



IJMPR



Copyright@IJMPR

## Evaluation of Nebulised Dexmedetomidine in Blunting Haemodynamic Response to Laryngoscopy and Intubation: Tertiary Care Centre

Dr Kirti Chavan<sup>1</sup>, Dr A.S.Tarkase<sup>1</sup>, Dr Mayuri Mali<sup>1</sup>

<sup>1</sup>Department of Anaesthesiology, SRTR Government Medical college, Ambajogai, Beed, Maharashtra

### ABSTRACT

**Introduction:** The process of laryngoscopy and endotracheal tube intubation causes immense sympathetic system activation which further causes intra-operative complications. In high risk patients, this pressor response can increase morbidity and mortality. Dexmedetomidine has good bioavailability and rapid absorption through nasal mucosa so this study evaluates effects of nebulised dexmedetomidine in blunting haemodynamic response to laryngoscopy and intubation. **Material and methods:** Analytical study was conducted in 100 ASA 1&2 patients. Study population was divided randomly into 2 groups. Control group B(n=50)received nebulisation with 5ml of normal saline and Study group A(n=50)received 1mics/kg dexmedetomidine in 5ml saline 10min before induction in sitting position. **Results:** Demographics are compared. Following laryngoscopy and intubation SBP, DBP, MAP, HR showed significant increase in control group B as compared to study group A. There was a dose sparing effect of propofol in group A. **Conclusion:** Nebulised dexmedetomidine effectively blunts the stress response to laryngoscopy and intubation with no adverse effects.

**Key Words:** Dexmedetomidine, intubation, laryngoscopy, premedication, sedation



\*Corresponding Author

Dr Kirti Chavan

Department of Anaesthesiology, SRTR Government Medical college, Ambajogai, Beed, Maharashtra

### INTRODUCTION

Direct laryngoscopy and intubation causes variable haemodynamic changes. Response occurs within 30sec after intubation and last less than 10min[1].Consequences:Ischaemia, arrhythmia, cerebrovascular stroke, pulmonary edema, raised ICT[2,3]. Dexmedetomidine: Selective short acting alpha 2 adrenoceptor agonist. It has sedative, hypnotic, anxiolytic, analgesic, antisialogogue, sympatholytic properties[4]. It promotes cardiac, respiratory, neurological stability. Dexmedetomidine has potential to produce bradycardia, hypotension when given as bolus so to circumvent this nebulisation route is chosen[5-8]. Dexmedetomidine has bioavailability of 65% through nasal mucosa and 82% through buccal mucosa. Nebulised route is chosen over intranasal as it avoids transient nasal irritation, vocal cord irritation, cough, laryngospasm. Primary aim of study is to evaluate it's role in blunting haemodynamic response to laryngoscopy and intubation. Secondary aim is to study adverse effect of drug such as bradycardia, hypotension, dose sparing effect of propofol

### MATERIAL AND METHODS

Prospective study design which recruited 100 adult patients. Study population: 18-60yr age and ASA 1&2 Place of study: Tertiary care hospital. Parameters observed: Heart rate, Systolic blood pressure, Diastolic blood pressure, Mean arterial pressure. Above findings will be observed till 10min after intubation. This was prospective study, study population was randomised into two groups Group B(control group) received 5 ml of normal saline nebulisation and Group A(Study group) received dexmedetomidine at a dose of 1mics/kg (mixed with saline to total volume of 5ml) nebulisation with nebuliser face mask with continuous flow of 100% o2 at 6L/min for 10min before induction in sitting position Premedication with Inj glyco 0.004mg/kg, inj Fentanyl 2mics/kg, induced with inj propofol 1-2mg/kg titrated to the loss of verbal response and the amount of drug administered was noted and atracurium 0.5mg/kg as a muscle relaxant to facilitate intubation with an aim to maintain both response entropy (RE) and state entropy (SE) around 40-60.Direct laryngoscopy and intubation done with appropriate size ETT and pt connected to ventilator Patient is undisturbed for 10 min HR, SBP, DBP, SPO2 noted at following time points: baseline after nebulisation, post intubation at 1,5,10 min and study ends here. Once surgical procedure done neuromuscular blockade was reversed with inj Neostigmine and glycopyrrolate then trachea was extubated

### RESULT

Demographics are compared. Following laryngoscopy and intubation SBP, DBP, MAP, HR showed significant increase in control group B as compared to treatment group A

