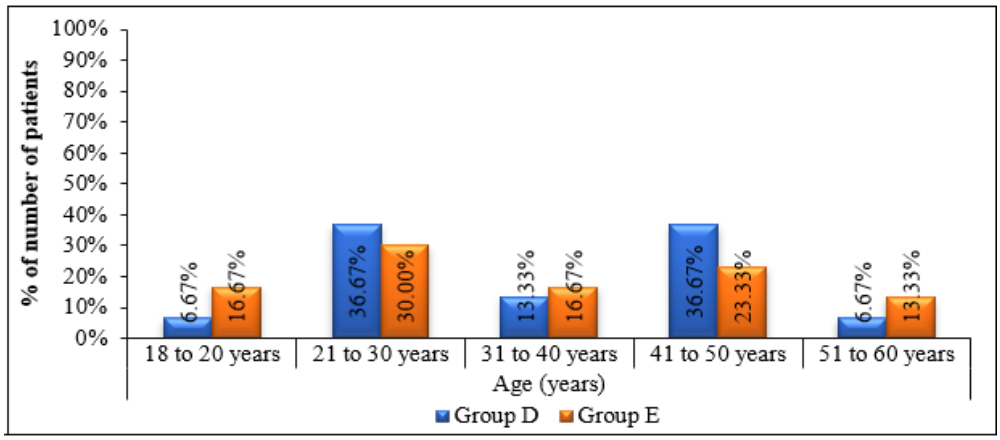

Figure 1: Comparison of age between group D and E

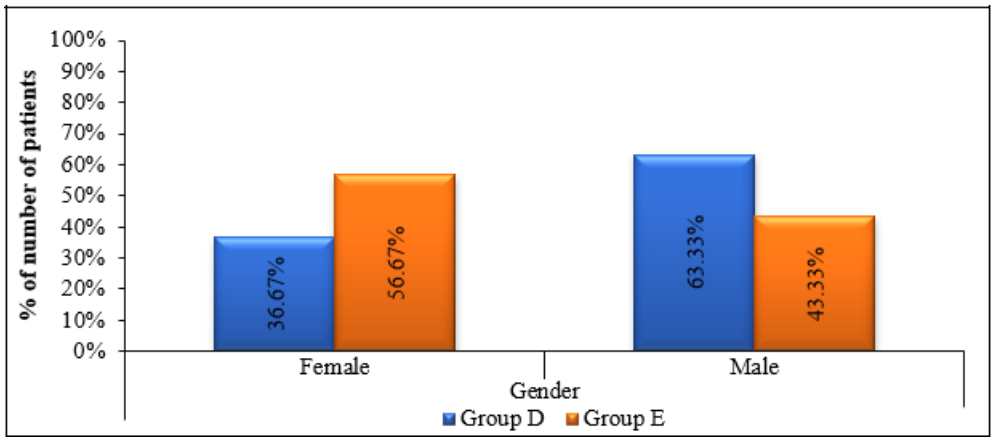

Figure 2: Comparison of gender between group D and E

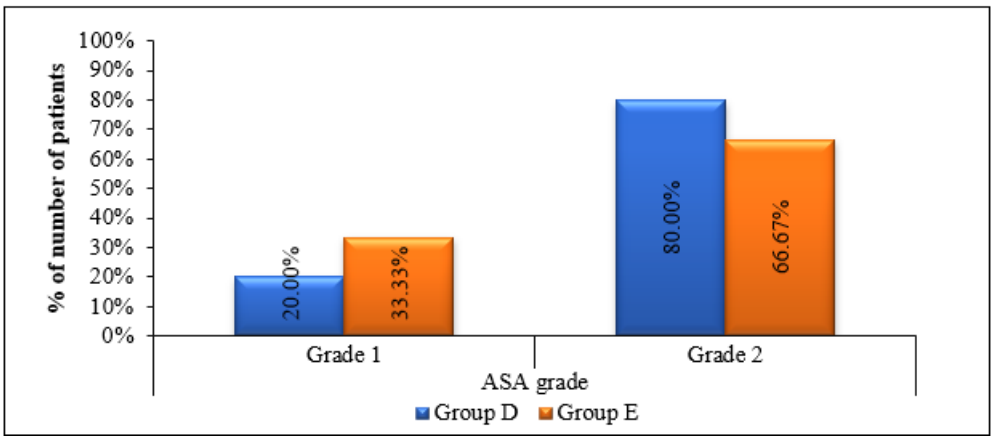

Figure 3: Comparison of ASA grade between group D and E

Heart rate

Baseline HR was similar in both groups (79.63 ± 12.93 vs. 80.23 ± 11.07 beats/min; P = 0.848). Following study drug administration, Group D showed a significant reduction in HR at induction (70.3 ± 7.85 vs. 79.33 ± 11.53 beats/min; P = 0.0008). Group D maintained significantly lower HR at all post-intubation time points compared to Group E (Table 2). In contrast, HR in Group E remained stable or showed a mild increase following intubation.

