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Original Research Article

Intravenous dexmedetomidine versus magnesium sulphate for attenuation of hemodynamic response to laryngoscopy and endotracheal intubation in adults undergoing cardiac valve replacement surgery at a tertiary care hospital in Udaipur, Rajasthan

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ABSTRACT

Background: Laryngoscopy and endotracheal intubation induce sympathetic activation (increased heart rate, blood pressure), which may be hazardous in patients undergoing cardiac valve replacement. This study aimed to compare intravenous dexmedetomidine (1 mcg/kg) and magnesium sulphate (20 mg/kg) in attenuating the hemodynamic response to laryngoscopy and intubation during elective cardiac valve replacement surgery.

Methods: This prospective, randomized, double-blind, parallel-group trial enrolled 66 adult patients (ASA II–III, aged 18–70 years) undergoing elective cardiac valve replacement surgery under general anaesthesia at a tertiary care centre. Patients were allocated to Group D (dexmedetomidine 1 mcg/kg, n=33) or Group M (magnesium sulphate 20 mg/kg, n=33), each infused over 10 minutes in 100 ml normal saline commencing 20 minutes before anticipated intubation. Haemodynamic parameters (heart rate [HR], systolic blood pressure [SBP], diastolic blood pressure [DBP], mean arterial pressure [MAP]) were recorded at baseline, after drug infusion, after induction, and at 1, 3, 5, 8, 10, and 15 minutes following intubation.

Results: Following drug infusion, dexmedetomidine reduced HR (84.3 ± 4.5 to 78.2 ± 4.3 bpm), whereas magnesium sulphate was associated with a paradoxical mild increase (85.8 ± 5.3 to 87.4 ± 5.1 bpm; $p < 0.001$ intergroup). At one-minute post-intubation, HR (99.4 ± 4.6 vs 104.5 ± 5.3 bpm), SBP (125.0 ± 5.7 vs 135.5 ± 6.2 mmHg), DBP (81.3 ± 3.9 vs 89.9 ± 4.0 mmHg), and MAP (95.9 ± 4.2 vs 105.1 ± 4.8 mmHg) were all significantly lower in Group D ($p < 0.001$ for all). These differences were sustained through 15 minutes.

Conclusions: Intravenous dexmedetomidine (1 mcg/kg) provides significantly superior attenuation of the haemodynamic response than magnesium sulphate 20 mg/kg in adults undergoing cardiac valve replacement surgery.

Keywords: Dexmedetomidine, Magnesium sulphate, Laryngoscopy, Endotracheal intubation, Haemodynamic response, Cardiac valve replacement surgery

INTRODUCTION

Direct laryngoscopy and endotracheal intubation trigger a transient sympathetic response in the form of increased heart rate and blood pressure. It is caused due to mechanical stimulation of the supraglottic and tracheal structures, which activates afferent pathways and sympathetic outflow. Mostly the patients tolerate this brief response, however it can be clinically significant in patients with cardiovascular diseases. Hence, during cardiac surgery procedures, it is important to alleviate such haemodynamic variations, during airway instrumentation.^{1,2} This becomes even more pertinent in patients undergoing cardiac valve replacement surgery, because such patients may have impaired ventricular function, and prone to have pulmonary hypertension and arrhythmias. Laryngoscopy and intubation can lead to a sudden change in sympathetic activity, subsequently leading to increased myocardial oxygen demand and put a strain on a heart with already limited cardiovascular reserve. Therefore, attenuating haemodynamic fluctuations during induction is a vital part of perioperative management of patients undergoing cardiac surgery.³

Researchers have studied various pharmacological strategies to attenuate the haemodynamic response to laryngoscopy and intubation. Dexmedetomidine is a highly selective α_2 -agonist which reduces the central sympathetic outflow, has sympatholytic function and provides sedation, analgesia without causing any significant respiratory depression. Clinical trials have reported that it attenuates the rise in both heart rate and blood pressure after airway instrumentation. However, studies also reveal that it can cause bradycardia and hypotension too, so dose selection is critical in patients, especially the ones with limited cardiovascular reserve.^{4,5} Magnesium sulphate (MgSO_4) is also an attenuator of haemodynamic response to intubation, but it has different mechanism of action. It acts by blocking the catecholamine release, relaxes blood vessels (blocks calcium channels) and blocks the NMDA receptors to reduce sympathetic activity.

