

Original Research Article

TO COMPARE THE EFFICACY OF 1MCG/KG AND 2 MCG/KG DOSES OF INTRANASAL DEXMEDETOMIDINE FOR ATTENUATION OF HEMODYNAMIC RESPONSES TO LARYNGOSCOPY AND INTUBATION IN ADULTS

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ABSTRACT

Background: Laryngoscopy and endotracheal intubation are associated with sympathetic stimulation, resulting in transient increases in heart rate and blood pressure. Dexmedetomidine, a selective alpha-2 adrenergic agonist, provides sedation and sympatholysis. The aim is to compare the efficacy and safety of intranasal dexmedetomidine 1 µg/kg and 2 µg/kg in attenuating haemodynamic responses to laryngoscopy and endotracheal intubation in adults.

Materials and Methods: This prospective, randomized, double-blinded study was conducted in the Department of Anaesthesiology, G S Medical College and Hospital, Pilkhuwa, Hapur, Uttar Pradesh. Ninety patients aged 18–60 years, belonging to ASA physical status I and II, were randomly allocated into two groups of 45 each. Group A received intranasal dexmedetomidine 1 µg/kg, while Group B received 2 µg/kg, administered 45 minutes before anaesthetic induction. Heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were recorded at baseline and at specified intervals following intubation.

Results: Heart rate was significantly lower in Group B at 5 minutes (79.27 ± 10.19 versus 84.50 ± 8.84 ; $p = 0.020$), 10 minutes ($p = 0.033$), and 15 minutes ($p = 0.034$). No significant intergroup differences were observed in systolic, diastolic, or mean arterial pressure. Adverse-effect distributions differed significantly ($p = 0.02$), whereas recovery times were comparable ($p = 0.62$).

Conclusion: Intranasal dexmedetomidine 2 µg/kg provided greater heart rate attenuation at later post-intubation intervals without significantly altering blood pressure or recovery time compared with 1 µg/kg. However, adverse-effect profiles differed between the groups.

Keywords: Intranasal dexmedetomidine; Haemodynamic response; Laryngoscopy; Endotracheal intubation; General anaesthesia.

INTRODUCTION

Laryngoscopy and endotracheal intubation are essential components of general anaesthesia, providing a secure airway and facilitating controlled ventilation during surgical procedures. However, these interventions are associated with considerable physiological stress due to mechanical stimulation of the laryngeal and tracheal structures. This stimulation activates the sympathetic nervous system, resulting in

the release of catecholamines and subsequent cardiovascular responses characterized by tachycardia, hypertension, and increased myocardial oxygen demand. Although these haemodynamic alterations are generally transient, exaggerated responses may produce serious complications, particularly in patients with cardiovascular or cerebrovascular disorders. Effective attenuation of the sympathetic response during airway instrumentation is therefore an important

consideration in anaesthetic management. Maintaining cardiovascular stability throughout induction and intubation contributes to patient safety and reduces the potential risk of perioperative complications.^[1] The haemodynamic response to laryngoscopy and endotracheal intubation is influenced by several factors, including the duration of laryngoscopy, intensity of airway stimulation, depth of anaesthesia, pre-existing cardiovascular conditions, and pharmacological agents administered before induction. Various medications, including opioids, beta-adrenergic blockers, vasodilators, local anaesthetics, and alpha-2 adrenergic agonists, have been employed to minimize these responses. However, individual agents have limitations related to their cardiovascular effects, duration of action, or potential respiratory complications. Dexmedetomidine has attracted considerable attention because of its selective alpha-2 adrenergic receptor agonist activity and its ability to produce sedation, anxiolysis, analgesia, and sympatholysis. Its central action reduces sympathetic outflow and modulates the cardiovascular response to stressful interventions without causing substantial respiratory depression at commonly used sedative doses.^[2] Dexmedetomidine exerts its pharmacological effects primarily through activation of alpha-2 adrenergic receptors within the central nervous system, particularly in the locus coeruleus. This mechanism produces a characteristic form of sedation while simultaneously reducing sympathetic nervous system activity. The drug also possesses analgesic-sparing properties and may decrease the requirements for other anaesthetic agents during general anaesthesia. Although intravenous administration is widely practiced, this route requires venous access, controlled administration, and careful cardiovascular monitoring. Alternative routes have consequently been explored to improve convenience and patient acceptance. Among these, intranasal administration offers a non-invasive technique that avoids intravenous injection and may provide a gradual onset of pharmacological effects. Its simplicity makes it a potentially useful method of preoperative medication in adult patients undergoing elective surgery.^[3] Intranasal administration of dexmedetomidine utilizes the highly vascular nasal mucosa to facilitate systemic absorption while avoiding gastrointestinal degradation and hepatic first-pass metabolism. Administration through a nasal atomizer promotes distribution of the medication over the mucosal surface, potentially improving drug absorption and consistency of delivery. This route is particularly advantageous when preoperative sedation and sympatholysis are desired before transferring patients for anaesthetic induction. Nevertheless, intranasal absorption may vary according to nasal mucosal conditions, formulation volume, administration technique, and individual pharmacokinetic differences. The timing of administration is therefore important to ensure adequate drug activity during laryngoscopy and

