e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2024; 16(10); 271-281

Original Research Article

Comparative Study between Intranasal and Intravenous Dexmedetomidine as a Premedication to Attenuate Hemodynamic Responses to Laryngoscopy and Endotracheal Intubation in Elective Surgery

Raju Kumar Choudhary[1], Ajay Kumar[2], Hari Damodar Singh[3], Subhojit Das[4]

[1]PG-Student, Department of Anaesthesiology & critical care, Darbhanga medical college & hospital, Laheriasarai, Darbhanga, Bihar, India

[2]Associate professor, Department of Anaesthesiology & critical care, Darbhanga medical college & hospital, Laheriasarai, Darbhanga, Bihar, India

[3]Professor and HOD, Department of Anaesthesiology & critical care, Darbhanga medical college & hospital, Laheriasarai, Darbhanga, Bihar, India

[4]PG -Student, Department of Anaesthesiology & critical care, Darbhanga medical college & hospital, Laheriasarai, Darbhanga, Bihar, India

Received: 12-08-2024 / Revised: 15-09-2024 / Accepted: 22-10-2024
Corresponding Author: Dr. Ajay Kumar
Conflict of interest: Nil

Abstract

Background: The induction of general anaesthesia and endotracheal intubation can activate the sympathetic nervous system, resulting in haemodynamic instability, notably tachycardia and hypertension, which presents a difficulty for patients with a history of hypertension. Pharmacological treatments such as dexmedetomidine, an alpha-2 adrenergic agonist, are employed to regulate these responses. This study evaluates the effectiveness of intranasal (IN) and intravenous (IV) dexmedetomidine in preserving haemodynamic stability throughout the induction of anaesthesia.

Methodology: This prospective, randomised, double-blind trial was performed at Darbhanga Medical College and Hospital in Bihar, India. A total of 120 patients (aged 18–55, ASA grade I/II) scheduled for elective surgery with general anaesthesia and endotracheal intubation were included. Participants were randomly assigned to two groups: Group A received intranasal dexmedetomidine at a dosage of 0.75 mcg/kg, whereas Group B received intravenous dexmedetomidine at the same dosage. Haemodynamic measures (heart rate, blood pressure, mean arterial pressure) and the Ramsay Sedation Scale (RSS) were documented at many times throughout the perioperative period.

Results: No significant changes were seen in systolic and diastolic blood pressure between the two groups ($p > 0.05$). Group A exhibited a markedly reduced heart rate at 20 minutes following injection ($p < 0.001$). The respiratory rate in Group A was markedly elevated at 20, 25, and 30 minutes ($p < 0.05$). No notable disparities in mean arterial pressure were seen across the groups.

Conclusion: Both intranasal and intravenous dexmedetomidine successfully mitigate sympathetic reactions during laryngoscopy without causing considerable haemodynamic instability. The intranasal approach shown benefits in lowering heart rate, indicating its potential as a feasible option for premedication in anaesthesia induction. Additional research is necessary to validate these findings in more extensive populations.

Keywords: Anaesthesia induction, Dexmedetomidine, Haemodynamic stability, Intranasal administration, Intravenous administration

Introduction

The sympathetic nervous system is stimulated during the induction period of general anaesthesia by intubation, potentially resulting in haemodynamic instability, including tachycardia and hypertension [1]. The heightened reflex haemodynamic responses in individuals with a history of hypertension pose a considerable issue for anaesthesiologists [2-5]. Anaesthesiologists have utilised several pharmacological agents to mitigate the haemodynamic response during airway manipulation in patients undergoing elective surgery under general anaesthesia. Limited research has assessed and contrasted the efficacy of antihypertensive agents such as nitroglycerin,

labetalol, and hydralazine, alongside opioids including remifentanil, fentanyl, and alfentanil, in managing blood pressure and heart rate, while also analysing the incidence of opposing effects or complications [6].

Numerous studies have examined the optimal drugs for individuals undergoing elective surgery that provide haemodynamic stability while providing enough sedation. Each experiment, however, was limited to the comparison of two or three drugs, and the results differed. Premedication is primarily aimed at alleviating anxiety, enhancing sedation, and stabilising haemodynamics. Introduction of two variables [7]. In premedication, sedative, analgesic, antisialagogue, and anxiolytic qualities are favoured. Optimal attributes would include a short half-life, rapid onset, non-parenteral delivery, and absence of negative haemodynamic effects [8]. Dexmedetomidine is a highly selective, rapid-acting alpha-2 agonist that possesses analgesic, sedative, and anxiolytic properties, however it does not induce respiratory depression. It is the ideal medication to administer before anaesthesia to alleviate any anxiety or apprehension. It is established that intravenous (IV) dexmedetomidine effectively mitigates the laryngoscopic stress response prior to operation [9].

