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Research Article

AN OBSERVATIONAL STUDY TO COMPARE THE TWO DOSES OF INTRAVENOUS DEXMEDETOMIDINE ON HEMODYNAMIC RESPONSES DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

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ABSTRACT

Objective: During general anesthesia, laryngoscopy and endotracheal intubation may cause hemodynamic disturbances such as tachycardia and hypertension, which may result in difficulties. Dexmedetomidine, an alpha 2 agonist, has been shown to attenuate these responses. However, the optimal dose for this effect is unclear.

Methods: This observational study consisted of 30 patients undergoing general anesthesia, randomly assigned to 2 groups with 15 patients in each group. Group A received intravenous dexmedetomidine 1 mcg/kg, whereas Group B received 0.5 mcg/kg over 20 min before induction. The study was conducted from August 2023 to December 2023.

Results: The results showed that dexmedetomidine 1 mcg/kg (Group A) provided better hemodynamic stability, with a more significant attenuation of systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) compared to the 0.5 mcg/kg dose (Group B). Group A showed significantly lower SBP at intubation (121.73±4.33 mmHg vs. 129.20±7.81 mmHg, p=0.0031) and at 7 min post-intubation (110.53±5.10 mmHg vs. 119.73±6.23 mmHg, p=0.0001). MAP was also significantly lower in Group A at intubation (92.40±4.23 mmHg vs. 98.53±6.54 mmHg, p=0.0050) and remained significantly different throughout the observation period. Heart rate showed no significant difference between groups (p>0.05).

Conclusion: Dexmedetomidine 1 mcg/kg is a more effective dose for maintaining hemodynamic stability in terms of SBP, DBP, and MAP during laryngoscopy and endotracheal intubation compared to 0.5 mcg/kg. This study suggests that the higher dose is a better option for patients undergoing general anesthesia.

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

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INTRODUCTION

Heart rate (HR) and arterial blood pressure (BP) always rise after tracheal intubation and laryngoscopy. Typically, the greatest increase in HR and BP happens 30 s after intubation and lasts <10 min. A few variables, including the degree of anesthesia, precautions taken before airway manipulation, the type of anesthetic used, and the length of laryngoscopy and intubation, can affect the severity of these hemodynamic alterations. The precise mechanism underlying the hemodynamic reactions to intubation and laryngoscopy remains unclear to this day. Increased catecholamine activity may trigger the sympathetic response, which is the main mechanism responsible for tachycardia and hypertension. Usually, the rise in BP and pulse rate is transient, erratic, and unpredictable [1,2].

Tachycardia, hypertension, or arrhythmia might result after laryngoscopy and tracheal intubation because of the sympathetic adrenergic output triggered by stimulation of the larynx [3-5].

In healthy people, transient hypertension and tachycardia are normally not dangerous, but they can be dangerous for those who have hypertension, heart failure, or cerebrovascular illnesses. In such cases, the development of myocardial insufficiency, cerebrovascular accidents, and pulmonary edema may be predisposed to by this laryngoscopic response. Endotracheal intubation-induced circulatory reactions are not always suppressed by intravenous (IV) anesthetic induction drugs. Thus, several researchers have tried additional pharmacological measures before starting laryngoscopy, including the use of inhalational anesthetics, opioids, topical and IV lidocaine, vasodilators (e.g., nitroglycerin), calcium channel blockers, and β-blockers. It has not been discovered that any of these medications can totally reduce the sympathetic reaction to intubation [6].

α2 agonists have been employed to reduce the sympathetic reaction, and dexmedetomidine and clonidine seem to satisfy all the requirements. Dexmedetomidine is a highly selective and specific α2 adrenoceptor agonist, with an α2: α1 binding selectivity ratio of 1620:1, compared to 220:1 for clonidine, even though they both act on both α1 and α2 receptors. Furthermore, many investigations have shown that dexmedetomidine reduces the hemodynamic response to intubation and laryngoscopy. When used as a premedication for esthesia, IV dexmedetomidine has the following advantages: less respiratory depression, increased hemodynamic stability, drowsiness, anxiolysis, and analgesia. These beneficial characteristics also result in a 90% reduction in the minimum alveolar concentration (MAC) of volatile anesthetics [2,7].

