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Original Research Article

Comparison of Hemodynamic Effects of Intravenous and Intranasal Dexmedetomidine in ENT Surgery Patients

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Abstract

Introduction: Dexmedetomidine is widely used as a premedication because of its sympatholytic and sedative properties, which help attenuate the hemodynamic response to laryngoscopy and endotracheal intubation. While intravenous dexmedetomidine provides rapid and predictable effects, intranasal administration has emerged as a non-invasive alternative with good bioavailability. The present study aimed to compare the hemodynamic effects of intravenous and intranasal dexmedetomidine in patients undergoing ENT surgeries under general anesthesia.

Materials and Methods: This prospective comparative study was conducted at Government Medical College, Mancherial, from January 2024 to June 2025. 100 adult patients (ASA I–II) undergoing elective ENT surgeries under general anesthesia were randomly allocated into two groups (n = 50 each). Group I received intravenous dexmedetomidine 1 µg/kg over 10 minutes, and Group II received intranasal dexmedetomidine 1 µg/kg 40 minutes before induction. Heart rate (HR) and mean arterial pressure (MAP) were recorded at baseline, at 10, 20, 30, and 40 minutes after drug administration, at induction, and at 1, 2, 4, 5, 7, and 10 minutes after intubation.

Results: Baseline variables were comparable between the groups. HR and MAP decreased progressively in both groups after drug administration. From 20 to 40 minutes and at induction, the IV dexmedetomidine group demonstrated significantly lower HR and MAP compared with the intranasal group ($p < 0.05$). Following intubation, transient increases in HR and MAP were observed in both groups, but values were consistently lower in the IV group, with significant differences at 7 and 10 minutes for MAP.

Conclusion: Both intravenous and intranasal dexmedetomidine effectively attenuate peri-intubation hemodynamic responses. However, intravenous dexmedetomidine provides faster and more pronounced early control of heart rate and mean arterial pressure, while intranasal dexmedetomidine offers a safe, non-invasive alternative when adequate premedication time is available.

Keywords: Dexmedetomidine; Intranasal Route; Intravenous Route; Hemodynamic Response; ENT Surgery.

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Introduction

Dexmedetomidine is a highly selective α2-adrenergic agonist that has gained wide acceptance in anesthetic practice because of its sedative, anxiolytic, and analgesic properties, along with its ability to attenuate sympathetic responses without causing significant respiratory depression [1]. Owing to these characteristics, dexmedetomidine has been increasingly used as a premedicant and as an adjuvant to general anesthesia for achieving hemodynamic stability during perioperative periods [2]. Its sympatholytic effect is particularly useful in suppressing stress responses associated with laryngoscopy, intubation, and surgical stimulation [3]. Endotracheal intubation and surgical manipulation during ENT procedures are well known to provoke marked cardiovascular responses such as tachycardia and hypertension due to intense sympathetic stimulation [4]. These hemodynamic fluctuations may be detrimental, especially in patients with limited cardiovascular reserve, and therefore strategies to blunt these responses are clinically important [5]. Several pharmacological

agents, including opioids, beta-blockers, and α2-agonists, have been employed to mitigate these responses; however, dexmedetomidine has emerged as a preferred agent because of its favourable pharmacodynamic profile and minimal effect on respiration [6,7].

Dexmedetomidine can be administered through various routes, most commonly intravenously, which provides rapid onset and predictable plasma concentrations [8]. Intranasal administration has recently gained interest as a non-invasive alternative that offers ease of administration, good bioavailability, and avoidance of first-pass metabolism [9]. Intranasal dexmedetomidine has been used successfully for premedication and sedation, particularly in outpatient and pediatric settings [10]. However, differences in onset time, peak effect, and overall hemodynamic control between intravenous and intranasal routes remain areas of ongoing investigation, especially in adult patients undergoing ENT surgeries where hemodynamic stability is crucial [8,11].

