Original Research Article

# A comparative study of intravenous dexmedetomidine and intravenous clonidine for attenuating pressor responses to laryngoscopy and endotracheal intubation

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## Abstract

**Introduciton:** Laryngoscopy and intubation are noxious stimuli that activate sympathetic nervous system causing tachycardia and hypertension. Both Clonidine and Dexmedetomidine are α2 agonist attenuate sympathoadrenal response by inhibiting norepinephrine release but dexmedetomidine has 8 times greater affinity than Clonidine. **Aims and objective**: The aim is to compare the effects of IV Clonidine 1µg/kg and IV Dexmedetomidine 1µg/kg in attenuating hemodynamic responses during laryngoscopy and intubation. **Material and method**: 90 patients of age 18-60 yr of either sex of ASA 1 & 2 physical status are randomly allocated into 3 groups (30 each). Group D and group C received IV Dexmedetomidine 1 µg/kg and IV Clonidine 1 µg/kg respectively diluted in 100 ml of normal saline and group P infused 100 ml of normal saline, over 10 minutes, 15 minutes before induction. HR, SBP, DBP and MAP are recorded at baseline, after drug administration & after induction and laryngoscopy then at 1, 3,5,10,15 minutes after intubation. **Results**: HR, SBP, DBP and MAP are lower in group D and C as compared to group P at all time intervals. However these hemodynamic parameters are significantly more stable in group D compared to group C at all time intervals. **Conclusion**: Both Dexmedetomidine and Clonidine are effective in attenuating hemodynamic responses to laryngoscopy and intubation but Dexmedetomidine is better than Clonidine.

**Keywords:** Dexmedetomidine, Clonidine, laryngoscopy, intubation.



## Introduction

With the advent of endotracheal anaesthesia in the last quarter of 19th century, endotracheal intubation has become one of the commonly performed procedures in the practice of anaesthesia. Endotracheal intubation is the translaryngeal placement of a tube into trachea via the nose or mouth, so it involves manipulation of airway and the sensory part of the airway is of great concern during the manipulation.[1]Both laryngoscopy and intubation are noxious stimuli and cause sympathetic stimulation which is manifested as hypertension and tachycardia.[1]The hemodynamic response during laryngoscopy and intubation is mediated by Vagus(X) and Glossopharyngeal nerve(IX) which carry the afferent stimulus from epiglottis and infraglottic region and activate vasomotor centre and hence result in release of adrenaline and noradrenaline which is responsible for variation in hemodynamic changes.[2]

The increase in hemodynamic responses are transitory, variable and unpredictable which may not be of much clinical significance in normal healthy individuals but in susceptible individuals, such as patients with untreated severe hypertension, coronary artery disease or intracranial aneurysm, this transient sympathetic response can lead to life threatening conditions.[3,4] Hence to overcome these undesirable responses multiple pharmacological and non pharmacological methods have been tried.[5]The non-pharmacological methods include smooth & gentle intubation with brief period of laryngoscopy (<15 seconds), use of supraglottic device such as laryngeal mask airway in place of endotracheal intubation and blocking glossopharyngeal and superior laryngeal nerves.[6-8]Pharmacological methods are evaluated either as premedication or during induction to attenuate these hemodynamic responses to laryngoscopy and intubation such as inhalational anesthetics , topical and intravenous lidocaine, opioids, topical anesthetics beta blockers, calcium channel blockers, vasodilators (Sodium nitroprussides, Nitroglycerines), Magnesium sulphateetc as these agents decrease stress induced catecholamine release during laryngoscopy and intubation.[9-13]But none of above methods and agents are ideal and completely effective, so there has been a focus on the use of α2-adrenergic agonists. These drugs by virtue of their sympatholytic action, attenuate the hemodynamic response following laryngoscopy and endotracheal intubation. These drugs also have sedative, anxiolytic and analgesic effects.[14]Dexmedetomidine and clonidine are two pharmacologically related to alpha 2 agonists, used to induce preoperative sedation, reduce anaesthetic and analgesic drugs requirement and also provide hemodynamic stability along

