DOI: https://doi.org/10.54393/pjhs.v7i2.3651

PAKISTAN JOURNAL OF HEALTH SCIENCES
(LAHORE)
https://thejas.com.pk/index.php/pjhs
ISSN (E): 2790-9352, (P): 2790-9344
Volume 7, Issue 02 (February 2026)

Original Article

OPEN ACCESS

Role of Intravenous Dexmedetomidine for Attenuation of Hemodynamic Response to Laryngoscopy and Intubation in Controlled Hypertensive Patients: A Prospective Cohort Study

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ARTICLE INFO

Keywords:
Dexmedetomidine, Hemodynamics, Laryngoscopy, Intubation, Intratracheal, Hypertension

How to Cite:
Saeed, A., Abbas, S. M., Abbas, M. Q., Malik, S., Kumar, V., & Khurana, S. (2026). Role of Intravenous Dexmedetomidine for Attenuation of Hemodynamic Response to Laryngoscopy and Intubation in Controlled Hypertensive Patients: A Prospective Cohort Study: Intravenous Dexmedetomidine: Laryngoscopy and Intubation in Controlled Hypertensive Patients. Pakistan Journal of Health Sciences, 7(2), 20-25. https://doi.org/10.54393/pjhs.v7i2.3651

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Received Date: 14th November, 2025
Revised Date: 23rd December, 2025
Acceptance Date: 8th January, 2026
Published Date: 28th February, 2026

ABSTRACT

Due to sympathetic stimulation, laryngoscopy and endotracheal intubation are known to cause brief but noticeable elevations in blood pressure and heart rate, which can be dangerous for hypertensive individuals. It has been demonstrated that the specific α2-adrenergic agonist dexmedetomidine reduces these reactions. **Objectives:** To evaluate whether intravenous dexmedetomidine could effectively reduce hemodynamic reactions to laryngoscopy and intubation in patients with controlled hypertension. **Methods:** This prospective cohort study was carried out at Sindh Institute of Urology and Transplantation (SIUT) and used a non-probability consecutive sampling technique for six months from January 1, 2025, to June 30, 2025. Eligible were ASA II patients with high blood pressure who had been planned for elective surgery while under the influence of general anesthesia. Individuals were randomly assigned to receive either a placebo (20 mL normal saline) or dexmedetomidine (0.5 µg/kg diluted to 20 mL in saline) ten minutes before induction. Repeated-measures ANOVA and the Student's t-test were used to examine the data; $p \leq 0.05$ was deemed significant. **Results:** The groups' baseline characteristics were similar. When compared to control, dexmedetomidine significantly reduced the increase in HR and SBP following laryngoscopy and intubation ($p < 0.001$ at all post-intubation intervals). Bradycardia (5.7%) and hypotension (2.9%) were rare but clinically treatable adverse effects. **Conclusions:** In hypertensive patients, intravenous dexmedetomidine (0.5 µg/kg) administered as a single pre-induction dosage is safe and efficient in reducing the hemodynamics of laryngoscopy and intubation while also enhancing cardiovascular stability throughout induction.

INTRODUCTION

Laryngoscopy and tracheal intubation are the tests that should also be conducted during general anesthesia, but which are also related to the extreme sympathetic reaction [1, 2]. The consequent outcome of such a reflex surge is an acute rise in heart rate (HR), systolic and diastolic blood pressure (SBP, DBP), as well as an increase in circulating catecholamine. Although these hemodynamic changes are tolerated by most healthy people, they expose patients with underlying cardiovascular or hypertension to the risk of myocardial ischemia, arrhythmias, left ventricular failure, or cerebrovascular accidents. The primary concern in the practice of anesthetics that must be attained to maintain cardiovascular stability in airway manipulation is the attenuation of these pressor reflexes [3]. There are various pharmacological interventions that have been tried over the years, and these are opioids, beta-blockers, calcium channel blockers, vasodilators, and local anesthetics [4-6]. Nevertheless, these drugs are not