Although previous studies have demonstrated the efficacy of dexmedetomidine in attenuating peri-intubation stress responses, limited data are available directly comparing the hemodynamic effects of intravenous and intranasal dexmedetomidine in adult patients undergoing ENT procedures [11,12]. A clearer understanding of the relative effectiveness of these two routes would help optimize premedication strategies and improve perioperative cardiovascular stability. Therefore, the present study was undertaken to compare the hemodynamic effects of intravenous and intranasal dexmedetomidine in patients undergoing ENT surgeries under general anesthesia, with the aim of evaluating their influence on heart rate and mean arterial pressure during the peri-induction and post-intubation periods.

Materials and Methods

This prospective comparative study was conducted in the Department of Anaesthesiology at Government Medical College, Mancherial, over a period of eighteen months from January 2024 to June 2025, after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to inclusion in the study.

A total of 100 adult patients of either sex, aged 18–60 years, scheduled for elective ENT surgeries under general anesthesia were enrolled. Only patients belonging to American Society of Anesthesiologists (ASA) physical status I and II were included. Patients with known hypersensitivity to dexmedetomidine, significant cardiovascular disease such as arrhythmias or heart block, respiratory disorders, hepatic or renal impairment, pregnancy or lactation, those receiving beta-blockers or other drugs affecting heart rate or blood pressure, and patients with nasal pathology such as deviated nasal septum, nasal polyps, or active nasal infection were excluded from the study.

Patients were randomly allocated into two equal groups of 50 each using a computer-generated randomization sequence. Group I received dexmedetomidine 1 µg/kg intravenously diluted in 50 mL of normal saline and administered over 10 minutes. Group II received dexmedetomidine 1 µg/kg intranasally, divided equally between both nostrils using a mucosal atomization device, 40 minutes before induction of anesthesia. All patients were kept nil per oral as per standard guidelines and did not receive any sedative premedication. On arrival in the operating room, standard monitoring including electrocardiography, non-invasive blood pressure, pulse oximetry, and heart rate was instituted. Baseline heart rate and mean arterial pressure were recorded before administration of the study drug.

Following administration of the study drug as per group allocation, patients were preoxygenated for three minutes. General anesthesia was induced with intravenous propofol 2 mg/kg and fentanyl 2 µg/kg. Neuromuscular blockade was achieved with succinylcholine 1.5 mg/kg to facilitate endotracheal intubation. Anesthesia was maintained with oxygen, nitrous oxide, and sevoflurane, with intermittent doses of vecuronium as required. Hemodynamic parameters, namely heart rate and mean arterial pressure, were recorded at baseline, at 10, 20, 30, and 40 minutes after administration of the study drug, at induction, and at 1, 2, 4, 5, 7, and 10 minutes following endotracheal intubation. Patients were observed for any adverse events.

Data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) software. Continuous variables were expressed as mean ± standard deviation and categorical variables as number and percentage. Intergroup comparison of continuous variables was performed using Student's t-test and repeated measures analysis of variance, whereas categorical variables were analyzed using the Chi-square test. A p-value of less than 0.05 was considered statistically significant.

Results

Baseline demographic and clinical characteristics were comparable between the IV dexmedetomidine and intranasal dexmedetomidine groups. The mean age was similar (34.6 ± 9.2 vs 35.1 ± 8.7 years), with a nearly equal gender distribution in both groups. Body weight and ASA physical status (I/II)

were also well matched between the two arms. The mean duration of surgery did not differ significantly between the groups (58.2 ± 10.4 vs 60.1 ± 11.2 minutes). These findings indicate that both groups were demographically and clinically comparable at baseline, minimizing confounding factors related to patient characteristics or surgical duration (Table 1).