Adverse haemodynamic outcomes, including a reduction in heart rate (HR), a decline in blood pressure, or even cardiac collapse, have likely been recorded. Intravenous dexmedetomidine has induced sedation, delaying recovery [10]. Alternative delivery systems have been proposed to alleviate the adverse effects of dexmedetomidine, rather than depending only on rapid IV infusion. Furthermore, dexmedetomidine is effective when administered orally, intramuscularly, or intranasally. Intranasal administration is more practical and effective than alternative methods [11]. Patients have reported that intranasal delivery of dexmedetomidine is well tolerated. Recent studies in paediatrics have shown that premedication with dexmedetomidine, compared to conventional premedication, yields favourable peri-operative results [12]. Research examinations do not provide clear evidence associating the sedative prodrug dexmedetomidine to prolonged anaesthesia recovery.

It is suggested that IND would be more efficacious in attaining stable haemodynamics under general anaesthesia compared to the intravenous route in patients undergoing elective surgery under general anaesthesia. We designed this study to evaluate if IND exhibited significantly superior haemodynamic characteristics with sufficient sedation compared to the intravenous route.This study was directed to evaluate the effects of dexmedetomidine administered by intranasal and intravenous methods. This study evaluated the impact of intranasal vs intravenous administration of dexmedetomidine on haemodynamic parameters, including heart rate (HR), mean arterial pressure (MAP), systolic and diastolic blood pressure, and the Ramsay sedation score.

Methodology

Study Area

This prospective randomised, double-blind controlled experiment was conducted over two years at the Department of Anaesthesiology and Critical Care at Darbhanga Medical College and Hospital, Laheriasarai, Darbhanga, Bihar, India. Approval from the institutional ethics committee was obtained before the commencement of the inquiry.

Sample Size

The study had a total sample size of 120 patients.

Inclusion and Exclusion Criteria

The research encompassed patients of both genders, aged 18 to 55 years, categorised as ASA Grade I or II, undergoing elective surgery with general anaesthesia and endotracheal intubation. Patients were excluded if they declined participation, had a documented allergy or hypersensitivity to Dexmedetomidine, or presented with serious cardiac, renal, neurological, hepatic, or respiratory disorders. Furthermore, people classified outside ASA Grades I or II, those anticipated to experience difficult intubation, or those presenting with intranasal pathology (including nasal ulcers, polyps, or septal deviation) were excluded. Patients were also excluded from the trial if the length of laryngoscopy and intubation over 15 seconds.

Intervention

Group	Intervention
Group A	Administered undiluted intranasal Dexmedetomidine (0.75 mcg/kg), derived from a parenteral formulation (100 mcg/mL) or IV Normal saline. Each nostril received half of the entire estimated amount administered using a syringe. The patient was advised against sneezing or swallowing following the administration of the medication.
Group B	Received intravenous Dexmedetomidine (0.75 mcg/kg) [diluted in 20 mL syringe with normal saline] through an infusion pump over 20 min before induction or IV Normal saline.

Procedure

The study's methodology focused standardised preoperative preparation and anaesthesia administration. Patients underwent preoperative preparation by adhering to a nil per os (NPO) condition for 6 hours before surgery. Upon arrival in the preoperative area, an 18/20G peripheral intravenous cannula was placed for venous access, and the patient was preloaded with Ringer's lactate at a rate of 10 mL/kg/hr, later adjusted to a maintenance rate of 6 mL/kg/hr. She had routine ASA monitoring, which included continuous assessment of heart rate, ECG, non-invasive blood pressure (systolic, diastolic, and mean arterial pressure), and oxygen saturation (SpO2). Basal haemodynamic parameters were recorded before the administration of premedication. Following the delivery of premedication, the patient was administered Dexmedetomidine. Anaesthesia induction commenced with pre-oxygenation, followed by IV Propofol (2 mg/kg) for sedation and IV Vecuronium (0.1 mg/kg) for muscular relaxation. Glycopyrrolate and Alprazolam were withheld to prevent interaction with Dexmedetomidine. To sustain anaesthesia following endotracheal intubation, a combination of oxygen, air, and isoflurane was employed to achieve a minimum alveolar concentration (MAC) of 1 promptly.

