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Original Article

Anaesthesia Section

Evaluation of Nebulised Dexmedetomidine in Blunting Haemodynamic Response to Laryngoscopy and Intubation in Adults Undergoing General Anaesthesia for Laparoscopic Surgeries: A Randomised Control Trial

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ABSTRACT

Introduction: Laryngoscopy and endotracheal intubation can trigger sympathetic stimulation, potentially leading to cardiovascular or cerebrovascular complications in high-risk patients. Dexmedetomidine, when administered via nebulisation, allows effective absorption without the adverse effects of intravenous administration (bradycardia and hypotension).

Aim: To assess the efficacy of nebulised dexmedetomidine in blunting haemodynamic response to intubation in adult patients undergoing general anaesthesia for laparoscopic surgery.

Materials and Methods: The present randomised control trial conducted during August 2023 to December 2024. A total of 60 American Society of Anaesthesiologists (ASA) I and II adult patients undergoing elective laparoscopic surgeries with tracheal intubation were randomised to receive either nebulised dexmedetomidine (1 µg/kg in 5 mL saline) or saline alone, 30 minutes prior to induction. Heart rate and Mean Arterial Pressure (MAP) were monitored for 10 minutes post-laryngoscopy every 15 minutes following pneumoperitoneum. Statistical analysis was done using Chi-square test and one way analysis of variance.

Results: There were no statistically significant differences between Group S and Group D in any of the demographic or surgical variables (p>0.05). A statistically significant difference was observed from 1-minute postintubation onward, with Group S showing consistently higher mean HR and MAP values than Group D. For HR, Group S had higher means at 1-min (105.36±19.58 vs 89.66±11.80 bpm, p=0.0004), 3-min (98.36±15.64 vs 85.40±12.59 bpm, p=0.0008), 5-min (p=0.0054), 10-min (p=0.0049), and all time points after CO_2 insufflation (p<0.0001). For MAP, Group S recorded higher values at 1-min (96.0±9.0 vs 90.0±10.09 mmHg, p=0.018), 3-min (93.0±8.23 vs 86.0±11.32 mmHg, p=0.008), 5-min (p=0.038), 10-min (p=0.014), and throughout CO_2 insufflation (p<0.0001).No significant differences were noted at baseline, after nebulisation, or after induction (p>0.05). It also attenuated response to pneumoperitoneum without any risk of adverse effects like bradycardia and hypotension.

Conclusion: Nebulised dexmedetomidine at a dose of 1 µg/kg effectively blunted the rise in heart rate and mean arterial pressure after laryngoscopy and minimised intraoperative haemodynamic fluctuations during pneumoperitoneum, without risk of adverse effects like bradycardia and hypotension.

Keywords: Adrenergic alpha-2 receptor agonists, Airway management, Blood pressure, Heart rate, Sympatholytics

INTRODUCTION

Direct laryngoscopy and tracheal intubation after anaesthesia induction can trigger significant haemodynamic responses due to increased sympathetic nervous system activity [1,2]. This typically results in a transient rise in blood pressure and heart rate, beginning within 30 seconds of intubation and generally resolving within 10 minutes [3]. Depending on the method of induction, and in the absence of specific interventions to blunt the haemodynamic response, laryngoscopy and intubation can lead to a rise in heart rate by approximately 26 to 66%, and an increase in blood pressure ranging from 36 to 45% [4]. In susceptible individuals, these acute changes may lead to serious complications such as myocardial ischaemia, cardiac arrhythmias, cerebrovascular events, pulmonary oedema, or elevated intracranial pressure [5]. To mitigate these risks, various pharmacological agents such as local anaesthetics, beta-blockers, calcium channel blockers, and opioid analgesics have been employed, although their effectiveness has been inconsistent across different studies [4,6,7].

