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Research Article

A PROSPECTIVE RANDOMIZED STUDY TO COMPARE THE EFFICACY OF TWO DIFFERENT DOSES OF INTRAVENOUS DEXMEDETOMIDINE IN ATTENUATING HEMODYNAMIC RESPONSES DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

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ABSTRACT

Objectives: The objective of this study was to compare the efficacy of two different doses of intravenous dexmedetomidine (0.75 mcg/kg vs. 1 mcg/kg) in attenuating the hemodynamic responses during laryngoscopy and endotracheal intubation in adult patients undergoing elective surgeries under general anesthesia. The study aimed to evaluate the optimal dose that balances effective cardiovascular stabilization with minimal adverse effects.

Methods: This prospective randomized study included 60 adult patients (American Society of Anesthesiologists I and II) aged 18–60 years undergoing elective surgeries under general anesthesia. Patients were randomly allocated into two groups: Group A (n=30) received dexmedetomidine 0.75 mcg/kg IV, and Group B (n=30) received dexmedetomidine 1 mcg/kg IV as an infusion in 100 mL normal saline over 15 min 30 min before induction. Hemodynamic parameters (heart rate [HR], systolic blood pressure, diastolic blood pressure, and mean arterial pressure [MAP]) were recorded at baseline before drug administration, at 15 and 30 min following administration of the study drug until induction, after induction, during laryngoscopy and intubation, and subsequently at 1, 3, 5, and 7 min after intubation. Post-operative sedation was assessed using the Ramsay Sedation Score.

Results: Both groups showed significant attenuation of hemodynamic responses during laryngoscopy and endotracheal intubation. Group B (1 mcg/kg) demonstrated significantly better control of HR and blood pressure during laryngoscopy and intubation compared to Group A (0.75 mcg/kg). The mean percentage increase in HR from baseline during laryngoscopy and intubation was 11.8±3.2% in Group A compared to 5.8±2.0% in Group B ($p<0.001$). Similarly, the mean percentage increase in MAP was 9.7±2.8% in Group A versus 4.5±1.8% in Group B ($p<0.001$). Group B showed a higher incidence of bradycardia (20.0% vs. 6.7%) and hypotension (10.0% vs. 0.0%) compared to Group A, though these were easily managed. Post-operative sedation scores were significantly higher in Group B for the first 90 min after extubation.

Conclusion: Dexmedetomidine at a dose of 1 mcg/kg IV provides more effective attenuation of hemodynamic responses to laryngoscopy and endotracheal intubation compared to 0.75 mcg/kg. However, it is associated with a higher incidence of bradycardia and hypotension, requiring vigilant monitoring. The choice of dose should be individualized based on the patient's cardiovascular status and clinical requirements.

Keywords: Dexmedetomidine, Hemodynamic response, Laryngoscopy, Endotracheal intubation, General anesthesia.

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INTRODUCTION

General anesthesia has revolutionized surgical procedures by inducing a state of controlled unconsciousness, making patients insensitive to pain and unaware of the events occurring during surgery. It is typically achieved through a combination of induction agents, intravenous sedatives, analgesics, and maintenance with volatile anesthetics [1,2]. However, the process of securing the airway through direct laryngoscopy and endotracheal intubation is associated with significant sympathetic stimulation.

This sympathetic discharge in response to laryngoscopy and intubation leads to increased plasma catecholamine concentrations, resulting in elevated arterial blood pressure, increased heart rate (HR), and higher oxygen consumption [3]. The hemodynamic response is initiated within seconds of direct laryngoscopy, further increases with the placement of the endotracheal tube, peaks within 1–2 min, and typically returns to baseline within 5 min [4]. While these changes are usually transient and well-tolerated by healthy individuals, they can precipitate adverse events in patients with pre-existing cardiovascular or cerebrovascular diseases, potentially leading to myocardial ischemia, ventricular dysrhythmias, ventricular failure, pulmonary edema, or cerebrovascular accidents [5].

Various pharmacological agents have been investigated to attenuate these stress responses, including lignocaine, opioids, nitroglycerine, calcium channel blockers such as diltiazem, and β-blockers such as esmolol [1,6]. Each has demonstrated varying degrees of efficacy, but many are associated with side effects or incomplete attenuation of the hemodynamic response.

