# EFFECT OF DEXMEDETOMIDINE NEBULIZATION ON THE HEMODYNAMIC PARAMETERS DURING LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

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## ABSTRACT

Dexmedetomidine is an alpha-2 agonist used to attenuate the hemodynamic response to laryngoscopy and endotracheal intubation via the intravenous route. Our study was performed in 390 patients undergoing general anesthesia to assess the effect of dexmedetomidine nebulization on the hemodynamic parameters during laryngoscopy and endotracheal intubation. Group D received 0.75 mcg/kg dexmedetomidine diluted in 0.9% normal saline and group S received 3 ml of normal saline 30 minutes before induction of anesthesia. After laryngoscopy and intubation there was significantly lower trend of increase in heart rate and systolic blood pressure in group D ($p<0.001$) at 2, 4 and 6 minutes. Similarly, there was lower trend of increase in diastolic blood pressure and mean arterial pressure in 2, 4 ,6, 8 and 10 minutes ($p <0.001$). Dexmedetomidine by nebulization route attenuated the increase HR, SBP, DBP and MAP after laryngoscopy and endotracheal intubation. There was a reduction in intraoperative anesthetic and analgesic use. There was no increase in the incidence of adverse effects such as hypotension and bradycardia. Nebulized dexmedetomidine may be used for stabilization of hemodynamic parameters during laryngoscopy and endotracheal intubation.

## KEYWORDS

Dexmedetomidine, nebulization, laryngoscopy, intubation, hemodynamic

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## INTRODUCTION

Laryngoscopy and endotracheal intubation are core skills in anesthetic management under general anesthesia.[1] Direct laryngoscopy and tracheal intubation conceivably increase sympathetic response.[2] Laryngoscopy and further placement of the endotracheal tube stimulate sympathoadrenal receptors, triggering catecholamine release into the bloodstream. This leads to a transient pressor response characterized by elevated systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR).[1] These transient hemodynamic responses may not pose significant risks in healthy individuals without comorbidities. However, patients with limited cardiovascular reserve, coronary artery disease, cardiac dysrhythmia, cardiomyopathy, congestive heart failure, hypertension, limited intracranial compliance, and geriatric population may land to life-threatening complications such as myocardial ischemia, acute cardiac failure, and cerebrovascular hemorrhage.[3,4]

Alpha-2 agonists like clonidine and dexmedetomidine have been used intravenously in various clinical settings in view of diverse actions which includes sedation, analgesia, anxiolysis, perioperative sympatholysis, cardiovascular stabilizing effects, reduced anesthetic requirement and preservation of respiratory function.[5] The efficacy of dexmedetomidine in decreasing the hemodynamic response to laryngoscopy and intubation has been studied through intravenous,[6-9] intranasal[10,11] and intramuscular[12] routes. Nevertheless, these routes are not free from adverse effects. Intravenous administration may cause bradycardia and hypotension, and intranasal administration may be associated with irritation.[13] An alternative route that has recently been studied is nebulization of dexmedetomidine.

Nebulization of dexmedetomidine allows rapid drug absorption and has been used effectively and safely in children and adults for sedation. Nebulized dexmedetomidine may offer an attractive alternative to both intravenous as well as intranasal routes of administration. The aim of our study is to evaluate the effect of dexmedetomidine nebulization on the hemodynamic parameters during laryngoscopy and endotracheal intubation.

## METHODS AND MATERIALS

This hospital-based descriptive cross-sectional study was conducted on patients scheduled for elective surgery under general anesthesia at Nepal Medical College Teaching Hospital over 12 months (May 2023 to February 2024). Ethical approval was taken from the Institutional Review Committee of Nepal Medical College (Ref. No.: 61-079/080).