Dexmedetomidine is a highly selective alpha-2 adrenergic agonist known for its sedative, analgesic, amnestic, and sympatholytic properties. These diverse effects have contributed to its growing use in the perioperative setting to reduce the need for anaesthetics and analgesics [8]. Its ability to attenuate the haemodynamic response to laryngoscopy and intubation has been evaluated via various administration routes, including intravenous, intranasal, and intramuscular [9-11]. While intravenous use can lead to adverse effects such as bradycardia and hypotension, and intranasal administration may cause local irritation, nebulised dexmedetomidine has emerged as a promising alternative. This route offers favourable bioavailability approximately 65% via the nasal and 82% via the buccal mucosa [12] and avoids the discomfort and complications often associated with intranasal delivery, such as nasal irritation, coughing, vocal cord irritation, or laryngospasm [13].

Despite evidence supporting dexmedetomidine's role in blunting the haemodynamic response to laryngoscopy and intubation, data on its nebulised form particularly in laparoscopic surgeries

involving pneumoperitoneum remain limited. Most existing studies involve heterogeneous surgical populations, short procedures, or brief monitoring periods, providing little insight into its sustained intraoperative effects. Furthermore, the optimal dose, route, and safety profile of nebulised dexmedetomidine in this setting are not well established. The present study aimed to evaluate the efficacy of nebulised dexmedetomidine in attenuating the haemodynamic responses associated with laryngoscopy and endotracheal intubation in adult patients undergoing general anaesthesia for laparoscopic surgeries

The primary objective of the present study was to compare the effects of nebulised dexmedetomidine and control on heart rate and blood pressure responses during laryngoscopy and intubation. The secondary objective was to assess its impact on haemodynamic changes during pneumoperitoneum and laparoscopic surgery, and to monitor for adverse effects, including bradycardia and hypotension.

MATERIALS AND METHODS

The present randomised, double-blind control study was conducted at a Tertiary Care Institution Government Medical College, Gondia, Maharashtra, India, following approval from the Institutional Ethics Committee (GMC/GONDIA/PHARMACOLOGY/IEC/07/2023). The study period spanned from August 2023 to December 2024. All procedures adhered to Good Clinical Practice guidelines and were in full compliance with the ethical principles outlined in the Declaration of Helsinki (1975), as revised in 2024. Patient safety and well-being were prioritized throughout the study. A total of sixty patients were enrolled in study.

Sample size calculation: The sample size was estimated based on a previous study by Shrivastava P et al.,[14] assuming: Formula used for sample size calculation:

$$N = \frac{(u+v)^2(\sigma_1^2+\sigma_0^2)}{(\mu_1-\mu_0)}$$

Where -

N = Sample size

μ_1 , μ_0 = Difference between the means (μ_1=125.92 and μ_0= 113.2)

σ_1, σ_0 = Standard deviations (σ_1=15.263 and σ_0 =14.503)

u = two sided percentage point of the normal distribution corresponding to 100 % - the power = 90%, u =1.282

v = Percentage point of the normal distribution corresponding to the (two sided) significance level for significance level = 5%, v = 1.960

$N = (1.282+1.960)^2(233.0+210.4)/(125.92–113.2)^2$

$N = 10.52\times443.4/(12.72)^2$

$N = 28.8 \approx 29$

$N_{adjusted}$= 29×100/90 =32-2´ 30 subjects per group(adjusted to 10% drop out)

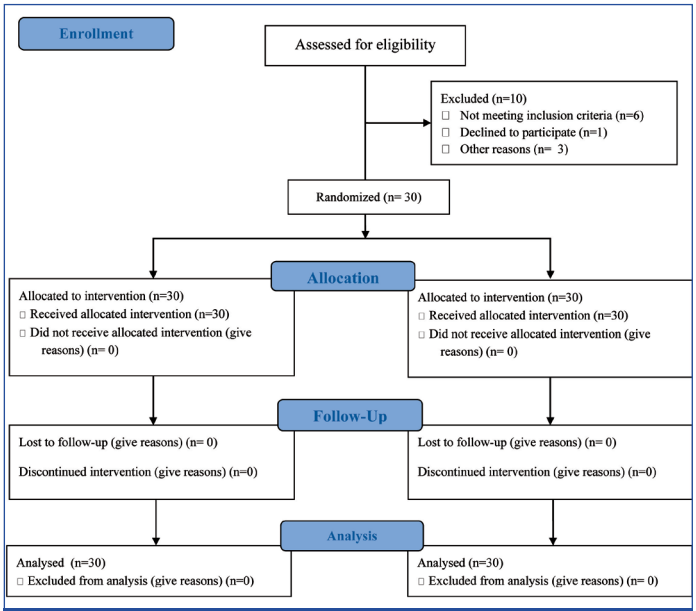
The sample size was calculated based on mean heart rate 5 minutes postintubation, aiming to detect a 20% difference between groups with 95% confidence and 90% power. Minimum of 26 patients per group was required and accounting for a 10% dropout, 30 patients were recruited per group.

