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Research Article

# A COMPARATIVE STUDY OF THE EFFECTS OF ORAL PREGABALIN, ORAL CLONIDINE, AND A CONTROL GROUP ON ATTENUATION OF STRESS RESPONSE TO NASOTRACHEAL INTUBATION

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**ABSTRACT**

**Objective:** Endotracheal intubation, particularly nasotracheal intubation, induces a pronounced sympathetic response characterized by tachycardia, hypertension, and elevated plasma catecholamines. This stress response may be deleterious in patients with cardiovascular or neurological comorbidities. Pharmacological agents such as clonidine and pregabalin have been investigated for their potential to attenuate this hemodynamic surge. The study aims to compare the efficacy and safety of oral clonidine (300 µg) and oral pregabalin (150 mg) in attenuating the hemodynamic response to nasotracheal intubation.

**Methods:** In this prospective randomized comparative interventional study, 120 American Society of Anesthesiologists I-II adult patients (18–60 years) scheduled for elective surgeries requiring nasotracheal intubation were divided into three groups using a computer-generated random sequence: Group A received oral clonidine, Group B received oral pregabalin, and Group C served as control. The study was single-blinded, with patients unaware of group allocation. Hemodynamic parameters were recorded at baseline, immediately before induction, immediately after intubation, and at 1, 3, 5, and 10 min post-intubation. Adverse events were also noted.

**Results:** Baseline demographic and clinical characteristics were comparable across all groups. Both clonidine and pregabalin significantly attenuated the post-intubation rise in heart rate, systolic blood pressure, and diastolic blood pressure compared to the control group ($p<0.001$). Adverse events were infrequent, mild, and self-limiting, with no serious complications reported.

**Conclusion:** Oral clonidine and pregabalin are effective and safe pre-medications for attenuating the hemodynamic response to nasotracheal intubation, enhancing peri-intubation stability and patient safety.

**Keywords:** Nasotracheal intubation, Clonidine, Pregabalin, Hemodynamic response.

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## INTRODUCTION

Endotracheal intubation is an integral component of administering general anesthesia, ensuring airway protection and adequate ventilation during surgery [1,2]. However, the act of laryngoscopy and endotracheal tube insertion is known to produce a pronounced sympathetic surge characterized by tachycardia, hypertension, and increased plasma catecholamine levels [3]. These hemodynamic fluctuations, often referred to as the "stress response," may be tolerated by healthy individuals but can prove deleterious in patients with cardiovascular disease, intracranial pathology, or other comorbid conditions [4,5]. Nasotracheal intubation, commonly used in maxillofacial and head and neck surgeries, is often more stimulating than orotracheal intubation, thereby amplifying the magnitude of this stress response [6].

Several pharmacological agents have been investigated to blunt the hemodynamic effects associated with intubation. These include opioids, beta-blockers, calcium channel blockers, vasodilators, and $\alpha$2-adrenergic agonists [7,8]. While many of these drugs are effective, their use is often limited by dose-related adverse effects such as bradycardia, hypotension, or prolonged sedation. As a result, the search for safe and reliable alternatives remains ongoing. Among the newer options, pregabalin and clonidine have emerged as promising pre-medication agents due to their anxiolytic, analgesic, and sympatholytic properties [9].

Pregabalin, a structural analog of gamma-aminobutyric acid, binds to the $\alpha$2-$\delta$ subunit of voltage-gated calcium channels, thereby modulating neuronal excitability and reducing sympathetic outflow [10]. It has been widely used in the management of neuropathic pain, seizures, and perioperative anxiety [11]. Evidence suggests that pregabalin can attenuate the hemodynamic response to laryngoscopy and intubation while also providing perioperative analgesic benefits [12]. Clonidine, on the other hand, is an $\alpha$2-adrenergic agonist with well-documented sedative, anxiolytic, and analgesic properties. By reducing central sympathetic activity, clonidine lowers heart rate (HR) and blood pressure, making it a valuable agent in blunting intubation-induced stress responses [13,14].