Informed consent was obtained. Pre-anesthetic checkup was done/reviewed on the previous day. All patients were kept fasting 6 hours for solid food and 2 hours for plain fluid. In the preoperative room, heart rate and blood pressure were recorded before intervention. Nebulization either with the study drug, dexmedetomidine 0.75 mcg/kg diluted in 3 ml of 0.9% saline or 0.9% normal saline (3ml) was done 30 minutes prior to the induction of anesthesia. In the operation theatre, standard monitoring (electrocardiogram, pulse oximetry, non-invasive arterial blood pressure) was attached. Baseline pulse, blood pressure and $SPO_2$ were recorded. Intravenous line was opened with 18/20G intravenous cannula on left arm. The anesthesia regimen and surgery was followed according to the hospital protocol. After preoxygenation, induction of anesthesia was done with injection midazolam 2mg, injection fentanyl 2 mcg/kg, injection propofol titrated to effect and rocuronium 0.8 mg/kg intravenously. Tracheal intubation was performed by an experienced anesthesiologist with at least two years of experience, followed by the initiation of intermittent positive pressure ventilation. The Patients received injection granisetron (40 mcg/kg) after induction of anesthesia. Anesthesia was maintained with oxygen, isoflurane and supplemental doses of injection vecuronium (0.01 mg/kg intravenously).

The Heart rate, systolic and diastolic blood pressure was recorded before administration of nebulization, after nebulization but before induction (baseline), and at every 2 minutes interval until 10 minutes of laryngoscopy. Inj. Propofol 20-30 mg was given intravenously if there was increase in heart rate and blood pressure of more than 20.0% within the first 10 minutes following laryngoscopy and intubation. Bradycardia was treated with inj. Atropine 0.6mg and hypotension was treated with injection mephenteramine 5mg intravenously. Administration of additional doses of fentanyl was given when there was increase in heart rate and/or blood pressure more than 20.0% of the baseline during the intraoperative period. The total doses of intraoperative fentanyl and propofol were recorded.

At the end of surgical procedure, residual neuromuscular block was adequately reversed using neostigmine (0.04 mg/kg) and

injection glycopyrrolate (0.2 mg for every 1 mg of neostigmine). Patients were extubated after meeting extubation criteria and shifted to post anesthesia care unit. The data was analysed by using SPSS-20.

*Inclusion Criteria:*

- Age between 16-65 years of either gender
- ASA I and II
- Patients scheduled for elective surgery under general anesthesia
- Patients who received nebulization either with dexmedetomidine or normal saline 30 minutes prior to induction of anesthesia

*Exclusion criteria:*

- Age less than 16 or more than 65 years
- ASA III or more
- Allergy to dexmedetomidine
- Patient refusal
- Pregnancy
- Emergency Surgery
- Laryngoscopy time more than 20 seconds
- Obese patients (Body Mass Index >30 kg/m$^2$)

## RESULTS

**Table 1: General characteristics**

| Variables | N (%) |
|---|---|
| **Age** | |
| 16-25 | 67 (17.2) |
| 26-35 | 83 (21.3) |
| 36-45 | 93 (23.8) |
| 46-55 | 66 (16.9) |
| 56-65 | 81 (20.8) |
| **Gender** | |
| Male | 131 (33.6) |
| Female | 259 (66.4) |
| **Group** | |
| D (Dexmedetomidine) | 195 (50.0) |
| S (Normal Saline) | 195 (50.0) |
| **ASA Grade** | |
| I | 244 (62.6) |
| II | 146 (37.4) |

This table shows a distribution across age, gender, group assignment, and ASA classification.