Inclusion and Exclusion criteria: A total of sixty patients aged 18 to 60 years, of either gender, and classified as ASA physical status I or II, scheduled to undergo elective laparoscopic surgery under general anaesthesia, were enrolled. Patients refusing to give consent, allergic to study drugs, anticipated difficult intubation, BMI >30 kg/m^2, pregnant patients, psychiatric, neuromuscular, cardiovascular, hepatic, renal impairments, seizure disorders, or uncontrolled hypertension, patients on antidepressants, antipsychotics, beta-blockers, ARBs, or ACE inhibitors were excluded.

Study Procedure

Patients were preoperatively evaluated and randomly assigned to two groups of 30 using block randomisation with a computer-generated sequence [Table/Fig-1]. Sequence generation was performed by an independent statistician, enrolment by study investigator, and group allocation was communicated to the Anaesthesiologist administering nebulisation. The study was double-blinded, with patients and the Anaesthesiologist recording haemodynamic data blinded to allocation. Anaesthesia was maintained as per institutional protocol, and postoperative monitoring included haemodynamic assessment and management of adverse events such as bradycardia or hypotension. Patients were randomly assigned to one of the two groups for preoperative nebulisation. A dose of 1 µg/kg dexmedetomidine was used, shown previously to be effective [15].

- Group S (Saline group): Received nebulisation with 5 mL of 0.9% saline, administered 30 minutes prior to induction of anaesthesia.
- Group D (Dexmedetomidine group): Received nebulisation with dexmedetomidine at a dose of 1 µg/kg, diluted in 5 mL of 0.9% saline, administered 30 minutes before induction.



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

Bias was minimised through block randomisation, double-blinding of patients and outcome assessors, standardised anaesthesia and monitoring protocols, and objective recording of haemodynamic parameters.

One day prior to surgery, a thorough preoperative evaluation was conducted for each patient, including detailed medical history and clinical examination. The study protocol was explained in detail, and informed consent was obtained from all participants. On the day of surgery, patients in Group D (study group) received dexmedetomidine nebulisation at a dose of 1 µg/kg, diluted with normal saline to a total volume of 5 mL. Patients in the control group received nebulisation with 5 mL of normal saline following the same method, in accordance with their assigned group. Nebulisation was performed using an electrical compressor nebulizer (Eco Smart, Saify Healthcare and Medi Devices, India), designed to generate a fine mist suitable for optimal drug delivery. The entire volume (approximately 5 mL) was typically nebulised within 15-20 minutes, and the process was considered complete when no further mist was observed after tapping the volume chamber. The independent investigator closely monitored the entire nebulisation procedure and intervened in the event of adverse effects.

On day of surgery, Nil Per Os (NPO) status 8 hours for solids and 2 hours for clear liquids before surgery and consent was checked. An i.v. line was secured and Ringer's Lactate (RL) was started. Patients

were attached with standard monitors including Electrocardiogram (ECG), SpO_2, Non-Invasive Blood Pressure (NIBP), End Tidal CO_2 ($ETCO_2$), temperature probe and baseline parameters were recorded. Patients were premedicated with midazolam 0.02 mg/kg, fentanyl 2 µg/kg and pantoprazole 40 mg IV.