intubation. Careful selection of the administered dose is also necessary to achieve desirable sedation and cardiovascular stability while minimizing adverse effects such as excessive sedation, bradycardia, and hypotension.^[4] The appropriate intranasal dose of dexmedetomidine remains an important consideration in perioperative anaesthetic practice. A dose of 1 µg/kg has been investigated as a premedication regimen for reducing the stress response associated with airway manipulation, whereas a higher dose of 2 µg/kg may produce more pronounced sympatholytic and sedative effects. However, an increase in dosage may also influence haemodynamic stability and the incidence of cardiovascular adverse effects. Determining the relative efficacy of these doses requires systematic assessment of heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure before and after endotracheal intubation. Evaluating these variables at sequential intervals can help characterize the intensity and persistence of the cardiovascular response and provide clinically relevant information regarding dose selection.^[5,6]

MATERIALS AND METHODS

The present prospective, randomized, double-blinded study was conducted in the Department of Anaesthesiology, G S Medical College and Hospital, Pilkhuwa, Hapur, Uttar Pradesh, after obtaining approval from the Institutional Ethics Committee. The study was carried out in accordance with the ethical principles outlined in the Declaration of Helsinki. A total of 90 patients were included in the study. Written informed consent was obtained from all participants after explaining the nature, purpose, potential benefits, and possible risks of the study in a language they understood. Patients aged between 18 and 60 years, belonging to American Society of Anesthesiologists (ASA) physical status I and II, and scheduled for elective surgical procedures under general anaesthesia requiring laryngoscopy and endotracheal intubation were included. Patients with known hypersensitivity to dexmedetomidine, pre-existing cardiac or respiratory disorders, anticipated difficult airway, nasal polyps, nasal ulcers, deviated nasal septum, and pregnant patients were excluded. Eligible patients were recruited directly from the Department of Anaesthesiology. Randomization was performed in a 1:1 ratio using a computer-generated random allocation sequence, and allocation concealment was maintained using sequentially numbered green envelopes opened by healthcare personnel not involved in patient care. Patients were randomly allocated into two groups of 45 patients each. Group A (n = 45) received intranasal dexmedetomidine 1 µg/kg diluted in 2 mL normal saline, whereas Group B (n = 45) received intranasal dexmedetomidine 2 µg/kg diluted in 2 mL normal saline.

Pre-anaesthetic Evaluation and Study Drug

Administration: All patients underwent a detailed pre-anaesthetic evaluation, including medical history, general physical examination, systemic examination, and airway assessment. Routine laboratory investigations, including haemoglobin, complete blood count, blood urea nitrogen, serum creatinine, random blood sugar, and liver function tests, were performed. Chest X-ray (posteroanterior view) and electrocardiography were also obtained, and the findings were recorded in a structured proforma. Patients were instructed to maintain fasting for at least 6 hours before surgery, remove dentures or loose teeth, and empty their bladder before the procedure. All patients received oral alprazolam 0.25 mg on the morning of surgery according to the standardized premedication protocol. Before shifting the patients to the operating theatre, the anaesthesia workstation, oxygen and nitrous oxide cylinders, suction apparatus, multiparameter monitors, electrical defibrillator, endotracheal tubes of different sizes with stylets, Macintosh laryngoscopes, oropharyngeal airways, face masks, intravenous cannulas, and emergency medications were checked and kept ready. The allocated dose of dexmedetomidine was administered intranasally using an atomizer 45 minutes before induction of general anaesthesia. Baseline haemodynamic parameters, including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), peripheral oxygen saturation (SpO₂), and respiratory rate (RR), were recorded before administration of the study drug and again 45 minutes after premedication. The Ramsay Sedation Score (RSS) was assessed at baseline and after 45 minutes to evaluate the sedative effects of the study drug.

Anaesthesia Technique and Endotracheal

Intubation: After arrival in the operating theatre, standard monitoring was established using electrocardiography, non-invasive blood pressure monitoring, and pulse oximetry. Baseline vital parameters were recorded, and an 18-gauge intravenous cannula was secured over the dorsum of the hand, followed by initiation of Ringer's lactate infusion. Five minutes before induction of general anaesthesia, all patients received intravenous glycopyrrolate 0.005 mg/kg, ondansetron 4 mg, and fentanyl 1.5 µg/kg. Preoxygenation was performed with 100% oxygen for 3 minutes using a tight-fitting face mask. General anaesthesia was induced with intravenous propofol 2 mg/kg until loss of verbal response, and neuromuscular blockade was achieved using intravenous vecuronium 0.08 mg/kg to facilitate endotracheal intubation. Bag-and-mask ventilation was performed with 100% oxygen. Direct laryngoscopy was performed by a single experienced anaesthesiologist 3 minutes after administration of vecuronium to minimize operator-related variations, and the duration of laryngoscopy was recorded. The trachea was intubated using an appropriately sized cuffed endotracheal tube. Anaesthesia was

maintained using oxygen 33%, nitrous oxide 66%, isoflurane 1%, and intravenous vecuronium bromide 0.08–0.12 mg/kg as required. Mechanical ventilation was maintained with a tidal volume of 6 mL/kg, respiratory rate of 15 breaths per minute, inspiratory-to-expiratory ratio of 1:2, and positive end-expiratory pressure of 5 cm H₂O. End-tidal carbon dioxide was maintained between 35 and 45 mmHg throughout the procedure. During laparoscopic procedures, intra-abdominal pressure was maintained between 12 and 14 mmHg.

