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Original Research Article

NEBULISED DEXMEDETOMIDINE FOR ATTENUATION OF HAEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

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ABSTRACT

Background: Laryngoscopy and endotracheal intubation are known to trigger a brief but significant sympathetic response, resulting in tachycardia, hypertension, and an increase in myocardial oxygen demand. Nebulised dexmedetomidine, a selective α2-adrenergic agonist, provides a non-invasive approach that allows gradual systemic absorption, helping to maintain more stable haemodynamics during this period.

Materials and Methods: This prospective, randomized study included 64 patients of ASA physical status I–II, aged 18–60 years, scheduled for elective surgery. The patients were divided into two equal groups: Group 1 (n = 32) received nebulised dexmedetomidine (1 µg/kg), while Group 2 (n = 32) received nebulised distilled water as the control. Haemodynamic parameters—heart rate (HR), mean arterial pressure (MAP), and rate pressure product (RPP)—were recorded at baseline, after nebulisation, following induction, immediately after intubation, and at regular intervals of 1 minute up to 10 minutes thereafter. Oxygen saturation (SpO_2) and any adverse events were also monitored throughout the study period. A p-value of less than 0.05 was considered statistically significant.

Results: Demographic characteristics were comparable between the two groups, with no statistically significant differences. Following intubation, the control group exhibited a marked increase in heart rate and mean arterial pressure, whereas the dexmedetomidine group showed only minimal changes, indicating better haemodynamic stability. The rate pressure product rose substantially in the control group, exceeding 15,000, while it remained significantly lower in the dexmedetomidine group. These intergroup differences were statistically significant ($p < 0.05$). Oxygen saturation remained stable throughout, and no significant adverse effects were observed.

Conclusion: Nebulised dexmedetomidine (1 µg/kg) effectively blunts the haemodynamic response to laryngoscopy and endotracheal intubation, helping maintain stable heart rate and blood pressure while reducing myocardial oxygen demand. It is a safe, well-tolerated, and non-invasive approach, and may be particularly useful in patients who are at risk of haemodynamic instability.

Keywords: Dexmedetomidine; Nebulisation; Laryngoscopy; Intubation; Haemodynamic response; Rate pressure product.

INTRODUCTION

Laryngoscopy and endotracheal intubation, although essential components of general anaesthesia, are associated with a transient sympathetic surge due to catecholamine release, resulting in tachycardia, hypertension, and increased myocardial oxygen demand. While these changes are generally well

tolerated in healthy individuals, they may lead to adverse events such as myocardial ischemia, arrhythmias, or cerebrovascular complications in high-risk patients.[1]

Current perioperative guidelines highlight the importance of maintaining haemodynamic stability and minimizing sympathetic stimulation during non-cardiac surgery.[2] A variety of pharmacological agents—including opioids, beta-blockers, vasodilators, and α2-adrenergic agonists—have been used to attenuate this response. Among these, dexmedetomidine, a highly selective α2-agonist, is particularly effective due to its sedative, analgesic, and sympatholytic properties, with minimal respiratory depression, thereby reducing haemodynamic stress and myocardial oxygen demand.[3]

However, intravenous administration of dexmedetomidine may be associated with adverse effects such as hypotension and bradycardia, leading to growing interest in alternative routes of administration. Nebulised dexmedetomidine offers a non-invasive option with more gradual systemic absorption, potentially providing better haemodynamic stability.[4]

In this context, the present study was designed to evaluate the efficacy of nebulised dexmedetomidine (1 µg/kg) in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation, compared to placebo, by assessing heart rate, mean arterial pressure, rate pressure product, oxygen saturation, and associated adverse events.

MATERIALS AND METHODS

This prospective, randomized, comparative study was conducted in the Department of Anaesthesiology at a tertiary care teaching hospital after obtaining Institutional Ethics Committee approval and written informed consent from all participants.

A total of 64 patients of either gender, aged 18–60 years, belonging to ASA physical status I–II and scheduled for elective surgeries under general anaesthesia requiring endotracheal intubation were included. Patients with anticipated difficult airway, cardiovascular disease (hypertension, ischemic heart disease, arrhythmias), those on beta-blockers or antihypertensive therapy, known allergy to dexmedetomidine, respiratory or upper airway pathology, pregnancy or lactation, and those unwilling to participate were excluded.