Table 1: Baseline Demographic and Clinical Characteristics

Variable	Group I (IV dexmedetomidine) (n = 50)	Group II (Intranasal dexmedetomidine) (n = 50)	p-value
Age (years), mean ± SD	34.6 ± 9.2	35.1 ± 8.7	0.78
Gender (M/F)	28 / 22	27 / 23	0.84
Weight (kg), mean ± SD	62.4 ± 8.1	63.0 ± 7.6	0.69
ASA physical status (I / II)	32 / 18	30 / 20	0.67
Duration of surgery (min), mean ± SD	58.2 ± 10.4	60.1 ± 11.2	0.38

Heart rate values were similar between the two groups at baseline and during the initial 10 minutes after drug administration. However, from 20 to 40 minutes, the IV dexmedetomidine group demonstrated significantly lower heart rates compared with the intranasal group, indicating a more pronounced sympatholytic effect with the intravenous route. At induction, heart rate remained significantly lower in the IV group. Following induction, transient increases in heart rate were observed in both groups at 1 and 2 minutes, with no statistically significant intergroup difference. Subsequently, heart rate gradually decreased and remained consistently lower in the IV group, with borderline significance at 10 minutes. Overall, IV dexmedetomidine provided better attenuation of heart rate compared with the intranasal route during the peri-induction period (Table 2).

Table 2: Heart Rate (beats/min) at Different Time Points

Time Point	Group I (IV dexmedetomidine) Mean ± SD	Group II (Intranasal dexmedetomidine) Mean ± SD	p-value
Baseline	82.3 ± 8.1	80.4 ± 7.2	0.39
10 min	75.6 ± 7.3	78.2 ± 8.0	0.12
20 min	70.4 ± 6.2	74.6 ± 7.1	0.04
30 min	66.8 ± 7.0	71.5 ± 6.3	0.02
40 min	64.2 ± 7.1	70.3 ± 7.2	0.01
Induction	68.5 ± 8.0	72.4 ± 7.1	0.03
1 min	85.6 ± 9.3	88.7 ± 10.2	0.18
2 min	82.4 ± 8.5	85.9 ± 9.3	0.14
4 min	79.3 ± 8.2	81.6 ± 8.4	0.26
5 min	76.5 ± 7.4	80.8 ± 8.3	0.08
7 min	74.2 ± 7.1	78.5 ± 7.3	0.07
10 min	72.1 ± 6.4	76.7 ± 8.2	0.05

Mean arterial pressure was comparable between the two groups at baseline and up to 20 minutes after drug administration.

From 30 minutes onward, the IV dexmedetomidine group exhibited significantly lower MAP values compared with the intranasal group, suggesting superior control of blood pressure with intravenous administration. At induction, MAP remained significantly lower in the IV group. Post-induction, both groups showed a rise in MAP at 1 and 2 minutes, without significant intergroup differences. Thereafter, MAP gradually declined, with significantly lower values observed in the IV group at 7 and 10 minutes.

These findings indicate that IV dexmedetomidine achieved more effective attenuation of pressor responses during laryngoscopy and intubation than the intranasal route (Table 3).

Table 3: Mean Arterial Pressure (MAP, mmHg) at Different Time Points

Time Point	Group I (IV dexmedetomidine) Mean ± SD	Group II (Intranasal dexmedetomidine) Mean ± SD	p-value
Baseline	95.4 ± 9.2	93.6 ± 8.4	0.45
10 min	88.6 ± 8.3	90.2 ± 9.1	0.30
20 min	84.5 ± 7.4	87.6 ± 8.2	0.17
30 min	80.3 ± 8.1	85.2 ± 7.3	0.03
40 min	78.4 ± 7.2	83.5 ± 8.1	0.02
Induction	82.6 ± 8.3	86.4 ± 7.2	0.04
1 min	100.8 ± 10.4	104.6 ± 11.3	0.15
2 min	97.5 ± 9.5	101.3 ± 10.2	0.13
4 min	92.4 ± 9.3	96.7 ± 9.4	0.09
5 min	89.6 ± 8.4	94.5 ± 10.3	0.06
7 min	87.3 ± 8.2	92.4 ± 9.2	0.04
10 min	85.2 ± 7.4	90.6 ± 9.1	0.03

The incidence of adverse effects was generally comparable between the two groups. Bradycardia and hypotension were more frequent in the IV dexmedetomidine group, though the differences did not reach statistical significance. Hypertension and tachycardia occurred infrequently and showed no significant intergroup variation. Nasal irritation was observed exclusively in the intranasal dexmedetomidine group and was statistically significant, reflecting a route-specific local adverse effect.