Dexmedetomidine has emerged as a promising agent in this context. As a highly selective, short-acting $\alpha 2$-adrenoreceptor agonist, it possesses sedative, analgesic, and anxiolytic properties without causing respiratory depression, making it an ideal agent for premedication [7]. Through pre-synaptic activation of $\alpha 2$-receptors in the locus coeruleus, dexmedetomidine inhibits noradrenaline release, producing sedation, and hypnosis. Post-synaptic activation of $\alpha 2$-receptors in the central nervous system decreases sympathetic activity, resulting in bradycardia and hypotension [8,9]. These pharmacological properties make dexmedetomidine particularly valuable for attenuating the hemodynamic response to laryngoscopy and intubation.

While multiple studies have demonstrated the efficacy of dexmedetomidine in this setting, there is ongoing debate regarding the optimal dosage that provides adequate hemodynamic stability

with minimal side effects. Sebastian *et al*. found that 0.75 mcg/kg was more effective than 0.5 mcg/kg in attenuating the stress response to laryngoscopy and intubation [10]. Yildiz *et al*. demonstrated that a single pre-induction dose of 1 mcg/kg dexmedetomidine reduced anesthetic requirements by 39% for thiopental and 92% for sevoflurane while effectively blunting hemodynamic responses [11]. Other studies have used doses ranging from 0.5 to 2 mcg/kg, with varying results [12-14].

Given this variation in dosing strategies and the need to balance efficacy with safety, our study aimed to compare the effectiveness of two clinically relevant doses of dexmedetomidine (0.75 mcg/kg vs. 1 mcg/kg) in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation in patients undergoing elective surgeries under general anesthesia.

MATERIALS AND METHODS

Study design and setting

This prospective randomized study was conducted in the Department of Anaesthesiology at Dhiraj Hospital, S.B.K.S. Medical Institute and Research Centre, Vadodara, Gujarat, India, after obtaining approval from the Institutional Ethics Committee (IEC approval number: SVIEC/ON/MEDI/BNPG21/D22001). The study was conducted over a period of 12 months from July 2023 to June 2024.

Sample size calculation

The sample size was calculated based on previous studies that reported a mean difference in HR of 8 beats/min between different doses of dexmedetomidine with a standard deviation of 7.5. Using a power of 90% and a significance level of 5%, the minimum required sample size was determined to be 28 patients per group. To account for potential dropouts, we included 30 patients in each group, resulting in a total sample size of 60 patients.

The inclusion criteria for the study required patients to provide written informed consent, be between 18 and 60 years of age, and be classified as the American Society of Anesthesiologists (ASA) physical status I or II. Eligible participants were those scheduled for elective surgeries under general anesthesia.

Patients were excluded if they were unwilling to participate, had a body mass index of 30 kg/m^2 or greater, a history of muscular disorders, myositis, or any local infection, cardiovascular diseases, arrhythmias, heart blocks, or were taking beta-blockers. Additional exclusion criteria included cerebrovascular, hepatic, or renal diseases, psychiatric illness, known allergy to α-2 adrenergic agonists, as well as pregnant and lactating women.

Study procedure

A total of 60 adult patients scheduled for elective surgeries under general anesthesia were enrolled in this study after obtaining written informed consent. Patients were randomly allocated using computer-generated random numbers into two groups of 30 patients each:

- Group A: Patients received injcetion dexmedetomidine 0.75 mcg/kg intravenously as an infusion in 100 mL normal saline over a period of 15 min and the infusion was completed 30 min before induction of general anesthesia
- Group B: Patients received injcetion dexmedetomidine 1.0 mcg/kg intravenously as an infusion in 100 mL normal saline over a period of 15 min and the infusion was completed 30 min before induction of general anesthesia.

Pre-operative assessment

All patients underwent a thorough pre-anesthetic check-up 1 day before surgery to assess their eligibility for the study. This included a detailed history, general physical examination, systemic examination, and airway assessment. All routine blood examination was performed.

Patients were advised to remain nil per oral for 6 h for solids and 2 h for clear liquids prior to surgery.

Anesthetic technique

On the day of surgery, patients were shifted to the pre-operative area one hour before the scheduled operation time. Standard monitors, including pulse oximeter, non-invasive blood pressure, and electrocardiogram (ECG), were attached, and baseline hemodynamic parameters were recorded. An 18-gauge peripheral venous cannula was secured, and intravenous Ringer's Lactate solution was administered as maintenance fluid (80 mL/h).