Table 2: Comparison of heart rate (beats/min) between Group D and Group E

Time Point	Group D (Dexmedetomidine) n = 30 Mean ± SD	Group E (Esmolol) n = 30 Mean ± SD	P value
Baseline	79.63 ± 12.93	80.23 ± 11.07	0.8
At induction	70.3 ± 7.85	79.33 ± 11.53	<0.001*
1 min post-intubation	70.73 ± 8.36	79.87 ± 11.9	0.001*
3 min post-intubation	68.37 ± 7.12	79.57 ± 10.97	<0.001*
5 min post-intubation	69.4 ± 9.69	79.3 ± 11.51	<0.001*
10 min post-intubation	68.57 ± 10.46	77.97 ± 12.13	0.002*

Values expressed as mean ± SD. *P < 0.05, statistically significant. P values by independent t-test

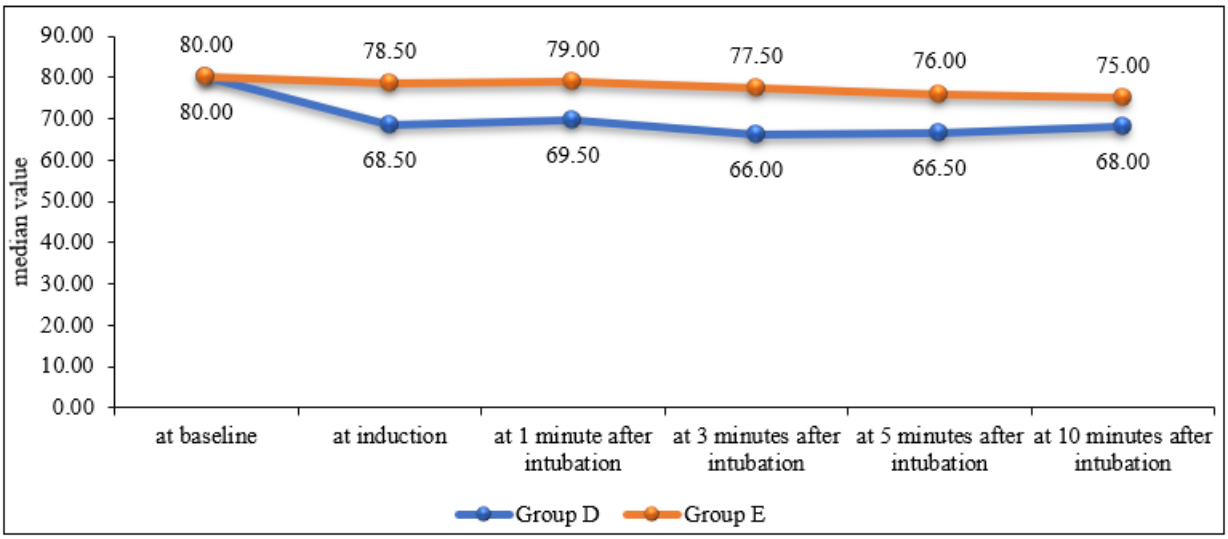

Figure 4: Comparison of heart rate (per minute) between group D and E

Systolic blood pressure

Baseline SBP was comparable between Group D and Group E (117.57 ± 11.96 vs. 117.93 ± 10.69 mmHg; P = 0.901). Following intubation, SBP rose in Group E while remaining stable or decreasing in Group D. Significant differences were observed at 1 minute, 3 minutes, and 10 minutes post-intubation (Table 3). At the 10-minute mark, Group E showed an SBP of 135.37 ± 12.36 mmHg compared to 117.37 ± 9.21 mmHg in Group D (P < 0.0001).