Assessment of Haemodynamic Parameters and Adverse Effects:

Haemodynamic parameters were recorded systematically in both groups to evaluate the efficacy of different doses of intranasal dexmedetomidine in attenuating cardiovascular responses associated with direct laryngoscopy and endotracheal intubation. The primary parameters included systolic blood pressure, diastolic blood pressure, mean arterial pressure, and heart rate. These parameters were recorded at baseline, 45 minutes before surgery and prior to study drug administration, at the time of endotracheal intubation, and subsequently at 1, 3, 5, 10, and 15 minutes after intubation. Changes in these parameters from baseline were assessed to determine the magnitude and duration of haemodynamic responses in the two groups. The measurements obtained at different time intervals were compared between Group A and Group B to assess the relative effectiveness of intranasal dexmedetomidine 1 µg/kg and 2 µg/kg in minimizing sympathetic stimulation during airway instrumentation. Oxygen saturation and respiratory rate were monitored to evaluate respiratory safety, while the Ramsay Sedation Score was used to assess the level of sedation achieved following intranasal premedication. Patients were continuously monitored for adverse effects, particularly hypotension, bradycardia, excessive sedation, respiratory depression, and oxygen desaturation. Hypotension was defined as a reduction in systolic blood pressure of 30% or more from baseline and was managed with an intravenous bolus of mephentermine 6 mg whenever required. Bradycardia was defined as a heart rate below 50 beats per minute and was treated with intravenous atropine 0.6 mg.

Statistical Analysis: Statistical analysis was performed using IBM Statistical Package for the Social Sciences (SPSS) Statistics version 29.0 (IBM Corp., Armonk, New York, USA). The collected data were entered, checked for completeness, and analyzed using appropriate descriptive and inferential statistical methods. The normality of continuous variables was assessed using the Shapiro–Wilk test. Quantitative variables, including age, heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure, were expressed as mean ± standard deviation, whereas categorical variables were presented as frequencies and percentages. Comparisons of continuous variables between the two independent groups were performed using the independent samples t-test. Categorical variables,

including demographic characteristics and the incidence of adverse effects, were compared using the Chi-square test. A p-value of less than 0.05 was considered statistically significant.

RESULTS

[Table 1] Distribution of Participants According to Study Group, Gender and ASA Physical Status (N = 90)

[Table 1] shows the distribution of study participants according to study group, gender, and American Society of Anesthesiologists (ASA) physical status. A total of 90 patients were included in the study and were equally divided into two groups of 45 patients each. Group A received intranasal dexmedetomidine 1 µg/kg, whereas Group B received intranasal dexmedetomidine 2 µg/kg, with each group representing 50.0% of the total study population. Regarding gender distribution, 47 patients (52.2%) were males and 43 patients (47.8%) were females. In Group A, 23 patients (51.1%) were males and 22 (48.9%) were females, whereas Group B comprised 24 males (53.3%) and 21 females (46.7%). The difference in gender distribution between the two groups was statistically insignificant ($p = 1.000$).

[Table 2] Comparison of Heart Rate (HR) Between the Two Groups at Different Time Intervals (n = 45 Each)

[Table 2] presents the comparison of mean heart rate between Group A and Group B at different time intervals following endotracheal intubation. At baseline, the mean heart rate was 76.93 ± 8.42 beats/minute in Group A and 72.63 ± 8.93 beats/minute in Group B, with a statistically significant difference between the groups ($t = 2.35$, $p = 0.021$). At 1 minute after intubation, the mean heart rate increased to 99.17 ± 9.68 beats/minute in Group A and 98.27 ± 12.53 beats/minute in Group B, with no statistically significant difference ($t = 0.38$, $p = 0.705$). At 3 minutes after intubation, the mean heart rate was 89.30 ± 11.87 beats/minute in Group A and 92.03 ± 11.83 beats/minute in Group B, and the difference remained statistically insignificant ($t = -1.09$, $p = 0.278$). At 5 minutes after intubation, the mean heart rate decreased to 84.50 ± 8.84 beats/minute in Group A and 79.27 ± 10.19 beats/minute in Group B, showing a statistically significant difference ($t = 2.60$, $p = 0.020$). Similarly, at 10 minutes, the mean heart rate was 79.83 ± 8.21 beats/minute in Group A compared with 75.93 ± 8.46 beats/minute in Group B, with a statistically significant difference ($t = 2.17$, $p = 0.033$). At 15 minutes after intubation, the corresponding values were 77.62 ± 7.98 beats/minute and 73.94 ± 8.02 beats/minute, respectively, and the difference was statistically significant ($t = 2.15$, $p = 0.034$).

