

INTRAVENOUS VERSUS INTRANASAL DEXMEDETOMIDINE: A COMPARATIVE ANALYSIS OF HEMODYNAMIC RESPONSES DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

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ABSTRACT

Objectives: Laryngoscopy and intubation causes a profound sympathetic response, causing significant increase in heart rate (HR) and blood pressure, potentially leading to complications like laryngospasm and bronchospasm, requiring close monitoring and expert anesthesia care. This study evaluated intranasal and intravenous effectiveness of dexmedetomidine in mitigating adverse hemodynamic consequences to laryngoscopy and intubation, aiming to discern the optimal route for hemodynamic stability.

Methods: This double-blinded, randomized study involved 72 adults (18–60 years, American Society of Anesthesiologists I/II) undergoing various surgeries requiring general anesthesia and endotracheal intubation. Group D_{IV} received Inj. Dexmedetomidine 1 µg/kg intravenously as an infusion in 100 mL Normal Saline over 10 min, 40 min before induction of general anesthesia. Group D_{IN} received Inj. Dexmedetomidine 2 µg/kg (1 µg/kg in each nostril) intranasally through atomizer 40 min before induction of general anesthesia. The study's primary objective was to optimize dexmedetomidine's route and dosage for minimizing laryngoscopy's stress response. Secondary outcomes included assessing incidence of any adverse event.

Results: Both study groups showed similar demographics, study duration, and baseline hemodynamics. Hemodynamic parameters decreased significantly 30–40 min post-administration of study drug and after induction ($p < 0.05$). Laryngoscopy and intubation increased HR and mean arterial pressure (MAP) where Group D_{IN} HR (98.86 ± 17.16 bpm), MAP (108.5 ± 15.69 mmHg) showed more increase than Group D_{IV} HR (88.89 ± 9.23 bpm) and MAP (102.36 ± 9.06 mmHg). Group D_{IV} showed greater attenuation of hemodynamic parameters at 1, 3, and 5 min post-laryngoscopy ($p < 0.05$).

Conclusion: Intravenous dexmedetomidine 1 µg/kg is more effective than intranasal dexmedetomidine 2 µg/kg in attenuating the hemodynamic stress response to laryngoscopy and intubation.

Keywords: Dexmedetomidine, Endotracheal intubation, Laryngoscopy response, Intravenous route, Intranasal route.

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INTRODUCTION

General anesthesia renders patients insensitive to pain and oblivious to the proceedings of the surgical operation, ensuring a more comfortable and safer experience [1] which is achieved through a combination of induction agent with intravenous sedatives and analgesics, followed by the use of volatile anesthetics for maintenance [2,3]. The hemodynamic response begins within seconds of direct laryngoscopy and further increases with the passage of the endotracheal tube. After 5 s of direct laryngoscopy, the arterial blood pressure begins to rise, peaking in the next 1–2 min and finally returning to baseline levels within 5 min. Such hemodynamic fluctuations pose a significant risk of myocardial ischemia/infarction in patients with pre-existing cardiac conditions but are generally well-tolerated in healthy individuals [4,5].

Other researchers have investigated numerous pharmacological approaches to blunt the cardiovascular response to laryngoscopy, including pre-treatment with intravenous (i.v.) lidocaine, narcotics, topical anesthesia, beta-blockers, calcium channel blockers, angiotensin-converting enzyme inhibitors, and vasodilators producing varied responses [6–12].

Dexmedetomidine is a highly selective alpha-2-adrenoreceptor agonist with sedative, analgesic, and anxiolytic properties. It exerts its sedative effect through stimulation of the alpha-2-adrenoreceptor in the locus coeruleus, which blunts excitation of the central nervous system. The purported benefits of dexmedetomidine includes a reduction in the surgical stress response [8] by decreasing the plasma catecholamine

levels and catecholamine release [12,13] hence stabilizing intraoperative hemodynamics. It also reduces concurrent need for analgesia.