The study drug (dexmedetomidine) was prepared by diluting the appropriate dose (based on the patient's weight) in 100 mL normal Saline. The drug was administered as an infusion over 30 min before induction of general anesthesia by an anesthesiologist who was not involved in the subsequent management or data collection.

After the completion of dexmedetomidine infusion, patients were transferred to the operating room and connected to a multichannel monitor for continuous recording of HR, non-invasive systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), ECG, and oxygen saturation (SpO_2).

Patients were premedicated with injcetion glycopyrrolate 0.004 mg/kg IV, injcetion ondansetron 0.1 mg/kg IV, and injcetion tramadol 2 mg/kg IV. After pre-oxygenation with 100% oxygen for 3 min, anesthesia was induced with injcetion propofol 2 mg/kg IV. Once bag-mask ventilation was confirmed to be adequate, injcetion succinylcholine 2 mg/kg IV was administered to facilitate endotracheal intubation.

Direct laryngoscopy was performed using a Macintosh laryngoscope, and the trachea was intubated with an appropriate-sized cuffed endotracheal tube. The position of the tube was confirmed by bilateral chest auscultation and capnography. Anesthesia was maintained with O_2 and N_2O in a 1:1 ratio along with isoflurane using a circle system. Neuromuscular blockade was achieved with injcetion atracurium at a loading dose of 0.5 mg/kg IV followed by maintenance doses of 0.1 mg/kg IV. Patients were mechanically ventilated to maintain normocapnia (end-tidal CO_2 35–40 mmHg).

Hemodynamic monitoring

Hemodynamic parameters, including HR, SBP and DBP, MAP, and oxygen saturation (SpO_2), were carefully recorded at several key intervals throughout the perioperative period. Measurements were taken at baseline before drug administration, at 15 and 30 min following administration of the study drug until induction, after induction, during laryngoscopy and intubation, and subsequently at 1, 3, 5, and 7 min after intubation.

Intraoperative hypotension (defined as SBP <80 mmHg) was treated initially with a bolus of 200 mL Ringer's Lactate solution. If hypotension persisted, incremental doses of injcetion mephentermine 6 mg IV were administered. Bradycardia (defined as HR <50 beats/min) was treated with injcetion atropine 0.6 mg IV. Any persistent increase in SBP >150 mmHg was treated with injcetion esmolol 1.5 mg/kg IV.

At the end of surgery, neuromuscular blockade was reversed with injcetion neostigmine 0.05 mg/kg IV and injcetion glycopyrrolate 0.008 mg/kg IV. Patients were extubated when they met the standard extubation criteria.

Post-operative assessment

Postoperatively, sedation status was assessed using the Ramsay Sedation Score (RSS) at 30-min intervals until a score of ≤2 was achieved. Post-operative complications, including nausea, vomiting, respiratory depression, and dryness of the mouth, were also noted.

Statistical analysis

Data were analyzed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as

mean±standard deviation, while categorical variables were presented as frequency and percentage. The normality of data was tested using the Shapiro–Wilk test. A $p<0.01$ was considered highly statistically significant. A $p<0.05$ was considered statistically significant for all tests. Given the exploratory nature of the multiple time-point comparisons, adjustments for multiple testing were not performed, and results should be interpreted with this in mind.

For between-group comparisons of numerical variables, the independent samples t-test or Mann–Whitney U test was used as appropriate. For within-group comparisons, repeated measures analysis of variance or the Friedman test was used. For categorical variables, the Chi-square test or Fisher's exact test was applied. A $p<0.05$ was considered statistically significant.

RESULTS

A total of 60 patients were enrolled in the study, with 30 patients in each group. All patients completed the study protocol without any dropouts.

Demographic characteristics

The demographic profiles of patients in both groups were comparable, with no statistically significant differences observed in age, gender distribution, weight, ASA physical status, or duration of surgery ($p>0.05$) (Table 1).

