RESEARCH PAPER

A Prospective Comparative Study of Two Different Doses of Intramuscular Dexmedetomidine in Attenuating Hemodynamic Responses to Laryngoscopy and Endotracheal Intubation

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ABSTRACT

Introduction: Laryngoscopy and endotracheal intubation provoke a marked sympathoadrenal response that may precipitate serious cardiovascular and cerebrovascular events in susceptible patients. Dexmedetomidine, a highly selective α_2-adrenergic agonist, attenuates this response, but the optimal intramuscular (IM) premedication dose remains undefined.

Aim: To compare the efficacy of two doses of IM dexmedetomidine (1 mcg/kg versus 1.5 mcg/kg) in attenuating heart rate and blood pressure responses to laryngoscopy and intubation, and to compare postoperative sedation.

Materials and Methods: In this prospective comparative study, 64 adults (ASA I–II, 18–60 years) undergoing elective surgery under general anaesthesia were randomly allocated into two equal groups. Group A received IM dexmedetomidine 1 mcg/kg and Group B received 1.5 mcg/kg, 60 minutes before induction. Heart rate (HR), systolic (SBP), diastolic (DBP) and mean arterial pressure (MAP), and SpO_2 were recorded at baseline, every 15 minutes until induction, after induction, at intubation and at 1, 3, 5 and 7 minutes post-intubation. Postoperative sedation was graded by the Ramsay Sedation Score (RSS). Data were analysed using the unpaired t-test, Mann–Whitney U test and chi-square test, with $p<0.05$ considered significant.

Results: The groups were comparable for demographic and baseline characteristics (all $p>0.05$). Group B showed significantly greater attenuation of HR at intubation (84.5±5.2 vs 69.1±4.8 bpm; $p<0.001$) and at all post-intubation time points, with a 58.5% smaller absolute tachycardic response than Group A. SBP, DBP and MAP were significantly lower in Group B from 30 minutes after the IM injection through the post-intubation period (all $p<0.001$). SpO_2 remained comparable and >98% in both groups. At 30 minutes post-extubation, sedation was deeper in Group B (RSS 3 vs 2; $p<0.001$) but equalised (RSS 2) by 60 minutes. No significant complications occurred in either group.

Conclusion: IM dexmedetomidine 1.5 mcg/kg provides superior attenuation of the hemodynamic response to laryngoscopy and endotracheal intubation compared with 1 mcg/kg, at the cost of transiently deeper early sedation. Both doses were haemodynamically safe; dose selection should be individualised..

Keywords: *Alpha-2 agonist; Dexmedetomidine; Endotracheal intubation; Hemodynamic response; Intramuscular; Laryngoscopy; Premedication.*
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INTRODUCTION

Modern general anaesthesia relies on a balanced, multimodal approach that combines intravenous induction agents, volatile anaesthetics, analgesics and muscle relaxants to provide adequate anaesthetic depth, haemodynamic stability and smooth recovery[1]. The perioperative stress response is characterised by activation of the sympathetic nervous system and the hypothalamic–pituitary–adrenal axis, raising plasma catecholamines and producing increases in arterial pressure, heart rate and myocardial oxygen consumption that may precipitate cardiovascular and cerebrovascular complications in patients with pre-existing disease[2].

Among the stimuli encountered during anaesthesia, laryngoscopy and endotracheal intubation evoke the most intense transient sympathoadrenal response, manifesting as tachycardia and hypertension. The reflex was first documented by Reid and Brace in 1940; it begins within seconds of laryngoscopy, peaks at one to two minutes and subsides over five to ten minutes, with systolic pressure rising by 40–50 mmHg and heart rate by 15–20 beats per minute at its peak[3]. In patients with coronary, cerebrovascular or aortic disease, these brief perturbations can trigger myocardial ischaemia, arrhythmia, aneurysm rupture or stroke.

Numerous agents have been used to blunt this response, including opioids, beta-adrenergic blockers, calcium-channel antagonists, local anaesthetics and α_2-agonists, each with its own limitations[4]. Intravenous lidocaine is inexpensive but inconsistent[5]; beta-blockers such as esmolol control heart rate but provide less reliable pressure control and risk bradycardia and bronchospasm[6]. α_2-adrenergic agonists produce sympatholysis, sedation and analgesia without respiratory depression and have an established place in the anaesthetic armamentarium[7]; clonidine, the prototype, is limited by slow onset and hypotension[8].