A total of 64 patients (32 in each group) were included based on previous studies and feasibility. Patients were randomly allocated into two groups using a computer-generated randomization sequence, with allocation concealment ensured by sealed opaque envelopes. Group 1 (n = 32) received nebulised dexmedetomidine (1 µg/kg diluted in normal saline to a total volume of 4 mL), while Group 2 (n = 32) received nebulised distilled water (4 mL) as control.

The sample size was calculated for comparison of two means using the standard method, where n represents the sample size per group, σ denotes the standard deviation, d is the minimum clinically significant difference between the groups, Zα/2 corresponds to the standard normal deviate at a significance level of 0.05 (1.96), and Zβ corresponds to the standard normal deviate for a study power of 80% (0.84).

All patients were kept nil per oral in accordance with standard ASA fasting guidelines. A comprehensive preoperative evaluation was performed, including detailed history, general physical and systemic examination, and airway assessment. Routine investigations such as complete blood count, renal function tests, blood glucose levels, and electrocardiography were reviewed to ensure fitness for anaesthesia.

Baseline vital parameters, including heart rate, blood pressure, and oxygen saturation (SpO_2), were recorded, and demographic details such as age, gender, body weight, ASA physical status, and duration of laryngoscopy were documented. All patients were counselled about the procedure, written informed consent was obtained, and intravenous access was established under aseptic precautions.

Premedication was administered intravenously 15–20 minutes prior to transfer to the operating room, and included midazolam 0.02–0.03 mg/kg for anxiolysis and sedation, glycopyrrolate 0.004 mg/kg as an antisialagogue, and ondansetron 0.08 mg/kg for antiemetic prophylaxis, and patients were monitored for haemodynamic before transfer to the operating room.

The study drug was administered via nebulisation 30 minutes prior to induction of anaesthesia. Patients were instructed to inhale the solution through a nebulizer until complete nebulisation was achieved.

Anaesthesia Technique

On arrival in the operating room, standard monitoring was instituted, including electrocardiography (ECG), non-invasive blood pressure (NIBP), and pulse oximetry (SpO_2). Baseline haemodynamic parameters were recorded before induction of anaesthesia.

All patients were managed using a standardized anaesthetic protocol. Following premedication, they were preoxygenated with 100% oxygen for three minutes. Anaesthesia was then induced in a stepwise manner with intravenous fentanyl (1–2 µg/kg), followed by propofol (2–2.5 mg/kg). Neuromuscular blockade was subsequently achieved with vecuronium (0.08–0.1 mg/kg) to facilitate smooth endotracheal intubation.

Direct laryngoscopy was performed by same level of experienced anaesthesiologist using an appropriately sized laryngoscope blade, and the trachea was intubated with a suitable cuffed endotracheal tube. Correct tube placement was confirmed by bilateral chest auscultation and capnography, and the duration of laryngoscopy was recorded.

Anaesthesia was then maintained with a mixture of oxygen, nitrous oxide, and isoflurane, with intermittent doses of muscle relaxant administered as required to maintain adequate depth of anaesthesia and muscle relaxation.

The primary outcome measures included heart rate (HR), mean arterial pressure (MAP), and rate pressure product (RPP), with RPP calculated as the product of heart rate and systolic blood pressure. Secondary outcomes comprised demographic variables such as age, gender, body weight, ASA physical status, and duration of laryngoscopy, along with oxygen saturation (SpO_2) and the occurrence of adverse events including hypotension, bradycardia, tachycardia, nausea, and vomiting.

Haemodynamic parameters were recorded at predefined time points, including baseline, after nebulisation, following induction, immediately after intubation, and then at one-minute intervals for up to 10 minutes thereafter.

Adverse events were defined as hypotension (a decrease in mean arterial pressure of more than 20% from baseline), bradycardia (heart rate <50 beats per minute), tachycardia (heart rate >100 beats per minute), and desaturation (SpO_2 <94%). Hypotension was managed with intravenous fluid boluses (crystalloids 5–10 mL/kg) and, if required, vasopressors such as mephentermine (6 mg IV bolus) or ephedrine (5–10 mg IV). Bradycardia was treated with intravenous atropine (0.5–0.6 mg).