Hemodynamic parameters

The hemodynamic responses at various time points are presented in Table 2. The hemodynamic trends are graphically represented in Figs. 1-4. Fig. 1 depicts heart rate trends. Fig. 2 shows systolic blood pressure trends. Fig. 3 illustrates diastolic blood pressure trends. Fig. 4 displays mean arterial pressure trends. Both groups showed effective attenuation of hemodynamic responses, with Group B demonstrating superior control. Statistical analysis revealed significant differences between groups at multiple time points. At intubation, the mean HR in Group B (82.70±6.46 bpm) was significantly lower than in Group A (90.00±6.21 bpm) ($p<0.001$). Similarly, at 1 min post-intubation, Group B maintained better control (78.60±6.15 bpm vs. 87.87±6.06 bpm, $p<0.001$).

Table 1: Demographic characteristics of study population

Parameter	Group A (0.75 mcg/kg) (n=30)	Group B (1.0 mcg/kg) (n=30)	p-value
Age (years), mean±SD	41.27±12.36	39.63±8.47	0.542
Gender distribution (%)			
Male	13 (43.3)	17 (56.7)	0.309
Female	17 (56.7)	13 (43.3)	
Weight (kg), mean±SD	67.43±8.21	68.97±9.15	0.487
ASA physical status (%)			
ASA I	26 (86.7)	20 (66.7)	0.061
ASA II	4 (13.3)	10 (33.3)	
Duration of Surgery (min), mean±SD	115.80±18.34	119.83±15.67	0.367

Data presented as mean±standard deviation or number (percentage).
ASA: American Society of Anesthesiologists, SD: Standard Deviation. $p<0.05$ considered statistically significant

Post-operative parameters

Post-operative sedation assessment revealed different recovery profiles between the groups (Table 3).

Complications

Group B (1.0 mcg/kg) showed a higher incidence of bradycardia (20.0% vs. 6.7%) and hypotension (10.0% vs. 0.0%) compared to Group A, with the differences being statistically significant ($p<0.05$). All episodes of bradycardia and hypotension were successfully managed with appropriate interventions as per protocol. No patients in either group experienced arrhythmias, dryness of mouth, or respiratory depression.

Oxygen saturation

Oxygen saturation remained stable throughout the study in both groups, with no significant differences observed (all values maintained at 99-100%).

DISCUSSION

In the present prospective randomized study demonstrated clear dose-dependent efficacy of dexmedetomidine in attenuating hemodynamic responses to laryngoscopy and endotracheal intubation. The key findings indicate that while both 0.75 mcg/kg and 1.0 mcg/kg doses effectively blunt the pressor response, the higher dose provides significantly superior hemodynamic control, albeit with increased manageable side effects.

The demographic profiles of both groups were comparable in terms of age, gender, weight, ASA physical status, and duration of surgery. This ensures that the observed differences in hemodynamic parameters can be attributed to the different dexmedetomidine doses rather than underlying patient characteristics, strengthening the validity of the present study findings and allowing for more robust conclusions.

The hemodynamic response to laryngoscopy and endotracheal intubation remains a critical concern in anesthetic practice, particularly in patients with cardiovascular comorbidities. Our results reveal that the 1.0 mcg/kg dose provided superior HR control, with a mean percentage increase of only 5.8±2.0% compared to 11.8±3.2% with the 0.75 mcg/kg dose ($p<0.001$). This finding establishes a clear dose–response relationship that has important clinical implications.

These findings align closely with Singh *et al*., who reported that patients receiving 0.5 μg/kg dexmedetomidine showed a mean HR increase of 7.05 bpm (6.99%) at 1 min post-intubation, while those receiving 1 μg/kg showed only 1.58 bpm (1.74%) increase ($p=0.01$) [15]. The superior attenuation within the present study 1 mcg/kg dose (5.8% increase) compared to their 0.5 μg/kg dose (6.99% increase), supports