The S-enantiomer, or dextroisomer, of medetomidine, a common anesthetic in veterinary medicine, is called dexmedetomidine [8,9]. The Food and Drug Administration first authorized dexmedetomidine in 1999 for use as a short-term (<24 h) sedative and analgesic for intensive care unit (ICU) patients receiving mechanical ventilation. Intramuscular/IV dexmedetomidine produces modest respiratory depression along with anxiolysis, sedation, analgesia, and sympatholysis. It is utilized in regional anesthesia, MAC, as a lone anesthetic drug, as a

premedication, and in the ICU. Dexmedetomidine is prescribed as an IV infusion of 0.5–1 μg/kg for 10 min, then 0.2–0.7 μg/kg/h for up to 24 h as a maintenance dose [2, 10,11].

METHODS

After approval of the Ethics Committee (SVIEC/ON/medi/SRP/July/23/126) and patient consent, this Observational study was carried out in patients undergoing general anesthesia. It examined how well two IV doses of dexmedetomidine reduced hemodynamic reactions to laryngoscopy and intubation. The study was conducted at a tertiary care hospital from August 2023 to December 2023.

The study included 30 adults, 18–60 years of age, with the American Society of Anesthesiologists (ASA) grade I and II, who underwent elective surgeries under general anesthesia. The inclusion criteria were: Adults aged 18–60 years, ASA physical status I or II, scheduled for elective surgery under general anesthesia, and written informed consent. The exclusion criteria included: patients who were not willing to undergo surgery, ASA Grade III or more, pregnant or lactating women, with known allergy to the study drug, patients having any cardiovascular diseases, anticipated difficult intubation, and patients on antihypertensive medications or beta-blockers.

For sample size calculation, the change in hemodynamic parameters to detect a 20% change in BP and HR among the groups was of clinical significance [11]. Considering this for our pilot data, we calculated that a minimum of 30 patients would be required for an experimental design incorporating equal-sized groups, using 0.05 and 0.2 alpha and beta errors. To minimize and effect of data loss, we recruited 35 patients in our study. Of 35 patients, 5 patients dropped out. Thirty patients were randomly divided into two groups of 15 patients each based on computer-coded sealed envelopes.

Formulas

This calculator uses the following formulas to compute sample size and power, respectively:

$$n_A = kn_B \text{ and } n_B = \left(\frac{p_A(1-p_A)}{k} + p_B(1-p_B)\right)\left(\frac{z_{1-\alpha/2} + z_{1-\beta}}{p_A - p_B}\right)^2$$

$$1-\beta = \phi(z - z_{1-\alpha/2}) + \phi(-z - z_{1-\alpha/2}), z = \frac{p_A - p_B}{\sqrt{\frac{p_A(1-p_A)}{n_A} + \frac{p_B(1-p_B)}{n_B}}}$$

Where
$\kappa = n_A/n_B$ is the matching ratio
Φ is the standard normal distribution function
Φ^{-1} is the standard normal quantile function
α is type I error
β is type II error, meaning $1 - \beta$ is power