Table 3: Comparison of systolic blood pressure (mmHg) between Group D and Group E

Time Point	Group D (Dexmedetomidine) n = 30 Mean ± SD	Group E (Esmolol) n = 30 Mean ± SD (mmHg)	P value
Baseline	117.57 ± 11.96	117.93 ± 10.69	0.901
At induction	115.97 ± 10.61	118.4 ± 9.96	0.363
1 min post-intubation	113.87 ± 11.92	127.83 ± 10.73	<0.001*
3 min post-intubation	114.6 ± 13.21	123.53 ± 6.43	0.002*
5 min post-intubation	116.83 ± 9.88	119.13 ± 7.38	0.311
10 min post-intubation	117.37 ± 9.21	135.37 ± 12.36	<0.001*

*Values expressed as mean ± SD. *P < 0.05, statistically significant. P values by independent t-test.*

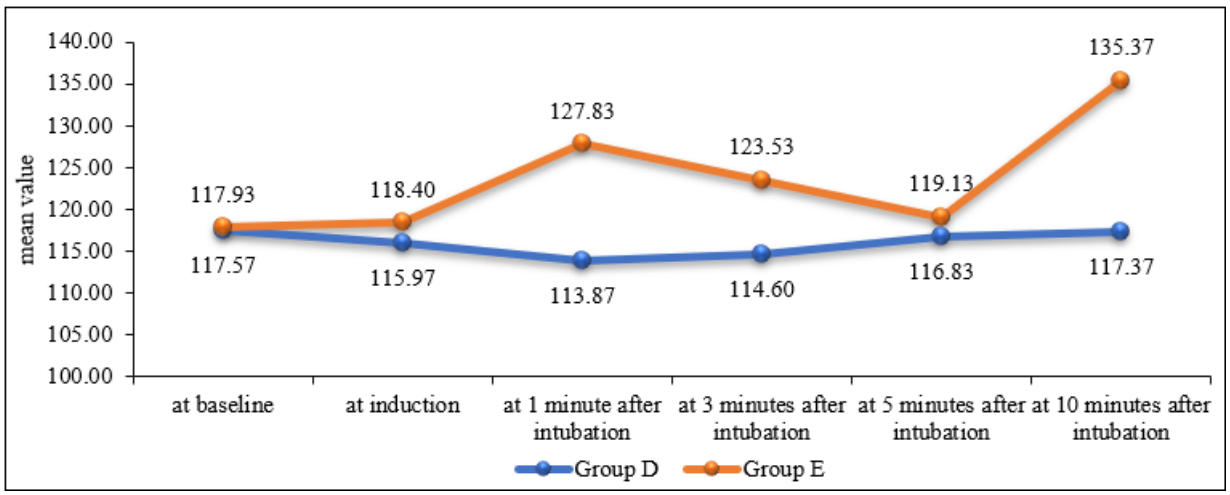

Figure 5: Comparison of systolic blood pressure (mmHg) between group D and E

Diastolic blood pressure

DBP was similar at baseline (77.4 ± 9.47 vs. 74.73 ± 6.88 mmHg; P = 0.217) and at induction (P = 0.986). Significant differences emerged at 1 minute post-intubation (74.33 ± 7.98 vs. 79.13 ± 6.43 mmHg; P = 0.013) and at 10 minutes (76.17 ± 7.42 vs. 84 ± 9.95 mmHg; P = 0.001), both times Group D showed a lower DBP which was stable. DBP values at 3 and 5 minutes post-intubation were comparable between the groups (Table 4, figure 6).

Table 4: Comparison of diastolic blood pressure (mmHg) between Group D and Group E

Time Point	Group D (Dexmedetomidine) n = 30, Mean ± SD (mmHg)	Group E (Esmolol) n = 30 Mean ± SD (mmHg)	P value
Baseline	77.4 ± 9.47	74.73 ± 6.88	0.2
At induction	75.1 ± 8.11	75.13 ± 6.16	0.9
1 min post-intubation	74.33 ± 7.98	79.13 ± 6.43	0.01*
3 min post-intubation	75.03 ± 9.54	75.47 ± 5.66	0.8
5 min post-intubation	75.4 ± 6.59	75.33 ± 5.81	0.9
10 min post-intubation	76.17 ± 7.42	84 ± 9.95	0.001*

*Values expressed as mean ± SD. *P < 0.05, statistically significant. P values by independent t-test.*