The present study aimed to compare the efficacy of oral pregabalin, oral clonidine, and a control regimen in attenuating the hemodynamic responses to nasotracheal intubation. By systematically evaluating changes in HR, systolic and diastolic blood pressures (SBP and DBP), as well as recording adverse events, this study seeks to provide clarity on the comparative effectiveness and safety of these agents. Identifying the superior option has important implications for improving perioperative hemodynamic stability and enhancing patient safety during surgeries requiring nasotracheal intubation.

## METHODS

This prospective randomized comparative interventional study was conducted at Apollo Institute of Medical Sciences and Research, Hyderabad, between April 2025 and September 2025. The study included adult patients aged 18–60 years, belonging to the American

Society of Anesthesiologists (ASA) grades I and II, who were scheduled to undergo elective surgical procedures under general anesthesia requiring nasotracheal intubation. Written informed consent was obtained from all participants before inclusion. Patients with a history of drug or alcohol abuse, chronic pain syndromes, psychiatric illness, peripheral vascular disease, anticipated difficult airway, pregnancy, or those already taking pregabalin or clonidine were excluded.

Sample size calculation was based on the variability reported by Kaur *et al*. [14], using percentage change in HR as the primary outcome. From the reference study (mean HR difference=8 beats/min, standard deviation [SD]=10 beats/min), the sample size required to detect a 10% difference in HR with 80% power and $\alpha$=0.05 (two-tailed) was calculated as 36 per group using G*Power version 3.1.9.2, which was increased to 40/group (total=120) to account for attrition.

Patients were randomly assigned to one of three groups using a computer-generated randomization sequence, ensuring comparable baseline characteristics. Group A received oral clonidine 300 µg, Group B received oral pregabalin 150 mg, and Group C received a matching placebo (a vitamin capsule identical in appearance). All study medications were prepared and coded by an independent nurse to maintain allocation concealment.

The study was conducted in a single-blind manner, where patients were unaware of their group allocation, whereas the anesthesiologist administering the drugs was not blinded. The absence of double-blinding is recognized as a methodological limitation. Study drugs were given with a sip of water 90–120 min before induction of anesthesia, following institutional protocol.

Hemodynamic parameters such as HR, SBP, and DBP were recorded at baseline (before drug administration), immediately before induction, immediately after nasotracheal intubation and cuff inflation, and at 1, 3, 5, and 10 min post-intubation. Peri-intubation adverse events such as hypotension, bradycardia, dizziness, or excessive sedation were also documented. The primary outcome was attenuation of the hemodynamic response to nasotracheal intubation, and secondary outcomes included peri-intubation stability and adverse-effect profile.

Data were analyzed using the Statistical Package for the Social Sciences version 26. Continuous variables were presented as mean±SD and compared using analysis of variance. Categorical variables were expressed as percentages and analyzed using the Chi-square test. P<0.05 was considered statistically significant.

## RESULTS

The demographic and baseline clinical characteristics were comparable across the three groups. The mean age was similar in Group A (38.5±10.2 years), Group B (37.6±9.8 years), and Group C (39.1±11.0 years). Gender distribution was nearly balanced, with males comprising 55.0%, 50.0%, and 57.5% of patients in Groups A, B, and C, respectively. The mean body weight was also comparable (65.2±8.5, 66.8±7.9, and 64.7±9.2 kg, respectively). ASA physical status was predominantly Class I in all three groups (60.0% in Group A, 62.5% in Group B, and 57.5% in Group C), with no statistically significant differences observed. The mean duration of surgery was comparable across groups, ranging from 112.3 to 115.7 min. These findings confirmed homogeneity of baseline variables between study groups (Table 1).