Dexmedetomidine is a highly selective α_2-adrenergic agonist introduced into clinical practice in 1999, with an α_2:α_1 selectivity ratio of approximately 1600:1—far higher than clonidine—translating into more specific effects with fewer α_1-mediated side effects[9-10]. Its sedative action arises from hyperpolarisation of locus coeruleus neurons, producing a uniquely arousable, cooperative sedation distinct from GABAergic agents[11]. The cardiovascular effects are biphasic: a rapid intravenous bolus causes transient hypertension with reflex bradycardia, followed by a fall in pressure and heart rate from central sympatholysis[12]. Importantly, respiratory drive is largely preserved[13].

The intramuscular route avoids the acute biphasic perturbation of an intravenous bolus by providing gradual absorption, while permitting drug administration without intravenous access—useful in anxious or uncooperative patients[14]. The principal adverse effects of dexmedetomidine are cardiovascular: bradycardia, occasionally severe[15], and dose-related hypotension[16]. Dose-ranging data suggest that an IM dose of 1–1.2 mcg/kg or lower is not associated with significant haemodynamic change, whereas 1.5–2.4 mcg/kg produces appreciable reductions in heart rate and mean arterial pressure[9]. The choice between 1 and 1.5 mcg/kg therefore represents a balance between adequate attenuation of the intubation response and the risk of excessive sedation and cardiovascular depression. The present study was designed to determine which of these two IM doses, administered 60 minutes before induction, provides superior attenuation of the hemodynamic response to laryngoscopy and endotracheal intubation while maintaining an acceptable safety and sedation profile.

AIM AND OBJECTIVES

The aim was to compare the efficacy of two different doses of intramuscular dexmedetomidine (1 mcg/kg versus 1.5 mcg/kg) in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation in patients undergoing general anaesthesia. The objectives were: (1) to compare attenuation of heart rate; (2) to compare attenuation of systolic, diastolic and mean arterial pressure; and (3) to compare postoperative sedation using the Ramsay Sedation Score.

MATERIALS AND METHODS

This prospective, comparative study was conducted in the Department of Anaesthesiology, Dhiraj Hospital, S.B.K.S. Medical Institute and Research Centre, Sumandeep Vidyapeeth (Deemed to be University), Piparia, Vadodara, over a period of 18 months after approval by the Sumandeep Vidyapeeth Institutional Ethics Committee. The study was conducted in accordance with the Declaration of Helsinki (2013 revision) and Good Clinical Practice guidelines, and was prospectively registered with the Clinical Trial Registry of India (CTRI/2024/12/078117). Written informed consent was obtained from all participants in their preferred language.

Sixty-four adults aged 18–60 years of ASA physical status I or II scheduled for elective surgery under general anaesthesia were enrolled and randomly allocated in a 1:1 ratio into two groups of 32 each, using a computer-generated random-number sequence created in SPSS version 25.0. Allocation concealment was maintained using sequentially numbered, opaque, sealed envelopes (SNOSE), opened only after enrolment and consent. Patients were blinded to the dose received and the statistician was blinded during analysis; the administering anaesthesiologist was necessarily aware of allocation, making this an open-label design. Group A received injection dexmedetomidine 1 mcg/kg intramuscularly and Group B received 1.5 mcg/kg intramuscularly, each 60 minutes before induction.

Inclusion criteria were patients aged 18–60 years of ASA grade I–II posted for elective surgery under general anaesthesia who were willing to provide consent. **Exclusion criteria** were body mass index ≥30 kg/m²; muscular disorder, myositis or local infection at the

injection site; cardiovascular disease, arrhythmia, heart block or beta-blocker use; cerebrovascular, hepatic or renal disease; psychiatric illness; known hypersensitivity to α_2-agonists; and pregnancy or lactation.

On arrival in the operating theatre, standard monitoring (electrocardiography, non-invasive blood pressure and pulse oximetry) was established and an 18-gauge intravenous cannula was secured with Ringer's lactate maintenance. After the allocated IM dose, all patients were premedicated with intravenous glycopyrrolate 0.004 mg/kg, ondansetron 0.1 mg/kg and tramadol 2 mg/kg. Following preoxygenation, anaesthesia was induced with propofol 2 mg/kg and succinylcholine 2 mg/kg to facilitate intubation with an appropriately sized cuffed endotracheal tube using a Macintosh laryngoscope. Anaesthesia was maintained with oxygen, nitrous oxide (1:1) and isoflurane, with atracurium for neuromuscular blockade. Bradycardia (heart rate <50 beats/min) was treated with atropine 0.6 mg and hypotension (systolic pressure <80 mmHg) with fluid and incremental mephentermine.

Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and oxygen saturation (SpO_2) were recorded at baseline, every 15 minutes after the IM injection until induction, after induction, during laryngoscopy and intubation, and at 1, 3, 5 and 7 minutes after intubation. The primary outcome was the comparative attenuation of HR and blood pressure at and after intubation. Secondary outcomes were postoperative sedation, assessed half-hourly by the Ramsay Sedation Score until a score ≤3 was reached, and the incidence of complications (bradycardia, hypotension, nausea, vomiting and dryness of mouth).

Statistical analysis was performed using SPSS version 25.0. Continuous variables were checked for normality with the Shapiro–Wilk test and expressed as mean ± standard deviation (or median with interquartile range when non-normal); categorical variables were expressed as frequency and percentage. Between-group comparisons used the unpaired Student's t-test or Mann–Whitney U test for continuous variables and the chi-square or Fisher's exact test for categorical variables. A p-value <0.05 was considered statistically significant.

RESULTS

All 64 enrolled patients completed the study without dropouts or protocol violations; the flow of participants is shown in Figure 1. The two groups were comparable with respect to age, weight, gender and ASA grade, confirming successful randomisation (Table 1).

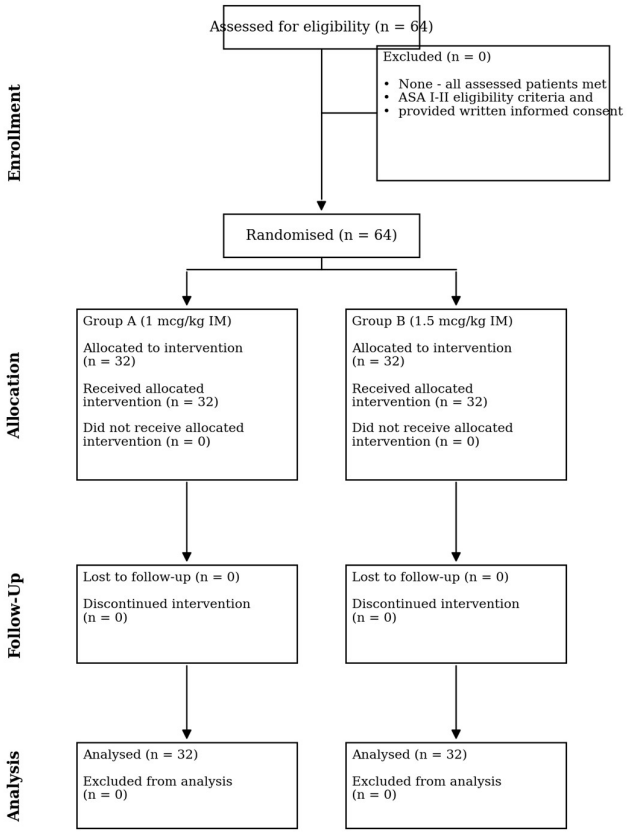

Figure 1: CONSORT 2010 flow diagram of participant enrolment, allocation, follow-up and analysis.

Table 1: Baseline demographic and clinical characteristics of the two groups

Characteristic	Group A (n=32)	Group B (n=32)	p-value
Age (years), mean ± SD	41.3 ± 7.8	41.8 ± 8.2	0.81
Weight (kg), mean ± SD	66.8 ± 5.4	67.2 ± 5.9	0.78
Male, n (%)	17 (53.1)	18 (56.3)	0.80
Female, n (%)	15 (46.9)	14 (43.7)	0.80
ASA I, n (%)	20 (62.5)	21 (65.6)	0.79
ASA II, n (%)	12 (37.5)	11 (34.4)	0.79

SD, standard deviation; ASA, American Society of Anesthesiologists. Age and weight compared by unpaired t-test; gender and ASA grade by chi-square test.

Following the IM injection, heart rate declined progressively in both groups, but the decline was significantly greater in Group B from 30 minutes onwards and after induction (Table 2, Figure 2). At intubation, heart rate rose transiently in both groups, but the increase was markedly smaller in Group B (69.1±4.8 bpm) than in Group A (84.5±5.2 bpm; p<0.001). Group A rose by 13.5 bpm (19.0%) from its post-induction value, whereas Group B rose by only 5.6 bpm (8.8%), a 58.5% smaller absolute tachycardic response. The difference remained highly significant at 1, 3, 5 and 7 minutes after intubation (all p<0.001).

