DOI: 10.7860/JCDR/2025/81360.21463

Original Article

Anaesthesia Section

Comparison of Clonidine versus Gabapentin as an Oral Premedication on Haemodynamic Response to Laryngoscopic and Tracheal Intubation in ENT Surgeries: A Double Blind, Randomised Clinical Study

PRIYA KISHNANI[1], DEVENDRA JOSHI[2], TEJASH H SHARMA[3], DINESH K CHAUHAN[4]

ABSTRACT

Introduction: Laryngoscopy and tracheal intubation are recognised as potent stimuli which cause significant haemodynamic responses. Various pharmacological strategies have been employed to attenuate the haemodynamic responses associated with this, including opioids, beta-blockers, calcium channel blockers, and α-2 adrenergic agonists.

Aim: The present study aimed to compare the efficacy of oral clonidine versus oral gabapentin in attenuating haemodynamic responses during laryngoscopy and endotracheal intubation.

Materials and Methods: The present study was a double blinded, randomised clinical study and it was conducted at Dhiraj Hospital from April 2024 to April 2025. The study was approved by the Institutional Ethics Committee (SVIEC/ON/Medi/SRP/April/24/76) and registered with Clinical Trials Registry - India (CTRI/2024/10/075366). Sixty patients aged 18-60 years of American Society of Anaesthesiologist physical status I-II, posted for elective surgeries under general anaesthesia were randomised into two groups (n=30 each): Group Dg received oral gabapentin 800 mg and Group Dc received oral clonidine 0.2 mg, 30 minutes prior to induction. Heart Rate (HR), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), and Mean Arterial Pressure (MAP) were noted at baseline, after medication, after induction, during intubation, and at 1, 3, 5, and 7 minutes post-intubation. Data were analysed using unpaired Student's t-test for numerical variables and Chi-square test for categorical variables. Statistical significance was set at $p<0.05$.

Results: Demographic profiles were comparable between groups. The mean age of patients was 39.97±9.92; 44.23±11.72 years (p=0.13), gender distribution showed 46.67%; 36.67% males (p=0.43), ASA I physical status 66.67%; 70% (p=0.78) in Group Dg and Group Dc, respectively. During intubation, HR was 91.83±6.84 bpm; 97.10±11.38 bpm, p=0.0338 in Group Dc and Group Dg, respectively. During intubation, 1, 3, 5 and 7 minutes post-intubation, SBP (143.20±7.30: 127.73±5.06, 137.40±7.22: 118.03±6.36, 131.33±5.93: 108.70±8.16, 127.80±6.65: 105.87±9.73, 123.73±6.33: 104.27±8.74, p<0.0001), DBP (105.23±6.04: 87.33±8.87, 102.23±5.30: 87.50±6.31, 98.77±5.26: 81.97±6.71, 94.83±5.76: 76.63±7.42, 91.33±6.21: 70.27±8.13) in Group Dc and Group Dg, respectively.

Conclusion: Oral clonidine is better than oral gabapentin at reducing the HR and especially the blood pressure spikes that occur during laryngoscopy and endotracheal intubation for general anesthesia. This makes clonidine a potentially better choice for premedication, particularly for patients at higher risk of heart problems.

Keywords: α2 agonist, Blood pressures, Ear nose throat surgery, Gamma-aminobutyric acid analogs, Intubation, Stress response