Dexmedetomidine is also effective when administered via intrathecal, intranasal, oral, intramuscular, and inhalational route [14–16]. Intranasal administration is preferred due to its ease, effectiveness, and high patient acceptance. It offers a pain-free, odorless, and tasteless experience without the need for intravenous (i.v.) infusion [17–20]. The intranasal route offers a safe, fast, and convenient method of drug administration, ensuring reliable effects, easy accessibility, simple application, and enhanced stability during anesthesia. This method allows direct access to the central nervous system through the cribriform plate, bypassing liver metabolism also due to the nasal mucosa's rich vascularization and microvilli. As a non-invasive approach, it enhances patient comfort and compliance, overcoming common challenges associated with injectable [18–20].

Intranasal dexmedetomidine is absorbed slowly, leading to a lower peak concentration in the bloodstream compared to the faster absorption seen with intravenous administration [16] but absorption is governed by other factors including the rate at which mucus clears from the nasal passages, the amount of nasal secretions, and the blood flow in the nasal lining. The volume of the drug administered is also crucial, as some of the solution may move into the throat and be swallowed [17].

Dexmedetomidine provides rapid onset, precise dosage control, 100% bioavailability, and predictable pharmacokinetics, with a metabolic pathway primarily involving UGT1A4-mediated glucuronidation, which

supports its safe use and reliable effects. However, its clinical utility is partially limited due to potential cardiovascular risks, including hypotension, bradycardia, and rare instances of cardiac arrest [21]. Furthermore, rapid infusion of dexmedetomidine intravenously can lead to biphasic changes in mean arterial pressure (MAP), which are undesirable in anesthesia [22].

Hence, we carried out this study with the primary objective to elucidate the optimal route of administering dexmedetomidine for attenuation of stress response to laryngoscopy and endotracheal intubation. The secondary outcome of this study was to assess the incidence of adverse events associated with the drug in both groups.

METHODS

After obtaining approval from the institutional ethical committee (SVIEC/ON/MEDI/BNPG21/OCT/22/69) and after obtaining a written and informed consent from patients, this study was conducted in the Anaesthesia Department of tertiary care hospital on 72 patients aged 18–60 years belonging to grade I and II of The American Society of Anesthesiologists (ASA) physical status classification system who underwent elective surgeries under general anesthesia with endotracheal intubation. The ASA physical status classification system offers a concise framework for perioperative teams to assess a patient's overall health and physical resilience, enabling more accurate predictions of surgical risks and informed decision-making. Patients were excluded if they declined participation, had a history of dexmedetomidine allergy/hypersensitivity, suffered from significant cardiac/respiratory disease, presented with intranasal pathology (ulcers, polyps, and septal deviation) during pre-operative evaluation. Exclusion criteria further included patients receiving β -blockers, or antipsychotics, as well as individuals with anticipated difficult airway management (Mallampati Grade III and IV), individuals having body mass index of $>30 \text{ kg/m}^2$, pregnant and lactating women.

Seventy-two patients who were posted for elective surgeries under general anesthesia with endotracheal intubation were randomized into two groups of 36 patients each with the help of computer-generated randomization in MS Excel and concealment was done using sealed envelope method. The sealed envelope was opened and the drugs were prepared by an anesthesiologist who was not involved in the study. The patients were randomly divided into:

- Group D_{IV} received Inj. Dexmedetomidine $1 \mu\text{g/kg}$ intravenously as an infusion in 100 mL Normal Saline over 10 min, 40 min before induction of general anesthesia. An equivalent volume of normal saline was administered intravenously to D_{IN} group [23].
- Group D_{IN} received Inj. Dexmedetomidine $2 \mu\text{g/kg}$ ($1 \mu\text{g/kg}$ in each nostril) intranasally through atomizer 40 min before induction of general anesthesia. An equivalent volume of normal saline was intranasally administered to the D_{IV} group. Patients were instructed to refrain from sucking or sneezing post intranasal administration of drug.

Throughout the study, a double-blind procedure was maintained, where both the participants as well as the anesthesiologist who assessed the outcome remained unaware of their respective group designations.

Detailed pre-anesthetic evaluation and necessary investigations were done for individual patients. They were kept nil per orally for 6 h and 2 h to solids and clear fluid respectively. They received tablet ranitidine (150 mg) and tablet alprazolam (0.5 mg) as premedication night before surgery.

In the preoperative room on the day of surgery, haemodynamic parameters like heart rate (HR), MAP, systolic blood pressure (SBP), diastolic blood pressure (DBP), and SpO_2 were noted at every 10 min intervals till induction of anesthesia.