Induction of anaesthesia was achieved using 2.5 mg/kg of propofol, titrated to the loss of verbal response. Once effective bag-mask ventilation was confirmed, neuromuscular blockade was facilitated with an intubating dose of vecuronium bromide (0.1 mg/kg). Confirmation of tube placement was done with five auscultation and capnography. Patient was maintained using oxygen: nitrous mixture 50:50% along with sevoflurane to maintain MAC of 1-1.2. with the Bispectral Index (BIS) monitored and maintained between 50-60 to ensure adequate depth. Mechanical ventilation settings were adjusted to maintain an end-tidal CO_2 of 32-35 mmHg. Vecuronium was given intermittently as and when required while fentanyl 1 mcg/kg was administered when there was increase in haemodynamic values i.e. rise in heart rate and blood pressure more than 20% from baseline values. All laryngoscopies and intubations were performed by Anaesthesiologists with over 5 years of clinical experience to ensure consistency. Haemodynamic responses within 10 minutes postintubation were managed with propofol (20-30 mg) for hypertension, ephedrine (6 mg) for hypotension, and atropine (0.6 mg) for bradycardia. At the end of surgery, neuromuscular blockade was reversed with neostigmine (0.05 mg/kg) and glycopyrrolate (0.02 mg/kg). Tracheal extubation was performed once patients were responsive to verbal commands. Postoperatively, all patients were monitored in the Post-Anaesthesia Care Unit (PACU) for 3 hours and discharged to the ward upon meeting standard discharge criteria.

The primary objective was to evaluate changes in heart rate and mean arterial pressure following laryngoscopy and intubation between the two study groups, which was recorded at multiple predefined time intervals: before nebulisation, immediately after nebulisation and at 1, 3, 5, and 10-minutes postintubation. The secondary objective was to assess its impact on haemodynamic changes during pneumoperitoneum and laparoscopic surgery, and to monitor for adverse effects, including bradycardia and hypotension. So heart rate and mean arterial pressure were also monitored following CO_2 insufflation, 15, 30, 45-minute following CO_2 insufflation and following exsufflation.

STATISTICAL ANALYSIS

The statistical software namely Statistical Package for the Social Sciences (SPSS) 22.0 (IBM Corp, Armonk, NY, USA) and R environment ver.3.2.2 were used for the analysis of the data. Microsoft word and excel have been used to generate graphs, tables, etc. Analysis of variance has been used to find the significance of study parameters between three or more groups of patients. Post hoc Tukey test has been used to find the group wise significance. Chi-square/Fisher's exact test has been used to find the significance of study parameters on categorical scale between two or more groups. Non-parametric setting for qualitative data analysis and Fisher's exact test was used when cell samples were very small. p-value <0.05 was considered statistically significant, and p-value <0.001 was considered highly significant.

RESULTS

Total 60 patients were enrolled in study with 30 patients in each group. All these patients completed study and had comparable demographic parameters [Table/Fig-2].

Heart Rate (HR) was comparable between Group S and Group D at baseline, after nebulisation, and post-induction (p>0.05). However, from 1minute postintubation onwards, Group S exhibited higher HR values than Group D. This difference was statistically significant at all time points, including 1,3,5, and10 minutes postintubation, as well as after CO_2 insufflation, at 15, 30, and 45 minutes, and at exsufflation (p<0.01 for all). These findings indicate better intraoperative heart rate control in Group D [Table/Fig-3].

Parameters	Group S (Mean±SD)	Group D (Mean±SD)	p-value
Age (years)	34.23±10.72	34.13±12.22	0.96
Female: male (%)	18 (60%): 12 (40%)	15 (50%): 15 (50%)	0.43
Weight (kg)	55±6.4	55.4±6.3	0.82
ASA I:II	22 (73.3%): 8 (26.7%)	20 (66.7%): 10 (33.3%)	0.57
Duration of surgery (in hours)	1.03±0.13	0.97±0.18	0.93
Surgery performed	30	30	0.51
Lap appendectomy	23 (76.6%)	25 (83.3%)	
Lap cholecystectomy	7 (23.4%)	5 (16.5%)	

