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Original Article

Anaesthesia Section

Intranasal Dexmedetomidine versus Magnesium Sulphate via Mucosal Atomisation Device for Attenuation of the Haemodynamic Stress Response to Laryngoscopy and Endotracheal Intubation: A Randomised Clinical Study

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ABSTRACT

Introduction: Laryngoscopy and endotracheal intubation are fundamental anaesthetic procedures performed millions of times annually worldwide. Both dexmedetomidine and magnesium sulphate have individually demonstrated efficacy in attenuating the haemodynamic stress response to laryngoscopy.

Aim: To compare the efficacy of intranasal dexmedetomidine (2 µg/kg) versus magnesium sulphate (1000 mg) delivered via Mucosal Atomisation Device (MAD) in attenuating the haemodynamic stress response to laryngoscopy and endotracheal intubation under General Anaesthesia (GA).

Materials and Methods: This randomised clinical study was conducted in the Department of Anaesthesiology at Dhiraj Hospital, Shrimati Bhikiben Kanjibhai Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed to be University, Piparia, Waghodia, Vadodara, Gujarat, India, from July 2024 to January 2026. Enrolled 60 patients with American Society of Anaesthesiology (ASA) physical status I and II, aged 18-65 years, undergoing elective surgery under GA. Patients were randomly allocated by the chit method into two equal groups. Group D received intranasal dexmedetomidine 2 µg/kg and Group M received intranasal magnesium sulphate 1000 mg, both via MAD 30 minutes before induction. Heart Rate (HR), blood pressure and SpO^2 were recorded from baseline through 10 minutes post-intubation, and postoperative sedation was assessed using the Ramsay Sedation Score (RSS). Continuous variables were compared using Welch's t-test, ordinal data using the Mann-Whitney U test and categorical data using the Chi-square test, with $p<0.05$ considered significant.

Results: The two groups were comparable in age, weight, height, Body Mass Index (BMI), gender and ASA status (all $p>0.05$). Group D showed significantly lower HR, Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP) and Mean Arterial Pressure (MAP) than group M from drug administration through 10 minutes post-intubation ($p<0.001$ at all time points). At laryngoscopy, HR was 72.5±4.0 versus 88.5±5.1 bpm and MAP 90.5±3.5 versus 101.9±3.7 mmHg in groups D and M, respectively. Postoperative RSSs were significantly higher in group D (3.8±0.7 versus 2.5±0.5 at 30 minutes, $p<0.001$). Bradycardia (33.3% vs 10.0%) and dry mouth (40.0% vs 23.3%) were more frequent in group D; no respiratory depression occurred.

Conclusion: Intranasal dexmedetomidine (2 µg/kg) via MAD is significantly more effective than magnesium sulphate (1000 mg) in attenuating the haemodynamic stress response to laryngoscopy and intubation. It also provides superior postoperative sedation with a favourable safety profile, and can be recommended as an effective, non invasive premedication strategy.

Keywords: Administration intranasal, Anaesthesia, Blood pressure, Conscious sedation, Heart rate

INTRODUCTION

Laryngoscopy and endotracheal intubation are among the most frequently performed procedures in anaesthetic practice, carried out millions of times each year in operating theatres, emergency departments and intensive care units. Although essential for securing the airway, these procedures invariably provoke a profound sympathetic and sympathoadrenal response, manifesting as transient yet marked increases in HR, SBP and DBP, and myocardial oxygen consumption [1,2]. The reflex arises from mechanical stimulation of the epipharyngeal and laryngopharyngeal receptors, which is transmitted through the glossopharyngeal and vagal afferents and provokes a surge in the release of circulating catecholamines from the adrenal medulla and sympathetic nerve endings. Although this transient stress response is generally well tolerated by healthy individuals, it can precipitate serious cardiovascular complications such as myocardial ischaemia, left ventricular dysfunction, cerebrovascular accidents, arrhythmias and acute heart failure in patients with pre-existing cardiovascular or hypertensive disease [3,4]. The response is rapid in onset, peaks within one to two minutes of airway instrumentation and usually resolves within five to ten minutes after intubation, but even this brief surge may be hazardous in vulnerable patients [5]. Effective attenuation of this pressor response is therefore an important goal of balanced anaesthesia.