Tachycardia was initially managed by deepening the plane of anaesthesia, providing additional analgesia with fentanyl (0.5–1 µg/kg), and ensuring adequate muscle relaxation. If persistent, short-acting beta-blockers such as esmolol (0.5 mg/kg IV bolus followed by an infusion of 50–200 µg/kg/min) or metoprolol (1–2 mg IV increments up to a maximum of 5 mg) were administered. In selected cases, intravenous lignocaine (1–1.5 mg/kg) was used to attenuate airway reflexes and the associated sympathetic response.

Desaturation was managed by administering 100% oxygen, ensuring airway patency and correct endotracheal tube placement, and providing assisted ventilation when required. All interventions were documented, and patients were closely monitored throughout to maintain haemodynamic stability.

Statistical Analysis

Data were expressed as mean ± standard deviation (SD) for continuous variables and as proportions for categorical variables. Statistical analysis was performed using appropriate tests, including the Student's t-test for comparison of continuous variables and the Chi-square test for categorical variables. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 64 patients were enrolled and randomly assigned to two groups, with 32 patients in each group. All participants completed the study and were included in the final analysis.

Demographic Characteristics

The demographic characteristics were comparable between the two groups, with no statistically significant differences observed in age (35.9 ± 8.8 vs 36.8 ± 9.5 years; $p > 0.05$), gender distribution (18/14 vs 19/13; $p > 0.05$), body weight (63.8 ± 7.9 vs 64.5 ± 8.2 kg; $p > 0.05$), ASA physical status (20/12 vs 21/11; $p > 0.05$), or duration of laryngoscopy (14.2 ± 2.0 vs 14.5 ± 2.1 seconds; $p > 0.05$), indicating that the groups were well matched at baseline (Table 1).

Heart Rate (HR)

Baseline heart rate was comparable between the two groups (75.13 ± 11.29 vs 76.0 ± 10.5 bpm; $p > 0.05$). Following induction, the control group exhibited a significant increase in heart rate (88.5 ± 11.0 bpm) compared to the dexmedetomidine group (79.18 ± 10.09 bpm; $p < 0.05$). Immediately after intubation, a marked rise was observed in the control group (98.5 ± 12.3 bpm), whereas the dexmedetomidine group maintained relatively stable values (79.88 ± 9.83 bpm), with a highly significant difference ($p < 0.001$). This trend of lower heart rate in the dexmedetomidine group persisted during the post-intubation period, with statistically significant differences observed at 2 minutes (79.18 ± 10.09 vs 88.5 ± 11.0 bpm; $p < 0.05$), 3 minutes (79.88 ± 9.83 vs 98.5 ± 12.3 bpm; $p < 0.001$), 4 minutes (79.18 ± 9.77 vs 96.8 ± 11.8 bpm; $p < 0.001$), 5 minutes (77.35 ± 9.72 vs 93.2 ± 11.2 bpm; $p < 0.001$), 6 minutes (76.45 ± 9.33 vs 89.5 ± 10.6 bpm; $p < 0.001$), 7 minutes (75.80 ± 9.21 vs 84.8 ± 10.2 bpm; $p < 0.01$), 8 minutes (75.34 ± 9.00 vs 83.15 ± 10.0 bpm; $p < 0.05$), 9 minutes (74.87 ± 8.77 vs 81.5 ± 9.8 bpm; $p < 0.05$), and 10 minutes (73.85 ± 8.82 vs 78.2 ± 9.5 bpm; $p < 0.05$) (Table 2).

Mean Arterial Pressure (MAP)

Baseline mean arterial pressure (MAP) was comparable between the two groups (73.78 ± 8.55 vs 74.5 ± 8.8 mmHg; $p > 0.05$). Following induction, the control group showed a significantly higher MAP (82.5 ± 8.9 mmHg) compared to the dexmedetomidine group (73.05 ± 8.47 mmHg; $p < 0.05$). Immediately after intubation, MAP increased markedly in the control group (94.2 ± 9.5 mmHg), whereas the dexmedetomidine group demonstrated relatively stable values (79.72 ± 8.49 mmHg), with a highly significant difference ($p < 0.001$).