[Table 3] Comparison of Systolic Blood Pressure (SBP) Between the Two Groups at Different Time Intervals (n = 45 Each)

[Table 3] demonstrates the comparison of systolic blood pressure between the two groups at different time intervals. At baseline, the mean systolic blood pressure was 119.20 ± 12.93 mmHg in Group A and 120.93 ± 12.26 mmHg in Group B, with no statistically significant difference between the groups ($t = -0.65$, $p = 0.51$). At 1 minute after endotracheal intubation, the mean systolic blood pressure increased to 137.30 ± 10.99 mmHg in Group A and 139.10 ± 10.08 mmHg in Group B, and the difference was statistically insignificant ($t = 0.81$, $p = 0.419$). At 3 minutes after intubation, the mean systolic blood pressure decreased to 127.20 ± 9.75 mmHg in Group A and 128.37 ± 12.08 mmHg in Group B, without a statistically significant difference ($t = -0.50$, $p = 0.618$). At 5 minutes, the mean systolic blood pressure was 119.33 ± 11.01 mmHg in Group A and 118.40 ± 11.05 mmHg in Group B, with a statistically insignificant difference ($t = 0.40$, $p = 0.69$). At 10 minutes after intubation, the mean values were 117.20 ± 10.54 mmHg in Group A and 115.83 ± 9.87 mmHg in Group B ($t = 0.63$, $p = 0.53$). At 15 minutes, the mean systolic blood pressure further decreased to 116.47 ± 9.82 mmHg in Group A and 114.92 ± 9.44 mmHg in Group B, with no statistically significant intergroup difference ($t = 0.76$, $p = 0.45$).

[Table 4] Comparison of Diastolic Blood Pressure (DBP) Between the Two Groups at Different Time Intervals (n = 45 Each)

[Table 4] illustrates the comparison of diastolic blood pressure between Group A and Group B at baseline and various intervals following endotracheal intubation. At baseline, the mean diastolic blood pressure was 74.47 ± 8.59 mmHg in Group A and 73.83 ± 7.99 mmHg in Group B, with no statistically significant difference ($t = 0.37$, $p = 0.71$). At 1 minute after intubation, the mean diastolic blood pressure increased to 86.50 ± 8.62 mmHg in Group A and 88.83 ± 9.18 mmHg in Group B, and the intergroup difference was statistically insignificant ($t = -1.24$, $p = 0.217$). At 3 minutes after intubation, the mean diastolic blood pressure was 85.60 ± 9.44 mmHg in Group A and 86.47 ± 7.62 mmHg in Group B, with no statistically significant difference ($t = -0.48$, $p = 0.631$). At 5 minutes, the mean diastolic blood pressure decreased to 73.23 ± 8.95 mmHg in Group A and 75.07 ± 9.44 mmHg in Group B, with a statistically insignificant difference ($t = -0.95$, $p = 0.346$). At 10 minutes after intubation, the mean values were 72.17 ± 8.12 mmHg in Group A and 73.40 ± 7.83 mmHg in Group B, without a statistically significant difference ($t = -0.73$, $p = 0.47$). At 15 minutes, the mean diastolic blood pressure was 71.63 ± 7.84 mmHg in Group A and 72.58 ± 7.66 mmHg in Group B, and the difference remained statistically insignificant ($t = -0.59$, $p = 0.55$).

[Table 5] Comparison of Mean Arterial Pressure (MAP) Between the Two Groups at Different Time Intervals (n = 45 Each)

[Table 5] presents the comparison of mean arterial pressure between the two study groups at different

time intervals. At baseline, the mean arterial pressure was 88.47 ± 10.32 mmHg in Group A and 90.90 ± 9.46 mmHg in Group B, with no statistically significant difference between the groups ($t = -0.17$, $p = 0.86$). At 1 minute after intubation, the mean arterial pressure increased to 103.70 ± 10.11 mmHg in Group A and 100.57 ± 10.96 mmHg in Group B, with a statistically insignificant difference ($t = 0.41$, $p = 0.61$). At 3 minutes after intubation, the mean arterial pressure was 93.17 ± 8.51 mmHg in Group A and 95.23 ± 8.78 mmHg in Group B, and the difference remained statistically insignificant ($t = -1.13$, $p = 0.262$). At 5 minutes, the mean arterial pressure decreased to 84.70 ± 8.81 mmHg in Group A and 81.93 ± 8.68 mmHg in Group B, with no statistically significant difference ($t = 1.51$, $p = 0.135$). At 10 minutes after intubation, the mean values were 83.85 ± 8.20 mmHg in Group A and 81.54 ± 7.96 mmHg in Group B, without a statistically significant difference ($t = 1.35$, $p = 0.18$). At 15 minutes, the mean arterial pressure further decreased to 82.58 ± 7.96 mmHg in Group A and 80.47 ± 7.84 mmHg in Group B, and the intergroup difference was statistically insignificant ($t = 1.27$, $p = 0.21$).