[Table/Fig-2]: Patient demographic parameters.
Values are expressed as mean±Standard Deviation (SD) or number (percentage). ASA: American society of anaesthesiologists physical status classification. Statistical analysis was performed using the independent t-test or Chi-square test as appropriate; p<0.05 was considered statistically significant

Heart Rate (HR beats/min)	Group S (Mean±SD)	Group D (Mean±SD)	p-value
Baseline HR	89.63±14.59	90.26±14.89	0.86
HR after nebulisation	90.43±14.65	90.1±16.85	0.45
HR After induction	87.80±7.09	88.33±7.07	0.77
HR 1-min postintubation	105.36±19.58	89.66±11.80	0.0003
HR 3-min postintubation	98.36±15.64	85.40±12.59	0.0008
HR 5-min postintubation	83.49±6.36	80.33±7.58	0.005
HR 10-min postintubation	88.0±12.06	79.56±10.22	0.004
HR after CO_2 Insufflation	86.80±5.47	74.60±4.32	<0.0001
HR after 15-min after CO_2 insufflation	85.60±5.43	73.03±4.10	<0.0001
HR after 30-min after CO_2 insufflation	85.47±6.47	73.20±5.19	<0.0001
HR after 45-min after CO_2 insufflation	85.67±9.68	73.73±6.97	<0.0001
At exsufflation	86.67±8.19	73.57±8.44	<0.0001

[Table/Fig-3]: Changes in Heart Rate (HR) in the dexmedetomidine group and the saline group.
Values are expressed as mean±Standard Deviation (SD). HR: Heart rate; CO_2: Carbon dioxide. Statistical analysis was performed using the independent t-test. p<0.05 was considered statistically significant

Mean Arterial Pressure (MAP) values were comparable between Group S and Group D at baseline and following nebulisation and induction, with no statistically significant differences (p>0.05). However, from 1-minute postintubation onward, Group D consistently exhibited lower MAP values compared to Group S. This difference reached statistical significance at 1-, 3-, 5-, and 10-minutes postintubation (p=0.018, 0.008, 0.038, and 0.014, respectively). Following CO_2 insufflation, MAP remained significantly lower in Group D at all recorded time points (15, 30, 45 minutes, and at exsufflation), with p-values<0.0001 [Table/Fig-4].

Group D had lower incidence of postoperative nausea and vomiting (1 vs. 6 patients; p=0.04) and shivering (0 vs. 2 patients; p=0.033) compared to Group S. Bradycardia, hypotension and sedation was not reported in any group [Table/Fig-5].

DISCUSSION

The present randomised control study is distinctive in utilising nebulised dexmedetomidine as a non-invasive technique to attenuate the stress response to laryngoscopy and intubation. Nebulisation provides a rapid onset of action and reliable systemic absorption owing to the large surface area of the respiratory mucosa. Compared with the intranasal route, it minimizes the risk of transient nasal irritation, coughing, or laryngospasm, and unlike the intravenous route, it reduces the likelihood of bradycardia and hypotension. This mode of delivery offers the advantage of a calm,

Mean arterial pressure (mmHg)	Group S (Mean±SD)	Group D (Mean±SD)	p-value
Baseline MAP	96.83±8.20	95.77±8.63	0.6276
MAP after nebulisation	96.0±8.18	94.0±10.71	0.4196
MAP after induction	94.0±8.10	92.0±08.70	0.3606
MAP 1-min postintubation	96.0±9.0	90.0±10.09	0.01819
MAP 3-min postintubation	93.0±8.23	86.0±11.32	0.008162
MAP 5-min postintubation	88.0±8.71	83.0±10.90	0.03786
MAP 10-min postintubation	85.0±8.29	79.0±9.98	0.01404
MAP after CO_2 insufflation	87.60±6.90	78.90±5.10	<0.0001
MAP after 15-min after CO_2 insufflation	88.30±6.26	78.67±4.11	<0.0001
MAP after 30-min after CO_2 insufflation	95.57±7.0	86.70±4.60	<0.0001
MAP after 45-min after CO_2 insufflation	97.90±7.35	87.40±5.0	<0.0001
At exsufflation	94.79±6.25	81.40±5.84	<0.0001