Table 2: Mean heart rate (beats/minute) in both groups at different time intervals.

Time point	Group A (mean ± SD)	Group B (mean ± SD)	t	p-value
Baseline	79.5 ± 2.8	80.7 ± 2.6	1.76	0.08
15 min after IM	77.4 ± 2.8	77.0 ± 2.7	0.60	0.55
30 min after IM	75.3 ± 2.8	72.9 ± 2.9	3.35	0.001*
45 min after IM	73.2 ± 2.8	68.9 ± 3.0	5.87	<0.001**
60 min after IM	72.1 ± 2.8	66.2 ± 3.1	7.91	<0.001**
After induction	71.0 ± 2.8	63.5 ± 3.2	9.88	<0.001**
At intubation	84.5 ± 5.2	69.1 ± 4.8	12.15	<0.001**
1 min post-intubation	82.3 ± 4.9	67.5 ± 4.6	12.30	<0.001**
3 min post-intubation	80.2 ± 4.7	65.9 ± 4.4	12.38	<0.001**
5 min post-intubation	78.8 ± 4.5	64.7 ± 4.3	12.65	<0.001**
7 min post-intubation	77.9 ± 4.4	64.1 ± 4.2	12.66	<0.001**

*IM, intramuscular. *p<0.05 (significant); **p<0.001 (highly significant).*

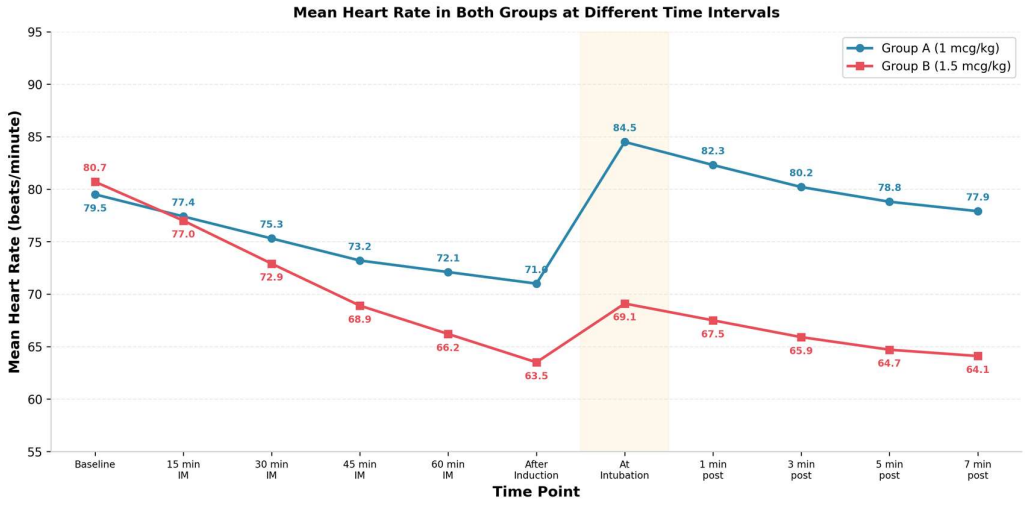

Figure 2: Mean heart rate in both groups at different time intervals (the shaded band marks the laryngoscopy/intubation event).

Systolic blood pressure followed a similar pattern (Table 3, Figure 3). It was significantly lower in Group B at 30, 45 and 60 minutes after the IM injection and after induction (all p<0.001). At intubation, SBP rose to 143.5±5.9 mmHg in Group A versus 124.2±5.5 mmHg in Group B (mean difference 19.3 mmHg; p<0.001), corresponding to an 18.0% rise in Group A and 13.7% in Group B—a 31.5% smaller absolute pressor response. The difference persisted at all post-intubation time points (all p<0.001).

Table 3: Mean systolic blood pressure (mmHg) in both groups at different time intervals

Time point	Group A (mean ± SD)	Group B (mean ± SD)	t	p-value
Baseline	132.1 ± 3.7	132.6 ± 3.6	-0.55	0.58
15 min after IM	130.0 ± 3.7	128.9 ± 3.6	1.20	0.23
30 min after IM	127.9 ± 3.7	122.9 ± 3.9	5.22	<0.001**
45 min after IM	125.8 ± 3.7	117.1 ± 4.1	8.84	<0.001**
60 min after IM	123.7 ± 3.7	113.3 ± 4.3	10.28	<0.001**
After induction	121.6 ± 3.7	109.2 ± 4.4	12.12	<0.001**
At intubation	143.5 ± 5.9	124.2 ± 5.5	13.40	<0.001**
1 min post-intubation	139.3 ± 5.6	120.6 ± 5.3	13.58	<0.001**
3 min post-intubation	136.2 ± 5.4	117.4 ± 5.1	14.15	<0.001**
5 min post-intubation	133.4 ± 5.2	115.3 ± 5.0	14.06	<0.001**
7 min post-intubation	131.8 ± 5.1	114.1 ± 4.9	14.04	<0.001**