INTRODUCTION

The advent of general anaesthesia marked a significant advancement in surgical medicine, enabling the induction of controlled unconsciousness and thereby ensuring patients remained insensible to pain and unaware of intraoperative events. However, laryngoscopy and endotracheal intubation are recognised as potent stimuli capable of provoking substantial haemodynamic changes, including tachycardia, hypertension, and dysrhythmias, which can be detrimental in patients with cardiovascular or cerebrovascular diseases [1]. These haemodynamic responses to laryngoscopy and intubation are mediated by sympathoadrenal discharge resulting from epipharyngeal and parapharyngeal stimulation, leading to increased plasma catecholamine levels [2]. The haemodynamic response typically begins within five seconds of laryngoscopy, reaches its peak within one to two minutes, and generally returns to baseline within five to seven minutes. While these transient changes are usually well-tolerated in healthy individuals, they may precipitate serious adverse events in patients with underlying comorbidities [3]. Various pharmacological strategies have been employed to attenuate the haemodynamic responses associated with this, including opioids, beta-blockers, calcium channel blockers, and α-2 adrenergic agonists [4]. Among these, clonidine, an α-2 adrenergic agonist, has been widely used due to its sedative, analgesic, and sympatholytic properties [5]. Gabapentin, a structural analog of Gamma-Aminobutyric Acid (GABA), originally used as an anticonvulsant, has also shown promise in preventing sympathetic response to laryngoscopy and intubation [6]. Clonidine acts by stimulating central α-2 adrenergic receptors, reducing sympathetic outflow and attenuating the stress response [7]. Gabapentin's mechanism in attenuating haemodynamic responses is less well understood but may involve inhibition of calcium influx through voltage-gated calcium channels, leading to reduced excitatory neurotransmitter release [8]. Several studies have compared various premedication drugs for decreasing the haemodynamic stress response associated to laryngoscopy and tracheal intubation [3,9-11]. The present study was evaluated and compared the effectiveness of oral clonidine (0.2 mg) and oral gabapentin (800 mg) in blunting the haemodynamic potent stress responses associated with laryngoscopy and

endotracheal intubation in patients scheduled for elective surgeries under general anaesthesia.

The primary objective was to compare the efficacy of oral clonidine and gabapentin in attenuating HR and secondary objectives were to compare blood pressure changes (SBP, DBP and MAP), during laryngoscopy and endotracheal intubation in patients undergoing ENT surgeries under general anaesthesia.

MATERIALS AND METHODS

The present study was a double-blinded, randomised clinical study conducted in the Department of Anaesthesiology at Smt BKS Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed to be University, Vadodara, Gujarat, India, following approval from the Institutional Ethics Committee (SVIEC/ON/Medi/SRP/April/24/76) with Clinical trial registry - India (CTRI) number CTRI/2024/10/075366. The study was conducted from April 2024 to April 2025. Informed and written consents were obtained from the patients.

Inclusion and Exclusion criteria: Total sixty adult patients aged 18 to 60 years, classified as American Society of Anaesthesiologists (ASA) physical status I or II, and scheduled for elective Ear Nose Throat (ENT) surgeries under general anaesthesia were enrolled in the study. Those patients had history of cardiovascular diseases, cerebrovascular diseases, hepatic or renal impairment, psychiatric disorders, or a history of allergy to study drugs, pregnant and lactating women, patients with anticipated difficult airway and those with a body mass index ≥30 kg/m² were excluded. All patients underwent a detailed pre-anaesthetic checkup a day before surgery.

Sample size calculation: Based on previous studies [12,13], a 15% difference in MAP between the two groups was considered clinically significant. With a significance level (α) of 0.05 and a power of 80% (β=0.20), the following formula was used to calculate the minimum required sample size for comparing two means:

$n = \{2 \times (Z\alpha/2 + Z\beta)^2 \times \sigma^2\}/\Delta^2$

Where:

$Z1\text{-}\alpha/2 = 1.96$ for 95% confidence;

$Z1\text{-}\beta = 0.84$ for 80% power;

σ = pooled standard deviation of MAP;

Δ = minimum clinically significant difference in MAP.

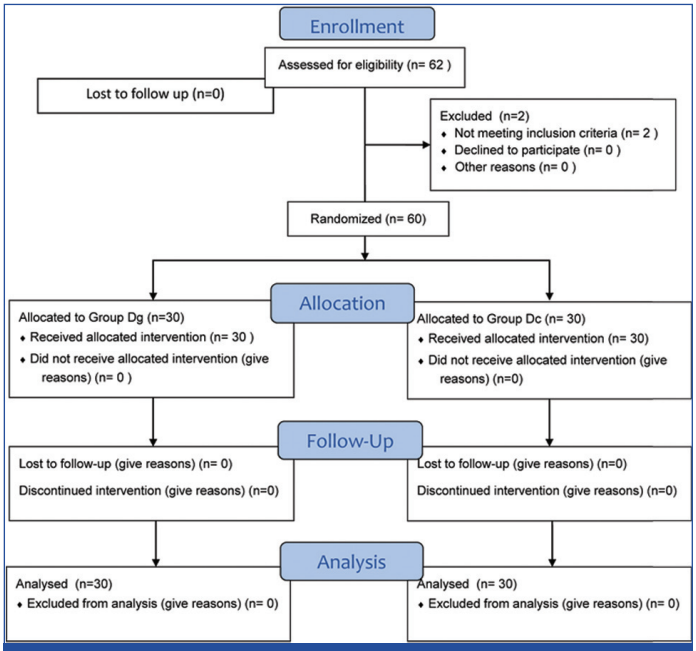
From pilot data and previous literature [12,13], the pooled standard deviation of MAP was estimated at approximately 6.27 mmHg. A 15% difference in MAP, with a baseline average of 98 mmHg, corresponds to d=14.7 mmHg. Substituting into the formula:

$n=\{2\times(1.96+0.84)^2\times18^2\}/13^2=30.05$

Based on the above calculation, the required sample size was approximately 30 per group, resulting in a total of 60 participants. This sample size allows for adequate statistical power to detect a clinically meaningful difference in haemodynamic response between the two interventions.

Study Procedure

Preoperative investigations were performed for all patients, which includes complete blood count, renal and liver function tests, Electrocardiogram (ECG), random blood sugar and chest X-ray. Accordance to standard preoperative fasting guideline, all patients were kept Nil Per Oral (NPO) or Nil By Mouth (NBM) for at least eight hours for solids and two hours for clear fluids prior to surgery [14]. On the day of surgery, patients were transferred to the pre-operative area one hour prior to the operative procedure. Baseline vital parameters were recorded in pre-operative area, which include HR, blood pressure, Respiratory Rate (RR), and Oxygen Saturation (SpO_2). An 18-gauge intravenous (i.v.) cannula was secured, and Ringer's lactate solution was started at according to case vignette [15]. Using a computer-generated block randomisation in 1:1 ratio with Microsoft excel version 16.0, patients were allocated by consultant anaesthesiologist not related to study into one of two groups [Table/Fig-1]: Group Dg (n=30) received oral gabapentin 800 mg and Group Dc (n=30) received oral clonidine 0.2 mg 30 minutes before induction [12,13]. The patients and investigators were blinded. The study drugs were concealed in opaque envelope, which was opened by the nurse in pre-operative area and was given 30 minutes prior to anaesthesia. Patients were shifted to operating room and standard monitoring- including Electrocardiogram (ECG), Non-Invasive Blood Pressure (NIBP), ETCO2 and pulse oximetry- monitors were attached. Premedication, glycopyrrolate 0.2 mg, pantoprazole 40 mg, and ondansetron 4 mg were given intravenously. Pre-oxygenation with 100% oxygen for three minutes, after that anaesthesia was induced with propofol 2 mg/kg, intravenously. As adequate mask ventilation was confirmed, succinylcholine 2 mg/kg was administered to perform laryngoscopy and intubation. Using a Macintosh laryngoscope, laryngoscopy was performed by an experienced anaesthesiologist. Appropriate size of endotracheal tube was use to secure airway and prevent injury. Maintenance of anaesthesia was done with isoflurane in mixture of oxygen and nitrous oxide with ratio 1:1. Muscle relaxation was achieved by atracurium, given intravenously with loading dose of 0.5 mg/kg and maintenance doses of 0.1 mg/kg as required intermittently. Reversal was achieved with inj. glycopyrrolate 0.4 mg + neostigmine 2.5 mg given by intravenously slowly after a Train Of Four (TOF) ratio of 0.9 [16]. In present study, HR, SBP, DBP and MAP were noted at baseline (before administration of the drug which had to be studied), 30 minutes after administration of the drug, after induction of anaesthesia, during laryngoscopy and tracheal intubation, and at 1, 3, 5, and 7 minutes post-intubation.



[Table/Fig-1]: Consolidate standards of reporting trials flow chart.

STATISTICAL ANALYSIS

Data were analysed using MedCalc software, version 12.5. Continuous variables were expressed as mean±standard deviation (Mean±SD) and compared between groups using the unpaired t-test. Categorical variables were presented as frequencies and percentages, with group comparisons performed using the Chi-square test. A p-value of <0.05 was considered statistically significant.

RESULTS

Both groups showed no significant difference in terms of demographic parameters. The mean age, gender distribution and ASA physical status distribution were comparable between the groups (p=0.7832) [Table/Fig-2].