**Table 2: Independent samples test for heart rate (HR) measurements before and after nebulization**

| Time Point | Group D (Dexmedetomidine) | Group S (Normal Saline) | Mean Difference ± SD | P-value (Independent t-test) |
|---|---|---|---|---|
| HR Before Nebulization | 75.68 ± 9.15 | 75.72 ± 9.18 | -0.04 ± 0.17 | 0.864 |
| HR After Nebulization (Pre-induction) | 76.88 ± 9.38 | 78.24 ± 9.28 | -1.36 ± 0.59 | 0.174 |
| HR at 2 Minutes | 74.13 ± 11.45 | 79.01 ± 11.14 | -4.88 ± 0.98 | <0.001 |
| HR at 4 Minutes | 75.30 ± 10.87 | 79.63 ± 10.53 | -4.33 ± 0.93 | <0.001 |
| HR at 6 Minutes | 75.10 ± 10.25 | 78.72 ± 10.50 | -3.62 ± 0.92 | <0.001 |
| HR at 8 Minutes | 74.10 ± 9.80 | 76.88 ± 10.25 | -2.78 ± 0.89 | 0.002 |
| HR at 10 Minutes | 73.20 ± 9.55 | 75.50 ± 9.90 | -2.30 ± 0.85 | 0.007 |

**Table 3: Group statistics for systolic blood pressure (SBP) measurements**

| Time Point | Group D (Dexmedetomidine) | Group S (Normal Saline) | Mean Difference ± SD | P-value (Independent t-test) |
|---|---|---|---|---|
| SBP Before Nebulization | 123.62 ± 10.42 | 122.39 ± 11.23 | 1.23 ± 1.01 | 0.131 |
| SBP Pre-Induction | 123.93 ± 10.83 | 122.86 ± 11.12 | 1.07 ± 1.01 | 0.169 |
| SBP at 2 Minutes | 122.37 ± 11.37 | 126.63 ± 12.14 | -4.26 ± 1.18 | <0.001 |
| SBP at 4 Minutes | 120.72 ± 10.76 | 125.22 ± 11.80 | -4.50 ± 1.15 | <0.001 |
| SBP at 6 Minutes | 120.09 ± 11.05 | 123.58 ± 10.73 | -3.49 ± 1.10 | <0.001 |
| SBP at 8 Minutes | 119.75 ± 11.32 | 122.85 ± 11.91 | -3.10 ± 1.09 | 0.004 |
| SBP at 10 Minutes | 120.05 ± 10.24 | 122.74 ± 11.01 | -2.69 ± 1.08 | 0.006 |

**Table 4: Group statistics for diastolic blood pressure (DBP) measurements**

| Time Point | Group D (Dexmedetomidine) | Group S (Normal Saline) | Mean Difference ± SD | P-value (Independent t-test) |
|---|---|---|---|---|
| DBP Before Nebulization | 79.44 ± 8.65 | 79.22 ± 7.50 | 0.22 ± 0.61 | 0.392 |
| DBP Pre-Induction | 79.37 ± 8.26 | 79.81 ± 7.09 | -0.44 ± 0.59 | 0.288 |
| DBP at 2 Minutes | 79.04 ± 8.75 | 82.45 ± 8.51 | -3.41 ± 0.88 | <0.001 |
| DBP at 4 Minutes | 76.95 ± 8.79 | 81.47 ± 8.34 | -4.52 ± 0.87 | <0.001 |
| DBP at 6 Minutes | 76.87 ± 8.63 | 80.82 ± 7.40 | -3.95 ± 0.86 | <0.001 |
| DBP at 8 Minutes | 76.94 ± 8.77 | 80.31 ± 7.93 | -3.37 ± 0.87 | <0.001 |
| DBP at 10 Minutes | 77.03 ± 8.41 | 80.16 ± 7.08 | -3.13 ± 0.86 | <0.001 |

**Table 5: Independent samples test for mean arterial pressure (MAP)**

| Time Point | Group D (Dexmedetomidine) | Group S (Normal Saline) | Mean Difference ± SD | P-value (Independent t-test) |
|---|---|---|---|---|
| MAP Before Nebulization | 94.03 ± 8.50 | 93.39 ± 8.17 | 0.64 ± 0.72 | 0.224 |
| MAP Pre-Induction | 94.16 ± 8.48 | 94.02 ± 8.50 | 0.14 ± 0.71 | 0.434 |
| MAP at 2 Minutes | 93.42 ± 8.81 | 97.03 ± 8.79 | -3.61 ± 0.89 | <0.001 |
| MAP at 4 Minutes | 91.38 ± 8.74 | 95.87 ± 9.14 | -4.49 ± 0.88 | <0.001 |
| MAP at 6 Minutes | 90.99 ± 8.61 | 94.93 ± 7.94 | -3.94 ± 0.85 | <0.001 |
| MAP at 8 Minutes | 90.85 ± 8.68 | 94.19 ± 8.60 | -3.34 ± 0.86 | <0.001 |
| MAP at 10 Minutes | 91.18 ± 8.29 | 94.09 ± 8.01 | -2.91 ± 0.84 | <0.001 |