Literature shows that it reduces peri-intubation rises in blood pressure and heart rate, however the doses, routes, timing and patient populations vary across studies.^{6,7} When both the drugs (Dexmedetomidine and Magnesium sulphate) are compared, it shows that both have attenuating response on heart rate and blood pressure after laryngoscopy and intubation, despite having different pharmacological profiles and haemodynamic effects. Randomized trials among patients of cardiac surgeries, have particularly supported the efficacy of dexmedetomidine in comparison to Magnesium sulphate.^{4,5} Despite growing evidence for both agents, comparative data on intravenous dexmedetomidine and magnesium sulphate in adults undergoing cardiac valve replacement surgery are limited. Establishing whether these two pharmacologically different agents give comparable haemodynamic attenuation under the same

anaesthetic and surgical conditions may therefore guide drug selection at induction. This study compared the efficacy of intravenous dexmedetomidine (1 $\mu\text{g/kg}$) and intravenous magnesium sulphate (20 mg/kg) in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation in adults undergoing cardiac valve replacement surgery.

METHODS

This prospective, randomized, double-blind, parallel-group clinical trial was conducted at the Department of Anesthesiology and Critical Care, at a tertiary care hospital in Udaipur, Rajasthan, after obtaining institutional ethics committee approval (GU/HREC/EC/2024/2492) and written informed consent from all participants. The study was conducted between March 2024 and January 2026.

Inclusion criteria and exclusion criteria

Adult patients aged 18–70 years of either sex, classified as ASA Physical Status II or III with left ventricular ejection fraction $\geq 40\%$, scheduled for elective cardiac valve replacement surgery under general anaesthesia and who were willing to participate in the study were included. However, the patients with anticipated or unanticipated difficult airway (requiring ≥ 2 intubation attempts or laryngoscopy duration >15 seconds); severe renal or hepatic dysfunction; active lower respiratory tract infection; known hypersensitivity to the study drugs; body weight outside 50–120 kg were excluded from the study.

In our study, a total of 102 patients were assessed for eligibility. Of these, 36 were excluded: 31 did not meet the inclusion criteria, 3 declined to participate, and 2 were excluded due to hepatic dysfunction. The remaining 66 eligible patients were enrolled and randomly allocated to one of two study groups (33 patients in each group) as shown in Figure 1.

Sample size and randomization

Sample size was calculated, assuming a two-sided significance level of 5% and 80% power, and taking value of Heart rate in the form of mean and the standard deviation from a study done by Choudhary N et al the minimum required sample size was 25 participants per group.⁸ After accounting for an anticipated 25% dropout rate, the sample size was increased to 33 participants per group, resulting in a total sample size of 66 participants. Allocation was by computer-generated random number tables.

Allocation concealment was maintained through sequentially numbered, sealed, opaque envelopes opened by a third-party anaesthesiologist who prepared and administered the study drug without being involved in data collection. Both the patient and the attending anaesthesiologist recording haemodynamic data were blinded to group assignment.

Interventions

On arrival in the operating theatre, firstly baseline haemodynamic parameters were recorded. All patients received peripheral intravenous access (20-gauge cannula), central venous line, and arterial line, followed by three minutes of preoxygenation with 100% oxygen.

Group D received intravenous dexmedetomidine 1 mcg/kg diluted in 100 ml normal saline, infused over 10 minutes initiating 20 minutes before anticipated intubation. Group M received intravenous magnesium sulphate 20 mg/kg diluted in 100 ml normal saline over the same period. Both infusions were prepared in identical syringes and bags by the unblinded third-party anaesthesiologist. A standardised induction protocol was used: intravenous midazolam 0.1 mg/kg, fentanyl 3 mcg/kg, etomidate 0.2 mg/kg and succinylcholine 1.5 mg/kg. Oro-tracheal intubation was

performed with an appropriately sized cuffed endotracheal tube using a Macintosh laryngoscope. Anaesthesia was maintained with 50% oxygen in air delivered via Bain's circuit using intermittent positive pressure ventilation, supplemented with etomidate 0.2 mg/kg as required. Muscle relaxation was maintained with vecuronium 0.1 mg/kg followed by incremental doses of 0.02 mg/kg.

Statistical analysis

Data were entered in Microsoft Excel and analysed using SPSS version 26.0. Continuous variables are expressed as mean $\pm$ SD. Intergroup comparisons of continuous variables were performed using the independent samples t-test. Categorical variables were compared using the chi-square test or Fisher's exact test where expected cell frequencies were <5 . A $p < 0.05$ was considered statistically significant.