Outcome Parameters

The subsequent criteria were carefully recorded during the investigation. Haemodynamic evaluation, encompassing systolic blood pressure, mean arterial pressure, diastolic blood pressure, respiration rate, heart rate, and sedation levels, was performed at various intervals (0, 5, 10, 15, 20, 25, and 30 minutes) subsequent to medication administration throughout the preoperative phase. Laryngoscopy was executed with a Macintosh blade, followed by endotracheal intubation with a suitably sized cuffed disposable ET tube by a seasoned anaesthesiologist once the train of four (TOF) count hit zero. The intubation duration was restricted to 15 seconds, excluding data from unsuccessful attempts in the study. Vital signs were measured continuously at specified intervals post-intubation (0, 1, 2, 5, 8, and 10 minutes). SpO2 was continually assessed with a pulse oximeter, whereas intraoperatively, end-tidal CO2 (EtCO2) and neuromuscular monitoring were documented. The Ramsay Sedation Scale (RSS) was employed to assess sedation levels at identical time intervals. Surgical intervention was not conducted until 10 minutes after intubation.

Statistical Analysis

Data from patients undergoing elective surgery with general anaesthesia were organised in tabular format using Microsoft Excel 365 and analysed using SPSS version 24. Continuous data (age, BMI, SBP, DBP, MAP, and heart rate) were presented as mean ± SD, and the differences between group A (intranasal) and group B (intravenous) were evaluated using an unpaired t-test. Categorical data (age group, gender, operation type, complications) were presented as percentages and frequencies, and analysed using 'chi-square or Fisher's exact test'. A p-value of less than 0.05 was deemed statistically substantial.

Results

The table 1 illustrates the age distribution of Group A (Intranasal Dexmedetomidine) with Group B (Intravenous Dexmedetomidine). In Group A, the predominant age group is 18-30 years (35%), followed by 41-50 years (31.67%). In Group B, the majority belong to the 18-30 years group (53.33%). The age ranges 31-40 and 51-55 are equally represented in both categories. The 'Chi-square test' yields a p-value of 0.12, signifying no statistically significant variance in age distribution between the two groups.

Table 1: Comparison of Age between Group A (IND) and Group B (Intravenous Dexmedetomidine)

Age Group	No. of Patients (%)		P Value (Chi- Square test)
	Group A	Group B	
18-30	21 (35.00)	32 (53.33)	0.12
31-40	11 (18.33)	11 (18.33)	
41-50	19 (31.67)	9 (15.00)	
51-55	9 (15.00)	8 (13.33)	

The table 2 outlines the gender distribution between Group A (Intranasal Dexmedetomidine) and Group B (Intravenous Dexmedetomidine). In Group A, 38.33% of patients are male and 61.67% are female; in Group B, 25% are male and 75% are female. The 'Fisher's Exact Test' produces a p-value of 0.17, signifying no statistically substantial disparity in gender distribution between the two groups.

Table 2. Comparison of Gender between Group A (IND) and Group B (Intravenous Dexmedetomidine)

Gender	No. of Patients (%)		P Value (Fisher's Exact Test)
	Group A	Group B	
Male	23 (38.33)	15 (25.00)	0.17
Female	37 (61.67)	45 (75.00)	

Table 3 compares heart rate (HR) between Group A (Intranasal Dexmedetomidine) and Group B (Intravenous Dexmedetomidine) over various time periods. The baseline heart rates were similar in both groups, with Group A at 83.34 ± 9.87 bpm and Group B at 84.56 ± 9.27 bpm ($p = 0.49$). No significant changes in heart rate (HR) were seen at consecutive time periods until 20 minutes, at which point Group A exhibited a substantially lower HR (73.89 ± 4.27 bpm) compared to Group B (78.59 ± 2.39 bpm, $p < 0.001$). At the remaining time intervals (5, 10, 15, 25, and 30 minutes), the variances in heart rate between the two groups were not statistically substantial ($p > 0.05$). The only significant difference came at 20 minutes, when Group A displayed a lower heart rate compared to Group B.