Parameters	Group Dg (n=30)	Group Dc (n=30)	p-value
Age (years) (Mean±SD)	39.97±9.92	44.23±11.72	0.13
Gender (Male: Female) {n (%)}	14:16 (46.67%:53.33%)	11:19 (36.67%:63.33%)	0.43
ASA (I/II) {n (%)}	20:10 (66.67%:33.33%)	21:9 (70%:30%)	0.78

[Table/Fig-2]: Demographic characteristics between Group Dg and Group Dc. Using Chi-square's test for categorical variables (Gender and ASA Grade). Student's t-test: Used for a continuous variable (Age). Statistically not significant-(NS), (p>0.05).

Heart Rate (HR) change

There was no any significant difference observed in baseline HRs between the two groups. After medication, during induction, at intubation, and at 1 and 3 minutes post-intubation, HR was significantly lower in Group Dc compared to Group Dg (p<0.05). The difference was most pronounced during intubation. At 5 and 7 minutes post-intubation, the difference was not statistically significant [Table/Fig-3].

Time point	Group Dg (n=30) (Mean±SD)	Group Dc (n=30) (Mean±SD)	p-value
Baseline	82.30±9.23	79.27±7.55	0.1693 (NS)
After medication	81.97±8.79	77.17±7.64	0.0278*
Induction	82.37±8.48	77.83±7.95	0.0366*
Intubation	97.10±11.38	91.83±6.84	0.0338*
1 minute	93.27±10.67	87.13±7.69	0.0132*
3 minute	91.23±9.10	86.10±8.76	0.0300*
5 minute	86.83±8.78	82.97±8.35	0.0863
7 minute	83.37±8.71	79.23±8.20	0.0630

[Table/Fig-3]: Heart Rate (HR) changes between Group Dg and Group Dc at baseline, 30 minutes after medication, induction, intubation, 1, 3, 5 and 7 minutes. Student's -t test, *p <0.0001- statistically significant, NS-not significant.

Systolic Blood Pressure (SBP) changes

Baseline SBP was comparable between the groups. During intubation and at 1, 3, 5, and 7 minutes post-intubation, SBP was significantly lower in Group Dc compared to Group Dg (p<0.0001 for all time points) [Table/Fig-4].

Time point	Group Dg (n=30) (Mean±SD)	Group Dc (n=30) (Mean±SD)	p-value
Baseline	124.17±6.15	121.77±6.65	0.1521 (NS)
After medication	125.83±6.49	123.00±6.36	0.0934 (NS)
Induction	126.07±8.20	124.47±6.45	0.4044 (NS)
Intubation	143.20±7.30	127.73±5.06	<0.0001*
1 minute	137.40±7.22	118.03±6.36	<0.0001*
3 minute	131.33±5.93	108.70±8.16	<0.0001*
5 minute	127.80±6.65	105.87±9.73	<0.0001*
7 minute	123.73±6.33	104.27±8.74	<0.0001*

[Table/Fig-4]: SBP changes between Group Dg and Group Dc at baseline, 30 minutes after medication, induction, intubation, and at 1, 3, 5 and 7 minutes. Student's –t-test, *p <0.0001- statistically significant, NS- Not significant

Mean Arterial Pressure (MAP) changes

Baseline MAP was comparable between the groups. MAP during intubation was low in Group Dc than Group Dg, (p<0.0001). Significant difference also seen at 1, 3, 5 and 7 minutes post-intubation (p<0.0001) [Table/Fig-5].

Time point	Group Dg (n=30) (Mean±SD)	Group Dc (n=30) (Mean±SD)	p-value
Baseline	98.40±8.05	96.43±5.67	0.2777 (NS)
After medication	96.97±5.33	95.10±4.94	0.1641 (NS)
Induction	92.13±4.97	90.90±4.71	0.3293 (NS)
Intubation	118.77±7.46	97.90±4.79	<0.0001*
1 minute	114.30±6.24	99.67±5.70	<0.0001*
3 minute	110.03±5.23	93.57±5.14	<0.0001*
5 minute	106.13±5.78	90.80±6.06	<0.0001*
7 minute	102.33±5.60	85.67±6.30	<0.0001*

[Table/Fig-5]: MAP changes between Group Dg and Group Dc at baseline, 30 minutes after medication, induction, intubation and at 1, 3, 5 and 7 minutes. Student's -t test, *p <0.0001- statistically significant, NS- Not significant

Intergroup MAP comparison shows statistically significant difference between clonidine and gabapentin during intubation and at 1, 3, 5 and 7 minutes post intubation, p<0.05.