[Table/Fig-4]: Changes in Mean Arterial Pressure (MAP) mmHg in the dexmedetomidine group and the saline group.
Values are expressed as mean±Standard Deviation (SD). MAP: Mean arterial pressure; CO_2: Carbon dioxide. Statistical analysis was performed using the independent t-test; p<0.05 was considered statistically significant

Adverse effects	Group S (n=30)	Group D (n=30)	p-value
Postoperative sedation	0	0	-
Postoperative nausea and vomiting	6	1	0.04
Bradycardia	0	2	0.14
Hypotension	0	0	-
Shivering	2	0	0.03

[Table/Fig-5]: Incidence of adverse effects in the dexmedetomidine group and the saline group.
Values are expressed as number of patients (n). Statistical analysis was performed using the Chi-square or Fisher's exact test as appropriate. p<0.05 was considered statistically significant

adequately sedated patient without significant respiratory depression or haemodynamic instability. The observed effective suppression of the pressor and tachycardic responses following intubation with nebulised dexmedetomidine highlights its potential as a safe and efficacious alternative to conventional routes of administration. The present randomised controlled trial demonstrated that preoperative nebulised dexmedetomidine effectively attenuated the haemodynamic response to laryngoscopy and intubation without significant adverse effects.

In the present study, demographic parameters such as age, gender, weight, ASA status, and duration of surgery were comparable between groups, indicating adequate randomisation. The mean age was 34.23±10.72 years in Group S and 34.13±12.22 years in Group D (p=0.96), comparable to Kumar NRR et al., [16] (40.66±11.55 vs. 37.16±11.63 years) and Misra et al., [17] (40.6±12.0 vs. 37.7±10.5 years). Gender distribution (S: 60% males; D: 50% males) and mean weight (55±6.4 vs. 55.4±6.3 kg; p=0.82) were also similar across groups. Most patients were ASA I (73.3% vs. 66.7%), consistent with previous reports [16,17]. The mean duration of surgery was 1.03±0.13 hours in Group S and 0.97±0.18 hours in Group D (p=0.93). These findings align closely with those of earlier studies, confirming demographic comparability and ensuring that observed haemodynamic differences were attributable to the study intervention rather than baseline variability [16,17].

In present study, heart rate and MAP were significantly lower in the dexmedetomidine group compared with controls at 1, 3, 5, and 10 minutes postintubation. This sustained attenuation, including during CO_2 insufflation (p<0.0001), is likely due to its selective α-adrenergic agonist action, which reduces sympathetic outflow and circulating norepinephrine, leading to dose-dependent decreases in heart rate and arterial pressure. The results of present study align with those reported by Kumar NRR et al., [16], who evaluated 100 ASA I-II patients and found significant attenuation of systolic, diastolic, and mean arterial pressures in the dexmedetomidine group following laryngoscopy and intubation. That study also noted a reduction in response and state entropy and demonstrated a dose-sparing effect of propofol without excessive sedation. Both studies used an identical dexmedetomidine dose (1 µg/kg in 5 mL) and timing before induction, which may explain the comparable suppression of the haemodynamic response. In contrast to the present study, their evaluation did not include pneumoperitoneum, which represents an additional sympathetic stimulus; hence, the present study findings extend the evidence by demonstrating that nebulised dexmedetomidine also mitigates the pressor response to intra-abdominal insufflation during laparoscopic procedures.

The observations of the present study also partly agree with the findings of Misra et al., [17], which investigated 120 ASA I–II patients and reported attenuation of heart rate but not systolic blood pressure after laryngoscopy with nebulised dexmedetomidine (1 µg/kg in 3-4 mL saline, 30-min prior to induction). The discrepancy in blood pressure response between the current study and theirs may be attributable to methodological differences, such as the smaller nebulisation volume (3-4 mL vs. 5 mL) and the inclusion of varied elective surgical procedures rather than laparoscopic cases alone. Additionally, their use of linear mixed-effects modelling and relatively shorter observation window (10-min post-laryngoscopy) might have limited the detection of more sustained haemodynamic changes. They also reported significant anaesthetic- and opioid-sparing effects, highlighting the systemic absorption and clinical efficacy of nebulised dexmedetomidine.