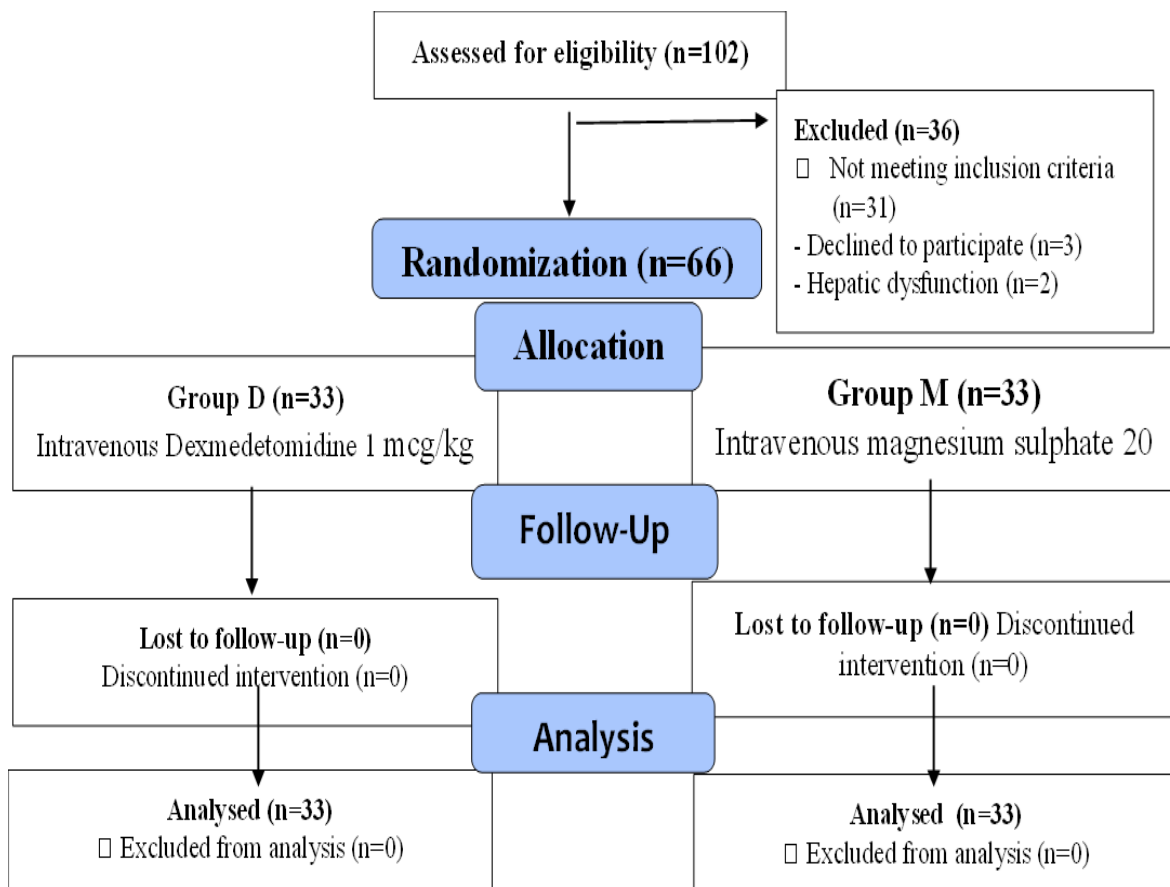


Figure 1: CONSORT (Consolidated Standards of Reporting Trials) flowchart of recruited participants.

RESULTS

A total of 66 patients were included in the study, with 33 in each group. All the demographic variables (including mean age, gender, BMI) and pre operative vital parameters were comparable between groups as shown in Table 1 and 2.

Following drug infusion (T1), dexmedetomidine produced a significant reduction in HR (78.2 ± 4.3 bpm), whereas magnesium sulphate was associated with a paradoxical mild increase (87.4 ± 5.1 bpm; intergroup $p < 0.001$). At one-minute post-intubation (T3), HR was significantly lower in Group D and this difference was maintained at all subsequent time points through 15 minutes (Table 3).

SBP at baseline was comparable between both groups, however a significantly greater post-intubation rise was seen in Group M at all time points. (Table 4).