**DISCUSSION**

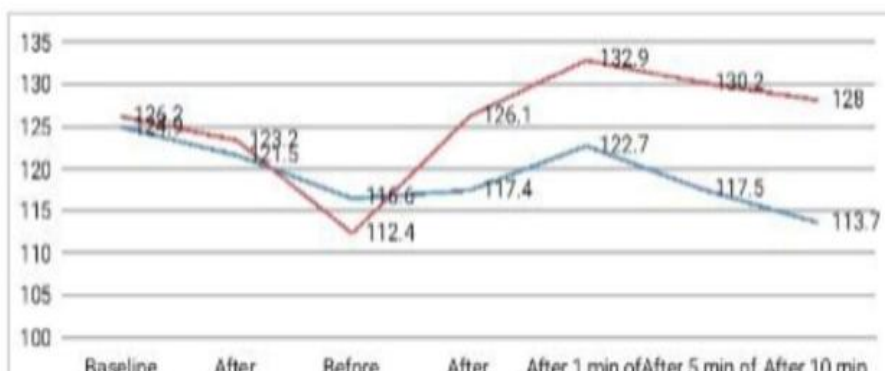
There is no significant association seen among cases and controls for gender (p value 0.6889) and age (p value was 0.3130).

***Distribution of Study subjects according to Systolic Blood pressure.***

We observed systolic BP. Baseline SBP was less in study group  $124.9 \pm 8.284$  than control group  $126.2 \pm 9.409$  with p value 0.479. After nebulization SBP was less in study group  $121.5 \pm 8.205$  than control group  $123.2 \pm 9.184$  with p value 0.3258. Before laryngoscopy SBP was more in study group  $116.6 \pm 5.753$  than control group  $112.4 \pm 7.002$  with p value 0.0015. After intubation SBP was less in study group  $117.4 \pm 6.608$  than control group  $126.1 \pm 10.99$  with p value 0.0001. After 1 min of intubation SBP was less in study group  $122.7 \pm 9.13$  than control group  $132.9 \pm 9.572$  with p value 0.0001. After 5 min of intubation SBP was less in study group  $117.5 \pm 9.177$  than control group  $130.2 \pm 9.323$  with p value 0.0001. After 10 min of intubation SBP was less in study group  $113.7 \pm 9.459$  than control group  $128 \pm 9.138$  with p value 0.0001. Kumar NR et al [9] in their study had similar observation that SBP values after nebulisation and immediately after intubation were comparable in both groups. The SBP values at 1, 5 and 10 min after intubation were lower in study group in a statistically significant manner with P values of 0.01, 0.02, 0.03, respectively (Graph 4)

**Table no.06: Distribution of study subjects according to systolic blood pressure.**

SBP	Study Group	Range	Control Group	Range	p value
Baseline	$124.9 \pm 8.284$	110-140	$126.2 \pm 9.409$	108-150	0.479
After nebulization	$121.5 \pm 8.205$	104-136	$123.2 \pm 9.184$	104-144	0.3258
Before Laryngoscopy	$116.6 \pm 5.753$	108-126	$112.4 \pm 7.002$	101-124	0.0015
After intubation	$117.4 \pm 6.608$	106-127	$126.1 \pm 10.99$	110-143	0.0001
After 1 min of Intubation	$122.7 \pm 9.13$	100-140	$132.9 \pm 9.572$	114-158	0.0001
After 5 min of Intubation	$117.5 \pm 9.177$	98-140	$130.2 \pm 9.323$	112-155	0.0001
After 10 min of Intubation	$113.7 \pm 9.459$	90-128	$128 \pm 9.138$	110-150	0.0001



**Graph 4: Distribution of study subjects according to systolic blood pressure**

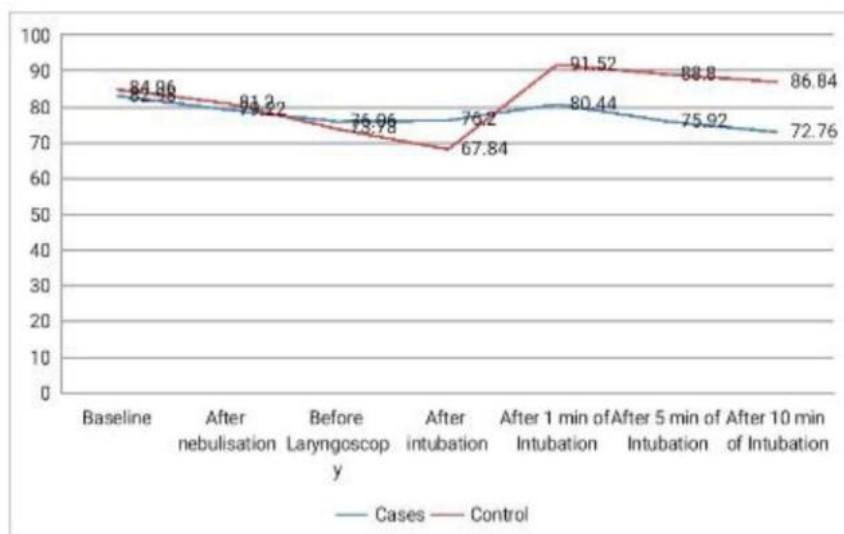
**Distribution of Study subjects according to Diastolic Blood Pressure**