A wide range of pharmacological agents has been evaluated to attenuate this pressor response, including topical and intravenous local anaesthetics, opioids, beta-blockers, calcium channel blockers, vasodilators and alpha-2 adrenergic agonists [6,7]. Each of these has limitations related to inadequate efficacy, haemodynamic instability or unwanted side-effects. Dexmedetomidine, a highly selective alpha-2 adrenoceptor agonist, attenuates both the tachycardic and hypertensive components of the response by reducing central

sympathetic outflow, while additionally providing sedation, anxiolysis and analgesia without clinically significant respiratory depression [8,9]. Magnesium sulphate, acting as a physiological calcium channel antagonist and N-Methyl-D-Aspartate (NMDA) receptor blocker, attenuates catecholamine release from the adrenal medulla and peripheral nerve terminals and reduces central sensitisation, thereby blunting the stress response [10,11].

Among the various routes of administration, the intranasal route delivered through a MAD offers a non invasive, painless and easily administered alternative that is particularly suited to the preoperative setting, avoiding the discomfort of intravenous cannulation and the cooperation required for nebulisation. It provides rapid systemic absorption through the highly vascularised nasal mucosa and avoids hepatic first-pass metabolism, resulting in a predictable onset of action [12,13]. The device fragments the drug into fine particles that are distributed over a large mucosal surface area, enhancing absorption and bioavailability compared with simple nasal drops, and requires no specialised training to use [13]. These properties make atomised intranasal premedication an attractive option for blunting the haemodynamic stress response before induction of anaesthesia.

Both dexmedetomidine and magnesium sulphate have individually demonstrated efficacy in attenuating the haemodynamic response to laryngoscopy and intubation. However, most published studies have employed the intravenous or nebulised routes and have compared each agent against placebo or against agents such as fentanyl rather than against each other [14]. As per knowledge, there are no studies comparing these two drugs administered via the intranasal atomiser route. This gap in the literature, together with the practical advantages of atomised intranasal premedication, provided the rationale for the present study.

The present study was therefore designed to compare the efficacy of intranasal dexmedetomidine (2 µg/kg) with that of intranasal magnesium sulphate (1000 mg), both delivered via a MAD, in attenuating the haemodynamic stress response to laryngoscopy and endotracheal intubation under GA. The primary objective was to compare the attenuation of HR, SBP, DBP and MAP between the two groups. The secondary objectives were to compare postoperative sedation, assessed by the RSS, and the incidence of adverse effects between the two groups.

MATERIALS AND METHODS

This randomised clinical study was conducted in the Department of Anaesthesiology at Dhiraj Hospital, Shrimati Bhikiben Kanjibhai Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed to be University, Piparia, Waghodia, Vadodara, Gujarat, India, over a period of 18 months from July 2024 to January 2026. The study was approved by the Institutional Ethics Committee (SVIEC/ON/Medi/BNPG23/July/24/222) and registered with the Clinical Trials Registry-India (CTRI/2024/11/076109), and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from every patient in English, Hindi or Gujarati according to the patient's preference.

Inclusion criteria: Patients of ASA physical status I and II, aged between 18 and 65 years, of either gender, who were posted for elective surgery under GA requiring tracheal intubation and who gave written informed consent, were included in the study.

Exclusion criteria: Patients who had a BMI greater than 30 kg/m^2, cardiac arrhythmias, a baseline HR below 70 beats per minute, psychiatric illness, pregnancy or lactation, ASA physical status III and above, a known allergy to the study drugs, or a serum magnesium concentration greater than 2.5 mEq/l, were excluded from the study.