DBP and MAP showed a pattern mirroring the SBP findings. Statistically significant intergroup differences were observed from the post-induction period (T2) through all post-intubation time points, as shown in Table 5 and 6. Peripheral oxygen saturation (SpO₂) remained well

maintained in both groups throughout the study period, with no significant intergroup differences at any

assessment point. However, sinus tachycardia was reported in Group M. Hypotension was almost similar and responded promptly to vasopressor therapy in both the groups. Flushing or warmth was noted in 9.1% of Group M patients only. No allergic reactions were recorded (Table 7).

Table 1: Comparison of demographic data between the two groups (n=66).

Parameter	Group D (DEX) n=33	Group M (MgSO ₄) n=33	P value
Age (years), Mean±SD	42.1±14.5	43.8±13.0	0.614
Gender: Male, N (%)	17 (51.5%)	18 (54.5%)	0.795
Weight (kg), Mean±SD	77.1±13.5	77.1±11.7	0.996
Height (cm), Mean±SD	168.5±7.5	168.0±7.3	0.793
BMI (kg/m ²), Mean±SD	27.1±2.8	27.4±2.6	0.631
ASA Grade II, N (%)	17 (51.5%)	16 (48.5%)	0.802
ASA Grade III, N (%)	16 (48.5%)	17 (51.5%)	0.802

Independent t-test for continuous variables; Chi-square test for categorical variables.

Table 2: Comparison of pre-operative vital parameters between the two groups.

Parameter	Group D (DEX) Mean±SD	Group M (MgSO ₄) Mean±SD	P value
HR (bpm)	78.0±7.8	78.4±6.8	0.823
SBP (mmHg)	127.0±10.8	128.9±9.4	0.412
DBP (mmHg)	83.1±7.4	83.9±6.9	0.628
MAP (mmHg)	97.8±8.3	98.9±7.7	0.563
SpO ₂ (%)	97.2±1.2	97.4±1.1	0.491

Independent t-test; HR: Heart rate; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure.

Table 3: Comparative analysis of heart rate (bpm) across time points between the two groups.

Time point	Group D Mean±SD (bpm)	Group M Mean±SD (bpm)	Mean Diff (D-M)	P value
T0 – Baseline	84.3±4.5	85.8±5.3	-1.5	0.218
T1 – Post-drug	78.2±4.3	87.4±5.1	-9.2	<0.001**
T2 – Post-induction	92.2±4.6	92.7±4.0	-0.5	0.641
T3 – 1 min post-intubation	99.4±4.6	104.5±5.3	-5.1	<0.001**
T4 – 3 min post-intubation	96.7±5.0	103.4±4.6	-6.7	<0.001**
T5 – 5 min post-intubation	92.7±4.0	99.4±4.7	-6.7	<0.001**
T6 – 8 min post-intubation	89.9±4.2	95.4±1.7	-5.5	<0.001**
T7 – 10 min post-intubation	87.3±4.5	92.2±4.6	-4.9	<0.001**
T8 – 15 min post-intubation	85.3±4.5	90.2±4.6	-4.9	<0.001**

**p<0.001; Independent t-test used for all comparisons.

Table 4: Comparative analysis of systolic blood pressure (mmHg) across time points.

Time point	Group D Mean±SD (mmHg)	Group M Mean±SD (mmHg)	Mean Diff (D-M)	p value
T0 – Baseline	123.2±5.7	123.8±6.3	-0.6	0.300
T1 – Post-drug	119.2±5.7	122.5±5.8	-3.4	0.024*
T2 – Post-induction	103.1±5.5	112.5±5.8	-9.5	<0.001**
T3 – 1 min post-intubation	125.0±5.7	135.5±6.2	-10.5	<0.001**
T4 – 3 min post-intubation	114.0±5.7	129.2±6.3	-15.2	<0.001**
T5 – 5 min post-intubation	107.2±5.7	122.6±5.9	-15.4	<0.001**
T8 – 15 min post-intubation	99.5±5.0	111.9±5.1	-12.4	<0.001**

*p<0.05; **p<0.001; Independent t-test used for all comparisons.

Table 5: Comparative analysis of diastolic blood pressure (mmHg) across time points.