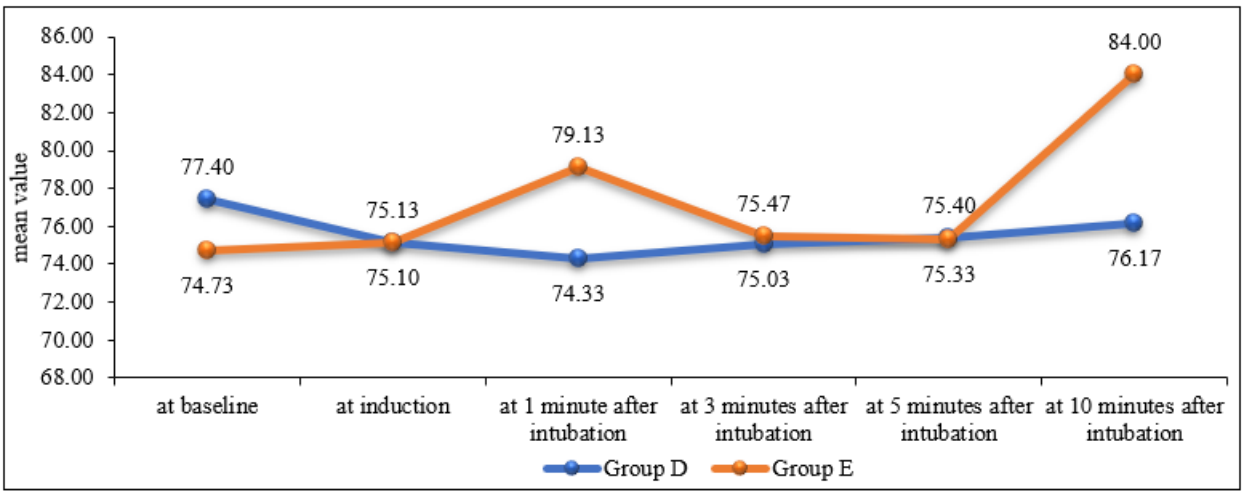

Figure 6: Comparison of diastolic blood pressure (mmHg) between group D and E

Mean arterial pressure

MAP values were comparable at baseline and induction in both groups. Following intubation, Group D exhibited significantly lower MAP at 1 minute (87.43 ± 8.58 vs. 94.83 ± 6.78 mmHg; P = 0.0005) and at 10 minutes (89.63 ± 7.59 vs. 100.07 ± 10.15 mmHg; P < 0.0001). MAP at 3 and 5 minutes post-intubation did not differ significantly between groups (Table 5, figure 7).

Table 5: Comparison of mean arterial pressure (mmHg) between Group D and Group E

Time Point	Group D (Dexmedetomidine) n = 30, Mean ± SD (mmHg)	Group E (Esmolol) n = 30 Mean ± SD (mmHg)	P value
Baseline	90.6 ± 9.6	89.43 ± 7.25	0.5
At induction	88.07 ± 9.19	89.43 ± 6.4	0.5
1 min post-intubation	87.43 ± 8.58	94.83 ± 6.78	<0.001*
3 min post-intubation	88.03 ± 10.34	91.13 ± 4.97	0.1
5 min post-intubation	88.9 ± 7.2	89.57 ± 5.68	0.6
10 min post-intubation	89.63 ± 7.59	100.07 ± 10.15	<0.001*

*Values expressed as mean ± SD. *P < 0.05, statistically significant. P values by independent t-test*