In the operating room, all ASA standard monitors were connected to the patients. They were premedicated with Inj. Glycopyrrolate

0.004 mg/kg i.v., Inj. Ondansetron 0.1 mg/kg i.v. and Inj. Tramadol 100 mg i.v. Following premedication, they underwent pre-oxygenation with 100% oxygen via face mask for 3 min.

Standard general anesthesia was administered using propofol at a dosage of 2 mg/kg i.v., supplemented with intravenous succinylcholine at a dose of 2 mg/kg to aid in endotracheal intubation after confirmation of ventilation.

Following tracheal intubation, the patient was put on mechanical ventilation in Volume Control mode, using a closed circuit with a 1:1 mixture of nitrous oxide and oxygen, along with isoflurane. Muscle relaxation was achieved through an initial loading dose of inj. Atracurium 0.5 mg/kg i.v. followed by a maintenance dose of 0.1 mg/kg i.v.

HR, MAP, and oxygen saturation were recorded at specific time points: Before induction, immediately after induction, during laryngoscopy and tracheal intubation, and at 1, 3, 5, and 7 min following intubation by the consultant anesthesiologist who was unaware of the route of drug administration. Any episodes of low blood pressure (mean blood pressure $<60 \text{ mmHg}$), bradycardia (HR $<45 \text{ beats/min}$), or low oxygen levels ($<95\%$) were noted.

After the surgery ended, the effects of anesthesia were reversed by giving glycopyrrolate 0.008 mg/kg and neostigmine 0.05 mg/kg intravenously. Extubation was done after ensuring that the patient's breathing was normal with sufficient tidal volume and rate and after which patients were shifted to the recovery room. Any complications such as nausea, vomiting, and sedation (Ramsay Sedation Score >2) were evaluated after extubation. At the end of the study, the decoding procedure was done.

Statistical analysis

A sample size calculation was performed using open Epi software, based on pilot study examining the difference in MAP between two groups following intubation. Assuming a standard deviation (SD) of 15 mmHg, calculations revealed that 36 participants per group would be necessary to detect a 10 mmHg difference in MAP with 80% statistical power and a 5% probability of Type I error. $N=72$; $n_{D_{IV}}=36$, $n_{D_{IN}}=36$. Fig. 1 depicts the consort flow diagram of the study's methodology.

Variables such as patient demographics, response to laryngoscopy and intubation, post-operative complications, and sedation scores were monitored tabulated, with numerical variables presented as mean and SD.

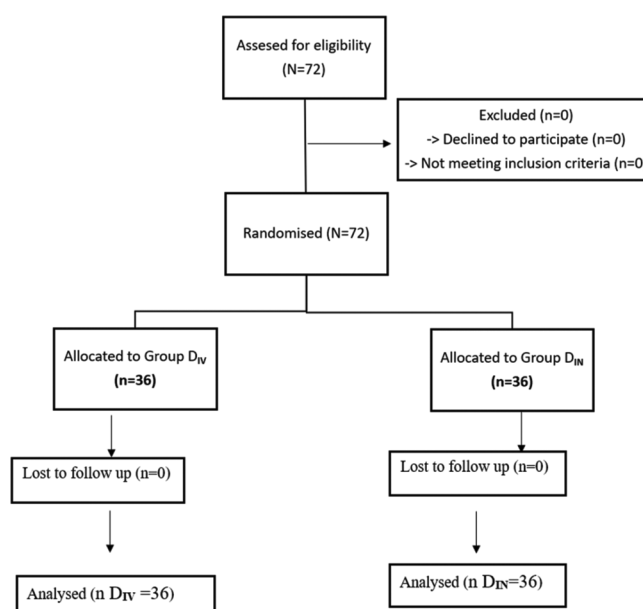


Fig. 1: Consort flow diagram

The data involved in our study research had a normal distribution. MedCalc statistical software version 12.5 was used for comparisons between groups, the unpaired Student's t-test was used for numerical variables, where appropriate. For categorical variables, the Chi-square test was employed.

A $p < 0.05$ was considered statistically significant, while a $p < 0.001$ was regarded as highly significant.

RESULTS

Both the groups were similar in terms of age ($p=0.542$), weight ($p=0.0739$), and duration of surgery in minutes ($p=0.8$), as shown in Table 1. Furthermore, no significant differences were observed in gender ($p=0.6341$) and ASA classification ($p=0.6308$), as shown in Table 2.