Time point	Group D Mean±SD (mmHg)	Group M Mean±SD (mmHg)	Mean Diff (D-M)	P value
T0 – Baseline	80.8±4.7	82.8±5.1	-2.0	0.095
T3 – 1 min post-intubation	81.3±3.9	89.9±4.0	-8.6	<0.001**
T5 – 5 min post-intubation	70.3±3.4	80.0±3.8	-9.7	<0.001**
T8 – 15 min post-intubation	64.3±2.9	72.0±2.9	-7.7	<0.001**

**p<0.001; Independent t-test used for all comparisons.

Table 6: Comparative analysis of mean arterial pressure (mmHg) across time points.

Time point	Group D Mean±SD (mmHg)	Group M Mean±SD (mmHg)	Mean Diff (D-M)	P value
T0 – Baseline	94.9±4.7	96.0±5.4	-1.1	0.100
T3 – 1 min post-intubation	95.9±4.2	105.1±4.8	-9.2	<0.001**
T5 – 5 min post-intubation	82.6±3.9	94.2±4.5	-11.6	<0.001**
T8 – 15 min post-intubation	76.1±3.5	85.3±3.8	-9.2	<0.001**

**p<0.001; Independent t-test used for all comparisons.

Table 7: Comparative analysis of adverse events between the two groups.

Adverse event	Group D N (%)	Group M N (%)	P value
Hypotension (MAP <60 mmHg or >20% fall)	5 (15.2%)	4 (12.1%)	0.720
Intraoperative arrhythmia	3 (9.1%)	6 (18.2%)	0.286
Sinus tachycardia (≥100 bpm)	0 (0.0%)	4 (12.1%)	0.038*
Ventricular ectopics	0 (0.0%)	2 (6.1%)	0.491
Nausea/vomiting	2 (6.1%)	1 (3.0%)	1.000
Flushing/warmth	0 (0.0%)	3 (9.1%)	0.238
Shivering	1 (3.0%)	0 (0.0%)	1.000
Allergic reaction	0 (0.0%)	0 (0.0%)	—

*p<0.05 (Fisher's exact test)

DISCUSSION

Both the groups were comparable in terms of age, gender, BMI, ASA status as well as pre operative vital parameters. The mean age of patients in both groups in our study was found to be around 43 years, which reflects the comparatively younger predominance of rheumatic aetiology in the Indian subcontinent, as compared to Western populations.⁹ Therefore, there is need to study the effective strategies for attenuating the haemodynamic responses to laryngoscopy and endotracheal intubation in this relatively young but high-risk population undergoing cardiac valve replacement surgeries in India. The study revealed that dexmedetomidine produced a clinically and statistically significant reduction in HR, consistent with presynaptic alpha-2 receptor-mediated inhibition of norepinephrine release at cardiac sympathetic terminals and reduced central sympathetic outflow from the locus coeruleus.¹⁰ However, magnesium sulphate was associated with a paradoxical mild HR increase, likely reflecting compensatory baroreflex-mediated sympathetic activation secondary to its vasodilatory effect. This observation is consistent with findings reported by Mahajan et al who demonstrated superior chronotropic control with dexmedetomidine under BI spectral index-controlled conditions.⁴

In our study, at the critical one-minute post-intubation time point, Group D (patients receiving dexmedetomidine) exhibited significantly lower heart rate, systolic and diastolic blood pressure and mean arterial pressure. These advantages were sustained throughout the 15-minute observation period, consistent with the terminal elimination half-life of dexmedetomidine (approximately two hours), which maintains therapeutic plasma concentrations across the peri-intubation window.¹¹ Our findings concur with those of Ali et al, Chickmath et al and Ghodki et al, all of whom documented superior HR control with dexmedetomidine in elective general surgical populations; the present study extends these observations to the more clinically challenging cardiac valve replacement cohort.¹²⁻¹⁴ The blood-pressure attenuation produced by magnesium sulphate, mediated through calcium channel blockade and inhibition of catecholamine release, appeared less complete at the 20 mg/kg dose employed.⁴ Investigations using higher magnesium doses (30–50 mg/kg) have reported more pronounced attenuation, and in some studies, a comparable performance to dexmedetomidine.⁴ Our conservative dosing choice reflected concern for hypotension and potentiation of neuromuscular blockade in haemodynamically vulnerable cardiac surgical patients, and may account for the relative inferiority of magnesium observed in our trial. The significantly higher incidence of sinus tachycardia in Group M (receiving magnesium sulphate) is an important safety finding. Tachycardia in the cardiac valve replacement population simultaneously increases myocardial oxygen demand while reducing diastolic filling time and sub-endocardial perfusion, representing a well-established substrate for perioperative

myocardial ischaemia in the hypertrophied and non-compliant ventricle. Hypotension rates were similar and manageable in both groups.