Table 3. Comparison of HR between Group A (IND) and Group B (Intravenous Dexmedetomidine)

Time	Heart Rate (bpm) in Mean ± SD		P Value (Unpaired test)
	Group A	Group B	
Baseline	83.34 ± 9.87	84.56 ± 9.27	0.49
5 Minutes	79.69 ± 8.33	78.86 ± 9.37	0.61
10 Minutes	76.26 ± 8.97	75.37 ± 9.08	0.59
15 Minutes	74.12 ± 9.49	72.36 ± 8.74	0.16
20 Minutes	73.89 ± 4.27	78.59 ± 2.39	<0.001
25 Minutes	73.17 ± 5.86	74.69 ± 5.39	0.14
30 Minutes	72.29 ± 9.73	71.38 ± 8.98	0.6
At induction	71.17 ± 9.68	70.34 ± 8.95	0.63
1 Minutes	84.29 ± 9.82	85.66 ± 10.03	0.45

2 Minutes	81.65 ± 9.72	82.83 ± 9.98	0.51
5 Minutes	78.37 ± 8.26	79.52 ± 9.63	0.48
8 Minutes	78.18 ± 9.03	80.08 ± 9.19	0.26
10 Minutes	78.82 ± 9.37	81.13 ± 8.32	0.16

Table 4 analyses systolic blood pressure (SBP) between Group A (Intranasal Dexmedetomidine) and Group B (Intravenous Dexmedetomidine) at several time intervals. The baseline systolic blood pressure levels were comparable across the two groups, with the initial group at 127.31 ± 10.85 mmHg and Group B at 125.83 ± 9.40 mmHg ($p = 0.43$). No substantial changes in SBP were seen between the two groups at any time point during the study, including 5, 10, 15, 20, 25, and 30 minutes, as well as during the induction and post-intubation periods ($p > 0.05$ for all comparisons). The data indicate that the two administration strategies exhibited similar effects on SBP over the observation period.

Table 4. Comparison of SBP between Group A (IND) and Group B (Intravenous Dexmedetomidine)

Time	**SBP (mmHg) in Mean ± SD**		**P Value**
	Group A	**Group B**	
Baseline	127.31 ± 10.85	125.83 ± 9.40	0.43
5 Minutes	124.15 ± 10.33	122.82 ±8.17	0.44
10 Minutes	122.57 ± 10.27	119.79 ± 9.23	0.12
15 Minutes	119.29 ± 10.60	117.98 ± 9.55	0.48
20 Minutes	117.36 ± 10.47	116.89 ± 9.34	0.80
25 Minutes	115.27 ± 10.21	115.91 ± 9.59	0.72
30 Minutes	116.39 ± 11.92	115.35 ± 9.76	0.60
At induction	109.94 ± 8.29	109.57 ± 7.86	0.80
1 Minutes	124.08 ± 10.42	121.54 ± 7.90	0.14
2 Minutes	120.14 ± 10.24	118.77 ± 8.66	0.43
5 Minutes	115.20 ± 9.19	113.62 ± 7.71	0.31
8 Minutes	114.77 ± 8.72	113.71 ±7.72	0.48
10 Minutes	1114.40 ± 8.01	114.94 ± 7.73	0.71

Table 5 compares diastolic blood pressure (DBP) between Group A (Intranasal Dexmedetomidine) and Group B (Intravenous Dexmedetomidine) at several time intervals. At baseline, diastolic blood pressure (DBP) was comparable across the two groups, with Group A exhibiting 78.86 ± 6.70 mmHg and Group B showing 77.63 ± 7.82 mmHg ($p = 0.36$). No significant variations in DBP were seen between the two groups at any time point, including 5, 10, 15, 20, 25, and 30 minutes, as well as throughout the induction and post-intubation phases ($p > 0.05$ for all comparisons). The results demonstrate that both delivery methods produced comparable effects on DBP over the observation period.

Table 5. Comparison of DBP between Group A (IND) and Group B (Intravenous Dexmedetomidine)

Time	DBP (mmHg) in Mean ± SD		P Value
	Group A	Group B	
Baseline	78.86 ± 6.70	77.63 ± 7.82	0.36
5 Minutes	74.40 ± 6.34	74.85 ± 8.45	0.74
10 Minutes	72.47 ± 5.82	72.97 ± 8.51	0.71
15 Minutes	70.78 ± 6.82	71.00 ± 8.55	0.88
20 Minutes	69.85 ± 7.61	70.89 ± 7.98	0.47
25 Minutes	68.57 ± 7.59	70.19 ± 7.56	0.24
30 Minutes	67.79 ± 7.56	69.71 ± 7.33	0.16
At induction	65.38 ± 5.33	66.14 ± 6.90	0.5
1 Minutes	74.34 ± 5.72	75.65 ± 6.66	0.54
2 Minutes	71.20 ± 5.04	71.91 ± 6.38	0.5
5 Minutes	68.57 ± 5.11	67.11 ± 5.11	0.12
8 Minutes	68.17 ± 5.47	66.62 ± 4.75	0.1
10 Minutes	66.45 ± 5.47	67.05 ± 6.01	0.54