Sample size calculation: Based on previous studies by Kochhar A et al., [15], taking the mean difference in SBP between groups as 10 mmHg with an SD of 12 mmHg, at 80% power (1-β=0.80) and a 5% significance level (α=0.05), using the following formula:

$n=2\times(Z\alpha/2+Z\beta)^2\times\sigma^2/d^2$

Where: $Z\alpha/2 = 1.96$ (for 95% confidence interval), $Z\beta=0.84$ (for 80% power),

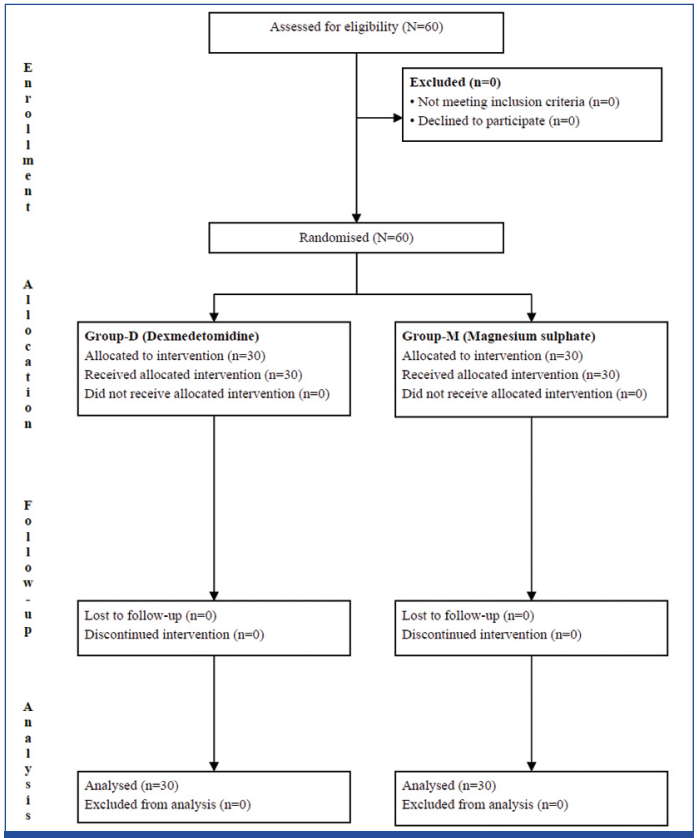
σ = pooled SD=12 mmHg,

d = minimum clinically significant difference=10 mmHg

$n=2\times(1.96+0.84)^2\times(12)^2/(10)^2=22.58\approx23$ per group

Adding 20% for possible dropouts: n=23+5=28 per group. However, 30 patients were enrolled per group to ensure adequate statistical power, giving a total sample size of 60 patients.

Eligible patients were randomly allocated into two equal groups by the chit (lottery) method. The allocation sequence was prepared by an anaesthesiologist who was not otherwise involved in the conduct of the study and who also prepared and administered the study drug; the investigator who recorded the haemodynamic parameters and the patients themselves were blinded to group allocation, giving the study an observer-blinded design. Standardised GA and a uniform postoperative protocol were followed for all patients to minimise bias. The flow of participants through the study is summarised in the Consolidated Standards of Reporting Trials (CONSORT) flow diagram [Table/Fig-1] [16].



[Table/Fig-1]: CONSORT flow diagram of the study.

Study Procedure

Sixty patients of ASA physical status I and II, aged 18 to 65 years and of either gender, undergoing elective surgery under GA requiring tracheal intubation, were enrolled. The elective procedures comprised general surgical, gynaecological, orthopaedic and otorhinolaryngological operations of short to intermediate duration performed under GA. All 60 patients assessed for eligibility met the selection criteria, and none were excluded before enrollment. Group D received intranasal dexmedetomidine 2 µg/kg diluted in 2 mL of 0.9% normal saline, and group M received intranasal magnesium sulphate 1000 mg in 2 mL, both administered bilaterally via a MAD 30 minutes before induction of anaesthesia; these doses were selected on the basis of previous studies [15].