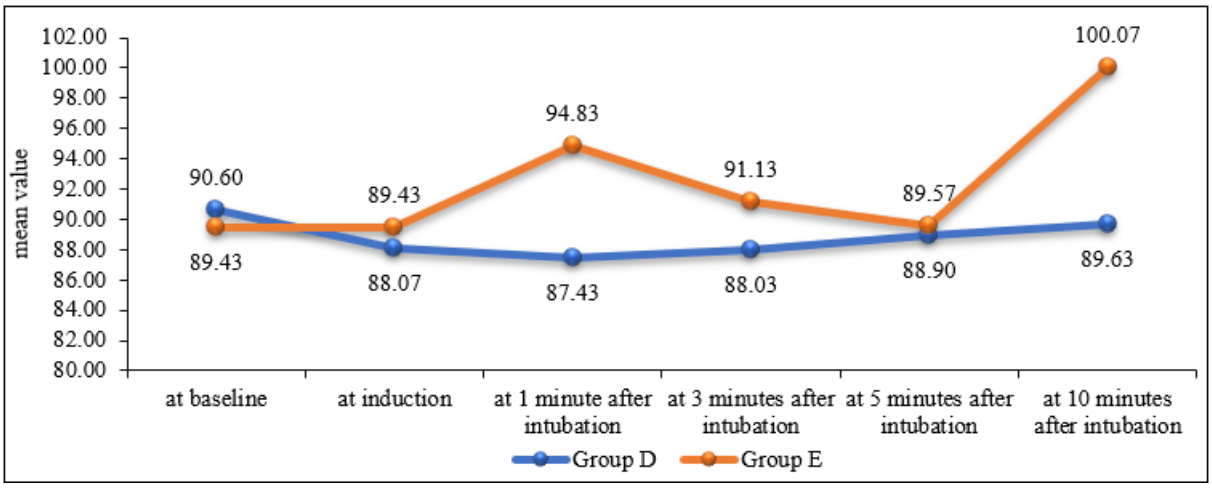

Figure 7: Comparison of mean arterial pressure (mmHg) between group D and E

Oxygen saturation

SpO_2 remained within the normal range (>97%) in both groups at all time points and did not differ significantly between the two groups throughout the observation period (Table 6, figure 8).

Table 6: Comparison of oxygen saturation (SpO_2, %) between Group D and Group E

Time Point	Group D (Dexmedetomidine) n = 30, Mean ± SD	Group E (Esmolol) n = 30 Mean ± SD (%)	P value
Baseline	97.5 ± 1.04	97.8 ± 1.16	0.2
At induction	97.63 ± 0.81	97.9 ± 0.99	0.2
1 min post-intubation	97.77 ± 1.38	98.3 ± 0.6	0.05
3 min post-intubation	98.03 ± 0.76	98.3 ± 0.6	0.1
5 min post-intubation	98 ± 0.79	98.27 ± 0.69	0.1
10 min post-intubation	98 ± 0.74	98.37 ± 0.81	0.07

Values expressed as mean ± SD. P values by independent t-test. No significant difference at any time point

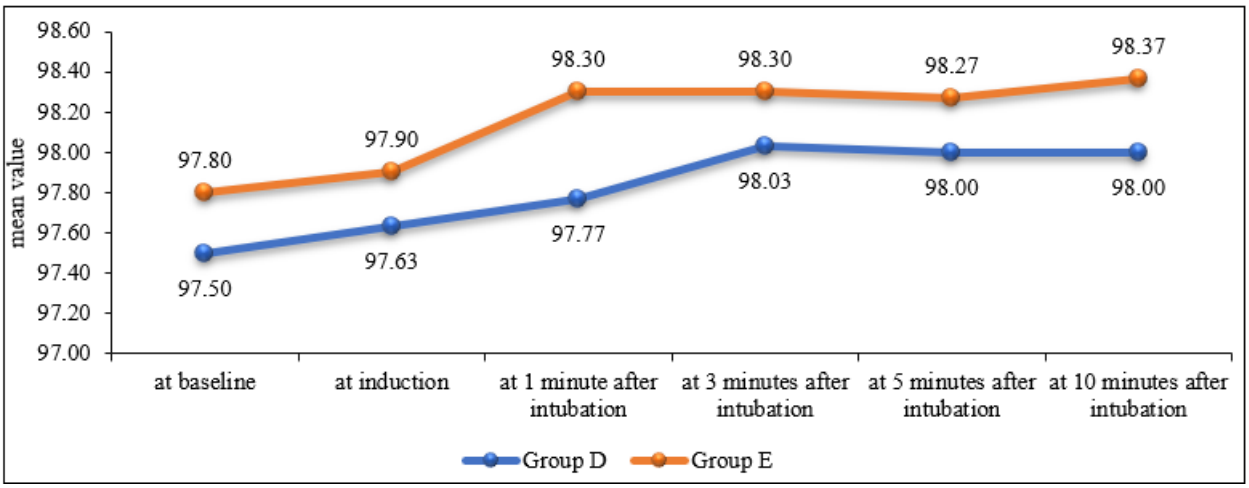

Figure 8: Comparison of SpO_2 (%) between group D and E

Adverse effects

No adverse effects, including bradycardia, hypotension, nausea, or vomiting, were recorded in either group during the study period. (Figure 9)