Table 6 shows MAP between Group A (Intranasal Dexmedetomidine) and Group B (Intravenous Dexmedetomidine) at different time intervals. At baseline, the MAP was comparable across the two groups, measuring 93.75 ± 7.01 mmHg for Group A and 92.18 ± 8.25 mmHg for Group B ($p = 0.26$). No substantial variations in MAP were seen between the two groups at any time point, including 5, 10, 15, 20, 25, and 30 minutes, as well as throughout induction and post-intubation periods ($p > 0.05$ for all comparisons). The data indicate that both routes of Dexmedetomidine delivery produced similar effects on MAP.

Table 6. Comparison of MAP between Group A (IND) and Group B (Intravenous Dexmedetomidine)			
Time	**MAP (mmHg) in Mean ± SD**		**P Value**
	Group A	**Group B**	
Baseline	93.75 ± 7.01	92.18 ± 8.25	0.26
5 Minutes	89.94 ± 7.50	89.46 ± 8.49	0.74
10 Minutes	87.56 ± 7.67	87.68 ± 8.39	0.93
15 Minutes	85.89 ± 7.97	85.80 ± 8.09	0.95
20 Minutes	83.86 ± 8.98	84.99 ± 5.99	0.42
25 Minutes	83.59 ± 8.63	83.67 ± 6.67	0.95
30 Minutes	83.08 ± 8.47	83.64 ± 6.78	0.69
At induction	78.85 ± 5.50	79.71 ± 7.05	0.46
1 Minutes	90.88 ± 6.72	90.17 ± 5.74	0.53
2 Minutes	87.74 ± 5.94	87.26 ± 6.92	0.68
5 Minutes	84.58 ± 6.17	83.06 ± 5.73	0.16
8 Minutes	84.63 ± 5.18	82.80 ± 5.20	0.06
10 Minutes	82.17 ± 4.72	83.75 ± 6.43	0.13

Table 7 compares the respiratory rate (RR) of Group A (Intranasal Dexmedetomidine) with that of Group B (Intravenous Dexmedetomidine) at different time intervals. At baseline, the respiratory rate was comparable in both groups (11.44 ± 1.03 breaths per minute for Group A and 11.96 ± 1.45 breaths per minute for Group B, $p = 0.11$). Notable disparities in respiratory rate (RR) between the two groups were detected at 20 minutes ($p = 0.002$), 25 minutes ($p < 0.001$), and 30 minutes ($p = 0.03$), with Group A exhibiting somewhat elevated RR values compared to Group B. No significant variations in RR were seen at subsequent time intervals, including 5, 10, 15, and induction ($p > 0.05$). The data indicate that although no significant differences were seen initially, Group A had a markedly elevated respiratory rate later in the research relative to Group B.

Table 7. Comparison of Respiratory Rate between Group A (IND) and Group B (Intravenous Dexmedetomidine)			
Time	**RR in Mean ± SD**		**P Value**
	Group A	**Group B**	

Baseline	11.44 ± 1.03	11.96 ± 1.45	0.11
5 Minutes	11.31 ±0.94	11.63 ± 0.88	0.18
10 Minutes	11.55 ± 0.62	11.21 ± 0.92	0.1
15 Minutes	11.56 ± 0.83	11.32 ± 0.81	0.26
20 Minutes	11.48 ± 0.36	11.08 ± 0.56	0.002
25 Minutes	11.73 ± 0.46	11.05 ± 0.68	<0.001
30 Minutes	11.68 ± 0.83	11.09 ± 1.21	0.03
At induction	11.53 ± 0.84	11.19 ± 1.15	0.2
1 Minutes	11.65 ± 0.59	11.6 ± 0.6	0.75
2 Minutes	11.47 ± 0.6	11.65 ± 0.53	0.26
5 Minutes	11.69 ±0.89	11.64 ± 0.61	0.72
8 Minutes	11.49 ± 0.79	11.59 ±0.81	0.5
10 Minutes	11.61 ± 0.73	11.57 ± 0.78	0.77