*IM, intramuscular. **p<0.001 (highly significant).*

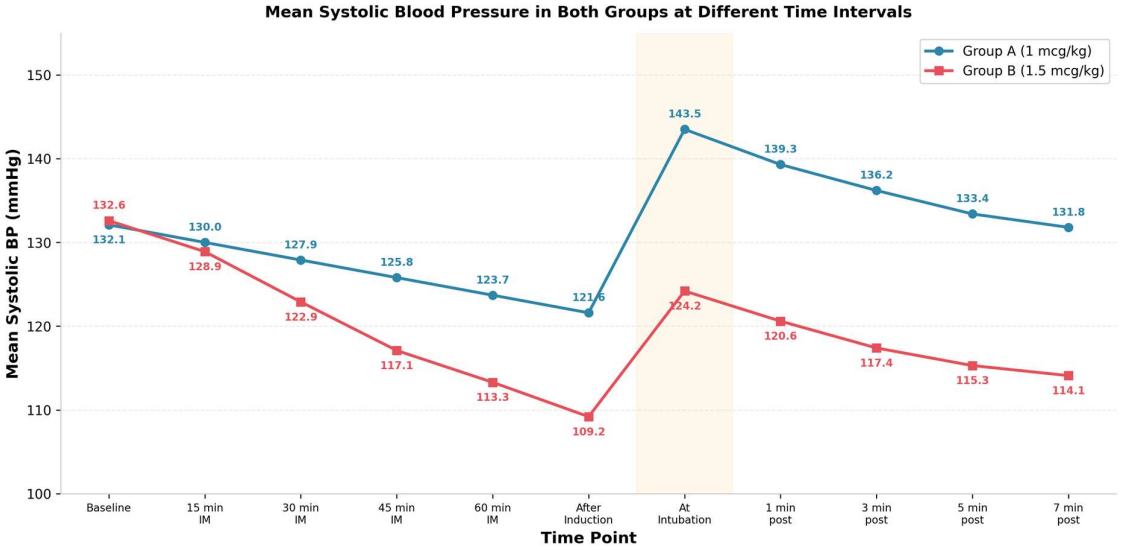

Figure 3: Mean systolic blood pressure in both groups at different time intervals.

Diastolic blood pressure was significantly lower in Group B at 30 minutes after the IM injection onwards and after induction (Table 4, Figure 4). At intubation, DBP was 87.3±4.2 mmHg in Group A versus 78.6±4.0 mmHg in Group B (p<0.001), and although the percentage rise was similar between groups, absolute values remained significantly lower in Group B at every post-intubation time point (all p<0.001).

Table 4: Mean diastolic blood pressure (mmHg) in both groups at different time intervals.

Time point	Group A (mean ± SD)	Group B (mean ± SD)	t	p-value
Baseline	83.2 ± 2.5	83.6 ± 2.7	-0.61	0.55
15 min after IM	81.2 ± 2.5	82.1 ± 2.9	-1.32	0.19
30 min after IM	79.3 ± 2.5	77.3 ± 3.0	2.87	0.005*
45 min after IM	77.4 ± 2.5	72.7 ± 3.1	6.61	<0.001**
60 min after IM	76.4 ± 2.5	69.7 ± 3.2	9.24	<0.001**
After induction	75.4 ± 2.5	67.3 ± 3.3	11.00	<0.001**
At intubation	87.3 ± 4.2	78.6 ± 4.0	8.40	<0.001**
1 min post-intubation	85.4 ± 4.0	76.9 ± 3.9	8.52	<0.001**
3 min post-intubation	83.6 ± 3.9	75.3 ± 3.8	8.54	<0.001**
5 min post-intubation	81.9 ± 3.8	73.9 ± 3.7	8.44	<0.001**
7 min post-intubation	80.8 ± 3.7	73.1 ± 3.6	8.34	<0.001**