After detailed preoperative assessment and standard fasting [17], the test drug was administered 30 minutes before induction in the preoperative area by a consultant anaesthesiologist. All patients were premedicated with inj. glycopyrrolate 0.2 mg, inj. ondansetron 4 mg, inj. ranitidine 50 mg, and inj. tramadol 2 mg/kg intravenously (i.v.). Patients were preoxygenated with 100% oxygen for three minutes and induced with inj. propofol 2 mg/kg; inj. succinylcholine 2 mg/kg facilitated intubation after confirming ventilation. The trachea was intubated with a cuffed endotracheal tube of appropriate size, bilateral air entry was checked, and the tube was secured.

Anaesthesia was maintained with oxygen, air/N2O (1:1), isoflurane, and atracurium guided by Train-Of-Four (TOF) monitoring [18]. HR, SBP, DBP, MAP, and SpO^2 were recorded at baseline (Tb), after drug administration (Td), after induction (Ti), during laryngoscopy and intubation (T0), and at 1, 3, 5, 7, and 10 minutes after intubation (T1, T3, T5, T7, T10). After surgery, neuromuscular blockade was reversed with neostigmine and glycopyrrolate, and the trachea was extubated after a TOF ratio of 0.9. Postoperative sedation was assessed every 30 minutes using the RSS, a standard six-point scale [19], until a score of 2 was achieved. Adverse effects including nausea, vomiting, respiratory depression, and dryness of mouth were recorded.

Parameters	Group D (n=30) Mean±SD	Group M (n=30) Mean±SD	p-value
Age (years)	40.5±9.5	40.8±9.3	0.881
Weight (kg)	67.9±7.0	67.4±6.1	0.770
Height (cm)	165.0±7.7	164.7±7.5	0.866
BMI (kg/m²)	24.8±0.4	24.8±0.3	0.65
Gender (M/F)	16 / 14	17 / 13	1.000
ASA (I/II)	6 / 24	10 / 20	1.000

[Table/Fig-2]: Demographic and baseline characteristics.
Unpaired t-test for continuous variables; Chi-square test for categorical variables. *p<0.05 considered significant

Time point	Group D (n=30) Mean±SD	Group M (n=30) Mean±SD	p-value
Baseline (Tb)	81.5±4.5	80.4±4.9	0.385
After drug (Td)	69.3±3.9	78.4±4.9	<0.001*
Induction (Ti)	66.0±3.5	76.9±4.5	<0.001*
Laryngoscopy (T0)	72.5±4.0	88.5±5.1	<0.001*
1 min (T1)	70.5±4.0	84.2±4.7	<0.001*
3 min (T3)	68.5±4.0	80.2±4.7	<0.001*
5 min (T5)	66.5±3.9	77.2±4.7	<0.001*
7 min (T7)	65.3±3.6	76.2±4.7	<0.001*
10 min (T10)	64.3±3.6	75.2±4.7	<0.001*

[Table/Fig-3]: Heart Rate (HR) (bpm) at all-time points.
Data presented as mean±SD. Welch's two-sample t-test. *p<0.05 considered significant.
Tb: baseline; Td: after drug; Ti: induction; T0: laryngoscopy; T1-T10: minutes post-intubation

STATISTICAL ANALYSIS

Data were tabulated in Microsoft Excel and analysed using Jamovi statistical software (version 2.3.28; The Jamovi project, Sydney, Australia). Continuous variables were expressed as Mean±SD and compared using Welch's two-sample t-test; categorical variables were expressed as frequency (percentage) and compared using the Chi-square test. The Mann-Whitney U test was used for ordinal data (RSS). A p-value <0.05 was considered statistically significant.