**Table 1: Baseline demographic and clinical characteristics**

| Variable | Group A (Clonidine) (n=40) | Group B (Pregabalin) (n=40) | Group C (Control) (n=40) | p-value |
|---|---|---|---|---|
| Age (years) | | | | |
| Mean±SD | 38.5±10.2 | 37.6±9.8 | 39.1±11.0 | 0.81 |
| Gender (%) | | | | |
| Male | 22 (55.0) | 20 (50.0) | 23 (57.5) | 0.79 |
| Female | 18 (45.0 | 20 (50.0) | 17 (42.5) | |
| Weight (kg) | | | | |
| Mean±SD | 65.2±8.5 | 66.8±7.9 | 64.7±9.2 | 0.62 |
| ASA physical status (%) | | | | |
| I | 24 (60.0) | 25 (62.5) | 23 (57.5) | 0.90 |
| II | 16 (40.0) | 15 (37.5) | 17 (42.5) | |
| Duration of surgery (min) | | | | |
| Mean±SD | 112.3±18.6 | 115.7±19.2 | 114.5±17.8 | 0.72 |

SD: Standard deviation, ASA: American Society of Anesthesiologists, ANOVA: Analysis of variance. Values are mean±SD or n (%). ANOVA used for continuous variables, $\chi 2$ test for categorical variables. Total n=120

HR response during nasotracheal intubation revealed significant attenuation in the clonidine and pregabalin groups compared to the control. While baseline and pre-induction HRs were comparable, a sharp rise was observed immediately after intubation in the control group (112.8±10.1 bpm), which was markedly blunted in Group A (92.6±8.9 bpm) and Group B (95.1±9.4 bpm). This attenuation persisted at 1, 3, 5, and 10 min post-intubation, with both clonidine and pregabalin groups maintaining significantly lower HRs than controls. The differences across groups were highly significant after intubation and during the early post-intubation period (p<0.001) (Table 2).

Similar trends were observed with SBP. Baseline and pre-induction values were statistically comparable; however, a marked surge occurred in the control group following intubation (158.7±12.4 mmHg), compared with attenuated rises in the clonidine (135.8±10.6 mmHg) and pregabalin groups (138.4±11.2 mmHg). The differences remained significant up to 5 min after intubation, with gradual normalization thereafter. Both study drugs demonstrated effective blunting of the pressor response when compared to control (Table 3).

DBP trends were consistent with the systolic changes. A significant elevation occurred in the control group after intubation (101.2±8.7 mmHg), whereas the clonidine and pregabalin groups showed only modest increases (86.9±7.8 mmHg and 88.1±8.1 mmHg, respectively). The differences remained statistically significant at 1, 3, and 5 min post-intubation, with all groups returning closer to baseline values by 10 min. Thus, both clonidine and pregabalin were effective in attenuating diastolic responses to intubation (Table 4).

The incidence of adverse events was low across all groups. Hypotension occurred in 5.0% of patients in the clonidine group and 7.5% in the pregabalin group, whereas none was observed in controls. Bradycardia was rare, occurring in only one patient (2.5%) receiving clonidine. Dizziness was reported in 7.5% of clonidine and 10.0% of pregabalin patients, whereas none occurred in controls. Excessive sedation was observed in a small proportion of patients (5.0% in Group A, 2.5% in Group B), but none in controls. All adverse events were mild, self-limiting, and not statistically significant across groups (Table 5).

## DISCUSSION

Endotracheal intubation, particularly nasotracheal intubation, is a potent noxious stimulus that triggers sympathetic activation, resulting in tachycardia and hypertension. Attenuating this hemodynamic stress response is critical to reduce perioperative morbidity, especially in patients with cardiovascular or neurological comorbidities. In this study, oral clonidine (300 µg) and oral pregabalin (150 mg) both effectively blunted the rise in HR and blood pressure compared to controls, demonstrating their clinical utility in mitigating intubation-induced stress responses.

Baseline demographic and clinical characteristics – including age, gender, body weight, ASA physical status, and duration of surgery – were comparable across groups, ensuring that observed differences in hemodynamics were attributable to the pharmacological interventions rather than confounding factors. Similar baseline homogeneity has been reported by Kaur *et al*., Murari *et al*., and Thengumgal *et al*. [14-16].