*IM, intramuscular. *p<0.05 (significant); **p<0.001 (highly significant).*

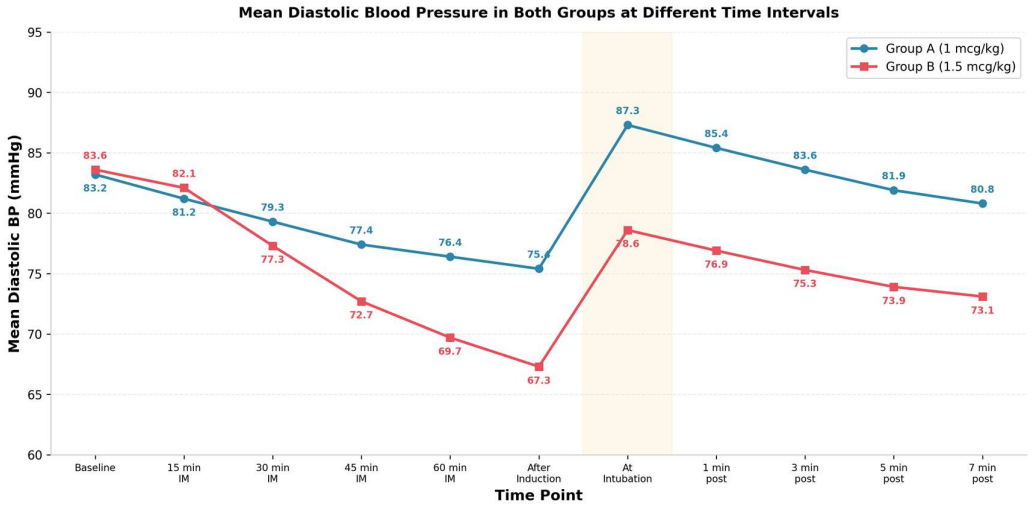

Figure 4: Mean diastolic blood pressure in both groups at different time intervals.

Mean arterial pressure showed the same dose-dependent pattern (Table 5, Figure 5), being significantly lower in Group B from 30 minutes after the IM injection. At intubation, MAP was 106.0±4.5 mmHg in Group A versus 93.8±4.3 mmHg in Group B (p<0.001). MAP in Group B remained below 100 mmHg at all time points beyond 30 minutes after injection, including during and after intubation, whereas Group A exceeded 100 mmHg at intubation and for the first three minutes thereafter.

Table 5: Mean arterial pressure (mmHg) in both groups at different time intervals.

Time point	Group A (mean ± SD)	Group B (mean ± SD)	t	p-value
Baseline	99.5 ± 2.5	99.9 ± 2.6	-0.62	0.54
15 min after IM	97.5 ± 2.5	97.7 ± 2.8	-0.30	0.76
30 min after IM	95.5 ± 2.5	92.5 ± 3.0	4.32	<0.001**
45 min after IM	93.5 ± 2.5	87.5 ± 3.2	8.29	<0.001**
60 min after IM	92.2 ± 2.5	84.2 ± 3.4	10.62	<0.001**
After induction	90.8 ± 2.5	81.3 ± 3.5	12.36	<0.001**
At intubation	106.0 ± 4.5	93.8 ± 4.3	10.93	<0.001**
1 min post-intubation	103.4 ± 4.3	91.5 ± 4.1	11.20	<0.001**

Time point	Group A (mean ± SD)	Group B (mean ± SD)	t	p-value
3 min post-intubation	101.1 ± 4.2	89.3 ± 4.0	11.41	<0.001**
5 min post-intubation	99.1 ± 4.1	87.7 ± 3.9	11.28	<0.001**
7 min post-intubation	97.8 ± 4.0	86.8 ± 3.8	11.16	<0.001**