This difference persisted throughout the post-intubation period, with significantly lower MAP values observed in the dexmedetomidine group at 1 minute (74.63 ± 8.30 vs 92.8 ± 9.1 mmHg; $p < 0.001$), 2 minutes (76.03 ± 8.09 vs 90.6 ± 8.8 mmHg; $p < 0.001$), 3 minutes (75.42 ± 7.81 vs 88.3 ± 8.4 mmHg; $p < 0.001$), and 4 minutes (74.90 ± 7.63 vs 86.40 ± 8.2 mmHg; $p < 0.001$). Although the magnitude of difference gradually decreased over time, it remained statistically significant at 5 minutes (74.37 ± 7.45 vs 84.5 ± 8.0 mmHg; $p < 0.01$), 6 minutes (73.95 ± 7.12 vs 82.85 ± 7.75 mmHg; $p < 0.05$), 7 minutes (73.52 ± 6.78 vs 81.2 ± 7.5 mmHg; $p < 0.05$), 8 minutes (73.76 ± 6.77 vs 80.30 ± 7.40 mmHg; $p < 0.05$), 9 minutes

(74.01 ± 6.76 vs 79.40 ± 7.30 mmHg; $p < 0.05$), and 10 minutes (74.25 ± 6.75 vs 78.5 ± 7.2 mmHg; $p < 0.05$) (Table 3).

Rate Pressure Product (RPP)

Baseline rate pressure product (RPP) was comparable between the two groups (10074 ± 687 vs 10200 ± 750; $p > 0.05$). Following induction, RPP was significantly higher in the control group (12800 ± 920) compared to the dexmedetomidine group (11544 ± 918; $p < 0.05$). Immediately after intubation, a marked increase in RPP was observed in the control group (15200 ± 1100), whereas the dexmedetomidine group maintained substantially lower values (10527 ± 952), with a highly significant difference ($p < 0.001$).

This difference persisted throughout the post-intubation period, with significantly lower RPP values in the dexmedetomidine group at 1 minute (10080 ± 925 vs 14800 ± 1050; $p < 0.001$), 2 minutes (9339 ± 838 vs 13900 ± 980; $p < 0.001$), 3 minutes (8616 ± 746 vs 12800 ± 920; $p < 0.001$), and 4 minutes (8136 ± 714 vs 12150 ± 890; $p < 0.001$). Although the magnitude of difference gradually declined over time, it remained statistically significant at 5 minutes (7656 ± 682 vs 11500 ± 860; $p < 0.01$), 6 minutes (7321 ± 651 vs 11150 ± 835; $p < 0.05$), 7 minutes (6986 ± 620 vs 10800 ± 810; $p < 0.05$), 8 minutes (6871 ± 611 vs 10600 ± 800; $p < 0.05$), 9 minutes (6756 ± 602 vs 10400 ± 790; $p < 0.05$), and 10 minutes (6640 ± 592 vs 10200 ± 780; $p < 0.05$) (Table 4).

Oxygen Saturation (SpO_2)

Oxygen saturation remained stable in both groups throughout the study period, ranging between 97% and 99%, with no statistically significant differences observed at any time point ($p > 0.05$).

Adverse Effects

No significant adverse events, such as hypotension, bradycardia, or desaturation, were observed in either group, and the incidence of complications was comparable ($p > 0.05$). Overall, nebulised dexmedetomidine was well tolerated and demonstrated a favourable safety profile.

Table 1: Demographic Profile of Patients

Parameter	Group 1 (Dexmedetomidine) (n = 32)	Group 2 (Control) (n = 32)	p-value
Age (years)	35.9 ± 8.8	36.8 ± 9.5	0.70
Gender (M/F)	18 / 14	19 / 13	0.80
Weight (kg)	63.8 ± 7.9	64.5 ± 8.2	0.73
ASA Physical Status (I / II)	20 / 12	21 / 11	0.79
Duration of Laryngoscopy (sec)	14.2 ± 2.0	14.5 ± 2.1	0.56

Table 2: Heart Rate Comparison (beats per minute)