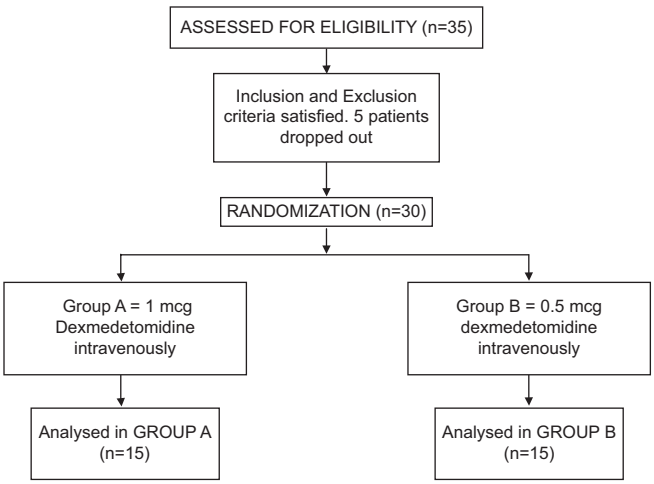
Group A: 15 patients were given Dexmedetomidine 1 mcg/kg intravenously in 100 mL of 0.9 % Normal Saline over 20 min before the induction. Group B: 15 patients were given dexmedetomidine 0.5 mcg/kg intravenously in 100 mL of 0.9 % normal saline over 20 min before the induction.

An in-depth pre-operative history was obtained the day before the procedure. Airway assessment, physical and systemic examination, and general examination were performed. All routine investigations were done. Written and informed consent was taken in their native language.

Patients were given 100% oxygen for 3 min before being induced with injection propofol 2 mg/kg IV, and after verifying ventilation, injection of succinylcholine (2 mg/kg) was given to aid in intubation. A cuffed endotracheal tube of the proper size was used to intubate the patient's trachea; the bilateral air entrance was checked, and the tube was fixed. O_2, N_2O at a 1:1 ratio, and isoflurane, utilizing the circle system, were used to maintain anesthesia. 0.5 mg/kg IV loading dose of atracurium was given and maintained with 0.1 mg/kg intravenously. To sustain normocapnia, patients underwent mechanical ventilation in volume control mode.

Parameters monitored were systolic arterial BP, HR, diastolic arterial BP, mean arterial pressure (MAP), and pulse oximetry. The measurements were recorded at baseline, just after drug administration, post-induction, at laryngoscopy and endotracheal intubation, and 1, 3, 5, and 7 min post-intubation.

Consort flow diagram



Statistical analysis

The sample size of 30 patients, in which 15 patients were present in each group, was determined using MedCalc 12.5 software. The categorical data were shown as percentages and frequencies, and the numerical variables as means and standard deviations. When appropriate, between-group comparisons were needed for numerical variables, the unpaired student t-test was employed; for categorical variables, the Chi-square test was employed. Statistical significance was defined as a difference with a significance level ($p<0.05$).

RESULTS

The study was conducted on 30 patients divided into two groups equally: Group A was given dexmedetomidine 1 mcg/kg intravenously in 100 mL of 0.9 % normal saline over 20 min before the induction. Group B was given dexmedetomidine 0.5 mcg/kg intravenously in 100 mL of 0.9% normal saline over 20 min before the induction.

Table 1 illustrates how both Group A and Group B were comparable and not significant in terms of age, weight, gender, and ASA grading.

Based on the above results, it was found that Group A and Group B were comparable in decreasing HR during intubation and 1 min, 3 min, 5 min, and 7 min after intubation ($p>0.05$), as shown in Fig. 1 and Table 2.

In case of systolic BP, Group A was very efficient than Group B in attenuating pressor response after drug administration, at intubation

Table 1: Demographic data

Parameter	Group A	Group B	p-value
Age	37.87±8.15	48.27±13.57	0.0167
Weight	59.47±9.20	61.20±5.53	0.5375
Gender (Male/Female)	10/5	7/8	

Data presented as mean±standard deviation or frequency; n=15 in each group

and 1 min, 3 min, 5 min, and 7 min after intubation with p<0.05 as shown in Fig. 2 and Table 3.

In case of diastolic BP, Group A was as efficient as Group B in attenuating pressor response 1, 3, and 7 min after intubation with p>0.05. But Group A showed more decrease in diastolic BP at intubation and 5 min after intubation with p<0.05, as shown in Fig. 3 and Table 4.