Table 2: Hemodynamic parameters at various time points

Time point	Heart rate (bpm)		Systolic BP (mmHg)		Diastolic BP (mmHg)		MAP (mmHg)	
	Group A	Group B	Group A	Group B	Group A	Group B	Group A	Group B
Baseline	78.63±5.76	79.37±6.58	127.23±7.84	128.47±7.56	80.13±5.83	80.63±5.62	95.83±6.21	96.57±5.87
After 15 min of drug	76.07±5.28	69.27±5.64	120.80±7.24	116.73±7.31	75.97±5.53	73.67±5.37	90.87±5.84	88.03±5.62
After 30 min of drug	71.13±4.92	61.77±4.80	114.83±6.86	108.37±6.74	72.33±5.25	69.27±5.10	86.50±5.57	82.33±5.39
After induction	66.77±4.62	58.50±4.32	108.67±6.53	102.80±6.38	68.63±4.96	64.70±4.78	81.97±5.28	77.43±5.16
During L&I	90.00±6.21	82.70±6.46	139.93±8.14	131.83±7.92	90.03±6.25	84.37±5.98	106.70±6.47	100.17±6.25
1 min post-intubation	87.87±6.06	78.60±6.15	135.67±7.94	125.63±7.53	87.33±6.13	80.73±5.73	103.47±6.32	95.70±6.13
3 min post-intubation	83.70±5.79	73.10±5.72	129.07±7.57	118.90±7.16	82.60±5.83	76.03±5.42	98.10±6.03	90.20±5.89
5 min post-intubation	79.03±5.43	66.87±5.21	123.80±7.25	112.70±6.74	78.67±5.54	71.27±5.16	93.73±5.79	85.07±5.62
7 min post-intubation	74.37±5.12	62.43±4.87	119.27±6.98	106.47±6.37	75.00±5.30	67.90±4.96	89.70±5.58	80.70±5.39

BP: Blood pressure, MAP: Mean arterial pressure

Table 3: Post-operative sedation assessment using Ramsay Sedation score

Time after extubation	Group A (0.75 mcg/kg) (n=30)	Group B (1.0 mcg/kg) (n=30)	p-value
Immediately	2	3	<0.05*
30 min	2	2	NS
60 min	2	2	NS
90 min	1	2	NS
120 min	1	1	NS

*Data presented as median. NS: Not significant. p<0.05 *considered statistically significant. Ramsay Sedation Score: 1*=Anxious/agitated; 2=Cooperative/tranquil; 3=Responds to commands only; 4=Brisk response to stimulus; 5=Sluggish response to stimulus; 6=No response

the dose–response relationship.

Sebastian *et al.* provided detailed comparative data showing baseline HR of 83.30±4.35 bpm in their 0.5 µg/kg group increasing to 74.57±3.13 bpm at intubation, actually demonstrating a decrease rather than an increase, with the 0.75 µg/kg dose completely obtunding the response [10]. In contrast, their control group showed an increase from 84.93±4.13 to 97.60±10.38 bpm, representing a 15% increase.

Kabara *et al.* found significant differences between 0.5 µg/kg and 0.75 µg/kg doses, with HR at 1 min post-intubation being 86.40±4.92 bpm versus 82.67±5.34 bpm, respectively (p=0.006), demonstrating approximately 4.3% better control with the higher dose [16]. This incremental improvement between 0.5 and 0.75 µg/kg supports the present study's finding of further benefit when comparing 0.75 and 1 mcg/kg.

Jain *et al.* under BIS monitoring reported even more pronounced effects, with their 1 µg/kg group showing HR decreasing from baseline to 70.93±4.13 bpm at intubation, while the control group increased from 89.03±11.90 to 97.60±10.38 bpm (p<0.001) [17]. The use of BIS monitoring in their study, ensuring adequate depth of anesthesia, may explain the more profound HR suppression compared to in the present study findings.

Kumari *et al.* similarly reported that preoperative dexmedetomidine significantly attenuated hemodynamic responses to direct laryngoscopy and tracheal intubation [20].

The SBP response in our study showed marked dose-dependent attenuation, with both groups demonstrating increases from baseline, but Group B showing significantly better control throughout the perioperative period.

Singh *et al.* reported that SBP increased by 3.38 mmHg (2.57%) in their 0.5 µg/kg group, while the 1 µg/kg group showed a decrease of 8.03 mmHg (5.7%) from baseline (p=0.01) [15]. This contrasts with the present study findings, where both groups showed increases, albeit significantly less in the 1 mcg/kg group, suggesting possible methodological differences or patient population variations.

Kabara *et al.* (2024) demonstrated SBP at 1 min post-intubation of 132.33±7.88 mmHg with 0.5 µg/kg versus 127.23±5.96 mmHg with 0.75 µg/kg (p=0.006), representing approximately 4% better control with the higher dose [16]. At 5 min, the difference persisted with 122.97±6.95 versus 112.87±5.85 mmHg (p=0.001), showing sustained benefit of the higher dose.