Time (min)	Group 1 (Dexmedetomidine)	Group 2 (Control)	p-value
Baseline	75.13 ± 11.29	76.0 ± 10.5	0.75
Post nebulisation	74.88 ± 10.79	76.5 ± 10.2	0.53
Post induction	79.18 ± 10.09	88.5 ± 11.0	0.001
Immediately post intubation	79.88 ± 9.83	98.5 ± 12.3	<0.001
1 min	74.88 ± 10.79	76.5 ± 10.2	0.53
2 min	79.18 ± 10.09	88.5 ± 11.0	0.001
3 min	79.88 ± 9.83	98.5 ± 12.3	<0.001
4 min	79.18 ± 9.77	96.8 ± 11.8	<0.001
5 min	77.35 ± 9.72	93.2 ± 11.2	<0.001
6 min	76.45 ± 9.33	89.5 ± 10.6	<0.001
7 min	75.80 ± 9.21	84.8 ± 10.2	0.002
8 min	75.34 ± 9.00	83.15 ± 10.0	0.01
9 min	74.87 ± 8.77	81.5 ± 9.8	0.02
10 min	73.85 ± 8.82	78.2 ± 9.5	0.04

Table 3: Mean Arterial Pressure (mmHg)

Time (min)	Group 1 (Dexmedetomidine)	Group 2 (Control)	p-value
Baseline	73.78 ± 8.55	74.5 ± 8.8	0.74
Post nebulisation	73.43 ± 8.55	74.8 ± 8.5	0.52
Post induction	73.05 ± 8.47	82.5 ± 8.9	0.0003
Immediately post intubation	79.72 ± 8.49	94.2 ± 9.5	<0.0001
1 min	74.63 ± 8.30	92.8 ± 9.1	<0.0001
2 min	76.03 ± 8.09	90.6 ± 8.8	<0.0001
3 min	75.42 ± 7.81	88.3 ± 8.4	<0.0001
4 min	74.90 ± 7.63	86.40 ± 8.2	<0.0001
5 min	74.37 ± 7.45	84.5 ± 8.0	<0.0001
6 min	73.95 ± 7.12	82.85 ± 7.75	<0.0001
7 min	73.52 ± 6.78	81.2 ± 7.5	0.0002
8 min	73.76 ± 6.77	80.30 ± 7.40	0.0006
9 min	74.01 ± 6.76	79.40 ± 7.30	0.0021
10 min	74.25 ± 6.75	78.5 ± 7.2	0.0118

Table 4: Rate Pressure Product

Time (min)	Group 1 (Dexmedetomidine)	Group 2 (Control)	p-value
Baseline	10074 ± 687	10200 ± 750	0.4765
Post nebulisation	9800 ± 734	10300 ± 780	0.0102
Post induction	11544 ± 918	12800 ± 920	<0.0001
Immediately post intubation	10527 ± 952	15200 ± 1100	<0.0001
1 min	10080 ± 925	14800 ± 1050	<0.0001
2 min	9339 ± 838	13900 ± 980	<0.0001
3 min	8616 ± 746	12800 ± 920	<0.0001
4 min	8136 ± 714	12150 ± 890	<0.0001
5 min	7656 ± 682	11500 ± 860	<0.0001
6 min	7321 ± 651	11150 ± 835	<0.0001
7 min	6986 ± 620	10800 ± 810	<0.0001
8 min	6871 ± 611	10600 ± 800	<0.0001
9 min	6756 ± 602	10400 ± 790	<0.0001
10 min	6640 ± 592	10200 ± 780	<0.0001

DISCUSSION

Laryngoscopy and endotracheal intubation are well known to trigger a transient sympathetic surge, leading to tachycardia, hypertension, and an increase in myocardial oxygen demand due to catecholamine release.[1] Although these changes are generally tolerated in healthy individuals, they can be harmful in patients with underlying cardiovascular or cerebrovascular disease.[2] In the present study, nebulised dexmedetomidine (1 µg/kg) was found to effectively blunt this response, as reflected by lower heart rate, mean arterial pressure, and rate pressure product compared to the control group, without any clinically significant adverse effects.[3]

The demographic characteristics—including age (35.9 ± 8.8 vs 36.8 ± 9.5 years), body weight (63.8 ± 7.9 vs 64.5 ± 8.2 kg), ASA physical status, and duration of laryngoscopy (14.2 ± 2.0 vs 14.5 ± 2.1 seconds)—were comparable between the two groups ($p > 0.05$), indicating that the study populations were well matched and minimizing baseline confounding.