[Table 6] Comparison of Adverse Effects and Recovery Time Between the Two Groups ($n = 45$ Each)

[Table 6] shows the distribution of adverse effects and comparison of recovery time between the two study groups. Among the 45 patients in Group A, 18 patients (40.0%) experienced no adverse effects, compared with 20 patients (44.4%) in Group B. Bradycardia was recorded in 11 patients (24.4%) in Group A and 3 patients (6.7%) in Group B. Hypotension was not observed in any patient in Group A, whereas 6 patients (13.3%) in Group B experienced hypotension. Desaturation was reported in 11 patients (24.4%) in Group A and 12 patients (26.7%) in Group B. Other adverse effects were recorded in 5 patients (11.1%) in Group A and 4 patients (8.9%) in Group B. The overall distribution of adverse effects differed significantly between the two groups, with a reported p -value of 0.02. Regarding the recovery profile, the mean recovery time was 12.23 ± 5.32 minutes in Group A and 12.80 ± 5.58 minutes in Group B. The mean recovery time was numerically higher in Group B than in Group A, with a difference of 0.57 minutes. However, this difference was statistically insignificant ($p = 0.62$).

Table 1: Distribution of Participants According to Study Group, Gender and ASA Physical Status (N = 90)

Characteristic	Category	Group A n (%)	Group B n (%)	Total n (%)	P value
Study group	Group A (IN Dex 1 µg/kg)	45 (50.0% of total)	–	45 (50.0%)	–
	Group B (IN Dex 2 µg/kg)	–	45 (50.0% of total)	45 (50.0%)	–
	Total	45	45	90 (100%)	–
Gender	Male	23 (51.1%)	24 (53.3%)	47 (52.2%)	1.000
	Female	22 (48.9%)	21 (46.7%)	43 (47.8%)	–
	Total	45 (100%)	45 (100%)	90 (100%)	–
ASA physical status	ASA I	23 (51.1%)	18 (40.0%)	41 (45.6%)	0.305
	ASA II	22 (48.9%)	27 (60.0%)	49 (54.4%)	–
	Total	45 (100%)	45 (100%)	90 (100%)	–

Table 2: Comparison of Heart Rate (HR) Between the Two Groups at Different Time Intervals (n = 45 Each)

Time interval	Group A (Mean ± SD)	Group B (Mean ± SD)	t value	P value
Baseline (45 min before surgery)	76.93 ± 8.42	72.63 ± 8.93	2.35	0.021
1 minute after intubation	99.17 ± 9.68	98.27 ± 12.53	0.38	0.705
3 minutes after intubation	89.30 ± 11.87	92.03 ± 11.83	-1.09	0.278
5 minutes after intubation	84.50 ± 8.84	79.27 ± 10.19	2.60	0.020
10 minutes after intubation	79.83 ± 8.21	75.93 ± 8.46	2.17	0.033
15 minutes after intubation	77.62 ± 7.98	73.94 ± 8.02	2.15	0.034

Table 3: Comparison of Systolic Blood Pressure (SBP) Between the Two Groups at Different Time Intervals (n = 45 Each)

Time interval	Group A (Mean ± SD)	Group B (Mean ± SD)	t value	P value
Baseline (45 min before surgery)	119.20 ± 12.93	120.93 ± 12.26	-0.65	0.51
1 minute after intubation	137.30 ± 10.99	139.10 ± 10.08	0.81	0.419
3 minutes after intubation	127.20 ± 9.75	128.37 ± 12.08	-0.50	0.618
5 minutes after intubation	119.33 ± 11.01	118.40 ± 11.05	0.40	0.69
10 minutes after intubation	117.20 ± 10.54	115.83 ± 9.87	0.63	0.53
15 minutes after intubation	116.47 ± 9.82	114.92 ± 9.44	0.76	0.45

Table 4: Comparison of Diastolic Blood Pressure (DBP) Between the Two Groups at Different Time Intervals (n = 45 Each)

Time interval	Group A (Mean ± SD)	Group B (Mean ± SD)	t value	P value
Baseline (45 min before surgery)	74.47 ± 8.59	73.83 ± 7.99	0.37	0.71
1 minute after intubation	86.50 ± 8.62	88.83 ± 9.18	-1.24	0.217
3 minutes after intubation	85.60 ± 9.44	86.47 ± 7.62	-0.48	0.631
5 minutes after intubation	73.23 ± 8.95	75.07 ± 9.44	-0.95	0.346
10 minutes after intubation	72.17 ± 8.12	73.40 ± 7.83	-0.73	0.47
15 minutes after intubation	71.63 ± 7.84	72.58 ± 7.66	-0.59	0.55

Table 5: Comparison of Mean Arterial Pressure (MAP) Between the Two Groups at Different Time Intervals (n = 45 Each)

Time interval	Group A (Mean ± SD)	Group B (Mean ± SD)	t value	P value
Baseline (45 min before surgery)	88.47 ± 10.32	90.90 ± 9.46	-0.17	0.86
1 minute after intubation	103.70 ± 10.11	100.57 ± 10.96	0.41	0.61
3 minutes after intubation	93.17 ± 8.51	95.23 ± 8.78	-1.13	0.262
5 minutes after intubation	84.70 ± 8.81	81.93 ± 8.68	1.51	0.135
10 minutes after intubation	83.85 ± 8.20	81.54 ± 7.96	1.35	0.18
15 minutes after intubation	82.58 ± 7.96	80.47 ± 7.84	1.27	0.21