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with post operative analgesia. These pharmacological effects made them useful as premedication drugs during general anaesthesia.[15]
Clonidine is centrally acting selective partial α2 agonist(220:1 α2 to α1) having elimination half life of 6-10 h. It decreases norepinephrine release and inhibits sympathetic activity. It also provides sympathoadrenal stability and suppresses renin angiotensin activity.[16]
It induces sedation, decreases anesthetics drug requirement and improves hemodynamics by blunting blood pressure and heart rate responses during laryngoscopy, intubation and surgical stimuli and thus protects against perioperative myocardial ischemia.
Dexmedetomidine is highly selective and potent α2 agonist(1620:1 α2 to α1) and eight times more selective for α2 receptors in comparison to clonidine. It has elimination half life of 2-3 hr and shorter duration of action, Like clonidine it also attenuates the hemodynamic response to endotracheal intubation, decreases catecholamine release during anesthesia, also decreases requirements of anaesthetics drugs.[16]
Thus considering the adverse effects associated with laryngoscopy and endotracheal intubation and at same time beneficial effects of both clonidine and dexmedetomidine in blunting hemodynamic response to laryngoscopy and endotracheal intubation, this double blind randomized study, has been attempted to compare the efficacy of dexmedetomidine (1 µg/kg IV) with clonidine (1µg/kg IV) for attenuating pressure responses to laryngoscopy and endotracheal intubation.

**Material and method**

After obtaining approval from the institutional ethical committee, the present prospective randomized controlled study was conducted from February 2019 to October 2020 in 90 patients of ASA grade I and II in the Department of Anesthesiology, J.A Group of Hospitals of G.R. Medical College, Gwalior (M.P) after obtaining well written informed consent .

**Inclusion criteria**

1. Adult patients aged between 18 and 60 yrs of either sex
2. ASA-I and II physical status
3. Patients Scheduled for elective surgery under GA.

**Exclusion criteria**

1. Unwilling patients
2. Patients with significant coronary artery disease or ischemic heart disease, chronic obstructive pulmonary disease, asthma, diabetes mellitus, renal failure, hepatic dysfunction, morbid obesity, hypertension.
3. Patients on psychotropic drugs or history of drug allergy.
4. Patients with ASA grade 3 or higher
5. Emergency surgeries.
6. Anticipated difficult intubation.
7. Pregnancy and lactating women.
8. Patients on Beta blockers and calcium channel blockers.

Detail preoperative history, clinical examination and relevant investigation for safe anesthesia were done. Upon arrival of the patient in operation theatre, hemodynamic parameters like-baseline pulse rate, systolic blood pressure, diastolic blood pressure , mean blood pressure and oxygen saturation were recorded by connecting monitor (Drager infinity kappa). Intravenous access was achieved with either 18G or 20G cannula in non dominant hand.
Patients were randomly allocated into 3 groups by using seal envelope method. Statistically valid sample size for each group was calculated, based on the previous study [17]in which pooled standard deviation (s) of systolic blood pressure at P0 between two groups was 6.90, assumption of clinical acceptable margin (δ) is 5 mm of Hg, alpha (α) = 0.05 and Power (β) = 0.80.

$$N = 2 \times \left( \frac{z_{1-\frac{\alpha}{2}} + z_{1-\beta}}{\delta_0} \right)^2 \times s^2$$

Sample size was calculated by using above formula was 30 in each group.
Group 'P' (n=30): Received normal saline 100 ml in 10 minutes.
Group 'D' (n=30):Received IV Dexmedetomidine(1 µg/kg) {**Dextomid**}diluted in 100 ml normal saline in 10 minutes.
Group 'C' (n=30) : Received IV Clonidine(1 µg/kg) {**Cloneon**}diluted in 100 ml normal saline in 10 minutes.
In all the groups above drugs were infused 15 minutes prior to induction. Then all the patients were premedicated with Inj.Glycopyrrolate 0.2 mg iv slowly and inj. Pentazocine 0.5 mg/kg iv. Patients were preoxygenated with 100 % oxygen for 3 min. General anesthesia induced with Inj. Thiopentone 3-5 mg/kg iv and Inj succinylcholine 2mg/ kg. Endotracheal intubation done with appropriate size ETT. Anaesthesia maintained on Nitrous oxide + Oxygen (66:33) along with intermittent inhalation anesthetic agents (Isoflurane) (0.5 MAC) and Non depolarizing muscle relaxants inj. Atracurium 0.25mg/kg (bolus) followed by 0.1mg/kg as maintenance dose. At the end of the procedure, patients reversed with Inj. Neostigmine (0.04- 0.08 mg/kg) + Glycopyrrolate (0.005-0.01 mg/kg), extubated and oxygenated with 100% oxygen for 10 min.
Heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) and Saturation were recorded at different time intervals as follows:
T0 - Before administration of the study drug (Baseline)
T1 - After completion of the administration of the study drug
T2 - After induction
T3 - After laryngoscopy and intubation
A1 - 1 minutes after intubation
A3 - 3 minutes after intubation
A5 - 5 minutes after intubation
A10 - 10 minutes after intubation
A15 - 15 minutes after intubation.