Comparison of HR

Mean HRs were comparable at the baseline in both the groups. We found that after test drug administration, HR decreased in both groups from baseline. However, when comparing between the groups, the decrease in HR was significantly more in D_{IV} group at 30, 40 min post-test drug administration and after induction as compared to D_{IN} group ($p=0.0444$, 0.0477 and 0.0497 respectively). During laryngoscopy, HR transiently increased in both groups, but the increase was more significant in the group D_{IN} as compared to the group D_{IV} ($p=0.0030$). After intubation, HR gradually decreased in both groups. However, during intergroup comparison, statistically significant decrease in HR was observed in group D_{IV} as compared to group D_{IN} at 1, 3, and 5 min after laryngoscopy ($p=0.0251$, 0.0292 and 0.0413), respectively, but was non-significant thereafter ($p=0.1662$). Results are summarized in Table 3.

Comparison of SBP

SBPs were comparable between the two groups at baseline. We found that after test drug administration, SBP was decreased in both the groups. There was a significant decrease in SBP in group DIV than group DIN seen at 30 and 40 min after test drug delivery and after induction of anesthesia ($p=0.0447$, 0.0199 and 0.0159 , respectively). SBP briefly rose during laryngoscopy in both groups, but the increase was significantly more pronounced in group DIN than in group DIV ($p=0.0425$).

Post-intubation SBP gradually decreased in both groups, but the decrease in SBP in group D IV was significant as compared to DIN at

Table 1: Inter group Comparison of demographic parameters: mean Age (in years), mean Weight (in kg) and duration of surgery (in minutes)

Parameter	Group D_{IV}	Group D_{IN}	p-value
	Mean \pm SD	Mean \pm SD	
Mean age (years)	40.67 \pm 10.35	39.06 \pm 11.89	0.542
Mean weight (kg)	63.31 \pm 10.85	67.33 \pm 7.68	0.0739
Duration of surgery (in minutes)	190.3	192.5	0.8

D_{IV} : Intravenous, D_{IN} : Intranasal, Mean \pm SD: Mean \pm standard deviation

Table 2: Comparison of gender distribution and ASA classification

Parameter	Group D_{IV}	Group D_{IN}	p-value
	n (%)	n (%)	
Gender: Male	22 (61.11)	19 (52.78)	0.6341
Gender: Female	14 (38.89)	17 (47.22)	
ASA I	23 (63.89)	20 (55.56)	0.6308
ASA II	13 (36.11)	16 (44.44)	

D_{IV} : Intravenous, D_{IN} : Intranasal, N: Absolute number (%) where n=36 in Group D_{IV} and n=36 in Group D_{IN}

1-, 3-, and 5-min post-intubation ($p=0.0276$, 0.0419 and 0.0366), respectively. At 7-min post-intubation, SBP between both the groups showed comparable results ($p>0.05$). The findings are presented in Table 4.

Comparing DBP

Both groups had comparable mean DBP at baseline. Our study revealed a decrease in DBP in both groups following test drug administration. Importantly, we observed a statistically significant decrease in group DIV than in group DIN at 30- and 40-min post-test drug administration and after induction of anesthesia ($p=0.0445$, 0.0398 , and 0.0347 , respectively). During laryngoscopy, DBP temporarily increased in both groups. However, the rise was notably more in Group D_{IN} (Mean \pm SD=95.5 \pm 13.94 mmHg) as compared to Group DIV (Mean \pm SD=89.28 \pm 7.91 mmHg, $p=0.0228$). This was followed by a gradual decline, which was significant in Group DIV at 1 min ($p=0.0265$), 3 min ($p=0.0202$), and 5 min ($p=0.0336$) post-intubation, indicating better blunting of DBP in Group DIV. Afterward, no further significant changes were observed between the groups ($p>0.05$). The key findings are summarized in Table 5.