**Table 2: Comparison of heart rate (beats/min)**

| Time point | Group A (Clonidine) (n=40) | Group B (Pregabalin) (n=40) | Group C (Control) (n=40) | p-value |
|---|---|---|---|---|
| Baseline | 82.4±8.2 | 81.9±7.6 | 83.1±7.9 | 0.84 |
| Before induction | 79.8±7.5 | 80.1±7.2 | 82.6±8.0 | 0.32 |
| After intubation+cuff inflation | 92.6±8.9 | 95.1±9.4 | 112.8±10.1 | <0.001 |
| 1 min post-intubation | 88.9±8.1 | 91.2±8.7 | 108.6±9.8 | <0.001 |
| 3 min post-intubation | 85.4±7.3 | 87.1±7.9 | 101.4±8.9 | <0.001 |
| 5 min post-intubation | 82.7±7.0 | 83.9±7.6 | 94.8±8.4 | <0.001 |
| 10 min post-intubation | 81.9±6.9 | 82.5±7.1 | 88.2±7.9 | 0.01 |

SD: Standard deviation, ANOVA: Analysis of variance. Values are mean±SD. ANOVA is used for continuous variables. Total n=120

**Table 3: Comparison of systolic blood pressure (mmHg)**

| Time Point | Group A (Clonidine) (n=40) | Group B (Pregabalin) (n=40) | Group C (Control) (n=40) | p-value |
|---|---|---|---|---|
| Baseline | 124.6±9.5 | 123.2±10.1 | 125.8±9.9 | 0.67 |
| Before induction | 121.9±8.7 | 122.1±9.0 | 124.2±8.8 | 0.49 |
| After intubation+cuff inflation | 135.8±10.6 | 138.4±11.2 | 158.7±12.4 | <0.001 |
| 1 min post-intubation | 131.7±9.8 | 134.2±10.1 | 152.9±11.8 | <0.001 |
| 3 min post-intubation | 127.5±9.2 | 128.8±9.6 | 144.6±10.7 | <0.001 |
| 5 min post-intubation | 124.2±8.9 | 125.6±9.3 | 136.1±9.8 | <0.001 |
| 10 min post-intubation | 122.8±8.4 | 123.5±8.6 | 128.4±9.0 | 0.02 |

SD: Standard deviation, ANOVA: Analysis of variance. Values are mean±SD. ANOVA is used for continuous variables. Total n=120

**Table 4: Comparison of diastolic blood pressure (mmHg)**

| Time point | Group A (Clonidine) (n=40) | Group B (Pregabalin) (n=40) | Group C (Control) (n=40) | p-value |
|---|---|---|---|---|
| Baseline | 78.2±6.8 | 77.6±7.1 | 78.9±6.9 | 0.83 |
| Before induction | 76.4±6.5 | 76.9±6.7 | 78.1±6.8 | 0.61 |
| After intubation+cuff inflation | 86.9±7.8 | 88.1±8.1 | 101.2±8.7 | <0.001 |
| 1 min post-intubation | 84.7±7.5 | 85.9±7.9 | 97.8±8.3 | <0.001 |
| 3 min post-intubation | 81.8±7.0 | 82.7±7.3 | 92.1±7.8 | <0.001 |
| 5 min post-intubation | 79.9±6.7 | 80.3±6.8 | 87.4±7.2 | <0.001 |
| 10 min post-intubation | 78.5±6.6 | 78.8±6.7 | 82.9±6.9 | 0.03 |

SD: Standard deviation, ANOVA: Analysis of variance. Values are mean±SD. ANOVA is used for continuous variables. Total n=120

**Table 5: Adverse events**

| Adverse events | Group A (Clonidine) (n=40) (%) | Group B (Pregabalin) (n=40) (%) | Group C (Control) (n=40) (%) | p-value |
|---|---|---|---|---|
| Hypotension | 2 (5) | 3 (7.5) | 0 (0) | 0.23 |
| Bradycardia | 1 (2.5) | 0 (0) | 0 (0) | 0.37 |
| Dizziness | 3 (7.5) | 4 (10) | 0 (0) | 0.13 |
| Excessive sedation | 2 (5) | 1 (2.5) | 0 (0) | 0.35 |