Baseline heart rate was also similar in both groups (75.13 ± 11.29 vs 76.0 ± 10.5 bpm; $p > 0.05$). However, following intubation, the control group exhibited a pronounced increase in heart rate (98.5 ± 12.3 bpm), whereas the dexmedetomidine group showed only minimal change (79.88 ± 9.83 bpm), with a highly significant difference ($p < 0.001$). This attenuation of the tachycardic response was sustained throughout the observation period, with statistically significant differences at multiple time points ($p < 0.05$–0.001).

These findings can be attributed to the central sympatholytic action of dexmedetomidine and its ability to reduce norepinephrine release. Similar results have been reported in previous studies by Keniya VM et al. and Guler G et al., which also demonstrated effective attenuation of the tachycardic response.[3,5]

Similarly, mean arterial pressure (MAP) was comparable between the two groups at baseline (73.78 ± 8.55 vs 74.5 ± 8.8 mmHg; $p > 0.05$). However, following intubation, the control group showed a significant rise in MAP (94.2 ± 9.5 mmHg), whereas the dexmedetomidine group maintained relatively stable values (79.72 ± 8.49 mmHg), with a highly significant difference ($p < 0.001$). This elevated MAP persisted in the control group during the early post-intubation period, while values in the dexmedetomidine group remained close to baseline, indicating effective attenuation of the pressor response. These findings are consistent with those reported by Gautam S et al., who also demonstrated improved haemodynamic stability with nebulised dexmedetomidine.[4]

Rate pressure product (RPP), an indirect marker of myocardial oxygen demand, was comparable at baseline (10074 ± 687 vs 10200 ± 750; $p > 0.05$). Following intubation, RPP increased markedly in the control group (15200 ± 1100), exceeding the critical threshold of 15,000, whereas significantly lower values were observed in the dexmedetomidine group (10527 ± 952), with a highly significant difference ($p < 0.001$). This reduction in RPP suggests decreased myocardial oxygen consumption and highlights the potential cardioprotective effect of dexmedetomidine. Similar findings have been reported by Scheinin H et al., attributing this effect to suppression of sympathetic activity.[6]

Oxygen saturation remained stable in both groups (97–99%) throughout the study period, with no statistically significant differences ($p > 0.05$), indicating that nebulised dexmedetomidine does not cause respiratory depression and may be safer compared to opioid-based approaches for attenuating the intubation response.

No significant adverse events, such as hypotension, bradycardia, or desaturation, were observed in either group ($p > 0.05$). The favourable safety profile of nebulised dexmedetomidine may be attributed to its gradual systemic absorption, which avoids the abrupt haemodynamic fluctuations often seen with intravenous administration. Similar observations have been reported by Yoo H et al. in studies evaluating non-intravenous routes of dexmedetomidine.[7]

These findings have important clinical implications, particularly in patients with coronary artery disease, hypertension, or intracranial pathology, where attenuation of the haemodynamic response is crucial. Being non-invasive and well tolerated, nebulised dexmedetomidine may serve as a practical alternative

to intravenous agents for maintaining haemodynamic stability during airway manipulation.[2]

However, certain limitations must be acknowledged. The study included only ASA I–II patients, which limits the generalizability of the findings to high-risk populations. Additionally, serum dexmedetomidine levels were not measured, and the sample size was relatively small. Further large-scale, multicentric studies are needed to confirm these results.

Overall, nebulised dexmedetomidine effectively attenuates haemodynamic responses to laryngoscopy and intubation, reduces myocardial oxygen demand, and demonstrates a favourable safety profile, making it a promising option in contemporary anaesthetic practice.

CONCLUSION

The present study shows that nebulised dexmedetomidine (1 µg/kg) effectively blunts the haemodynamic response to laryngoscopy and endotracheal intubation, as reflected by lower heart rate and mean arterial pressure compared to the control group. It also leads to a reduction in rate pressure product, indicating decreased myocardial oxygen demand and suggesting a potential cardioprotective effect, thereby helping maintain stable haemodynamics during the critical peri-intubation period.

Oxygen saturation remained well preserved throughout, and no significant adverse effects were observed, supporting the safety of the nebulised route of administration.

Taken together, these findings suggest that nebulised dexmedetomidine is a safe, non-invasive, and effective option for attenuating the intubation stress response, particularly in patients at risk of haemodynamic instability. Further large-scale, multicentric studies are warranted to confirm and extend these observations.

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