We observed diastolic BP. Baseline DBP was less in study group  $82.98 \pm 8.16$  than control group  $84.96 \pm 6.543$  with p value 0.1838. After nebulization DBP was less in study group  $79.22 \pm 7.802$  than control group  $81.20 \pm 9.502$  with p value 0.2576. Before laryngoscopy DBP was more in study group  $76.06 \pm 3.766$  than control group  $73.78 \pm 6.926$  with p value 0.0435. After intubation DBP was more in study group  $76.2 \pm 6.051$  than control group  $67.84 \pm 7.17$  with p value 0.0001. After 1 min of intubation DBP was less in study group  $80.44 \pm 8.947$  than control group  $91.52 \pm 6.109$  with p value 0.0001. After 5 min of intubation DBP was less in study group  $75.92 \pm 8.6$  than control group  $88.8 \pm 5.813$  with p value 0.0001.

After 10 min of intubation DBP was less in study group  $72.76 \pm 8.916$  than control group  $86.84 \pm 5.884$  with p value 0.0001. (Graph 3) Shrivastava P et al 2022[10] in their study stated that there was a significant difference in mean DBP at time intervals before laryngoscopy ( $p < 0.001$ ), after intubation ( $p = 0.034$ ), after one minute ( $p = 0.011$ ), after five minutes ( $p < 0.005$ ), and after 10 minutes ( $p = 0.009$ ) of intubation when compared between the two groups; however, at baseline, no significant difference was observed between the two groups ( $p = 0.201$ ).

**Table no.07: Distribution of study subjects according to diastolic blood pressure.**

DBP	Study Group	Range	Control Group	Range	p value
Baseline	$82.98 \pm 8.16$	65-96	$84.96 \pm 6.543$	70-100	0.1838
After nebulisation	$79.22 \pm 7.802$	60-90	$81.20 \pm 9.502$	60-96	0.2576
Before Laryngoscopy	$76.06 \pm 3.766$	70-82	$73.78 \pm 6.926$	63-85	0.0435
After intubation	$76.2 \pm 6.051$	65-85	$67.84 \pm 7.17$	56-80	0.0001
After 1 min of Intubation	$80.44 \pm 8.947$	60-98	$91.52 \pm 6.109$	78-108	0.0001
After 5 min of Intubation	$75.92 \pm 8.6$	58-97	$88.8 \pm 5.813$	76-104	0.0001
After 10 min of Intubation	$72.76 \pm 8.916$	54-92	$86.84 \pm 5.884$	72-102	0.0001



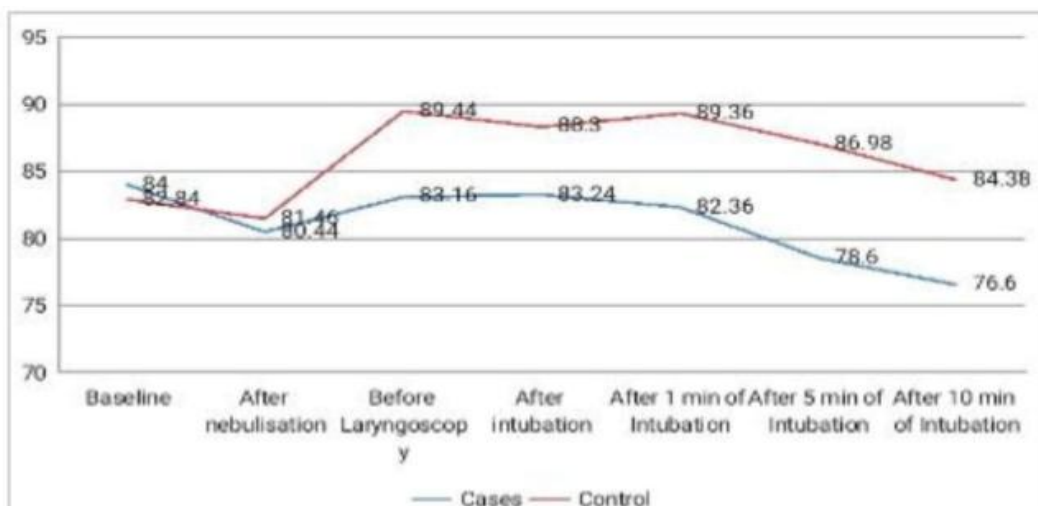
**Graph 3: Distribution of study subjects according to diastolic blood pressure**

### Distribution of Study subjects according to Heart rate

We observed heart rate. Baseline HR was more in study group  $84 \pm 5.657$  than control group  $82.84 \pm 5.347$  with p value 0.2558. After nebulization HR was less in study group  $80.44 \pm 5.647$  than control group  $81.46 \pm 5.16$  with p value 0.348. Jambhure N et al 2020[11] in their study found that mean heart rate of Lignocaine group was significantly ( $p < 0.01$ ) higher than dexmedetomidine group throughout the procedure. Compared to baseline value mean heart rate rise in lignocaine patients were more than that dexmedetomidine group. (graph 2)

Table no.09: Distribution of study subjects according to heart rate.