Regarding systolic blood pressure, baseline SBP was comparable between the dexmedetomidine and magnesium sulphate groups (123.2 ± 5.7 vs. 123.8 ± 6.3 mmHg; $p=0.300$). However, SBP was significantly lower with dexmedetomidine after drug administration and at all post-intubation time points ($p<0.001$). These findings are consistent with Mohan A et al and Joshi C et al who reported better attenuation of the post-intubation SBP rise with dexmedetomidine compared with magnesium sulphate.^{15,16} Mahajan L et al similarly observed a greater reduction in SBP with dexmedetomidine.⁴ In contrast, studies by Chaithanya K et al and Chandrakala M et al reported no significant intergroup difference in SBP from pre-induction to 5 minutes after intubation.^{17,18} The differences between these findings and our results may be related to variations in study population, drug dosage, timing of administration and anaesthetic protocol. Overall, our findings indicate that dexmedetomidine provided greater attenuation of the SBP response to laryngoscopy and intubation than magnesium sulphate. In our study, baseline diastolic blood pressure was significantly lower in the dexmedetomidine group at 1, 5 and 15 minutes after intubation ($p<0.001$). These findings are consistent with Mohan A et al who reported lower DBP with dexmedetomidine during induction and after intubation, with a greater rise in the magnesium sulphate group. Similarly, Mahajan L et al observed a significant reduction in DBP with dexmedetomidine and magnesium sulphate, with significant intergroup differences.^{4,15}

Our study reported that mean arterial pressure (MAP) was significantly lower in the dexmedetomidine group at 1, 5 and 15 minutes after intubation ($p<0.001$), with a smaller rise at 1-minute post-intubation. These findings are consistent with Mohan A et al who reported significantly lower MAP with dexmedetomidine during induction and after intubation, along with a significantly smaller post-intubation rise compared with magnesium sulphate.¹⁵ Thus, our findings support the superior efficacy of dexmedetomidine in attenuating the MAP response to laryngoscopy and endotracheal intubation.

In our study, both dexmedetomidine and magnesium sulphate attenuated the haemodynamic response to laryngoscopy and endotracheal intubation. However, dexmedetomidine demonstrated significantly greater control of HR, SBP, DBP and MAP at critical post-intubation time points, with a significantly lower incidence of post-intubation sinus tachycardia. Both agents were well tolerated, with no clinically significant adverse effects observed during the study period. These findings highlight the importance of effective pretreatment for maintaining haemodynamic stability during laryngoscopy and intubation in patients undergoing cardiac valve replacement surgery. Although magnesium sulphate remains a potential alternative, our findings support

dexmedetomidine 1 µg/kg as a more effective pretreatment agent, particularly when tight control of the chronotropic and pressor responses is desired.

Limitations

This study carries several limitations. It is a single-centre trial, which may limit external generalizability. The magnesium dose of 20 mg/kg may be suboptimal compared with the 30–50 mg/kg range used in most published comparative studies. The sample included heterogeneous valvular pathologies with differing haemodynamic profiles. Biochemical markers of sympathoadrenal activation (plasma catecholamines, troponin, BNP) were not assessed. The observation window was limited to 15 minutes post-intubation, and longer-term outcomes (ICU stay, postoperative myocardial injury, major adverse cardiac events) were not evaluated.

CONCLUSION

Intravenous dexmedetomidine at 1 mcg/kg, infused over 10 minutes prior to induction of general anaesthesia, provides significantly superior attenuation of the haemodynamic pressor and tachycardic response to laryngoscopy and endotracheal intubation compared with intravenous magnesium sulphate at 20 mg/kg in adults undergoing elective cardiac valve replacement surgery. Dexmedetomidine offers superior control of HR, SBP, DBP, and MAP at all critical post-intubation time points, with a significantly lower incidence of post-intubation sinus tachycardia. Both agents carry acceptable safety profiles. Dexmedetomidine is recommended as the preferred pretreatment agent before laryngoscopy and intubation in this high-risk surgical population, particularly when tight chronotropic control is the primary clinical objective. In resource-constrained settings where dexmedetomidine is unavailable, magnesium sulphate at higher doses (30 mg/kg) remains a viable alternative warranting further prospective evaluation.

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