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always effective, and the majority of them have dose effects, including bradycardia, hypotension, or slow recuperation. A new drug, dexmedetomidine, a robust selective agonist that acts on the 2 adrenergic receptors, is one of the most promising drugs due to its dose-dependent sedation, anxiolysis, sympatholytic effects, and minimal respiratory depression [7, 8]. Dexmedetomidine has a central locus coeruleus in the brainstem to counter sympathetic discharge and norepinephrine discharge, which results in a regulated drop in HR and blood pressure [9, 10]. Several randomized controlled trials and meta-analyses that have been performed over the recent decade have found that the pre-induction routine of dexmedetomidine is effective in counteracting the tachycardic and hypertensive effects of intubation [11, 12]. Nonetheless, their application can sometimes lead to bradycardia or hypotension, and there are still inconsistencies in the best dosage schedule, particularly in patients with regulated hypertension, a category that is especially susceptible to the exaggeration of pressor reactions during laryngoscopy [13, 14].

Although extensive data have been collected in normotensive or mixed patients, large-scale studies on the target population, namely controlled hypertensive people, are quite scarce. Since there is the possibility of overstimulation of sympathetic activity in such patients, the effectiveness of dexmedetomidine and its safety profile in this patient group need to be evaluated further. Controlled hypertension patients are a high-risk population in which peri-intubation hemodynamic surges may have severe clinical implications. The sympatholytic and cardioprotective properties of dexmedetomidine could provide a middle ground between these responses without affecting respiratory activity or hemodynamic stability. Nevertheless, the best dose and infusion rate for hypertensive patients is unclear. The current research was conducted to compare the effect of intravenous dexmedetomidine on the hemodynamic response to laryngoscopy and endotracheal intubation in patients with controlled hypertension.

METHODS

A prospective cohort study was conducted at the Department of Anesthesiology in the Sindh Institute of Urology and Transplantation (SIUT) from January 1st, 2025, to June 30th, 2025. The SIUT Ethical Review Committee (ERC) granted ethical approval, with approval number SIUT-ERC-2024/A-488. Before their involvement in the study, all individuals provided written informed consent. The sample size was calculated using Open Epi Version 3.01 for comparing two means, with an expected heart rate reduction of 7.04%, an estimated SD of 8 beats/min, alpha = 0.05, and power = 80%, assuming equal group sizes [15]. The formula used was $n=((Z1_{-\alpha/2}+Z_{1-\beta})^2\times2\times SD^2)/\Delta^2$, where Δ represents the expected group difference. To account for potential dropouts, 35 participants per group were recruited, totaling 70 participants. The method of non-probability consecutive sampling was utilized to select the eligible participants until the necessary sample size was obtained. The patients who were aged 20-70 years, who had controlled hypertension under the antihypertensive therapy, and who were ASA II were included [14]. Hypertension was recognized as a measurement of systolic blood pressure surpassing 140 mmHg or diastolic blood pressure over 90 mmHg, which was sufficiently controlled with medication. The exclusion criteria were severe cardiac arrhythmias, a history of myocardial infarction, severe valvular heart disease, uncontrolled diabetes, severe hepatic or renal dysfunction, use of beta-blockers, pregnancy, known allergy to study drugs, and an expected difficult airway. Baseline monitoring included non-invasive blood pressure, 5-lead ECG, pulse oximetry, end-tidal CO2, and capnography of all the participants. In 10 minutes, 0.5mg/kg dexmedetomidine in 20 mL 0.9per cent saline was given to the dexmedetomidine group, and 20 mL 0.9per cent saline was given to the control group. Infusions were made and administered by an anesthesiologist not involved in the care and data collection of the patients. Anesthesia induction was done with three minutes of mask ventilation of lignocaine. Laryngoscopy and tracheal intubation were done by experienced anesthesiologists directly, and the duration of the laryngoscopy was documented. Airoxygen mixture of isoflurane and mechanical ventilation was used to sustain anesthesia with an EtCO2 target of 35-40mmHg. Atropine 0.5 mg IV was given to control bradycardia (HR less than 50 bpm), and ephedrine 5mg IV was given to control hypotension (MAP less than 60 mmHg or more than 20% vs baseline). The parameters in the hemodynamic measurements were recorded at the baseline (T0), after the infusion (T1), before the intubation (T2), and 1, 3, and 5minutes following the intubation (T3, T4, and T5). The primary outcome was the change in HR one minute following intubation, and the secondary outcomes were the change in SBP and the adverse events or the necessity of vasoactivity interventions. The data were analyzed with SPSS version 26.0 (IBM Corp., Armonk, NY, USA). The Shapiro-Wilk test was used to test the normality of the data. The report on the continuous variables indicated the mean with standard deviation (SD), and the group difference was determined with the independent-samples t-test when the data were normally distributed. ANOVA was applied repeatedly in the study of heart rate and systolic blood pressure with the passage of time. The assumption of sphericity was tested using the Mauchly test, and when the assumption of