Heart Rate	Study Group	Range	Control	Range	p value
Baseline	$84 \pm 5.657$	74-102	$82.84 \pm 5.347$	74-92	0.2558
After nebulization	$80.44 \pm 5.647$	60-94	$81.46 \pm 5.16$	72-90	0.348
Before Laryngoscopy	$83.16 \pm 7.901$	72-95	$89.44 \pm 10.05$	73-103	0.0008
After intubation	$83.24 \pm 7.021$	71-95	$88.3 \pm 8.299$	76-102	0.0014
After 1 min of Intubation	$82.36 \pm 5.721$	72-104	$89.36 \pm 5.487$	82-98	0.0001
After 5 min of Intubation	$78.6 \pm 5.778$	70-98	$86.98 \pm 5.27$	80-96	0.0001
After 10 min of Intubation	$76.6 \pm 6.108$	68-96	$84.38 \pm 4.865$	78-94	0.0001



Graph 2: Distribution of study subjects according to heart rate

### CONCLUSION

Dexmedetomidine safe and effective agent in controlling the increase in the heart rate and blood pressure in response to laryngoscopy and tracheal intubation. Dexmedetomidine decreases the heart rate and blood pressure to a lower level and maintains it at these lower values even after laryngoscopy and intubation. Nebulised dexmedetomidine effectively blunts the stress response to laryngoscopy and intubation and with no adverse effects. It reduces the induction dose of propofol. A lower dose besides being cost-effective is also free of side effects such as hypotension and bradycardia.

Nebulized dexmedetomidine may represent a favorable alternative to the intravenous route in adult patients undergoing short-duration surgeries. Dexmedetomidine also provides adequate and longer postoperative analgesia.

#### REFERENCES

1. Low, J. M., Harvey, J. T., Prys-Roberts, C., & Dagnino, J. (1986). Studies of anaesthesia in relation to hypertension: VII: Adrenergic responses to laryngoscopy. *British Journal of Anaesthesia*, 58(5), 471-477.
2. Eilers, H., & Niemann, C. U. (2003). Clinically important drug interactions with intravenous anaesthetics in older patients. *Drugs & aging*, 20, 969-980.
3. Fox, E. J. (1977). Complications related to the pressor response to endotracheal intubation. *Anesthesiology*, 47, 524-525.
4. Khan, F. A., & Ullah, H. (2013). Pharmacological agents for preventing morbidity associated with the haemodynamic response to tracheal intubation. *Cochrane Database of Systematic Reviews*, (7).
5. Sale, H. K., & Shendage, V. J. (2015). Lignocaine and dexmedetomidine in attenuation of pressor response to laryngoscopy and intubation: a prospective study. *International Journal of Scientific Study*, 3(9), 155-160.
6. Paranjpe JS(2013). Dexmedetomidine: Expanding role in anesthesia. *Med J DY Patil Univ*; 6:5- 13.
7. Qiu, J., & Luo, Z. (2019). The comparison of dexmedetomidine and ketamine for pediatric dental surgery: A meta-analysis of randomized controlled studies. *Medicine*, 98(17).
8. Malhotra, P. U., Thakur, S., Singhal, P., Chauhan, D., Jayam, C., Sood, R., & Malhotra, Y. (2016). Comparative evaluation of dexmedetomidine and midazolam-ketamine combination as sedative agents in pediatric dentistry: A double-blinded randomized controlled trial. *Contemporary Clinical Dentistry*, 7(2), 186.
9. Kumar, N. R., Jonnavithula, N., Padhy, S., Sanapala, V., & Naik, V. V. (2020). Evaluation of nebulised dexmedetomidine in blunting haemodynamic response to intubation: A prospective randomised study. *Indian Journal of Anaesthesia*, 64(10), 874.
10. Shrivastava, P., Kumar, M., Verma, S., Sharma, R., Kumar, R., Ranjan, R., & Prakash, J. (2022). Evaluation of Nebulised Dexmedetomidine Given Pre-operatively to Attenuate Hemodynamic Response to Laryngoscopy and Endotracheal Intubation: A Randomised Control Trial. *Cureus*, 14(5).
11. Jambure NP(2021). Evaluation of intranasal dexmedetomidine as a premedicant in attenuating hemodynamic stress response to laryngoscopy and intubation. *MedPulseInternational Journal of Anesthesiology*. 18(3):113-116.