Jain *et al.* provided comprehensive data showing baseline SBP of 121.37±10.06 mmHg increasing to 131.63±5.81 mmHg in controls (8.4% increase, p<0.001), while their 0.5 µg/kg group increased to 130.80±8.41 mmHg (p=0.034) and the 1 µg/kg group maintained significantly lower values throughout [17]. The propofol requirement in their study decreased from 120.75±14.21 mg in controls to

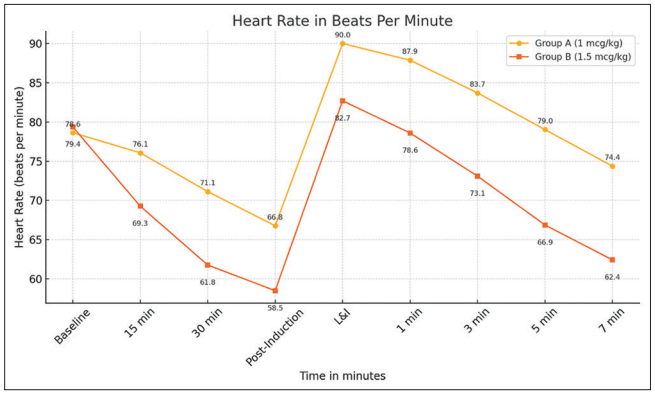

Fig. 1: Line graph showing heart rate trends over time for both groups. The graph displays mean heart rate (bpm) on the y-axis and time points on the x-axis, with Group A (0.75 mcg/kg) represented by a blue line with circle markers and Group B (1.0 mcg/kg) represented by a red line with square markers. Error bars represent standard error of mean. The graph demonstrates the superior attenuation of heart rate response in Group B, particularly during and after laryngoscopy and intubation

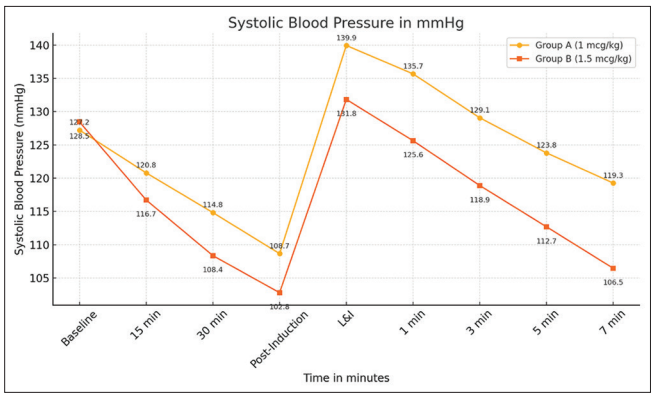

Fig. 2: Line graph showing systolic blood pressure trends over time for both groups. The graph displays mean systolic blood pressure (mmHg) on the y-axis and time points on the x-axis, with Group A (0.75 mcg/kg) shown in blue with circle markers and Group B (1.0 mcg/kg) in red with square markers. Error bars represent standard error of mean. The graph demonstrates better control of the pressor response in Group B throughout the perioperative period

80.50±10.61 mg with 0.5 µg/kg and 68.75±10.42 mg with 1 µg/kg (p<0.001), demonstrating significant anesthetic-sparing effects.

The MAP response in our study revealed clinically significant differences, with a 9.7±2.8% increase in Group A versus 4.5±1.8% in Group B (p<0.001), confirming superior hemodynamic stability with the higher dose.

Uysal *et al.* demonstrated that in hypertensive patients, dexmedetomidine was as effective as esmolol and sufentanil in attenuating hemodynamic responses [21].

Agrawal *et al.* reported detailed MAP changes, with their control group showing an increase of 3.25 mmHg (3.26%) while the 1 µg/kg dexmedetomidine group showed a decrease of 3.05 mmHg (2.93%) from baseline (p=0.01) [19]. The difference in absolute values between studies may reflect variations in baseline blood pressure or anesthetic technique.