Table 6: Comparison of Adverse Effects and Recovery Time Between the Two Groups (n = 45 Each)

Parameter	Category	Group A	Group B	P value
Adverse effects	None	18 (40.0%)	20 (44.4%)	0.02
	Bradycardia	11 (24.4%)	3 (6.7%)	–
	Hypotension	0 (0.0%)	6 (13.3%)	–
	Desaturation	11 (24.4%)	12 (26.7%)	–
	Other	5 (11.1%)	4 (8.9%)	–
	Total	45 (100%)	45 (100%)	–
Recovery profile	Recovery time (min), Mean ± SD	12.23 ± 5.32	12.80 ± 5.58	0.62

DISCUSSION

The present study included 90 adult patients who were randomly allocated into two equal groups of 45 patients each. Group A received intranasal dexmedetomidine 1 µg/kg, whereas Group B received intranasal dexmedetomidine 2 µg/kg. The overall study population comprised 47 males (52.2%) and 43 females (47.8%), with no statistically significant difference in gender distribution between the groups ($p = 1.000$). Similarly, 41 patients (45.6%) belonged to ASA physical status I and 49 patients (54.4%) belonged to ASA physical status II, without a significant intergroup difference ($p = 0.305$). These findings were compared with the study conducted by Safavi et al. (2022),^[7] who evaluated 88 patients receiving intranasal dexmedetomidine 1 µg/kg, 2 µg/kg, or placebo. Their study included 59 females (67.1%) and 29 males (32.9%), with a mean age of 41.89 ± 13.97 years. The gender distribution between their intervention groups was statistically comparable ($p = 0.377$), although the baseline heart rate differed significantly between the three study groups ($p = 0.008$). In the present study, heart rate increased in both groups immediately after endotracheal intubation, followed by a gradual reduction during subsequent observations. At 1 minute after intubation, the mean heart rate was 99.17 ± 9.68 beats/minute in Group A and 98.27 ± 12.53 beats/minute in Group B ($p = 0.705$). At 5 minutes, the mean heart rate decreased to 84.50 ± 8.84 and 79.27 ± 10.19 beats/minute, respectively, showing a statistically significant difference ($p = 0.020$). Similar significant differences were observed at 10 minutes ($p = 0.033$) and 15 minutes ($p = 0.034$), with lower mean heart rates in Group B. These findings were compared with those of Kochhar et al. (2021),^[8] who conducted a prospective randomized study involving 90 adult patients divided into three groups of 30 patients each. The groups received intranasal normal saline, dexmedetomidine 1 µg/kg, or dexmedetomidine 2 µg/kg before induction of anaesthesia. The investigators reported significantly greater increases in heart rate and blood pressure at 1,

3, and 5 minutes after intubation in the control group than in the dexmedetomidine groups. They also reported significantly higher sedation scores and lower propofol requirements in both dexmedetomidine groups ($p < 0.0001$). The heart rate findings were also compared with the study conducted by Niyogi et al. (2019),^[9] who evaluated 70 adult patients randomized into two groups of 35 patients each. One group received intravenous dexmedetomidine 0.5 µg/kg, while the other received intranasal dexmedetomidine 1 µg/kg approximately 40 minutes before induction of anaesthesia. The investigators observed that haemodynamic parameters remained within 20% of baseline values throughout the study period. No statistically significant difference in mean arterial pressure was observed between the two groups ($p > 0.05$), although the preoperative sedation score was significantly higher in the intravenous group ($p = 0.014$). In the present study, the mean heart rate at 10 minutes after intubation was 79.83 ± 8.21 beats/minute in Group A and 75.93 ± 8.46 beats/minute in Group B ($p = 0.033$). At 15 minutes, the corresponding values were 77.62 ± 7.98 and 73.94 ± 8.02 beats/minute ($p = 0.034$). Regarding systolic blood pressure, the present study demonstrated an increase immediately after intubation in both groups, followed by a gradual reduction. The baseline mean systolic blood pressure was 119.20 ± 12.93 mmHg in Group A and 120.93 ± 12.26 mmHg in Group B. At 1 minute after intubation, the corresponding values increased to 137.30 ± 10.99 mmHg and 139.10 ± 10.08 mmHg, respectively ($p = 0.419$). At 5 minutes, the mean values decreased to 119.33 ± 11.01 mmHg in Group A and 118.40 ± 11.05 mmHg in Group B ($p = 0.69$). At 15 minutes, the systolic blood pressure further decreased to 116.47 ± 9.82 mmHg and 114.92 ± 9.44 mmHg, respectively ($p = 0.45$). These findings were compared with those reported by Kumari et al. (2015),^[10] who conducted a randomized study involving 80 adult patients equally divided into dexmedetomidine and placebo groups. The dexmedetomidine group received a single intravenous dose of 0.5 µg/kg before induction. The