RESULTS

All 60 patients completed the study protocol without any dropouts or protocol violations. The demographic and baseline characteristics were comparable between the two groups, with no significant difference in age (40.5±9.5 vs 40.8±9.3 years, p=0.881), weight (p=0.770), height (p=0.866), BMI (p=0.65), gender distribution, or ASA status (all p>0.05) [Table/Fig-2]. Baseline haemodynamic parameters were also statistically similar between groups.

The HR was the primary parameter evaluated and was comparable between the two groups at baseline (p=0.385). From drug administration onwards, Group D maintained a significantly lower HR than group M at every recorded time point (p<0.001). The clinically decisive difference occurred at laryngoscopy, where group M rose well above its own baseline while group D remained below baseline, giving a between-group difference of 16 bpm. Dexmedetomidine therefore abolished, rather than merely blunted, the tachycardic response to airway instrumentation [Table/Fig-3].

The SBP, DBP and MAP were all comparable between the groups at baseline (p=0.598, 0.466 and 0.944, respectively). After drug administration, every pressure variable was significantly lower in group D at all-time points (p<0.001), and all three followed an identical trajectory: group M rose above its baseline at laryngoscopy, whereas group D remained at or below baseline. No patient in group D exceeded a SBP of 140 mmHg at laryngoscopy, compared with eight patients (26.7%) in group M [Table/Fig-4].

Oxygen saturation remained at or above 97% in every patient at all time points, with no episode of clinically significant desaturation and no difference between the groups (p=0.647 at laryngoscopy), confirming the respiratory safety of both agents. Postoperative sedation was significantly deeper in group D at every time point (p<0.001) and declined steadily in both groups as recovery progressed. No patient in either group recorded a score of 6, indicating that sedation remained rousable and within the safe clinical range throughout [Table/Fig-5].

Bradycardia occurred more often in group D (33.3% vs 10.0%), although this did not reach statistical significance (p=0.057); it was self-limiting and required no atropine. Dry mouth was more common in group D (40.0% vs 23.3%). Hypotension (23.3% vs 16.7%) and nausea/vomiting (23.3% in both) were comparable, and no respiratory depression occurred in either group [Table/Fig-6].

Time point	SBP			DBP			MAP		
	Group D (n=30) Mean±SD	Group M (n=30) Mean±SD	p-value	Group D (n=30) Mean±SD	Group M (n=30) Mean±SD	p-value	Group D (n=30) Mean±SD	Group M (n=30) Mean±SD	p-value
Baseline (Tb)	128.4±4.4	128.9±3.2	0.598	79.6±3.8	78.9±3.2	0.466	96.0±4.1	95.9±3.2	0.944
After drug (Td)	115.7±4.0	122.9±3.3	<0.001*	71.8±3.4	74.9±3.3	<0.001*	86.4±3.6	90.9±3.3	<0.001*
Induction (Ti)	109.8±3.8	119.9±3.3	<0.001*	68.2±3.0	72.9±3.3	<0.001*	82.0±3.1	88.9±3.3	<0.001*
Laryngoscopy (T0)	120.9±4.1	137.9±3.7	<0.001*	75.1±3.2	83.9±3.7	<0.001*	90.5±3.5	101.9±3.7	<0.001*
1 min (T1)	117.9±4.0	130.9±3.7	<0.001*	73.1±3.2	79.9±3.7	<0.001*	88.0±3.4	96.9±3.7	<0.001*
3 min (T3)	114.9±4.0	124.8±3.7	<0.001*	71.1±3.2	76.0±3.4	<0.001*	85.6±3.4	92.0±3.4	<0.001*
5 min (T5)	111.9±4.0	120.8±3.7	<0.001*	69.1±3.2	73.3±3.3	<0.001*	83.5±3.5	89.3±3.3	<0.001*
7 min (T7)	109.9±4.0	119.8±3.7	<0.001*	68.1±3.2	73.0±3.4	<0.001*	82.0±3.4	88.8±3.7	<0.001*
10 min (T10)	107.9±4.0	118.3±3.2	<0.001*	67.1±3.2	72.3±3.2	<0.001*	80.6±3.4	87.3±3.2	<0.001*

[Table/Fig-4]: Systolic Blood Pressure (SBP) (mmHg), Diastolic Blood Pressure (DBP) (mmHg), Mean Arterial Pressure (MAP) (mmHg) at all-time points.