Comparison of MAP

The initial MAP was comparable between both the groups. We found a decrease in MAP in both groups following test drug administration. Notably, there was a statistically significant decrease in MAP in group DIV than in group DIN at 30- and 40-min post-test drug administration and after anesthesia induction ($p=0.0461$, 0.0340 , and 0.0056 , respectively). There

Table 3: Inter group Comparison of Heart Rate (in beats/minute) at different time intervals

Different time intervals (in minutes)	Heart rate (in beats/minute)		
	Group D_{IV}	Group D_{IN}	p-value
	Mean \pm SD	Mean \pm SD	
Baseline	84.06 \pm 6.62	86.28 \pm 14.36	0.4024
10 min	82.14 \pm 6.49	85.92 \pm 10.33	0.0672
20 min	81.78 \pm 6.45	85.61 \pm 10.7	0.0701
30 min	80.25 \pm 6.81	85 \pm 12.14	0.0444
40 min	79.31 \pm 7.09	84.22 \pm 12.78	0.0477
After induction	79.03 \pm 6.93	83.94 \pm 13.02	0.0497
At laryngoscopy and intubation	88.89 \pm 9.23	98.86 \pm 17.16	0.0030
1 min	87.17 \pm 9.49	94.08 \pm 15.43	0.0251
3 min	84.86 \pm 9.07	91.42 \pm 15.18	0.0292
5 min	81.92 \pm 9.1	87.39 \pm 12.9	0.0413
7 min	81.33 \pm 8.2	84.83 \pm 12.57	0.1662

Table 4: Inter group Comparison of SBP (in mmHg) at different time intervals

Different time intervals (in minutes)	SBP (in mmHg)		
	Group D_{IV}	Group D_{IN}	p-value
	Mean \pm SD	Mean \pm SD	
Baseline	123.19 \pm 5.81	122.72 \pm 8.59	0.7865
10 min	121.89 \pm 6.25	122.11 \pm 9.03	0.9047
20 min	120.81 \pm 6.31	121.56 \pm 8.35	0.6685
30 min	115.56 \pm 6.02	119.17 \pm 8.72	0.0447
40 min	113.97 \pm 6.57	118.25 \pm 8.54	0.0199
After induction	113.06 \pm 5.95	118.22 \pm 11.02	0.0159
At laryngoscopy and intubation	132.06 \pm 5.94	138.33 \pm 17.21	0.0425
1 min	126.97 \pm 5.17	133.78 \pm 17.41	0.0276
3 min	122.83 \pm 5.99	127.81 \pm 13.11	0.0419
5 min	114.86 \pm 6.8	119.03 \pm 9.57	0.0366
7 min	119.86 \pm 5.91	122.78 \pm 10.51	0.1507

was an increase in MAP during the stress response to laryngoscopy and intubation in both groups. The D_{IV} group (Mean±SD=102.36±9.06 mmHg) showed better blunting of the stress response to MAP than the D_{IN} group (Mean±SD=108.5±15.69 mmHg, $p=0.0458$). Following intubation, MAP decreased significantly in Group D_{IV} at 1 min ($p=0.0412$), 3 min ($p=0.0297$), and 5 min ($p=0.0440$) post-intubation, compared to Group D_{IN}. Subsequently, the response remained comparable between the groups. The findings are compiled in Table 6.

Post-operative sedation score

On comparing the MODE of Ramsay Sedation Score in both the groups, it was 2 at extubation and thereafter. And hence there was no statistically significant difference in Ramsay Sedation scores between the groups ($p>0.05$) as shown in Table 7.

Side effects and complications

In our study, there were no observed incidents of hypotension, bradycardia, nausea, vomiting, or bronchospasm noted.

DISCUSSION

Laryngoscopy and endotracheal intubation during general anesthesia trigger a transient yet pronounced sympathetic response, characterized by increased blood pressure and HR. This activation of the sympathetic nervous system can have detrimental hemodynamic effects on the myocardium. To mitigate these effects, adjunctive medications that modulate adrenergic receptors, such as β -blockers, or alternative agents such as opioids, calcium channel blockers, and α_2 agonists, can be employed to blunt the sympathetic response. Hence, minimizing stress from laryngoscopy and intubation is crucial in general anesthesia. This is vital to maintain stable hemodynamics during induction and reversal, preventing complications.