Diastolic Blood Pressure (DBP) changes

The DBP during intubation was lower in Group Dc than Group Dg, (p<0.0001). The difference in blood pressure parameters between the groups remained significant during intubation and at 1, 3, 5 and 7 minutes after intubation, with Group Dc consistently showing lower values than Group Dg [Table/Fig-6].

Time point	Group Dg (n=30) (Mean±SD)	Group Dc (n=30) (Mean±SD)	p-value
Baseline	82.17±5.77	80.33±5.92	0.2277 (NS)
After medication	81.87±6.80	79.30±5.61	0.1157 (NS)
Induction	79.57±6.22	81.27±5.84	0.2796 (NS)
Intubation	105.23±6.04	87.33±8.87	<0.0001*
1 minute	102.23±5.30	87.50±6.31	<0.0001*
3 minute	98.77±5.26	81.97±6.71	<0.0001*
5 minute	94.83±5.76	76.63±7.42	<0.0001*
7 minute	91.33±6.21	70.27±8.13	<0.0001*

[Table/Fig-6]: DBP changes between Group Dg and Group Dc at baseline, 30 minutes after medication, induction, intubation, and at 1, 3, 5 and 7 minutes. [Student's t-test, *p <0.0001- statistically significant, NS: Not significant

Percentage Changes in Haemodynamic Parameters from Baseline to Intubation in both groups

Percentage changes from baseline to intubation in haemodynamic para meters, both drugs attenuated the increase in haemodynamic parameters like HR, SBP, DBP, MAP. Statistically significant difference between clonidine and gabapentin is seen (p<0.05) [Table/Fig-7].

Parameters	Group Dg (n=30) (%)	Group Dc (n=30) (%)	p-value
HR	18.0%	15.8%	<0.821
SBP	15.3%	4.9%	<0.001*
DBP	28.1%	8.7%	<0.001*
MAP	20.7%	1.5%	<0.001*

[Table/Fig-7]: Percentage Changes in haemodynamic parameters from baseline to intubation in both groups. Student's -t test, *p <0.0001) statistically significant, NS-not significant.

Respiratory Rate (RR) changes after medication

Both groups showed no significant difference in terms of RR changes or requirements after medication and intubation, p>0.05 [Table/Fig-8].

Time point	Group Dg (n=30) (Mean±SD)	Group Dc (n=30) (Mean±SD)	p-value
Baseline	13.77±1	13.81±0.74	0.49
After medication	13.84±1.28	13.87±0.67	0.56
Induction	13.88±0.79	13.93±0.56	0.25
Intubation	13.94±0.96	13.99±0.49	0.26
1 minute	13.98±0.57	14.04±0.35	0.16
3 minute	13.9±0.74	14.04±0.36	0.11
5 minute	13.91±0.64	14.07±0.42	0.08
7 minute	13.86±0.71	14.11±0.5	0.13

[Table/Fig-8]: RR changes between Group Dg and Group Dc at baseline, 30 minutes after medication, induction, intubation, and at 1, 3, 5 and 7 minutes. Student's t-test, NS: Not significant

SpO_2 (%) changes after medication

Both groups showed no significant difference in terms of SpO_2 (%) changes or requirements after, medication and intubation, p>0.05 [Table/Fig-9].

Time Point	Group Dg (n=30) (Mean±SD)	Group Dc (n=30) (Mean±SD)	p-value
Baseline	99.79±0.83	99.85±0.4	0.73
After medication	99.85±0.58	99.89±0.33	0.75
Induction	99.91±0.55	99.95±0.23	0.72
Intubation	99.96±0.42	99.97±0.17	0.90
1 minute	100±0	100±0	NA
3 minute	100±0	100±0	NA
5 minute	100±0	100±0	NA
7 minute	100±0	100±0	NA

[Table/Fig-9]: SpO_2 (%) changes between Group Dg and Group Dc at baseline, 30 minutes after medication, induction, intubation, and at 1, 3, 5 and 7 minutes. Student's t-test, NSL: not significant

DISCUSSION

Laryngoscopy and endotracheal intubation are associated with significant haemodynamic responses due to sympathoadrenal stimulation [17]. These responses, though transient, can have deleterious effects in patients who had already cardiovascular or cerebrovascular diseases. Various pharmacological agents have been used to decrease these potent stress responses, but the search for an ideal agent continues.