MAP monitored at intubation and 1, 3, 5, and 7 min after intubation showed that Group A was very efficient than Group B in controlling MAP with p<0.05, as shown in Fig. 4 and Table 5.

There was no statistically significant difference between Group A and Group B in pulse oximetry monitored at the time of intubation and 1, 3, 5, and 7 min after intubation, as shown in Fig. 5 and Table 6. In none of the patients, the SpO_2 fall below 95%. No oxygen supplementation was required.

Complications

There were no episodes of bradycardia and hypotension seen in any patients from any group.

Table 2: Intraoperative vitals: Pulse rate (beats/minutes)

Time point	Group A	Group B	p-value
	Mean±SD	Mean±SD	
Baseline	84.80±7.08	83.60±10.18	0.7106
After drug administration	76.13±5.48	82.27±9.97	0.0458
After induction	77.87±4.56	80.27±9.44	0.3828
At Laryngoscopy and Intubation	83.47±6.12	88.00±9.77	0.1393
1 min after intubation	82.40±5.82	86.93±10.08	0.1429
3 min after intubation	81.47±6.91	86.53±10.57	0.1319
5 min after intubation	77.47±7.65	84.00±10.53	0.0621
7 min after intubation	75.80±6.37	81.47±9.12	0.0600

Data presented as mean±SD; n=15 in each group
SD: Standard deviation

Table 3: Intraoperative vitals: Systolic blood pressure (mmHg)

Time point	Group A	Group B	p-value
	Mean±SD	Mean±SD	
Baseline	124.13±5.48	121.07±7.92	0.2287
After drug administration	110.67±3.83	121.73±9.82	0.0004
After induction	115.20±4.71	116.80±10.28	0.5880
At laryngoscopy and intubation	121.73±4.33	129.20±7.81	0.0031
1 min after intubation	120.53±6.02	127.33±7.16	0.0088
3 min after intubation	118.13±4.93	124.00±7.25	0.0150
5 min after intubation	113.07±8.00	122.00±6.76	0.0026
7 min after intubation	110.53±5.10	119.73±6.23	0.0001

Data presented as mean±SD; n=15 in each group
SD: Standard deviation

Table 4: Intraoperative vitals: Diastolic blood pressure (mmHg)

Time point	Group A	Group B	p-value
	mean±SD	mean±SD	
Baseline	79.33±4.82	77.87±6.78	0.5023
After drug administration	74.40±5.51	77.87±6.48	0.1253
After induction	75.07±5.55	74.67±6.44	0.8567
At laryngoscopy and intubation	77.73±5.50	83.20±7.81	0.0349
1 min after intubation	78.80±4.77	82.00±7.56	0.1766
3 min after intubation	76.40±4.73	80.13±6.95	0.0968
5 min after intubation	74.00±4.72	78.53±7.11	0.0492
7 min after intubation	72.53±5.32	76.93±7.13	0.0657