maximum percentage increase in systolic blood pressure after intubation was 12.38% in the dexmedetomidine group compared with 45.63% in the placebo group. Similarly, the maximum increase in heart rate was 12.96% in the dexmedetomidine group compared with 32.57% in the placebo group. The investigators also observed a reduction in propofol requirements from 2.01 mg/kg in the placebo group to 1.04 mg/kg in the dexmedetomidine group ($p < 0.001$). The diastolic blood pressure findings in the present study demonstrated a similar pattern of initial elevation followed by progressive reduction. At baseline, mean diastolic blood pressure was 74.47 ± 8.59 mmHg in Group A and 73.83 ± 7.99 mmHg in Group B ($p = 0.71$). At 1 minute after intubation, the values increased to 86.50 ± 8.62 mmHg and 88.83 ± 9.18 mmHg, respectively ($p = 0.217$). At 3 minutes, the corresponding values were 85.60 ± 9.44 mmHg and 86.47 ± 7.62 mmHg ($p = 0.631$), whereas at 15 minutes, they decreased to 71.63 ± 7.84 mmHg and 72.58 ± 7.66 mmHg, respectively ($p = 0.55$). These results were compared with those of Sebastian et al. (2017),^[11] who conducted a prospective randomized, double-blind study involving 90 ASA I patients distributed equally into three groups of 30 patients each. The groups received normal saline, intravenous dexmedetomidine 0.5 µg/kg, or intravenous dexmedetomidine 0.75 µg/kg. The investigators reported statistically significant differences in heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure between the dexmedetomidine and control groups at 1, 3, and 5 minutes after intubation ($p < 0.05$). The 0.75 µg/kg dose produced greater attenuation of the haemodynamic response, and no episodes of bradycardia, hypotension, respiratory depression, or oxygen desaturation were observed among the 90 participants. In the present study, mean arterial pressure increased immediately following endotracheal intubation in both groups and subsequently decreased during the observation period. At baseline, the mean arterial pressure was 88.47 ± 10.32 mmHg in Group A and 90.90 ± 9.46 mmHg in Group B ($p = 0.86$). At 1 minute after intubation, the corresponding values increased to 103.70 ± 10.11 mmHg and 100.57 ± 10.96 mmHg, respectively ($p = 0.61$). At 5 minutes, mean arterial pressure decreased to 84.70 ± 8.81 mmHg and 81.93 ± 8.68 mmHg ($p = 0.135$). At 10 minutes, the corresponding values were 83.85 ± 8.20 mmHg and 81.54 ± 7.96 mmHg ($p = 0.18$), while at 15 minutes, they were 82.58 ± 7.96 mmHg and 80.47 ± 7.84 mmHg, respectively ($p = 0.21$). These findings were compared with the study conducted by Padmasree and Nelamangala (2023),^[12] who evaluated 106 adult patients receiving intravenous dexmedetomidine 0.5 µg/kg or intranasal dexmedetomidine 1 µg/kg. Their study reported baseline mean arterial pressure values of 93.76 ± 8.77 mmHg in the intravenous group and 91.03 ± 8.15 mmHg in the intranasal group ($p = 0.09$). At 10 minutes after intubation, the

corresponding mean arterial pressures were 84.35 ± 4.49 mmHg and 81.54 ± 7.21 mmHg, respectively, with a statistically significant difference ($p = 0.012$). In the present study, the difference in mean arterial pressure at 10 minutes was statistically insignificant ($p = 0.18$), although Group B showed a numerically lower value. Regarding adverse effects, the present study demonstrated a statistically significant difference in their overall distribution between the two groups (reported $p = 0.02$). Bradycardia was observed in 11 patients (24.4%) in Group A and 3 patients (6.7%) in Group B, whereas hypotension was absent in Group A but occurred in 6 patients (13.3%) in Group B. Oxygen desaturation was recorded in 11 patients (24.4%) in Group A and 12 patients (26.7%) in Group B. Other adverse effects were reported in 5 patients (11.1%) and 4 patients (8.9%), respectively. Overall, 18 patients (40.0%) in Group A and 20 patients (44.4%) in Group B experienced no adverse effects. These findings were compared with the study conducted by Kohaf et al. (2024),^[13] who evaluated 60 adult patients undergoing functional endoscopic sinus surgery, with 30 patients receiving intranasal dexmedetomidine 1 µg/kg and 30 receiving intravenous dexmedetomidine 1 µg/kg. Their study reported a significantly lower atropine requirement in the intranasal group than in the intravenous group (0.20 ± 0.34 mg versus 0.70 ± 0.55 mg; $p < 0.001$). The recovery profile in the present study showed that the mean recovery time was 12.23 ± 5.32 minutes in Group A and 12.80 ± 5.58 minutes in Group B, with a mean difference of 0.57 minutes. The difference was statistically insignificant ($p = 0.62$), indicating comparable recovery times between the two intranasal dexmedetomidine dose groups. These findings were compared with those reported by Lu et al. (2016),^[14] who conducted a prospective randomized, double-blind study involving 81 adult patients undergoing suspension laryngoscopy. Patients received intranasal dexmedetomidine 1 µg/kg or placebo approximately 45–60 minutes before induction of anaesthesia. Their study demonstrated significantly improved preoperative sedation ($p < 0.001$) and reduced anxiety ($p = 0.001$) following intranasal dexmedetomidine administration. However, no statistically significant differences were observed in the time to spontaneous breathing, emergence, or tracheal extubation between the two groups.