Time point	Group D (n=30) Mean±SD	Group M (n=30) Mean±SD	p-value
30 min	3.8±0.7	2.5±0.5	<0.001*
60 min	3.0±0.6	1.7±0.4	<0.001*
90 min	2.4±0.5	1.0±0.2	<0.001*
120 min	1.8±0.4	1.0±0.0	<0.001*

[Table/Fig-5]: Ramsay Sedation Score (RSS) at postoperative time points.

Adverse effect	Group D n (%)	Group M n (%)	p-value
Bradycardia	10 (33.3)	3 (10.0)	0.057
Hypotension	7 (23.3)	5 (16.7)	0.748
Nausea/vomiting	7 (23.3)	7 (23.3)	1.000
Respiratory depression	0 (0.0)	0 (0.0)	-
Dry mouth	12 (40.0)	7 (23.3)	0.267

[Table/Fig-6]: Incidence of adverse effects.
Data presented as n (%). Chi-square/Fisher's-exact test. *p<0.05 considered significant

DISCUSSION

The present study demonstrates that intranasal dexmedetomidine (2 µg/kg) delivered via a mucosal atomisation device provides statistically and clinically superior attenuation of all four haemodynamic parameters - HR, SBP, DBP, and MAP - at laryngoscopy and throughout the post-intubation period, compared with intranasal magnesium sulphate (1000 mg). The demographic and baseline characteristics were well matched, ensuring that the observed differences are attributable to the pharmacological interventions rather than baseline variation.

The chronotropic superiority of dexmedetomidine was sustained from drug administration through 10 minutes post-intubation, with the maximum difference at laryngoscopy. These findings are supported by Niyogi S et al., who reported effective haemodynamic attenuation with intranasal dexmedetomidine [12], and by Kochhar A et al., who showed that intranasal dexmedetomidine at 1 and 2 µg/kg provided significant attenuation [15], with the 2 µg/kg dose associated with bradycardia (p<0.0001 at all time points), validating our dosing. Kumar NRR et al., and Misra S et al., reported effective HR attenuation with nebulised dexmedetomidine (p<0.012) [20,21], although Misra S et al., noted attenuation of HR but not SBP, suggesting that the higher dose and more efficient atomiser delivery used here account for the broader attenuation observed.

The SBP, DBP, and MAP responses showed the same pattern of sustained superiority for dexmedetomidine. Abraham V and Paul NS reported significant reduction in SBP and DBP with nebulised dexmedetomidine versus saline, and Sabu NA et al., reported significant blunting of DBP and MAP in the first minute (p<0.05) after intubation, mirroring our early-peak attenuation [22,23]. Fateh SG et al., reported significantly lower DBP and MAP during and after intubation with nebulised dexmedetomidine compared with nebulised magnesium sulphate, a finding our study reproduces and extends to the intranasal atomiser route [24]. Godbole S et al., and Elmeligy MSM et al., confirmed that magnesium sulphate itself attenuates the pressor response, consistent with the effect observed in our Group-M (p<0.001), although the effect size remained inferior to dexmedetomidine [25,26]. Chaudhary AV et al., demonstrated lower MAP at 10 minutes post (p<0.0001) intubation and higher sedation scores with nebulised dexmedetomidine, and Grover N et al., found both dexmedetomidine and magnesium sulphate superior to fentanyl [27,28].