The present study compared the efficacy of oral clonidine (0.2 mg) and oral gabapentin (800 mg) in attenuating the haemodynamic responses to laryngoscopy and endotracheal intubation.

The demographic profiles of both groups were comparable and did not show any significant difference [Table/Fig-2]. In both the groups, baseline HR, SBP, MAP, DBP, RR, SpO_2 (%) were comparable [Table/Fig-3-6,8,9]; it gives proper start point of comparison. RR and SpO_2 (%) were not changed after study drug and were comparable in both groups, p>0.05. Percentage-wise, the difference between both groups regarding the rise in HR from baseline to intubation was statistically insignificant, while the difference in the rise in SBP, DBP, and MAP was significant [Table/Fig-7].

The HR-lowering effect of clonidine could be attributed to its action as α-2 adrenergic agonist, which reduces sympathetic outflow and increases parasympathetic tone [18]. Gabapentin, on the other hand, primarily acts on voltage-gated calcium channels and has less direct effect on sympathetic activity [8].

In the present study, HR increased in both groups during intubation, but the increase was less pronounced in the clonidine group. Clonidine was more effective than gabapentin in controlling HR during laryngoscopy, intubation, at 1 and 3 minutes [Table/Fig-3]. This finding aligns with Singhal SK et al., Flor CAG et al., Nagaraja AS et al., who reported that HR rise was attenuated in clonidine group than group gabapentin, till 10 minutes after laryngoscopy, p<0.05 [12,19,20]. Contrary to this, Saravandi RY et al., findings for HR changes after laryngoscopy were comparable between both groups (Group G- Gabapentin 900 mg and Group C- Clonidine 200 µg), p>0.05 [21]. It may be due to use of 1-2 µg/kg inj. fentanyl as premedication, which itself is a potent attenuator of laryngoscopic stress response and stable haemodynamics [11].

The most striking difference between the two groups was observed in blood pressure parameters. During intubation and throughout the post-intubation period, SBP, DBP, and MAP were significantly lower in the clonidine group compared to the gabapentin group. When considering percentage changes from baseline to intubation, both drugs attenuated the increase in haemodynamic parameters, but clonidine showed a more profound effect. In Group Dc and Group Dg there was a 3.84 % and 17.2% rise in SBP after intubation, respectively, subsequently falling below baseline from one minute onwards but not requiring any intervention, while in Group Dg it shot up after intubation and touched baseline after seven minutes, remained consistent throughout, suggesting clonidine's superiority over gabapentin [Table/Fig-4-6]. This profound effect of clonidine on blood pressure could be explained by its action on α-2 adrenergic receptors in the brainstem, which reduces sympathetic outflow, leading to decreased peripheral resistance and blood pressure [22]. Clonidine also enhances baroreceptor sensitivity, further contributing to its hypotensive effect [10]. Gabapentin, while showing some ability to attenuate the pressor response, was less effective than clonidine. This finding is consistent with Montazeri K et al., who found that clonidine 0.3 mg was more effective than gabapentin 800 mg in blunting the haemodynamic response to laryngoscopy and intubation [13].

The percentage increase in MAP from baseline to intubation was only 1.5% in the clonidine group compared to 20.7% in the gabapentin group, highlighting the superior efficacy of clonidine in controlling blood pressure during this critical period [Table/Fig-5]. DBP during intubation was higher in Group Dg versus Group Dc, (p<0.0001). The difference in DBP between the groups remained significant during intubation and 1, 3, 5, 7 minutes after intubation, with Group Dc consistently showing lower values than Group Dg, which subsequently decrease below baseline after five minutes not requiring any intervention and remained stable, while in Group Dg it was consistently higher throughout [Table/Fig-6]. Similarly, Waikar C et al., reported that while both clonidine and gabapentin decrease the stress or pressor response, clonidine was good and superior in controlling HR [7]. Flor CAG et al., found that there was significant difference at three minutes after intubation, Group-C (clonidine 0.2 mg) showed a mean of 101.02 mmHg±10.14 and Group-G (gabapentin 600 mg) 104.32 mmHg±10.94 rise in SBP, p<0.05 and then after gradual drop in SBP in both groups, where Group-G showed average higher SBP. Also, MAP was significantly lower in Group-C, p<0.007 [19]. On the contrary, Sharma V et al., found that after laryngoscopy, HR, SBP, and DBP were reduced at 1, 5, 10, and 15 minutes in Group-G (gabapentin 900 mg) more than in Group-C (clonidine 0.3 mg), but statistically not significant except at 15 minutes [5]. It may be due to study drugs had been given 120 minutes prior to induction and also inj. fentanyl 3 µg/kg was given as premedication [11]. Bafna U et al., found that gabapentin at a higher dose of 1000 mg was effective in decreasing both HR and blood pressure responses [9]. This discrepancy might be due to the higher dose of gabapentin used in their study compared to the present study 800 mg dose.