The α_2 -adrenergic agonist dexmedetomidine has demonstrated efficacy as an adjunct for laryngoscopy and intubation, providing sedation, analgesia, and sympatholysis [24]. Its unique mechanism reduces stress response and catecholamine release, enabling smooth induction and intubation. Research comparing intranasal and intravenous administration highlights route-specific benefits: Intranasal dexmedetomidine offers a more gradual onset and reduced systemic exposure, while intravenous dexmedetomidine provides rapid effects and flexible dosing. This comparison underscores the importance of selecting the optimal administration route to maximize benefits and minimize risks in laryngoscopy and intubation.

As the primary outcome of interest of our study, we observed a significant decrease in hemodynamic parameters in the intravenous group of dexmedetomidine D_{IV} at various time intervals after drug administration, during laryngoscopy, intubation, and post-intubation. A comparison of percentage fall from baseline till 7 min after laryngoscopy is depicted in Table 8.

Similar to our study, Kohaf *et al.* [25] research in 2024 comparing dexmedetomidine administration routes revealed intravenous delivery decreased HR and blood pressure significantly. They demonstrated that intravenous dexmedetomidine 1 $\mu\text{g/kg}$ led to a significant decrease in HR compared to intranasal administration 1 $\mu\text{g/kg}$ at 10, 15, and 30 min after administration ($p<0.05$). In addition, SBP in the intravenous group decreased more substantially at 10-, 15-, 30-, and 45-min post-administration ($p<0.05$), with corresponding significant decreases also observed in DBP and MAP ($p<0.05$).

Our study's findings on HR changes align with Niyogi *et al.*, who administered dexmedetomidine intranasally 1 $\mu\text{g/kg}$ and intravenously 0.5 $\mu\text{g/kg}$ to 70 patients undergoing general anesthesia. Both studies showed a significant decrease in HR at 30 and 40 min after administration ($p=0.004$ and 0.001). However, in contrast to our findings, Niyogi *et al.* found minimal and non-significant changes in blood pressure (<20% from baseline, $p>0.05$).

Table 5: Inter group comparison of DBP (in mmHg) at different time intervals

Different time intervals (in minutes)	DBP (in mmHg)		p-value
	Group D _{IV}	Group D _{IN}	
	Mean±SD	Mean±SD	
Baseline	82.97±5.62	82.42±8.75	0.7519
10 min	82.33±6.35	81.42±8.14	0.5986
20 min	81.64±6.12	81.39±8.31	0.8849
30 min	77.11±9.79	81.89±10.03	0.0445
40 min	76.81±10.26	81.69±9.49	0.0398
After induction	76.61±8.43	80.97±8.74	0.0347
At laryngoscopy and intubation	89.28±7.91	95.5±13.94	0.0228
1 min	85.25±7.48	90.67±12.24	0.0265
3 min	80.39±10.63	87.64±14.89	0.0202
5 min	78.64±10.99	84.31±11.21	0.0336
7 min	77.22±9.89	80.83±11.21	0.1518

Table 6: Inter group comparison of MAP (in mmHg) at different time intervals

Different time intervals (in minutes)	MAP (in mmHg)		p-value
	Group D _{IV}	Group D _{IN}	
	Mean±SD	Mean±SD	
Baseline	94.61±7.33	94.78±10.56	0.9370
10 min	92.89±7.6	93.17±8.52	0.8834
20 min	90.42±6.71	92.67±9.56	0.2517
30 min	88.36±10.03	93.64±11.95	0.0461
40 min	87.86±10.2	93.53±11.98	0.0340
After induction	86.39±6.71	92.22±10.23	0.0056
At laryngoscopy and intubation	102.36±9.06	108.5±15.69	0.0458
1 min	99.75±8.74	105.47±14	0.0412
3 min	95.69±9.52	101.86±13.69	0.0297
5 min	92.28±9.66	97.17±10.55	0.0440
7 min	91.22±10.3	93.78±9.67	0.2807

Table 7: Ramsay sedation scores in both the groups at different time intervals

Time after extubation	Group D _{IV}	Group D _{IN}	p-value
	MODE	MODE	
0 min	2	2	N.A.
30 min	2	2	N.A.