Several studies [18,19] have reported that intravenous administration of dexmedetomidine, particularly when given 10 minutes prior to induction, can lead to adverse effects such as bradycardia, hypotension, hypertension, and respiratory depression. In contrast, the current study found that nebulised dexmedetomidine did not result in any significant change in heart rate at any time point during the observation period. The absence of bradycardia may be attributed to the avoidance of an intravenous bolus dose, which is often associated with a rapid onset of these adverse effects. Bradycardia was reported in 2 patients in dexmedetomidine group however it was not significant statistically.

These findings were similar to study conducted by Misra et al [17] and Kumar NRR et al., [16] demonstrated excellent haemodynamic stability, with no significant bradycardia or hypotension observed at any time point. Further, in the present study Postoperative Nausea and Vomiting (PONV) occurred in 6 patients (20%) in Group S and 1 patient (3.3%) in Group D (p=0.04), indicating a favourable antiemetic profile, possibly due to reduced opioid use and preoperative ondansetron administration. Misra et al., [17] similarly reported no significant difference in 2-hour postoperative PONV (p=0.612) or sore throat (p=0.741). These results suggest that nebulised dexmedetomidine offers haemodynamic stability with fewer adverse effects compared to the intravenous route.

Dexmedetomidine exerts its haemodynamic stabilising effects primarily through its highly selective agonist activity on presynaptic α2-adrenergic receptors, which leads to inhibition of norepinephrine release from the locus coeruleus - a mechanism widely proposed as the key factor in mitigating the stress response to airway manipulation [20]. When administered intravenously, dexmedetomidine typically has an onset of action within 5 minutes and a duration lasting up to 2 hours following a single dose, as reported in existing literature. In contrast, nebulised dexmedetomidine shows an onset of action around 15 minutes. However, it is hypothesized that the duration of its effect may extend beyond that of the intravenous route due to the gradual absorption of the drug through the respiratory mucosa into the systemic circulation [5]. The heart rate safety profile of nebulised dexmedetomidine may offer advantages over the intravenous route, particularly in patients with low baseline heart rates, such as those receiving preoperative beta-blocker therapy [16,21]. Additionally, nebulised dexmedetomidine has been associated with reduced postoperative sedation compared to its intravenous counterpart.

This characteristic may be especially beneficial in resource-limited settings where postoperative monitoring is insufficient, as well as in patients with conditions like obstructive sleep apnoea or chronic obstructive pulmonary disease, where excessive sedation could lead to adverse respiratory outcomes [21].

The findings of Systematic Review and Meta-Analysis (SRMA) done by Gupta M et al., [15] suggest that premedication with nebulised dexmedetomidine significantly attenuates the haemodynamic response to laryngoscopy and Endotracheal Intubation (ETI) compared to nebulised normal saline. All six Randomised Controlled Trials (RCTs) included in the analysis utilised a standardised dose of 1 µg/kg of dexmedetomidine, with a total of 480 patients evaluated across the studies.

Limitation(s)

Patients with anticipated difficult airways were excluded, which may limit the generalisability of the findings to broader clinical scenarios. Additionally, the duration of laryngoscopy and intubation was not accounted for, which could have influenced the haemodynamic responses observed. Furthermore, the results may not be applicable to high-risk populations, particularly those with significant comorbidities, as such patients were not included in the study cohort.

CONCLUSION(S)

Our findings reinforce that nebulised dexmedetomidine (1 µg/kg) effectively blunts the rise in HR and MAP after laryngoscopy and intubation, with additional attenuation of the haemodynamic response to pneumoperitoneum in laparoscopic surgeries. The drug was well tolerated, without any risk of adverse effects like bradycardia and hypotension. Collectively, these results strengthen the evidence supporting nebulised dexmedetomidine as a safe, effective, and patient-friendly alternative to the intravenous route for preoperative attenuation of airway and surgical stress responses.

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