Both agents maintained SpO_2 at or above 97% throughout, confirming respiratory safety. The respiratory safety of dexmedetomidine is attributed to its action through the locus coeruleus rather than GABAergic or opioid pathways [9], while magnesium sulphate did not produce clinically significant respiratory depression at the doses used [10]. The significantly higher postoperative sedation scores in Group-D reflect a beneficial residual sedative effect, consistent with Chaudhary AV et al., [27] (p<0.0001) and Sabu NA et al., (p<0.05) [23]. The mucosal atomisation device produces fine particles that enhance absorption and bioavailability and provides predictable pharmacokinetics, making it practical in the preoperative setting [13].

The pharmacokinetic behaviour of intranasally atomised drugs offers a mechanistic explanation for these findings. Atomisation produces a fine mist that deposits uniformly across the highly vascular nasal mucosa, from where the drug is rapidly absorbed into the systemic circulation while bypassing hepatic first-pass metabolism. Intranasal dexmedetomidine achieves a bioavailability of approximately 65%, with sedation beginning within 15 to 30 minutes and peak plasma concentrations at a median of 38 minutes [29], corresponding well with the 30-minute pre-induction interval used in this study. Magnesium sulphate is comparatively poorly absorbed across the nasal mucosa which, together with the fixed rather than weight-based dose used, may partly account for its weaker attenuation of the pressor response in Group-M. These pharmacokinetic differences provide a plausible basis for the superior haemodynamic control observed with dexmedetomidine.

Adverse effects were mild and self-limiting in both groups in the present study. Bradycardia occurred more often with dexmedetomidine (33.3% versus 10.0%) but did not reach statistical significance, resolved spontaneously and required no atropine, and dry mouth followed the same pattern (40.0% versus 23.3%). These observations accord with Kochhar A et al., who reported bradycardia at the 2 µg/kg intranasal dose without haemodynamic compromise [15], and with Lu C et al., who found intranasal dexmedetomidine premedication to be well tolerated in adults undergoing suspension laryngoscopy, with fewer episodes of tachycardia and hypertension after intubation and no increase in clinically important adverse events [30]. The bradycardic and xerostomic effects are the predictable consequence of central alpha-2 mediated sympatholysis and reduced salivary secretion [9]. Hypotension and nausea or vomiting occurred with similar frequency in the two groups and no patient in either Group-Developed respiratory depression, supporting the safety of both agents at the doses studied [10].

Limitation(s)

This study has several limitations. It was a single-centre study with a modest sample size that may be insufficient to detect rarer adverse effects. Only low-risk patients (ASA I and II) were included, excluding the high-risk hypertensive and cardiac patients who might benefit most from attenuation of the stress response. A fixed rather than weight-based dose of magnesium sulphate was used, and serum drug concentrations were not measured. Potential confounders such as the type and duration of surgery, blood loss and the experience of the intubating anaesthesiologist were not recorded or adjusted for. Recovery characteristics, postoperative analgesia and patient satisfaction were also not assessed. Future multi-centre, double-blind randomised controlled trials with larger samples, weight-based dosing, inclusion of high-risk populations and assessment of these additional outcomes are warranted.

CONCLUSION(S)

Intranasal dexmedetomidine (2 µg/kg) administered via a MAD produced significantly greater attenuation of the haemodynamic stress response to laryngoscopy and endotracheal intubation than intranasal magnesium sulphate (1000 mg). The superiority of dexmedetomidine was consistent across HR and SBP, DBP and MAP, and was sustained from drug administration through ten minutes after intubation. Dexmedetomidine also provided significantly better postoperative sedation, while both agents maintained adequate oxygen saturation without respiratory depression. The bradycardia seen more frequently with dexmedetomidine was mild, self-limiting and required no intervention. Intranasal atomised dexmedetomidine can therefore

be recommended as an effective, non-invasive and well-tolerated premedication strategy for blunting the pressor response to airway instrumentation. Its role in high-risk patients should be confirmed in larger multi-centre randomised controlled trials.

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AI use statement: No Artificial Intelligence (AI) or AI-assisted tools were used in the writing, data analysis, or creation of figures for this manuscript.

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