Discussion

This randomised, double-blind research examines the haemodynamic effects of intranasal (IN) and intravenous (IV) administration of dexmedetomidine (DEX) as premedication to reduce stress reactions during laryngoscopic intubation [13]. The study is to assess the effectiveness of both methods in mitigating sympathetic activation that commonly results in increased heart rate and MAP during intubation. Previous study by Niyogi et al. (2019) demonstrated that both intranasal DEX (1 μg/kg) given 40 minutes before induction and intravenous DEX (0.5 μg/kg) resulted in comparable decreases in laryngoscopic stress responses, without notable elevations in blood pressure or heart rate. In both cohorts, haemodynamic parameters fluctuated within ±20% of baseline values. Nonetheless, the sedation score was significantly elevated in the IV group, signifying that IV treatment produced more sedation compared to the intranasal route [13].

Laryngoscopic intubation, a prevalent operation performed under general anaesthesia, elicits a deleterious sympathetic response, characterised by a substantial elevation in heart rate and MAP due to catecholamine release. This reaction reaches its maximum after 1-2 minutes following intubation and subsides within 5-10 minutes. Effective premedication must alleviate this sympathetic activation. Historically, many therapies including opioids (e.g., fentanyl), adrenergic antagonists (e.g., esmolol), and local anaesthetics (e.g., IV lidocaine) have been employed to manage these haemodynamic alterations. Nevertheless, these medicines do not entirely eradicate the stress reaction [14,15].

Dexmedetomidine, a centrally acting α2 adrenergic agonist, has become notable for its sedative, anxiolytic, analgesic, and sympatholytic properties. In contrast to other sedatives, DEX facilitates conscious sedation without inducing respiratory depression, rendering it optimal for premedication [16]. DEX functions by suppressing noradrenaline production, thereby diminishing sympathetic activity, including tachycardia and hypertension, frequently observed during intubation [17,18].

The intranasal method has several benefits compared to intravenous administration, such as simplicity of application, non-invasive delivery, and direct access to the central nervous system via the

nasal mucosa's extensive vascular network. This enables DEX to circumvent the liver's first-pass metabolism, leading to rapid systemic absorption. Besides serving as an effective sedative and sympatholytic drug, DEX also promotes cooperative behaviour during post-operative neurological assessments [19-21]. Keniya et al. (2011) provide more evidence for the effectiveness of DEX in reducing the sympathoadrenal response to tracheal intubation [19]. Their investigation shown that DEX decreased the elevation in SBP and DBP by around 8-11% during intubation, whereas the control group saw rises of 40% and 25%, respectively. In the DEX group, heart rate increased by 7%, but the control group had a 21% increase [20,21].

Nonetheless, a disadvantage of IV DEX is its sedative properties, which may result in bradycardia and hypotension, especially at elevated dosages (>0.5 μg/kg). The rapid intravenous injection of DEX may elicit a biphasic response in mean arterial pressure (MAP), potentially resulting in adverse effects [22]. To mitigate these adverse effects, current research is investigating alternate administration methods, such as prolonged intravenous infusions and intranasal delivery. Intranasal DEX demonstrates comparable haemodynamic effects to slow IV infusion, while offering the advantage of a slower onset, thereby mitigating the deleterious cardiovascular effects associated with rapid IV boluses.

Research indicates that IN DEX is especially efficacious in paediatric populations, proving to be more effective than alternative sedative drugs in facilitating sleep and sedation [30]. In adult patients, IN DEX has demonstrated a reduction in the requirement for supplementary anaesthetic drugs during both local and general anaesthesia [23]. A recent study by HrishiPA et al. shown that IN DEX markedly decreased haemodynamic instability during transnasal transphenoidal skull base surgery, without significant alterations in heart rate or blood pressure [24].

Further study, including those conducted by Wang et al. (2014), validated that IN DEX substantially reduces the increase in MAP during the intubation reaction [25]. Moreover, Yuen et al. (2010) noted that IN DEX elicited drowsiness after 25-30 minutes, with effects persisting for 35-100 minutes, rendering it appropriate for operations necessitating sedation during the perioperative phase [26]. Research indicates that delivering IN DEX 25-40 minutes prior to surgery yields the most favourable outcomes [25]. Chengxiang Lu et al. (2016) corroborated these findings by demonstrating that DEX effectively mitigated tachycardia and hypertension post-intubation, with those receiving DEX displaying less cardiovascular changes compared to those administered a placebo [27]. The findings of this investigation corroborate that both IN and IV DEX efficiently attenuate the haemodynamic spikes commonly observed during laryngoscopic intubation.