Limitation(s)

The present study was limited by inclusion of only ASA I and II patients, so the findings may not be generalisable to patients with significant comorbidities. Also, there was use of a fixed dosage of clonidine and gabapentin rather than weight-based dosing, which might have influenced the results.

CONCLUSION(S)

Oral clonidine 0.2 mg administered 30 minutes prior to laryngoscopy is more effective than oral gabapentin 800 mg for controlling haemodynamic responses during laryngoscopy and endotracheal intubation. Clonidine offers superior blood pressure control, particularly during and immediately after intubation, which may be useful in patients at risk of adverse cardiovascular events.

REFERENCES

[1] Henderson J. Airway management in the adult. In: Miller RD, editor. Miller's Anaesthesia. 7th ed. Philadelphia: Churchill Livingstone; 2010. p. 1573-610.
[2] Prys-Roberts C, Greene LT, Meloche R, Foëx P. Studies of anaesthesia in relation to hypertension. II: Haemodynamic consequences of induction and endotracheal intubation. Br J Anaesth. 1971;43(6):531-47.

[3] Kovac AL. Controlling the hemodynamic response to laryngoscopy and endotracheal intubation. J Clin Anaesth. 1996;8(1):63-79.
[4] Gupta A, Rai AS, Sharma DK. Effect of oral melatonin and oral clonidine premedication on attenuation of hemodynamic response to direct laryngoscopy and endotracheal intubation: A randomized controlled trial. Trends Anaesth Crit Care. 2023;5:101324.
[5] Sharma V, Fotedar K, Goel R. Comparison of oral clonidine and gabapentin premedication for attenuation of pressor response to laryngoscopy and endotracheal intubation. Anaesth Essays Res. 2020;14(3):412-19.
[6] AS N, Chetan L. A comparative study between oral clonidine and oral gabapentin as premedicants in attenuating the haemodynamics response to laryngoscopy and endotracheal intubation in normotensive patients. Eur J Cardiovasc Med. 2023;13(3):198-205.
[7] Waikar C, Singh J, Gupta D, Agrawal A. Comparative study of oral gabapentin, pregabalin, and clonidine as premedication for anxiolysis, sedation, and attenuation of pressor response to endotracheal intubation. Anaesth Essays Res. 2017;11(3):558.
[8] Bhandari G, Shahi KS. Effect of gabapentin on pressor response to laryngoscopy and tracheal intubation: A double blind randomized placebo controlled study. People's J Sci Res. 2013;6(1):01-06.
[9] Bafna U, Goyal VK, Garg A. A comparison of different doses of gabapentin to attenuate the haemodynamic response to laryngoscopy and tracheal intubation in normotensive patients. J Anaesthesiol Clin Pharmacol. 2011;27(1):43.
[10] Choudhary S, Sharma S, Kumari I, Kalluraya S, Meena K, Dave T. Comparative evaluation of oral melatonin and oral clonidine for the attenuation of haemodynamic response to laryngoscopy and tracheal intubation—A prospective randomised double blind study. Indian J Anaesth. 2020;64(8):696.
[11] Teong CY, Huang CC, Sun FJ. The haemodynamic response to endotracheal intubation at different time of fentanyl given during induction: A randomised controlled trial. Sci Rep. 2020;10:8829.
[12] Singhal SK, Kaur K, Arora P. Oral clonidine versus gabapentin as premedicant for obtunding hemodynamic response to laryngoscopy and tracheal intubation. Saudi J Anaesth. 2014;8(2):172.
[13] Montazeri K, Kashefi P, Honarmand A, Safavi M, Hirmanpour A. Attenuation of the pressor response to direct laryngoscopy and tracheal Intubation: Oral clonidine vs. oral gabapentin premedication. J Res Med Sci. 2011;16(Suppl1):S377.