Data presented as mean±SD; n=15 in each group
SD: Standard deviation

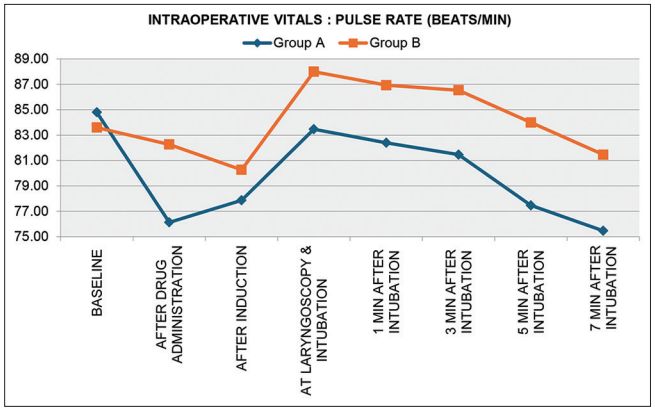


Fig. 1: Intraoperative pulse rate

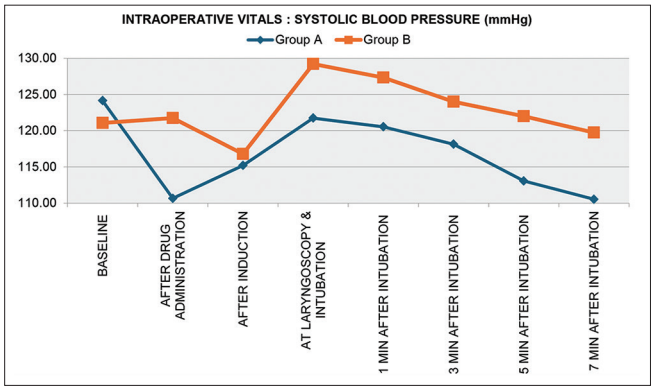


Fig. 2: Intraoperative systolic blood pressure

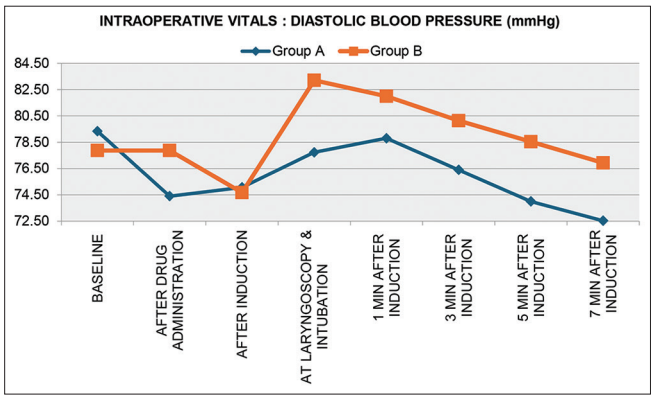


Fig. 3: Diastolic blood pressure

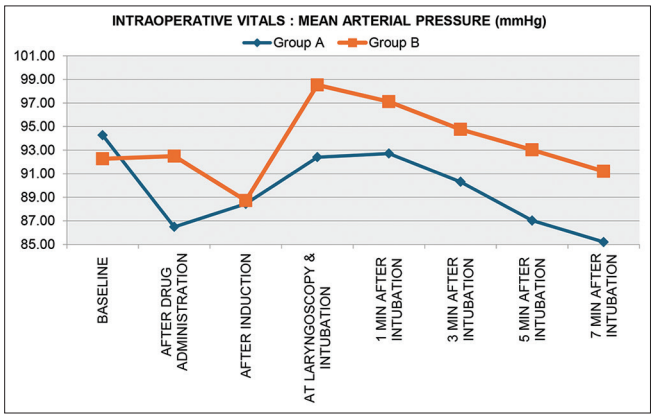


Fig. 4: Mean arterial pressure

Table 5: Intraoperative vitals: Mean arterial pressure (mmHg)

Time point	Group A	Group B	p-value
	mean±SD	mean±SD	
Baseline	94.27±4.51	92.27±5.41	0.2808
After drug administration	86.49±4.34	92.49±5.58	0.0027
After induction	88.44±4.31	88.71±6.14	0.8901
At laryngoscopy and intubation	92.40±4.23	98.53±6.54	0.0050
1 min after intubation	92.71±3.77	97.11±5.84	0.0207
3 min after intubation	90.31±3.71	94.76±5.57	0.0156
5 min after intubation	87.02±4.44	93.02±5.47	0.0027
7 min after intubation	85.20±4.49	91.20±4.78	0.0014