Li et al. (2018) investigated the pharmacokinetics and pharmacodynamics of IN DEX, demonstrating that intranasal administration exhibits a prolonged start and duration of activity relative to intravenous administration, which facilitates more rapid entry of the drug into the circulation [28]. Although intravenous dexamethasone achieves elevated plasma concentrations rapidly, intranasal dexamethasone provides a more delayed start, hence mitigating the risk of side effects including hypertension and bradycardia linked to fast intravenous delivery.

This study concludes that both IN and IV dexmedetomidine effectively mitigate haemodynamic stress responses during laryngoscopic intubation. The intravenous method produces enhanced sedation, but the intranasal route offers a practical option owing to its simplicity, low adverse effects, and similar effectiveness. Both methods significantly lower central catecholamine levels, alleviating the sympathetic response linked to intubation. Considering the benefits of the intranasal technique, especially regarding its simplicity and less cardiovascular adverse effects, IN DEX may be seen as a superior choice for premedication in several therapeutic settings.

Conclusion

In the current study, both intranasal and intravenous dexmedetomidine were found to be effective in attenuating sympathetic responses during laryngoscopic intubation, with no substantial variances in blood pressure or heart rate between the two groups. The intranasal route demonstrated a more favorable heart rate reduction at 20 minutes, suggesting its potential advantages in providing faster onset and ease of administration. Both routes maintained haemodynamic stability without inducing significant adverse effects. However, while the intranasal method appears promising, further large-scale, controlled studies are warranted to validate its comparative effectiveness and establish it as a viable alternative to intravenous dexmedetomidine in clinical practice.

References

1. King BD, Harris LC, Greifenstein FE, Elder JD, Dripps RD. Reflex circulatory responses to direct laryngoscopy and tracheal intubation performed during general anesthesia. The Journal of the American Society of Anesthesiologists. 1951 Sep 1;12(5):556-66.
2. Low JM, Harvey JT, Prys-Roberts C, Dagnino J. Studies of anaesthesia in relation to hypertension: VII: Adrenergic responses to