Bhagat *et al.* demonstrated remarkably stable hemodynamics with 1 µg/

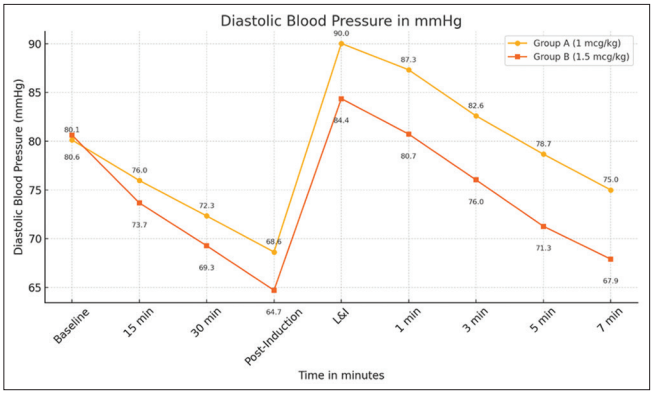

Fig. 3: Line graph showing diastolic blood pressure trends over time for both groups. The graph displays mean diastolic blood pressure (mmHg) on the y-axis and time points on the x-axis. Group A (0.75 mcg/kg) is represented by a blue line with circle markers and Group B (1.0 mcg/kg) by a red line with square markers. Error bars represent standard error of mean

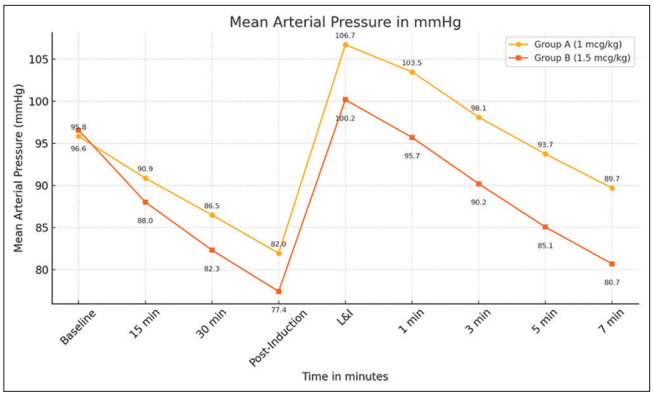

Fig. 4: Line graph showing mean arterial pressure trends over time for both groups. The graph displays mean arterial pressure (MAP) (mmHg) on the y-axis and time points on the x-axis. Group A (0.75 mcg/kg) is shown in blue with circle markers and Group B (1.0 mcg/kg) in red with square markers. Error bars represent standard error of mean. The graph clearly demonstrates the dose-dependent effect of dexmedetomidine in attenuating the MAP response to laryngoscopy and intubation

kg dexmedetomidine during laparoscopic surgery, with MAP increasing by only 2.1% in the dexmedetomidine group versus 8.4% in controls ($p<0.0001$) despite the additional stress of pneumoperitoneum [18]. Their study also showed a 36% reduction in MAC requirement (0.76±0.24 vs. 1.12±0.07, $p<0.0001$) and 43.99% reduction in isoflurane consumption (6.91 ml vs. 12.39 ml, $p<0.0001$).

Jain *et al*. reported baseline MAP of 91.90±7.92 mmHg increasing to 98.86±4.69 mmHg at intubation in controls, while the 1 µg/kg group showed an insignificant increase to 92.93±6.80 mmHg ($p=0.173$) [17]. The superior MAP control with 1 µg/kg in their study using BIS monitoring supports the present study findings of better hemodynamic stability with higher doses.

The DBP findings in our study demonstrated similar dose-dependent patterns. Singh *et al*. reported DBP increases of 3.64 mmHg (4.28%) with 0.5 µg/kg versus a minimal decrease of 0.25 mmHg (0.28%) with 1 µg/kg ($p=0.01$) [15]. Kabara *et al*. found DBP at 1 min post-intubation of 84.43±4.71 mmHg with 0.5 µg/kg versus 80.83±3.74 mmHg with 0.75 µg/kg ($p=0.001$), with the difference persisting at 5 min (77.00±4.01 vs. 72.13±4.11 mmHg, $p=0.001$) [16].

Swarnamba *et al.* found dexmedetomidine superior to clonidine in attenuating hemodynamic responses to laryngoscopy and intubation [22].

The anesthetic-sparing properties of dexmedetomidine demonstrated clear dose-dependence across multiple studies, supporting our findings of superior efficacy with higher doses. Yildiz *et al*. reported dramatic reductions with 1 µg/kg dexmedetomidine: thiopental requirements decreased by 39% and sevoflurane by 92%, with fentanyl requirements of 74.20±10.53 µg versus 84.00±27.04 µg in placebo ($p<0.05$) [11].