[14] Joshi GP, Abdelmalak BB, Weigel WA, Harbell MW, Kuo CI, Soriano SG, et al. 2023 American society of anesthesiologists practice guidelines for preoperative fasting: Carbohydrate-containing clear liquids with or without protein, chewing gum, and pediatric fasting duration—A modular update of the 2017 American society of anesthesiologists practice guidelines for preoperative fasting*. Anesthesiology. 2023;138(2):132-51.
[15] Choudhuri AH, Kiro K. Perioperative fluid manangement. In: Malbrain ML, Wong A, Nasa P, Ghosh S. (eds) Rational use of intravenous fluids in critically ill patients. Springer, Cham. 2024.
[16] Thilen SR, Weigel WA, Todd MM, Dutton RP, Lien CA, Grant SA, et al. 2023 American society of anesthesiologists practice guidelines for monitoring and antagonism of neuromuscular blockade: A report by the American society of anesthesiologists task force on neuromuscular blockade. Anesthesiology. 2023;138(1):13-41.
[17] Chauhan V, Kumar A. Comparative study of gabapentin, clonidine and placebo in alleviating the hemodynamic changes due to tracheal intubation and laryngoscopy. Cureus. 2023;15(4):e37898.
[18] Raval DL, Mehta MK. Oral clonidine pre medication for attenuation of haemodynamic response to laryngoscopy and intubation. Indian J Anaesth. 2002;46(2):124-29.
[19] Flor CAG et al. Comparison between oral gabapentin and oral clonidine as premedicant in obtunding haemodynamic response to laryngoscopy and endotracheal intubation. Journal of Chemical Health Risks. 2024;14(6):1041-51.
[20] Nagaraja AS, Chetan L, Komala B, Priyanka S. A comparative study between oral clonidine and oral gabapentin as premedicants in attenuating the haemodynamics response to laryngoscopy and endotracheal intubation in normotensive patients. European Journal of Cardiovascular Medicine. 2024;13(3):198-04.
[21] Saravandi RY, Reshma M, Mahadev MP, Nadaf MN. comparison between oral clonidine (0.3 mg) and oral gabapentin (900 mg) in adult patients undergoing elective surgeries under general anaesthesia with respect to attenuation of hemodynamic responses: A prospective randomised double blinded study. European Journal of Cardiovascular Medicine. 2024;14(3):513-19.
[22] Parveen S, Negi DS, Kumar R, Bagwan MC. Oral clonidine vs oral pregabalin premedication to attenuate pressor response to direct laryngoscopy in patients undergoing laparoscopic cholecystectomy: A randomized double blind study. J Clin Diagn Res. 2016;10(9):UC21.

PARTICULARS OF CONTRIBUTORS:
1. Associate Professor, Department of Anaesthesiology, Shrimati Bhikhiben Kanjibhai Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth (Deemed to be University), Vadodara, Gujarat, India.
2. Third Year Resident, Department of Anaesthesiology, Shrimati Bhikhiben Kanjibhai Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth (Deemed to be University), Vadodara, Gujarat, India.
3. Professor, Department of Anaesthesiology, Shrimati Bhikhiben Kanjibhai Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth (Deemed to be University), Vadodara, Gujarat, India.
4. Professor and Medical Superintendent, Department of Anaesthesiology, Shrimati Bhikhiben Kanjibhai Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth (Deemed to be University), Vadodara, Gujarat, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Devendra Joshi,
Third Year Resident, Anaesthesia Department, General OT Complex, 2nd Floor, Dhiraj Hospital, Smt. Bhikiben Kanjibhai Shah Medical Institute and Research Centre, Wagodhia, Pipariya, Vadodara-391760, Gujarat, India.
E-mail: devendra.joshi1995@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Jul 03, 2025
- Manual Googling: Jul 19, 2025
- iThenticate Software: Jul 22, 2025 (13%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Jun 17, 2025**
Date of Peer Review: **Jul 05, 2025**
Date of Acceptance: **Jul 24, 2025**
Date of Publishing: **Sep 01, 2025**