laryngoscopy. British Journal of Anaesthesia. 1986 May 1;58(5):471-7.
3. Prys-Roberts C, Meloche R, Foex P, Ryder A. Studies of anaesthesia in relation to hypertension I: cardiovascular responses of treated and untreated patients. BJA: British Journal of Anaesthesia. 1971 Feb 1;43(2):122-37.
4. Prys-Roberts C, Greene LT, Meloche R, Foex P. Studies of anaesthesia in relation to hypertension II: haemodynamic consequences of induction and endotracheal intubation. British Journal of Anaesthesia. 1971 Jun 1;43(6):531-47.
5. Lawes EG, Downing JW, Duncan PW, Bland B, Lavies N, Gane GA. Fentanyl-droperidol supplementation of rapid sequence induction in the presence of severe pregnancy-induced and pregnancy-aggravated hypertension. British Journal of Anaesthesia. 1987 Nov 1;59(11):1381-91.
6. Yoon SW, Choi GJ, Seong HK, Lee MJ, Kang H. Pharmacological strategies to prevent haemodynamic changes after intubation in parturient women with hypertensive disorders of pregnancy: A network meta-analysis. International Journal of Medical Sciences. 2021;18(4):1039.
7. Padmasree MK, Nelamangala K. A comparative study between intranasal and intravenous dexmedetomidine and hemodynamic responses during endotracheal intubation. Cureus. 2023 Feb;15(2).
8. Malde AD. Dexmedetomidine as premedication in children: Status at the beginning of 2017. Indian Journal of Anaesthesia. 2017 Feb 1;61(2):101-2.
9. Sebastian B, Talikoti AT, Krishnamurthy D. Attenuation of haemodynamic responses to laryngoscopy and endotracheal intubation with intravenous dexmedetomidine: A comparison between two doses. Indian journal of anaesthesia. 2017 Jan 1;61(1):48-54.
10. Bharati S, Pal A, Biswas C, Biswas R. Incidence of cardiac arrest increases with the indiscriminate use of dexmedetomidine: a case series and review of published case reports. Acta Anaesthesiologica Taiwanica. 2011 Dec 1;49(4):165-7.
11. Yuen VM, Irwin MG, Hui TW, Yuen MK, Lee LH. A double-blind, crossover assessment of the sedative and analgesic effects of intranasal dexmedetomidine. Anesthesia & Analgesia. 2007 Aug 1;105(2):374-80.
12. Talon MD, Woodson LC, Sherwood ER, Aarsland A, McRae L, Benham T. Intranasal dexmedetomidine premedication is comparable with midazolam in burn children undergoing reconstructive surgery. Journal of burn care & research. 2009 Jul 1;30(4):599-605.
13. Gupta M, Rohilla R, Gupta P, Tamilchelvan H, Joshi U, Kanwat J. Nebulized dexmedetomidine for attenuating hemodynamic response to laryngoscopy and endotracheal intubation in adult patients undergoing surgeries under general anaesthesia: a systematic review and meta-analysis of randomized controlled trials. BMC anesthesiology. 2023 Dec 11;23(1):406.
14. Khan FA, Ullah H. Pharmacological agents for preventing morbidity associated with the haemodynamic response to tracheal intubation. Cochrane Database of Systematic Reviews. 2013(7).
15. Figueredo E, Garcia-Fuentes EM. Assessment of the efficacy of esmolol on the haemodynamic changes induced by laryngoscopy and tracheal intubation: a meta-analysis. Acta Anaesthesiologica Scandinavica. 2001 Sep;45(8):1011-22.
16. Yabeck-Karam VG, Aouad MM. Perioperative uses of dexmedetomidine. Middle East Journal of Anesthesiology. 2006 Oct 1;18(6):1043.
17. Karhuvaara S, Kallio A, Salonen M, Tuominen J, Scheinin M. Rapid reversal of alpha 2-adrenoceptor agonist effects by atipamezole in human volunteers. British journal of clinical pharmacology. 1991 Feb;31(2):160-5.
18. Gerlach AT, Dasta JF. Dexmedetomidine: an updated review. Annals of Pharmacotherapy. 2007 Feb;41(2):245-54.
19. Keniya VM, Ladi S, Naphade R. Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anaesthetic requirement. Indian journal of anaesthesia. 2011 Jul 1;55(4):352-7.
20. Tanskanen PE, Kyttä JV, Randell TT, Aantaa RE. Dexmedetomidine as an anaesthetic adjuvant in patients undergoing intracranial tumour surgery: a double-blind, randomized and placebo-controlled study. BJA: British Journal of Anaesthesia. 2006 Nov 1;97(5):658-65.
21. 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 journal of anaesthesia. 2019 Nov 1;63(11):915-23.
22. Sağıroğlu AE, Celik M, Orhon Z, Yüzer S, Sen B. Dıfferent doses of dexmedetomidine on controlling haemodynamic responses to tracheal intubation. Internet J Anesthesiol. 2010;27(2).
23. Sulaiman S, Karthekeyan RB, Vakamudi M, Sundar AS, Ravullapalli H, Gandham R. The effects of dexmedetomidine on attenuation of stress response to endotracheal intubation in patients undergoing elective off-pump coronary

artery bypass grafting. Annals of cardiac anaesthesia. 2012 Jan 1;15(1):39-43.

24. Hrishi AP, Lionel KR, Nair P. A novel use of a novel drug: Preoperative nasal preparation with dexmedetomidine for transnasal transsphenoidal neurosurgery approach in skull base neurosurgery. Indian Journal of Neurosurgery. 2017 Dec;6(03):170-5.

25. Wang SS, Zhang MZ, Sun Y, Wu C, Xu WY, Bai J, Cai MH, Lin L. The sedative effects and the attenuation of cardiovascular and arousal responses during anesthesia induction and intubation in pediatric patients: a randomized comparison between two different doses of preoperative intranasal dexmedetomidine. Pediatric Anesthesia. 2014 Mar;24(3):275-81.

26. Kumar L, Kumar A, Panikkaveetil R, Vasu BK, Rajan S, Nair SG. Efficacy of intranasal dexmedetomidine versus oral midazolam for paediatric premedication. Indian journal of anaesthesia. 2017 Feb 1;61(2):125-30.

27. Lu C, Zhang LM, Zhang Y, Ying Y, Li L, Xu L, Ruan X. Intranasal dexmedetomidine as a sedative premedication for patients undergoing suspension laryngoscopy: a randomized double-blind study. PloS one. 2016 May 19;11(5):e0154192.

28. Li A, Yuen VM, Goulay-Dufaÿ S, Sheng Y, Standing JF, Kwok PC, Leung MK, Leung AS, Wong IC, Irwin MG. Pharmacokinetic and pharmacodynamic study of intranasal and intravenous dexmedetomidine. British Journal of Anaesthesia. 2018 May 1;120(5):960-8.