**Table 6: Group* added propofol cross-tabulation**

| Group | 0 mg | 20 mg | 30 mg | 50 mg | Total |
|---|---|---|---|---|---|
| Group D | 180 | 12 | 2 | 1 | 195 |
| Group S | 155 | 38 | 2 | 0 | 195 |
| P-value (Chi-square test) | <0.001 | | | | |

A total of 390 cases were enrolled, with participants evenly divided (n = 195 per group) between Group D (Dexmedetomidine) and Group S (normal saline).

Participants span a broad age range, with the largest age group being 36-45 years (23.8%), followed by 26-35 years (21.3%) and 56-65 years (20.8%), ensuring diverse age representation. The gender distribution is skewed towards females, who comprise 66.4% of the sample, compared to 33.6% males. Most participants are classified as ASA Grade I (62.6%), indicating they are generally healthy, while 37.4% fall into ASA Grade II. Overall, the sample's demographic and baseline characteristics provide a diverse and balanced foundation for analysing the effects of dexmedetomidine.

There were no significant differences in baseline HR between the two groups (Table 2). Following nebulization, the group D demonstrated a statistically significant reduction in HR at 2, 4 and 6 minutes post-intubation compared to group S (p <0.001 for each time point, Table 1). Similarly, the increase in heart rate was significantly less in group D at 8 and 10 minutes with p value 0.002 and 0.007, respectively.

No significant differences were observed in baseline SBP between groups. Post-nebulization, Group D exhibited a significantly lower increase in SBP at 2, 4, and 6 minutes post-intubation compared to Group S (p <0.001; Table 3), as well as at 8 minutes (p =0.004) and 10 minutes (p =0.006).

Similarly, the rise in diastolic blood pressure after intubation was significantly lower in the group D at 2, 4, 6, 8, and 10 minutes (p <0.001, Table 4). These results align with the SBP findings, suggesting that dexmedetomidine helps mitigate blood pressure fluctuations.

MAP levels followed a similar trend, with significant reduction in the trend of increase in MAP at 2, 4, 6, 8 and 10 minutes in group D (p <0.001; Table 5).

The group D required significantly less propofol compared to the group N (p <0.001, Table 6), with over 92% of the group D needing no propofol, compared to 79.5% in the group N.

| Table 7: Group * added fentanyl cross-tabulation | | | |
|---|---|---|---|
| **Group** | **0 mcg** | **25 mcg** | **Total** |
| Group D | 190 | 5 | 195 |
| Group S | 179 | 16 | 195 |
| P-value (Chi-sq. test) | 0.014 | | |

A similar pattern was observed for fentanyl, with significantly fewer group participants requiring supplemental doses (p =0.014, Table 7). Five patients required additional fentanyl in group D whereas 16 patients required additional fentanyl in group S.

## DISCUSSION

Direct laryngoscopy and endotracheal intubation following induction of anesthesia causes hypertension and/or tachycardia due to increased sympathoadrenal activity. The stimulation of the supraglottic region results in the somatovisceral kind of reflex.[1] Modern anesthesia practices plan to prevent sympathetic discharge and provide hemodynamic stability perioperatively. Various strategies with drugs and non-pharmacological techniques have been carried out for obtunding the stress response to laryngoscopy and intubation, including opioids, barbiturates, benzodiazepines, beta-blockers, calcium channel blockers, and vasodilators.[14]