CONCLUSION

The present study concluded that both intranasal dexmedetomidine 1 µg/kg and 2 µg/kg attenuated haemodynamic responses following laryngoscopy and endotracheal intubation in adult patients. Intranasal dexmedetomidine 2 µg/kg was associated with significantly lower heart rates at 5, 10, and 15 minutes after intubation compared with 1 µg/kg. However, no statistically significant differences were observed in systolic blood pressure, diastolic blood pressure, or mean arterial pressure between the two

groups. The incidence of adverse effects differed significantly between the groups, while recovery times remained comparable.

REFERENCES

- Misra S, Behera BK, Mitra JK, Sahoo AK, Jena SS, Srinivasan A. Effect of preoperative dexmedetomidine nebulization on the hemodynamic response to laryngoscopy and intubation: a randomized control trial. *Korean J Anesthesiol*. 2021;74(2):150-157. doi:10.4097/kja.20153.
- Singh V, Pahade A, Mowar A. Comparison of intravenous versus nebulized dexmedetomidine for laryngoscopy and intubation-induced sympathoadrenal stress response attenuation. *Anesth Pain Med*. 2022;12(5):e132607. doi:10.5812/aapm-132607.
- Ankita, Kumar H, Kumar M. Comparison of intranasal versus intravenous dexmedetomidine to attenuate haemodynamic response of laryngoscopy and endotracheal intubation in elective lumbar spine surgery: a randomized controlled study. *Future Health*. 2023;1(1):50-54. doi:10.25259/FH_20230101_43.
- Singla A, Saraswat RK, Bharadwaj A, Singh S. Nebulized versus intravenously administered dexmedetomidine for obtunding hemodynamic responses to laryngoscopy and tracheal intubation: a randomized double-blind comparative study. *Cureus*. 2024;16(2):e54768. doi:10.7759/cureus.54768.
- Deshmukh KM, Bhargava SV, Bhure AR, Mone AA, Dhoble KR, Rode AA. Effectiveness of intranasal versus intravenous dexmedetomidine for attenuation of hemodynamic responses of laryngoscopy and endotracheal intubation in patients undergoing general anaesthesia: randomized controlled trial. *Indian J Clin Anaesth*. 2025;12(1):50-58. doi:10.18231/j.ijca.2025.008.
- Singh G, Lnu H, Verma R, Shukla A, Singh P, Kohli M. Effects of intranasal and intravenous dexmedetomidine on hemodynamic responses to tracheal intubation and skull pin holder fixation: a double-blinded, randomized controlled trial. *Cureus*. 2025;17(1):e76980. doi:10.7759/cureus.76980.
- Safavi SMR, Honarmand A, Nazemroaya B, Ataie AM, Kamran Z. The effect of intranasal dexmedetomidine on hemodynamic disturbances caused by laryngoscopy and endotracheal intubation. *Int J Physiol Pathophysiol Pharmacol*. 2022;14(4):225-232.
- Kochhar A, Panjari P, Butt KM. Intranasal dexmedetomidine for attenuation of hemodynamic response to laryngoscopy and intubation in adults. *Acta Anaesthesiol Belg*. 2021;72(1):1-6. doi:10.56126/72.1.1.
- Niyogi S, Biswas A, Chakraborty I, Chakraborty S, Acharjee A. Attenuation of haemodynamic responses to laryngoscopy and endotracheal intubation with dexmedetomidine: a comparison between intravenous and intranasal route. *Indian J Anaesth*. 2019;63(11):915-923. doi:10.4103/ija.IJA_320_19.
- Kumari K, Gombar S, Kapoor D, Sandhu HS. Clinical study to evaluate the role of preoperative dexmedetomidine in attenuation of hemodynamic response to direct laryngoscopy and tracheal intubation. *Acta Anaesthesiol Taiwan*. 2015;53(4):123-130. doi:10.1016/j.aat.2015.09.003.
- Sebastian B, Talikoti AT, Krishnamurthy D. Attenuation of haemodynamic responses to laryngoscopy and endotracheal intubation with intravenous dexmedetomidine: a comparison between two doses. *Indian J Anaesth*. 2017;61(1):48-54. doi:10.4103/0019-5049.198404.
- Padmasree MK, Nelamangala K. A comparative study between intranasal and intravenous dexmedetomidine and hemodynamic responses during endotracheal intubation. *Cureus*. 2023;15(2):e35196. doi:10.7759/cureus.35196.
- Kohaf NA, Harby SA, Abd-Ellatief AF, Elsaid MA, Abdelmottaleb NA, Abd Elsalam TF. Premedication with intranasal versus intravenous dexmedetomidine for hypotensive anesthesia during functional endoscopic sinus surgery in adults: a randomized triple-blind trial. *Heliyon*. 2024;10(3):e25175. doi:10.1016/j.heliyon.2024.e25175.
- Lu C, Zhang LM, Zhang Y, Ying Y, Li L, Xu L, et al. Intranasal dexmedetomidine as a sedative premedication for patients undergoing suspension laryngoscopy: a randomized double-blind study. *PLoS One*. 2016;11(5):e0154192. doi:10.1371/journal.pone.0154192.