Then all hemodynamic parameters were monitored at interval of 5 minutes throughout the procedure for the safety of patients.
The observations were recorded, tabulated and subjected to statistical analysis using paired 't' test , un- paired 't' test and Chi square test by SPSS-20 software. P-value≤0.05 and p-value<0.01 were considered as statistically significant and highly significant respectively.

**Observation and result:**

There is no significant differences between three groups with regards to demographic data. (As shown in Table no. 1)

**Table 1:**Demographic profile (Mean±SD)

| | Group P (N=30) (Mean±SD) | Group D (N=30) (Mean±SD) | Group C (N=30) (Mean±SD) | P Value |
|---|---|---|---|---|
| Age (Years) | 37.46±12.43 | 35±12.64 | 36.43±10.37 | 0.950 |
| Sex (M/F) | 14:16 | 16:14 | 15:15 | 0.875 |
| Weight (kgs) | 59.26±6.49 | 57.83±6.52 | 60.13±8.09 | 0.448 |

In this study it was observed that as compared to baseline, increase in HR, SBP, DBP and MAP in Placebo group at the time of laryngoscopy and intubation were statistically highly significant(p<0.00). Hemodynamic stability was statistically significant in Dexmedetomidine 1µg/kg IV and Clonidine 1µg/kg IV group as compared to placebo group as shown in table no.2 and 3.

**Table 2:** Intergroup statistical analysis of mean pulse rate(bpm) and MAP(mmhg) in Placebo and Dexmedetomidine group

| Time | PR | | | MAP | | |
|---|---|---|---|---|---|---|
| | Group P Mean±SD | Group D Mean±SD | P value | Group P Mean±SD | Group D mean±SD | P value |
| T0 | 85.83±9.46 | 88.13±11.31 | 0.397 | 93.53±5.25 | 91.47±9.22 | 0.291 |
| T1 | 89.07±8.57 | 71±10.97 | 0.000** | 96.90±5.85 | 82.67±9.66 | 0.000** |
| T2 | 91.60±11.23 | 70.9±10.87 | 0.000** | 93.37±5.06 | 81.43±8.94 | 0.000** |
| T3 | 119.77±10.79 | 80.13±11.28 | 0.000** | 111.23±3.10 | 83.33±9.19 | 0.000** |
| A1 | 125.23±8.38 | 77.8±10.62 | 0.000** | 118.23±3.87 | 80.57±7.61 | 0.000** |
| A3 | 115.77±8.08 | 75.73±9.77 | 0.000** | 111.43±6.55 | 79.43±7.36 | 0.000** |
| A5 A5 | 110.20±7.73 | 75.2±10.39 | 0.000** | 103.37±6.23 | 78.37±7.58 | 0.000** |
| A10 | 96.03±8.21 | 75.9±10.41 | 0.000** | 95.20±5.65 | 79.87±7.35 | 0.000** |
| A15 | 91.83±8.03 | 76±9.85 | 0.000** | 93.13±4.80 | 79.50±7.20 | 0.000** |