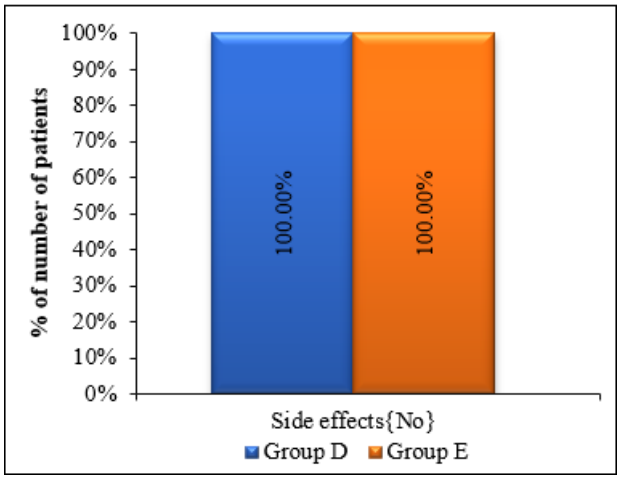

Figure 9: Comparison of side effects between group D and E

4. Discussion

The present randomized double-blind study compared IV dexmedetomidine 0.6 µg/kg with IV esmolol 1 mg/kg for attenuation of the cardiovascular stress response to laryngoscopy and endotracheal intubation. The two groups were well-matched at baseline for all demographic, clinical, and haemodynamic variables, ensuring that observed outcome differences were attributable to the study drugs.

The principal finding was that Group D demonstrated significantly lower HR from the time of induction through all post-intubation time points up to 10 minutes, whereas Group E showed comparatively higher heart rates throughout, reflecting incomplete attenuation of sympathetic drive. These findings are consistent with prior comparative studies. One study reported significantly lower HR in the dexmedetomidine group (1 µg/kg) compared to esmolol (1.5 mg/kg) at all post-intubation time points in neurosurgical patients.[12] A meta-analysis similarly found dexmedetomidine superior to esmolol in controlling post-intubation HR.[13] The mechanistic basis lies in the distinction between central and peripheral adrenergic inhibition: dexmedetomidine suppresses sympathetic outflow from the locus coeruleus globally,[12,14] while esmolol competitively blocks β_1 receptors on the heart without influencing central adrenergic tone,[15,16] resulting in more limited and shorter-lived heart rate control.

Regarding SBP, Group D maintained lower values at 1, 3, and 10 minutes post-intubation, with the most marked divergence at 10 minutes. The sustained elevation of SBP in Group E beyond the immediate post-intubation period is consistent with esmolol's ultra-short half-life (~9 minutes),[16] which results in waning efficacy within the observation window. Reddy SV et al. similarly found significantly higher SBP in the esmolol group from the time of intubation onwards.[14] Singh et al. reported an higher SBP in the esmolol group versus with dexmedetomidine at 1 minute post-intubation a

larger absolute difference than observed in the present study, likely attributable to the higher dexmedetomidine dose (1 µg/kg) used in that study.[17] The dose of 0.6 µg/kg employed here achieved clinically meaningful attenuation while potentially reducing the risk of adverse hemodynamic effects. DBP and MAP showed a similar pattern, with Group D recording significantly lower values at 1 minute and 10 minutes post-intubation.[12] The absence of mean between the 2 groups at 3 and 5 minutes likely reflects a nadir in the pressor response in both groups as the acute intubation stimulus subsides; the re-emergence of divergence at 10 minutes highlights dexmedetomidine's sustained sympatholytic effect beyond the waning of esmolol's β_1 blockade.[12,14]

SpO_2 was well-maintained in both groups throughout, confirming the established respiratory safety profile of dexmedetomidine at sedative doses. These findings are consistent Damarla R et al., who similarly reported no significant SpO_2 differences between dexmedetomidine and esmolol groups and no episodes of oxygen desaturation.[18]

Neither group experienced bradycardia, hypotension, nausea, or vomiting. This is in contrast to some prior studies that reported bradycardia requiring atropine in the dexmedetomidine group, particularly at doses of 1 µg/kg.[12,19,20] The use of the lower dose of 0.6 µg/kg in the present study may account for this favourable safety profile.[10] The absence of adverse effects in the esmolol group was consistent with its established safety record.[14]

Limitations of the study include its single-centre design, which may limit generalisability; the absence of a placebo control arm; hemodynamic follow-up restricted to 10 minutes post-intubation; and enrolment limited to ASA I–II patients. Plasma catecholamine levels were not measured. Future studies should examine dexmedetomidine in higher-risk patients (ASA III–IV), include longer hemodynamic monitoring, and assess postoperative recovery parameters such as sedation scores and extubation time.

5. Conclusion

Intravenous dexmedetomidine 0.6 µg/kg provides superior attenuation of the cardiovascular stress response to laryngoscopy and endotracheal intubation compared to IV esmolol 1 mg/kg in ASA I–II patients undergoing elective surgery. Dexmedetomidine maintained significantly lower HR, SBP, DBP, and MAP at multiple post-intubation time points without compromising oxygenation or producing adverse hemodynamic effects. These findings support the use of IV dexmedetomidine as a safe and effective option for blunting intubation-related cardiovascular responses.

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