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sphericity was violated, the test applied was the Greenhouse-Geisser correction. The intergroup difference and time x group interaction were assessed, and significant p-values of interaction demonstrated that attenuation of hemodynamic responses took place with time in the dexmedetomidine group. To adjust Type, I error caused by the multiple comparisons over six time points, Bonferroni post-hoc corrections were applied in all the pair-wise comparisons. The Chi-square and Fisher's exact test were used to analyse categorical data, which were later presented as frequencies and percentages. A p-value of less than 0.05 was used as a statistically significant value.

RESULTS

The dexmedetomidine group's mean age was 57.8 ± 7.9 years, while the non-dexmedetomidine groups was 56.9 ± 8.3 years (p=0.620). The baseline characteristics of both groups were comparable, indicating baseline comparability between groups. The p-values for age, sex distribution, body mass index (BMI), duration of laryngoscopy, baseline heart rate (HR), and baseline systolic blood pressure (SBP) were 0.620, 0.620, 0.660, 0.420, 0.680, and 0.630 respectively (Table 1).

Table 1: Demographic Characteristics of the Study Population (n=103)

Variables	Dexmedetomidine Group (n=35)	Control Group (n=35)	p-value
Age (Years)	57.8 ± 7.9	56.9 ± 8.3	0.620
Male	23 (65.7%)	21 (60.0%)	0.620
Female	12 (34.3%)	14 (40.0%)	
BMI (kg/m^2)	27.2 ± 3.8	27.6 ± 4.1	0.660
Duration of Laryngoscopy (s)	12.2 ± 1.8	12.5 ± 1.9	0.420
Baseline HR (beats/min)	78.6 ± 7.9	79.4 ± 8.1	0.680
Baseline SBP (mmHg)	138.0 ± 11.8	139.2 ± 12.1	0.630

At baseline, there was no difference in heart rate and systolic blood pressure between the groups. The dexmedetomidine group had a significantly lower heart rate and systolic blood pressure than the control group during infusion and the peri-intubation period (T1-T5) (p<0.001). Repeated-measures ANOVA demonstrated both variables to have a significant time × group interaction (p<0.001), indicating that there was a steady attenuation of hemodynamic responses over time in dexmedetomidine-treated patients (Table 2).

Table 2: Comparison of Hemodynamic Parameters Over Time among Study Participants

Time Point	HR Dexmed (beats/min)	HR Control (beats/min)	p-value (Between Groups)	Mean Difference (95% CI)	SBP Dexmed (mmHg)	SBP Control (mmHg)	p-value (Between Groups)	Time × Group Interaction p-value
T0 – Baseline	78.6 ± 7.9	79.4 ± 8.1	0.68	0.8 (–2.7 to 4.3)	138.0 ± 11.8	139.2 ± 12.1	0.630	–
T1 – Post-Infusion	72.4 ± 7.3	80.1 ± 8.2	<0.001	7.7 (4.1 to 11.3)	133.5 ± 11.2	140.0 ± 12.0	0.010	<0.001
T2 – Pre-Intubation	70.8 ± 7.0	81.5 ± 8.6	<0.001	10.7 (6.8 to 14.6)	131.8 ± 10.8	142.0 ± 12.5	<0.001	<0.001
T3 – 1 Min Post-Intubation	77.2 ± 9.0	95.2 ± 10.1	<0.001	17.9 (13.2 to 22.6)	138.6 ± 12.9	165.3 ± 15.8	<0.001	<0.001
T4 – 3 Min Post-Intubation	74.1 ± 8.2	88.6 ± 9.5	<0.001	14.5 (10.5 to 18.5)	135.0 ± 12.1	153.7 ± 14.2	<0.001	<0.001
T5 – 5 Min Post-Intubation	72.9 ± 7.8	83.5 ± 8.9	<0.001	10.6 (6.8 to 14.4)	133.2 ± 11.6	147.5 ± 13.7	<0.001	<0.001