Seangrung *et al.* compared dexmedetomidine with IV lidocaine combined with propofol and found dexmedetomidine provided superior hemodynamic stability [23].

Bhagat *et al*. quantified the volatile anesthetic sparing more precisely, showing isoflurane dial settings of 1.0±0.11% with dexmedetomidine versus 1.8±0.13% in controls ($p<0.0001$), representing a 44% reduction [18]. The mean isoflurane consumption calculated by Dion's formula was 6.91 mL versus 12.39 mL, a 43.99% reduction with associated cost savings despite the expense of dexmedetomidine.

Kunisawa *et al.* demonstrated that dexmedetomidine suppresses BP decrease during anesthetic induction while blunting cardiovascular response to intubation [24].

The incidence of adverse effects in our study showed clear dose-dependency, with bradycardia occurring in 20% of the 1 mcg/kg group versus 6.7% in the 0.75 mcg/kg group. This aligns with the literature. Bhagat *et al*. reported 8.33% incidence of bradycardia requiring atropine with 1 µg/kg dexmedetomidine [18]. Sebastian *et al*. reported no bradycardia or hypotension with doses up to 0.75 µg/kg, supporting the better safety profile of lower doses [10].

Regarding recovery profiles, our findings of prolonged sedation with the higher dose align with previous studies. Yildiz *et al*. found Steward scores >6 at 5 min in 56% of dexmedetomidine patients versus only 4% of controls ($p<0.05$), though sedation scores remained ≥4 in all dexmedetomidine patients at 10 min ($p<0.05$) [11]. This biphasic recovery pattern, with initial rapid emergence followed by sustained sedation, mirrors the present study findings of prolonged elevated sedation scores for 90 min in the 1 mcg/kg group.

Agrawal *et al.* confirmed the efficacy of single premedication dose of IV dexmedetomidine in attenuating pressor response to laryngoscopy and intubation [25].

Clinical Implications

The comprehensive evidence from our study and the supporting literature establishes several important clinical principles for dexmedetomidine use in attenuating hemodynamic responses to laryngoscopy and intubation:

First, there is a clear dose–response relationship, with 1.0 mcg/kg providing superior hemodynamic control compared to 0.75 mcg/kg. This comes at the cost of increased but manageable side effects, including bradycardia and hypotension. Second, the choice of dose should be individualized based on patient factors. For patients with significant cardiovascular comorbidities where tight hemodynamic control is paramount, the 1.0 mcg/kg dose may be preferred. For healthier patients or when rapid recovery is prioritized, 0.75 mcg/kg offers effective attenuation with fewer side effects. Third, the anesthetic-sparing effects of dexmedetomidine, particularly at higher doses, have important implications for reducing overall anesthetic exposure and potentially improving recovery profiles. Finally, appropriate monitoring and preparedness to manage bradycardia and hypotension are essential when using dexmedetomidine, particularly at doses of 1.0 mcg/kg or higher.

CONCLUSION

Both doses of dexmedetomidine (0.75 mcg/kg and 1 mcg/kg) effectively attenuated the hemodynamic response to laryngoscopy and endotracheal intubation in patients undergoing elective surgeries under general anesthesia. However, the higher dose (1 mcg/kg) provided significantly better control of HR and blood pressure during these stimulating procedures. This improved efficacy was associated with a higher incidence of bradycardia and hypotension, although these side effects were generally mild and easily managed.

The choice of dexmedetomidine dose should be individualized based on the patient's cardiovascular status, the anticipated difficulty of laryngoscopy and intubation, and the clinical setting. For patients with significant cardiovascular disease or those in whom a robust hemodynamic response to laryngoscopy and intubation could be particularly detrimental, the higher dose (1 mcg/kg) may be preferred, provided that close hemodynamic monitoring and prompt intervention for side effects are available. For patients with less cardiovascular reserve or in settings where extended post-operative sedation might be undesirable, the lower dose (0.75 mcg/kg) might be more appropriate.

AUTHOR CONTRIBUTIONS

Dr. Sara Mary Thomas: Conceptualization, methodology, supervision, manuscript review, and editing. Dr. Harsh Rathod: Data collection, analysis, manuscript writing, and revision. Dr. Kalpesh Patil: Data collection, statistical analysis, manuscript review. Dr. Jigisha Mehta: Methodology, data interpretation, manuscript review.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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