Values are n (%). $\chi^2$ test for categorical variables. Total n=120

The sympatholytic and analgesic mechanisms of clonidine and pregabalin explain these effects. Clonidine, an α2-adrenergic agonist, reduces central sympathetic outflow and provides sedation and anxiolysis, while pregabalin binds to the α2-δ subunit of voltage-gated calcium channels, decreasing excitatory neurotransmitter release and sympathetic drive. These findings are consistent with previous studies demonstrating effective attenuation of the pressor response to intubation by both agents [14,15,17].

In the present study, clonidine appeared to show slightly better control of tachycardia immediately following intubation, whereas pregabalin provided comparable attenuation of systolic and DBP. Immediately post-intubation, HR increased sharply in the control group (112.8±10.1 bpm) but was blunted to 92.6±8.9 bpm with clonidine and 95.1±9.4 bpm with pregabalin (p<0.001). Attenuation persisted at 1, 3, and 5 min (HRs of 88.9±8.1 and 91.2±8.7 bpm vs. 108.6±9.8 bpm in controls at 1 min). By 10 min, HRs approached baseline but remained lower in the drug groups (81.9±6.9 vs. 82.5±7.1 vs. 88.2±7.9 bpm; p=0.01). Murari *et al*. [16] reported similar reductions in HR and DBP with clonidine, whereas Kaur *et al*. [14] observed better mean arterial pressure stabilization with clonidine. Although the difference between clonidine and pregabalin was not statistically compared, the trend toward better tachycardia control with clonidine should be interpreted cautiously and considered descriptive rather than conclusive.

Adverse events were infrequent and self-limiting. Hypotension occurred in 5% of clonidine and 7.5% of pregabalin patients; bradycardia in 2.5% of clonidine patients; dizziness in 7.5% and 10%; and mild sedation in 5% and 2.5%, respectively. While these differences were not statistically significant (p>0.05), the study's total sample size (n=120) was likely underpowered to detect statistically significant differences in the incidence of relatively rare adverse events.

Compared with other strategies such as opioids, beta-blockers, or intravenous dexmedetomidine, oral pregabalin and clonidine offer practical advantages, including ease of administration, predictable onset, and minimal cardiorespiratory depression, making them well-suited for elective surgical settings.

**Limitations**

This study included only ASA I-II patients undergoing elective procedures, limiting generalizability to high-risk populations. Only a single dose of each drug was evaluated, and dose–response relationships were not explored. The study was single-blind, which may

have introduced some observer bias. The sample size was adequately powered for hemodynamic outcomes but not for detecting uncommon adverse events, limiting the ability to identify smaller yet clinically relevant safety differences. Plasma catecholamine levels were not measured, which could have provided a biochemical correlation for the observed hemodynamic effects. Future larger, double-blind trials with broader inclusion criteria and biochemical assessments are warranted.

## CONCLUSION

Both oral clonidine (300 μg) and oral pregabalin (150 mg), when administered before nasotracheal intubation, effectively attenuated the sympathetic stress response, producing stable hemodynamic parameters compared to the control group. Clonidine demonstrated a trend toward greater control of tachycardia, whereas pregabalin provided comparable attenuation of blood pressure changes. Adverse events were infrequent, mild, and self-limiting, but the study was not powered to detect rare side effects, and these results should therefore be interpreted with caution. Overall, both clonidine and pregabalin are safe and effective pre-medication options for minimizing peri-intubation hemodynamic fluctuations and enhancing patient safety.

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None.

## AUTHOR'S CONTRIBUTIONS

All the authors equally contributed to designing, collecting the data, analyzing the results, and writing the study.

## CONFLICT OF INTEREST

None declared.

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