*IM, intramuscular. **p<0.001 (highly significant).*

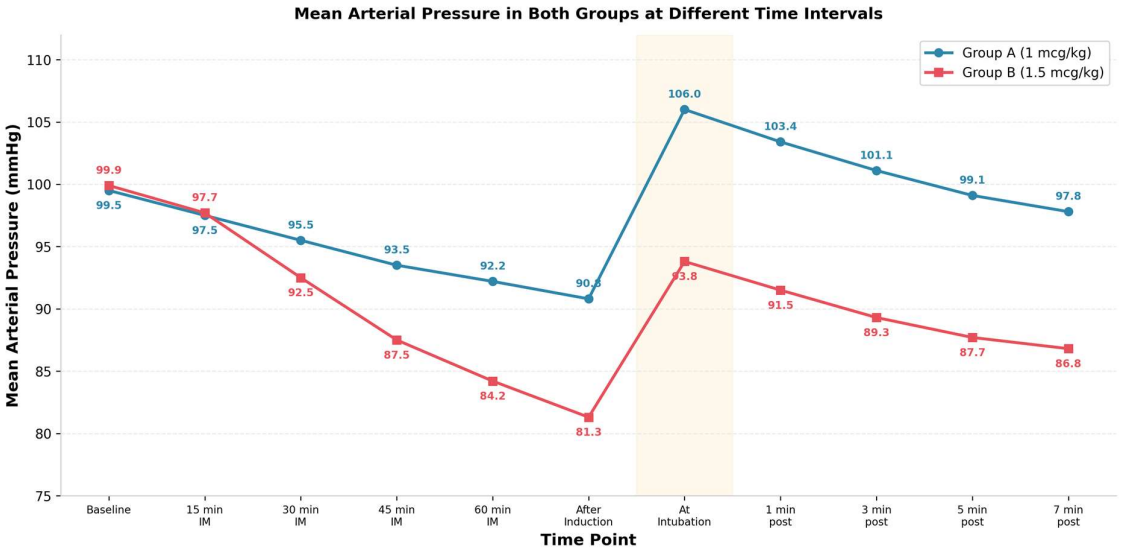

Figure 5: Mean arterial pressure in both groups at different time intervals.

Oxygen saturation remained comparable between the groups at every time point and consistently above 98% (Table 6), indicating that neither dose produced clinically significant respiratory depression in spontaneously breathing patients during the preoperative period.

Table 6: Mean oxygen saturation (%) in both groups at representative time intervals.

Time point	Group A (mean ± SD)	Group B (mean ± SD)	p-value
Baseline	98.7 ± 0.5	98.6 ± 0.6	0.45
60 min after IM	98.6 ± 0.5	98.6 ± 0.6	1.00
After induction	98.5 ± 0.5	98.4 ± 0.6	0.48
At intubation	98.3 ± 0.6	98.2 ± 0.6	0.51
1 min post-intubation	98.7 ± 0.5	98.7 ± 0.5	1.00
7 min post-intubation	98.7 ± 0.5	98.7 ± 0.5	1.00

IM, intramuscular. Differences not significant at any time point (p>0.05).

Postoperative sedation differed between the groups only early in recovery (Table 7). At 30 minutes after extubation, all Group A patients had a Ramsay Sedation Score of 2 (cooperative, oriented, tranquil) while all Group B patients had a score of 3 (responsive to commands only; p<0.001). By 60 minutes both groups had returned to a score of 2, which was maintained at 90 and 120 minutes (p=1.00). No significant complication—including clinically relevant bradycardia, hypotension, nausea, vomiting or dryness of mouth—was observed in either group (p>0.05).

Table 7: Ramsay Sedation Score (median, IQR) in both groups after extubation.

Time point	Group A, median (IQR)	Group B, median (IQR)	p-value
30 min post-extubation	2 (2–2)	3 (3–3)	<0.001**
60 min post-extubation	2 (2–2)	2 (2–2)	1.00
90 min post-extubation	2 (2–2)	2 (2–2)	1.00
120 min post-extubation	2 (2–2)	2 (2–2)	1.00

*IQR, interquartile range. Compared by Mann–Whitney U test. **p<0.001 (highly significant).*

Table 8 summarises the percentage rise of each haemodynamic parameter at intubation relative to the respective post-induction baseline. The tachycardic and systolic pressor responses were substantially smaller in Group B, while diastolic and mean arterial percentage rises were comparable between groups despite consistently lower absolute values in Group B.

Table 8: Percentage rise in haemodynamic parameters at intubation (versus post-induction baseline)

Parameter	Group A % rise	Group B % rise	Mean difference	p-value
Heart rate	19.0%	8.8%	10.2%	<0.001**
Systolic BP	18.0%	13.7%	4.3%	<0.001**
Diastolic BP	15.8%	16.8%	1.0%	<0.001**
Mean arterial pressure	16.7%	15.4%	1.3%	<0.001**

*BP, blood pressure. **p<0.001 for the corresponding absolute between-group comparison.*

DISCUSSION

This study demonstrated a clear dose-dependent effect of intramuscular dexmedetomidine on the hemodynamic response to laryngoscopy and endotracheal intubation, with the 1.5 mcg/kg dose providing superior control of heart rate and blood pressure compared with 1 mcg/kg, at the cost of transiently deeper early sedation. The two groups were well matched at baseline, supporting the attribution of these differences to the intervention.