Data presented as mean±SD; n=15 in each group
SD: Standard deviation

Table 6: Intraoperative vitals: SpO_2

Time point	Group A	Group B	p-value
	mean±SD	mean±SD	
Baseline	98.67±1.23	98.67±1.23	1.0000
After drug administration	98.40±1.12	98.67±1.23	0.5347
After induction	98.53±0.92	98.80±1.26	0.5082
At laryngoscopy and intubation	98.93±1.03	98.80±1.26	0.7593
1 min after intubation	98.13±1.19	98.53±1.19	0.3652
3 min after intubation	98.13±1.41	98.67±1.23	0.2732
5 min after intubation	98.67±1.45	98.27±1.03	0.3911
7 min after intubation	98.13±1.60	98.00±1.51	0.8206

Data presented as mean±SD; n=15 in each group
SD: Standard deviation

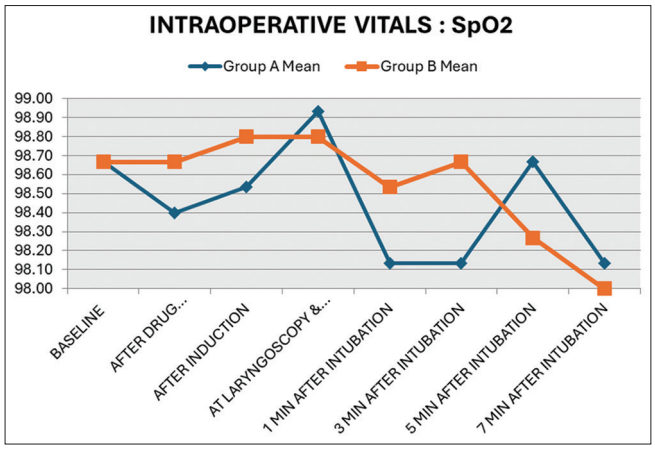


Fig. 5: SPO_2

DISCUSSION

Laryngoscopy and endotracheal intubation produce transient but significant sympathetic responses that can precipitate cardiovascular complications. Our study demonstrated that dexmedetomidine 1 mcg/kg provided superior hemodynamic attenuation compared to 0.5 mcg/kg, with enhanced cardiovascular stability and no adverse events.

The safety profile revealed no episodes of bradycardia, hypotension, or arrhythmias in either group, contrasting with Lawrence and De Lange [6] who reported increased complications at 2 µg/kg doses. This suggests our maximum 1 µg/kg dose remained within safe therapeutic limits while optimizing efficacy. Liu *et al.* [12] FAERS database analysis showed 10.7% hypotension and 3.7% bradycardia rates across all doses, providing context for our zero adverse event rate, likely reflecting controlled study conditions and careful patient selection. Similar findings have been documented by Gertler *et al.* [13].

HR responses showed no significant differences between groups (p>0.05), with Group A maintaining 83.47±6.12 beats/min versus Group B's 88.00±9.77 beats/min during intubation (p=0.1393). These findings corroborate Celik *et al.*, [14] who reported comparable HRs between 1.0 µg/kg and 0.5 µg/kg doses. However, Sebastian *et al.* [15] demonstrated dose-dependent differences with rsuperior to 0.5 µg/kg, possibly due to their placebo-controlled design, highlighting dose effects more clearly. Both doses prevented severe tachycardia, crucial for patients with cardiovascular comorbidities. Kabara *et al.* [16] recently confirmed comparable HR attenuation between doses, supporting our findings. The mechanism, as described by Weerink *et al.* [17], involves presynaptic α2-adrenoceptor activation, reducing norepinephrine release.

Systolic blood pressure (SBP) showed marked dose-dependent attenuation. Group A maintained SBP at 121.73±4.33 mmHg during intubation versus 129.20±7.81 mmHg in Group B (p=0.0031), with maximum difference of 9.2 mmHg at 7 min post-intubation (110.53±5.10 vs. 119.73±6.23 mmHg, p=0.0001). These findings align with Rachit *et al.* [2], who demonstrated superior SBP control with 1 µg/kg across all time points. Group A maintained a narrower SBP range (110–122 mmHg) compared to Group B (117–129 mmHg), indicating a better hemodynamic stability. The pharmacodynamic basis involves enhanced α2-receptor occupancy producing more complete central sympatholysis, as documented by Gertler *et al.* [13].