**P<0.01- Highly significant ,*P<0.05- Significant

**Table 3:** Intergroup statistical analysis of mean pulse rate(bpm) and MAP(mmhg) in Placebo and Clonidine group

| Time | PR | | | MAP | | |
|---|---|---|---|---|---|---|
| | Group P Mean±SD | Group C Mean±SD | P value | Group P Mean±SD | Group C mean±SD | P value |
| T0 | 85.83±9.46 | 82.56±10.53 | 0.211 | 93.53±5.25 | 92.87±4.42 | 0.597 |
| T1 | 89.07±8.57 | 83.26±10.43 | 0.022* | 96.90±5.85 | 94.13±3.91 | 0.036* |
| T2 | 91.60±11.23 | 82.03±10.60 | 0.001** | 93.37±5.06 | 92.17±3.93 | 0.310 |
| T3 | 119.77±10.79 | 87.6±10.45 | 0.000** | 111.23±3.10 | 97.93±3.94 | 0.000** |
| A1 | 125.23±8.38 | 91.9±10.84 | 0.000** | 118.23±3.87 | 101.90±5.28 | 0.000** |
| A3 | 115.77±8.08 | 88.06±9.68 | 0.000** | 111.43±6.55 | 97.87±4.03 | 0.000** |
| A5 | 110.20±7.73 | 85.66±8.98 | 0.000** | 103.37±6.23 | 96.33±3.88 | 0.000** |
| A10 | 96.03±8.21 | 83.46±7.88 | 0.000** | 95.20±5.65 | 94.77±3.35 | 0.719 |
| A15 | 91.83±8.03 | 81.8±7.80 | 0.000** | 93.13±4.80 | 93.03±3.40 | 0.926 |

**P<0.01- Highly significant,*P<0.05- Significant

Clonidine in a dose of 1µg/kg although reduces heart rate, SBP, DBP and MAP as compared to control group but compared to baseline statistically significant rise in HR, SBP,DBP and MAP is seen at the time of laryngoscopy and intubation which continued upto10 minutes after intubation. Baseline hemodynamic parameters are achieved at 15 min after intubation.( as shown in table no-3)

It was observed that Dexmedetomidine is statistically highly significant in attenuating hemodynamic responses to laryngoscopy and intubation as compared to Clonidine as shown in table no.4.

**Table 4:** Intergroup statistical analysis of mean pulse rate(bpm) and MAP(mmhg) in Clonidine and Dexmedetomidine group

| Time | PR | | | MAP | | |
|---|---|---|---|---|---|---|
| | Group D Mean±SD | Group C Mean±SD | P value | Group D Mean±SD | Group C Mean±SD | P value |
| T0 | 88.13±11.31 | 82.56±10.53 | 0.053 | 91.47±9.22 | 92.87±4.42 | 0.457 |
| T1 | 71±10.97 | 83.26±10.43 | 0.000** | 82.67±9.66 | 94.13±3.91 | 0.000** |
| T2 | 70.9±10.87 | 82.03±10.60 | 0.000** | 81.43±8.94 | 92.17±3.93 | 0.000** |
| T3 | 80.13±11.28 | 87.6±10.45 | 0.010** | 83.33±9.19 | 97.93±3.94 | 0.000** |
| A1 | 77.8±10.62 | 91.9±10.84 | 0.000** | 80.57±7.61 | 101.90±5.28 | 0.000** |
| A3 | 75.73±9.77 | 88.06±9.68 | 0.000** | 79.43±7.36 | 97.87±4.03 | 0.000** |
| A5 A5 | 75.2±10.39 | 85.66±8.98 | 0.000** | 78.37±7.58 | 96.33±3.88 | 0.000** |
| A10 | 75.9±10.41 | 83.46±7.88 | 0.000** | 79.87±7.35 | 94.77±3.35 | 0.000** |
| A15 | 76±9.85 | 81.8±7.80 | 0.014* | 79.50±7.20 | 93.03±3.40 | 0.000** |

*P<0.01- Highly significant,*P<0.05- Significant

**Discussion**

Direct laryngoscopy and orotracheal intubation are noxious stimuli that activate sympathetic nervous system causing transient tachyarrhymias and hypertension. Various conventional methods of treatment like lignocaine spray, inhalational, intravenous agents or opioids, calcium channel blockers, vasodilators like sodium nitroprusside and nitroglycerine etc. have been used to attenuate these hemodynamic responses to laryngoscopy and intubation but none of above methods have been proven ideal. Dexmedetomidine and Clonidine are two pharmacologically related to alpha 2 agonists, which are used to induce preoperative sedation, reduce the requirements of anaesthetic drugs and analgesic and also provide hemodynamic stability along with post operative analgesia. These pharmacological effects made them useful as premedication drugs during general anaesthesia. The cardiovascular response (hypertension and tachycardia) during laryngoscopy and intubation is mediated by Vagus (X) and Glossopharyngeal nerve (IX) which carry the afferent stimulus from epiglottis and infraglottic region and activate vasomotor centre and result in release of adrenaline and noradrenaline which causes the effect. They act by inhibiting