Table 8: Comparison of percentage fall from baseline of HR, SBP, DBP, and MAP post-intubation at 7 min where n=36 in Group D_{IV} and n=36 in Group D_{IN}

Parameters	Group D _{IV} (%)	Group D _{IN} (%)
Heart rate	3.24	1.68
SBP	2.70	-0.04
DBP	6.93	1.92
MAP	3.58	1.05

HR: Heart rate, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure

Another study done by Ankita and Kumar in 2023 [26] observed a significant decrease in HR with i.v. dexmedetomidine (1 $\mu\text{g/kg}$) administration who demonstrated a significant decrease in HR in the i.v. dexmedetomidine group compared to the intranasal group (1 $\mu\text{g/kg}$) at 30 and 40 min after administration ($p<0.05$), aligning with

our conclusions of significant decrease in HR in the i.v. group at 30 and 40 min ($p=0.0477$ and 0.0497) respectively.

Consistent with our results, Padmasree and Nelamangala [27] study in 2023 similarly observed significant attenuation of blood pressure and HR following dexmedetomidine administration. They found that intravenous dexmedetomidine $0.5 \mu\text{g/kg}$ resulted in a significant decrease in SBP at 40 min after administration compared to intranasal administration $1 \mu\text{g/kg}$ ($p<0.05$).

Deshmukh *et al.*'s [28] study in January 2025 on 90 participants compared intranasal ($1 \mu\text{g/kg}$, 40-min pre-induction) and intravenous ($0.5 \mu\text{g/kg}$, 10-min pre-induction) dexmedetomidine for hemodynamic stability during laryngoscopy and intubation. Post-operative sedation was similar. The study concluded that both routes are effective and prevented significant fluctuations in hemodynamics.

The study's findings are constrained by potential differences in anesthetic response among various ethnic populations and the single-center research design.

The major limitations of intravenous administration of dexmedetomidine as observed by other researchers includes rapid administration intravenously of dexmedetomidine causing biphasic alteration -involving an initial vasoconstrictive phase (0–15 min) with increased blood pressure and systemic vascular resistance, followed by a secondary vasodilatory phase (30–60 min) with decreased blood pressure, systemic vascular resistance, and potential bradycardia. This unique response is characterized by a rapid increase in mean arterial blood pressure, followed by a gradual decline, posing a challenge in anesthesia management.

Intravenous Dexmedetomidine has been explored for various uses beyond its traditional applications like locoregional anesthesia as adjuvant in transverse abdominis plane block for pain management in cesarean section [27], adjuvants as analgesia in various blocks, comprising safe and effective procedural sedation, neuroprotective efficacy in neuroanesthesia, and multidimensional symptom relief in intensive care patients receiving palliative and hospice care [28-34].

The intranasal route for dexmedetomidine administration provides a comfortable and convenient option, leveraging direct access to the central nervous system while minimizing patient discomfort. Nevertheless, the absorption process is influenced by various factors, including nasal physiology and clearance mechanisms, which may impact the rate and extent of drug absorption [17].

No sedation was observed in either of the groups according to the Ramsay Sedation Score.

Limitations

While our study provides valuable insights but due to small sample size, it necessitates further investigation through larger and more extensive RCTs. Since this is a hospital-based study, it has limited generalisability. Second, we have considered hemodynamic changes up to 7 min only after intubation so effect of dexmedetomidine through different routes on hemodynamic parameters is not considered throughout the intraoperative time.

CONCLUSION

We came to the conclusion that administering $1 \mu\text{g/kg}$ of dexmedetomidine intravenously would be more effective and would cause superior blunting of the hemodynamic response to laryngoscopy and intubation, with a statistically significant difference than $2 \mu\text{g/kg}$ intranasally ($p<0.05$). Intranasal is an emerging route of administration which warrants additional research to optimize its use in reducing hemodynamic responses during laryngoscopy and intubation.

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AUTHORS' CONTRIBUTIONS

Sara Mary Thomas: Conceived and designed the study, developed the methodology, conducted the experiments, analyzed the data, provided critical intellectual input, and ensured the accuracy, reliability, and integrity of the research findings. Paras Anand*: Assisted in study design, data acquisition, and provided input in data interpretation and drafted the manuscript. literature review, and refined the scientific content. Dushyant BharatBhai Chavda: Contributed to methodology development, data validation, statistical analysis, and resources. Kalpesh Patil: Provided overarching guidance and supervision, assisted in finalizing the manuscript, ensuring adherence to journal guidelines, and contributed to critical discussions and interpretation of findings.

CONFLICTS OF INTERESTS

Nil.

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