Overall, IV dexmedetomidine was associated with a higher trend toward hemodynamic adverse events, whereas intranasal administration was associated with local nasal discomfort (Table 4).

Table 4: Adverse Effects

Adverse Event	Group I (IV dexmedetomidine)	Group II (Intranasal dexmedetomidine)	p-value
Bradycardia	6 (12%)	2 (4%)	0.07
Hypotension	5 (10%)	2 (4%)	0.15
Hypertension	1 (2%)	2 (4%)	0.56
Tachycardia	2 (4%)	3 (6%)	0.64
Nasal irritation	0 (0%)	3 (6%)	0.04

Discussion

The present study demonstrated that both intravenous and intranasal dexmedetomidine effectively attenuated the hemodynamic response to laryngoscopy and endotracheal intubation in patients undergoing ENT surgeries. In both groups, heart rate and mean arterial pressure remained within clinically acceptable limits throughout the perioperative period. However, intravenous dexmedetomidine produced a faster onset and a more pronounced reduction in heart rate and MAP during the pre-induction period and early post-intubation phase compared with the intranasal route, indicating superior immediate hemodynamic control. These findings can be attributed to the rapid achievement of peak plasma concentrations following intravenous administration, leading to earlier sympatholysis.

The results of the present study are comparable to those reported by Ankita et al., who observed a progressive decline in heart rate and blood pressure in both intranasal and intravenous dexmedetomidine groups, with a more marked reduction in the intravenous group during the preoperative period [13]. Similarly, Cheung et al. reported a greater fall in heart rate with intravenous dexmedetomidine compared with the intranasal route before induction [14]. The transient rise in heart rate and MAP observed immediately after intubation in both groups in the present study is consistent with the physiological stress response however, values returned to near baseline within 10 minutes, suggesting effective attenuation of the pressor response by dexmedetomidine. This pattern closely parallels the observations of Ankita et al., who also noted maximal increases at intubation with gradual normalization thereafter in both groups [13].

Intranasal dexmedetomidine has gained interest because of its non-invasive nature and favorable pharmacokinetic profile. Yuen et al. demonstrated that intranasal dexmedetomidine at doses of 1–2 μg/kg produced satisfactory sedation without adverse hemodynamic effects in children, while Iriola et al. reported that peak plasma concentrations are achieved approximately 38–45 minutes after intranasal administration, supporting the timing of 40 minutes used in the present study [15,16]. Nevertheless, the slower absorption

through the nasal mucosa explains the comparatively lesser and delayed pre-induction reduction in heart rate and MAP observed in the intranasal group when compared with the intravenous route.

With regard to adverse effects, the present study noted a higher incidence of bradycardia and hypotension in the intravenous group and nasal irritation confined to the intranasal group. These findings are in agreement with those reported by Ankita et al. and Lakshmi et al., who also observed that intravenous dexmedetomidine is more likely to produce bradycardia, whereas intranasal administration may be associated with mild local irritation [13,17]. Overall, the present study confirms that both intravenous and intranasal dexmedetomidine are effective in attenuating peri-intubation hemodynamic responses; however, intravenous dexmedetomidine provides faster and more reliable early control of heart rate and mean arterial pressure, while intranasal dexmedetomidine serves as a safe and useful non-invasive alternative when sufficient premedication time is available.

Conclusion

Both intravenous and intranasal dexmedetomidine were effective in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation in patients undergoing ENT surgeries. However, intravenous dexmedetomidine provided a faster onset and more pronounced early reduction in heart rate and mean arterial pressure, resulting in better peri-intubation hemodynamic stability. Intranasal dexmedetomidine, while slower in onset, offered satisfactory and clinically acceptable hemodynamic control with the advantage of being non-invasive and well tolerated. Thus, intravenous dexmedetomidine may be preferred when immediate hemodynamic control is required, whereas intranasal dexmedetomidine can be considered a useful alternative when adequate premedication time is available.

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