sympathetic activity by decrease in norepinephrine release, thus attenuate hemodynamic responses following laryngoscopy and intubation. They activate post synaptic α2 receptor which results in decrease in sympathetic nervous system outflow from CNS to peripheral tissue and this lead to bradycardia and hypotension. In addition these drugs have sedative, anxiolytic and analgesic effect without respiratory depression. Dexmedetomidine has 8 times greater affinity for α2 receptors as compared to Clonidine. So Dexmedetomidine is more effective in attenuating hemodynamic responses as compared to Clonidine which was also observed in our study.

Studies conducted by Keshri R K et al [18] and Shilpa AR et al[19] compared the effects of two different doses of iv Dexmedetomidine(1 µg/kg and 0.5µg/kg) on hemodynamic responses(HR,SBP,DBP and MAP) to laryngoscopy and endotracheal intubation and found that Dexmedetomidine in dose of 1 µg/kg was more effective in attenuating presser responses to laryngoscopy and intubation as compared to Dexmedetomidine in dose of 0.5 µg/kg. Thus dose of Dexmedetomidine as 1 µg/kg was selected in this study in accordance with above studies. Various studies by Hazra R et al[17] ,Gupta N et al[20], Ahmed ALM, Bora J[21], AgrawalS et al[22], and Hussain SY et al [23] also used IV Clonidine in a dose of 1 µg/kg to investigate its effects on hemodynamic responses during laryngoscopy and intubation. Based on the results of above studies the optimum dose of1 µg/kg Clonidine for attenuating hemodynamic response to laryngoscopy and intubation with minimum adverse effects, was chosen in our study.

Studies by Yildiz et al[24], Ferdi et al[25], Keniya et al[26] and Laha A et al[27] also investigated the effects of IV Dexmedetomidine(1µg/kg) and Placebo(Normal saline) on hemodynamic responses during laryngoscopy and intubation and concluded that IV Dexmedetomidine in dose of 1 µg/kg was effective in blunting sympathoadrenal responses(HR) to laryngoscopy and intubation.

This study was undertaken to evaluate the efficacy of IV Dexmedetomidine (1µg/kg) vs IV Clonidine in attenuating hemodynamic responses to laryngoscopy and intubation. It was found that HR, SBP, DBP and MAP were lower in group D and group C group as compared to group P at all time intervals. However these hemodynamic parameters are significantly more stable in group D as compared to group C at all time intervals. This signifies that both Clonidine and Dexmedetomidine act as α2 agonist that decrease norepinephrine release, thus blunt the sympathoadrenal responses to laryngoscopy and intubation. But Dexmedetomidine was more effective than Clonidine in attenuating stress responses.

Similar to our study Ahmed ALM, Bora J[21], Hazra R et al[17] and Arora S et al[28] also found that Dexmedetomidine was more effective than Clonidine in attenuating hemodynamic responses to Laryngoscopy and intubation.Bharti D et al[29] found that Clonidine (3µg/kg) was more effective than Dexmedetomidine(0.2 µg/kg) in blunting hemodynamic responses to laryngoscopy. Better hemodynamic response to Clonidine as compared to Dexmedetomidine in their study might be due to higher dose of Clonidine (3 µg/kg) used, while dose used for Dexmedetomidine was 0.2 µg/kg which was lower than used in our study((1 µg/kg).In this study there were no significant side effects observed among study drugs.

**Limitations of our study**

- Mallampatti grade III and IV patients were not included in our study.
- Patients with comorbid conditions like hypertension and diabetes, etc. were not included in the study. Hence, the advantages of using Clonidine or dexmedetomidine in patients having co morbid diseases could not be evaluated.
- Factors other than laryngoscopy may have contributed to hemodynamic findings.
- The depth of anaesthesia or adequacy of muscle relaxation which might affect hemodynamic changes were not monitored in our study.

**Conclusion**

Based on the present clinical comparative studyit was concluded that IntravenousDexmedetomidine(1µg/kg) is superior than Clonidine(1µg/kg) in attenuating hemodynamic responses during laryngoscopy and intubation. No significant side effects were observed among study drugs.

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