Dexmedetomidine, a selective α2 agonist with eight times greater affinity for α2-adrenergic receptors than clonidine, possesses all the properties of an α2 agonist without causing respiratory depression. Intravenous use of dexmedetomidine in the perioperative period has been found to decrease serum catecholamine levels by 90.0% which helps to blunt the haemodynamic response to laryngoscopy and endotracheal intubation.[14] A biphasic response on blood pressure occurs with a bolus or loading dose. Initially, there is hypertension followed by hypotension which is thought to be due to stimulation of $\alpha_2$ B receptors in the vascular smooth muscle. It also causes bradycardia when used intravenously requiring management with injection atropine at times.

Dexmedetomidine nebulization allows rapid drug absorption, with a bioavailability of 65.0% through the nasal mucosa and 82.0% through the buccal mucosa.[15] Nebulization is easier, provides high bioavailability and maintains hemodynamic parameters better than the intravenous routes.[17] It avoids transient nasal irritation, coughing and vocal cord irritation associated with intranasal administration.[18] Hence nebulization is an attractive alternative to both intravenous and intranasal routes of administration.

The dose of dexmedetomidine that we used in our study was 0.75 mcg/kg which is lesser than the dose used by Mishra *et al.*[16] They analysed the effect of dexmedetomidine nebulization using 1 mcg/kg and concluded that it attenuated the increase in heart rate but not the systolic blood pressure following laryngoscopy. In contrast to that our study showed that dexmedetomidine nebulization significantly attenuated the rise of heart rate and SBP at 2, 4 and 6 minutes of laryngoscopy and intubation. It also attenuated the rise of DBP and MAP at 2, 4, 6, 8 and 10 minutes. Fasil *et al*[19] used 1mcg/kg of dexmedetomidine nebulization 10 minutes before intubation and concluded that there was fall in HR in the dexmedetomidine group as compared to saline group but overall the HR was stable during post intubation period and pneumoperitoneum period. Similar to above mentioned study, our study showed stable heart rate even with 0.75 mcg/kg dose. Six patients in their study developed bradycardia and hypotension and were treated with single dose of injection atropine 0.6 mg intravenously. In contrast to that no event of bradycardia was observed in our study. This may be due to the lower dose of dexmedetomidine that we used. Our observation was similar to that of Srimaka *et al*[18] who found that SBP was better controlled post-nebulization and no patient developed bradycardia or hypotension requiring treatment.

Dexmedetomidine has been proven to consistently reduce the requirement of opioids, propofol and benzodiazepines.[19] In our study, 92% of the patients did not require added doses of propofol as compared to 79.5% patients requiring added doses of propofol in group S. Identical result was seen in the study done by Mishra *et al*[16] who observed that less dose of propofol (1.5±0.6) mg/kg was required in dexmedetomidine group as compared to 1.9±0.6 mg/kg of propofol in the saline group (p <0.001). Similarly the rescue analgesia was required early (55.5 mins) in saline group as compared to dexmedetomidine group (173 minutes in dexmedetomidine 0.2 and 249 minutes in dexmedetomidine 0.4 group) in the study done by Manne *et al.*[14] In our study 5 patients required additional fentanyl in group D whereas 16 patients required additional fentanyl in group S.

Nebulisation is painless, comfortable, provides higher bioavailability and maintains haemodynamic parameters better than the intravenous route. It avoids transient nasal irritation and coughing associated with intranasal administration. Dexmedetomidine nebulization has good bioavailability due to large mucosal surface and has short half-life. It is effective in reducing the stress response

to laryngoscopy and intubation. It decreases overall use of anaesthetic and analgesic agents and has fewer side effects.

In conclusion, dexmedetomidine nebulization at a dose of 0.75 mcg/kg helps maintain hemodynamic stability during laryngoscopy and intubation without adverse effects. It also helped to decrease the doses of anesthetic agents and opioid use. Nebulization of dexmedetomidine is a good alternative route for maintenance of hemodynamic stability during laryngoscopy and endotracheal intubation.

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