Diastolic BP showed selective efficacy, with significant differences only during intubation (77.73±5.50 vs. 83.20±7.81 mmHg, p=0.0349) and at 5 min post-intubation (74.00±4.72 vs. 78.53±7.11 mmHg, p=0.0492). This partially aligns with Shin *et al.* [11] who found significant diastolic blood pressure (DBP) reduction at similar time points using 0.5 µg/kg in elderly patients. The selective DBP effects relate to differential impact on arterial compliance versus venous capacitance, mediated through complex interactions between central sympatholysis and peripheral α2-activation.

MAP provided the strongest evidence for higher dose superiority, with consistent significant differences throughout the study. At intubation, MAP was 92.40±4.23 mmHg in Group A versus 98.53±6.54 mmHg in Group B (p=0.0050), with maximum difference at 7 min post-intubation (85.20±4.49 vs. 91.20±4.78 mmHg, p=0.0014). The 6–7 mmHg difference represents a clinically meaningful improvement in hemodynamic stability, particularly important for patients with compromised autoregulation. These findings corroborate Celik *et al.* [14], establishing clear dose–response relationships. MAP represents tissue perfusion pressure, making it the most important parameter for organ perfusion assessment.

Oxygen saturation remained above 98% in all patients without significant differences between groups, confirming dexmedetomidine's respiratory safety profile. This aligns with extensive literature documenting minimal respiratory depression as dexmedetomidine's distinguishing feature. The preservation occurs because dexmedetomidine mimics natural sleep patterns without affecting medullary respiratory centers, providing significant advantages over opioid-based strategies. Niyogi *et al.* [8] confirmed respiratory safety across administration routes, whereas Chen *et al.* [18] identified preserved respiratory function as a key advantage in their bibliometric analysis.

Clinical implications suggest 1 mcg/kg represents optimal dosing for most patients undergoing general anesthesia, balancing superior efficacy with excellent safety. The marginal cost difference is negligible compared to potential complications from inadequate hemodynamic control. While our single-center design focusing on ASA I-II patients limits generalizability, the results provide strong evidence supporting higher dose selection within the therapeutic range. Future multicenter trials incorporating diverse populations and longer monitoring periods could further refine dosing recommendations, particularly for special populations where individualized approaches remain relevant.

Previous studies have similarly demonstrated dexmedetomidine's efficacy in perioperative hemodynamic management, with Raghu *et al*. [19] confirming stable hemodynamics in laparoscopic procedures. The clinical utility of dexmedetomidine over alternative agents like midazolam for hemodynamic stability has been further established by comparative studies [20], reinforcing our findings that appropriate dexmedetomidine dosing provides reliable cardiovascular protection during the stress of laryngoscopy and intubation.

CONCLUSION

Compared to 0.5 mcg/kg intravenously, the hemodynamic effect of laryngoscopy and intubation can be significantly decreased with the use of 1 mcg/kg of dexmedetomidine in terms of SBP, DBP, and MAP.

Limitation

The limitation of our study is: First, the study was carried out at a single centre with a small sample size, focusing only on stable patients classified as ASA class I or II. As a result, our conclusions may not be applicable to patients with significant co-morbidities. Second, adverse events such as arrhythmias, hypotension, hypertension, vomiting, and dry mouth were not observed during the post-operative period. Thirdly, to validate the findings presented in this paper, larger multicenter studies should be conducted. Fourthly, the duration of monitoring is short, only till 7 min post-induction, so the effect of dexmedetomidine on hemodynamic parameters is not considered intraoperatively.

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CONFLICT OF INTEREST

No conflicts of interest was present.

SOURCE OF FUNDING

Nil.

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