The intramuscular route was chosen deliberately. Dyck et al. showed that intramuscular dexmedetomidine avoids the acute hypertension and reflex bradycardia of an intravenous bolus by providing gradual absorption to peak concentration at about 1.6–1.7 hours[14]. Our timing of administration 60 minutes before induction exploits this pharmacokinetic profile, and the smooth pre-induction decline in heart rate and pressure in both groups is consistent with it.

Our heart-rate findings align closely with earlier dose-ranging work. Aho et al. compared intramuscular dexmedetomidine 0.6, 1.2 and 2.4 mcg/kg with placebo before gynaecological laparoscopy and reported a clear dose-dependent reduction in the heart-rate response to intubation, with doses ≥1.2 mcg/kg providing clinically meaningful blunting[17]. Sun et al. found that low-dose intramuscular dexmedetomidine 1 mcg/kg significantly attenuated, but did not abolish, the heart-rate response to suspension laryngoscopy—mirroring our Group A[18], while Menda et al. demonstrated effective blunting of the heart-rate rise and rate–pressure product at intubation with dexmedetomidine 1 mcg/kg in cardiac surgical patients[19]. Our 58.5% smaller absolute tachycardic response with 1.5 mcg/kg places the higher dose firmly within this dose–response continuum.

The blood-pressure findings are equally consistent with the literature. Scheinin et al., in a 192-patient multicentre study of intramuscular dexmedetomidine premedication, found marked attenuation of the pressor response, though hypotension requiring intervention was more frequent at higher (2.5 mcg/kg) doses[20]. Dogru et al. studied controlled hypertensive patients and reported that intramuscular dexmedetomidine 2.5 mcg/kg attenuated but did not abolish the hypertensive response (systolic pressure 142 mmHg at intubation versus 168 mmHg in controls)[21]; our 1.5 mcg/kg group achieved a comparable mean systolic pressure at intubation (124.2 mmHg) in a normotensive population, suggesting the chosen dose is well matched to the elective adult surgical patient. Niyogi et al. showed that route influences the haemodynamic profile, the intravenous bolus producing transient hypertension that a non-intravenous route avoids[22], supporting our intramuscular approach.

Comparisons with other agents reinforce these observations. Kumari et al. found that intravenous dexmedetomidine produced significantly better control of systolic and mean arterial pressure at intubation than esmolol[23], and Takita et al. showed that tracheal lidocaine offered less consistent and shorter-lasting pressure control than α_2-agonists[5]. The superior control we observed reflects dexmedetomidine's mechanism: presynaptic and central α_2 activation reduces sympathetic outflow and circulating catecholamines—Aantaa et al. reported a 50% fall in plasma noradrenaline with dexmedetomidine premedication[24]. Higher receptor occupancy at 1.5 mcg/kg produces more complete sympatholysis and hence better haemodynamic control, but the same mechanism explains the greater bradycardia and deeper early sedation at the higher dose. Sebastian et al. similarly found a dose-dependent benefit when comparing two intravenous doses of dexmedetomidine[25], and a recent meta-analysis confirmed that dexmedetomidine premedication reliably attenuates the intubation response across routes and doses[26].

The sedation data showed a transient, dose-related effect: Group B patients were deeper at 30 minutes after extubation (Ramsay 3 versus 2) but both groups reached full alertness (Ramsay 2) by 60 minutes, with no delayed extubation. This favourable recovery profile is in keeping with the easily arousable, cooperative sedation characteristic of α_2-agonists and supports the clinical usability of both doses.

LIMITATIONS

This was a single-centre study with an open-label design, since preparation of different doses precluded blinding of the administering anaesthesiologist; patients and the analysing statistician were, however, blinded. The population was restricted to ASA I–II adults aged 18–60 years, which may limit generalisability to elderly, paediatric or higher-risk patients. Plasma catecholamine concentrations were not measured, and the study was not powered to detect rare adverse events. Larger, multicentre, double-blind trials would further define the optimal intramuscular dose.

CONCLUSION

Premedication with intramuscular dexmedetomidine 1.5 mcg/kg, administered 60 minutes before induction, attenuates the hemodynamic response to laryngoscopy and endotracheal intubation more effectively than 1 mcg/kg, with significantly lower heart rate and blood pressure at and after intubation. The higher dose is associated with transiently deeper early sedation and a slightly greater tendency to bradycardia, but both doses are haemodynamically safe without significant hypotension or respiratory depression. The choice of dose should be individualised to patient characteristics and clinical requirements.

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