The incidence of adverse events was low and comparable between the two groups, with no major complications reported. Bradycardia occurred in 5.7% of patients in the dexmedetomidine group and none in the control group (p=0.150), while hypotension was observed in 2.9% versus none (p=0.310). The requirement for atropine was slightly higher in the dexmedetomidine group (5.7%) compared to the control (0%) (p=0.150). Similarly, vasopressor use was noted in 2.9% of dexmedetomidine patients and 8.6% of controls (p=0.300) (Table 3).

Table 3: Incidence of Adverse Events among Study Participants (n=70)

Events	Dexmed Group (n=35)	95% CI	Control Group (n=35)	95% CI	p-value
Bradycardia (HR <50 bpm)	2 (5.7%)	0–13.4%	0 (0%)	0–0%	0.150
Hypotension (MAP <60 mmHg)	1 (2.9%)	0–8.4%	0 (0%)	0–0%	0.310
Atropine Use	2 (5.7%)	0–13.4%	0 (0%)	0–0%	0.150
Vasopressor Use	1 (2.9%)	0–8.4%	3 (8.6%)	0–17.8%	0.300
Major Adverse Events	0 (0%)	0–0%	0 (0%)	0–0%	–

DISCUSSION

The hemodynamic reaction to laryngoscopy and endotracheal intubation was significantly and clinically reduced by a single pre-induction infusion of dexmedetomidine at a dose of 0.5 µg/kg in this prospective cohort study of 70 ASA II patients with controlled hypertension. Heart rate and systolic blood pressure were considerably lower in the dexmedetomidine group from immediately following infusion to five minutes post-intubation, but initial findings were similar in both groups. These results demonstrate that a modest dose of dexmedetomidine reliably blunts peri-intubation sympathetic surges in patients with controlled hypertension, with a low incidence of major adverse events. [16, 17]. Current findings are consistent with several randomized trials and systematic reviews showing that dexmedetomidine reduces the tachycardic and hypertensive responses associated with airway manipulation. Misra et al. reported reduced HR and BP after nebulized dexmedetomidine compared with placebo

DOI: https://doi.org/10.54393/pjhs.v7i2.3651

during laryngoscopy and intubation, supporting the concept that dexmedetomidine, administered by different routes, attenuates the pressor response. Xiong and colleagues similarly found that dexmedetomidine premedication increased sedation and inhibited intubation-related stress in adults. A study by Jain et al. and other randomized trials have likewise reported significant reductions in peri-intubation HR and BP with doses in the 0.5–1.0 µg/kg range, corroborating our observation that 0.5 µg/kg is effective in the peri-intubation window [16, 18]. Several dose-comparison studies and meta-analyses have addressed the dose-response relationship of dexmedetomidine for attenuating intubation responses. While our study used 0.5 µg/kg, some trials found greater suppression with higher doses, suggesting a dose-dependent effect. Nevertheless, higher doses are also more likely to produce clinically relevant bradycardia or hypotension. The balance between efficacy and safety, therefore, supports use of moderate dosing (0.5 µg/kg) in many elective settings, especially when treating patients with controlled hypertension where excessive hypotension or bradycardia is undesirable. Our low rates of bradycardia (2/35) and hypotension (1/35), none of which led to major complications, echo the safety signals reported in other moderate-dose trials [19, 20]. Comparative and alternative strategies have also been evaluated in recent years. Trials comparing dexmedetomidine to other agents such as fentanyl, magnesium sulfate, esmolol, or labetalol have produced mixed results: some studies show comparable or superior attenuation with dexmedetomidine, whereas others found similar efficacy with shorter-acting agents but with different side-effect profiles. For example, head-to-head comparisons with beta-blockers or esmolol report that these drugs can blunt HR peaks rapidly but may be less effective on blood pressure or have different cardiovascular safety considerations. These different findings show that agent selection can also be tailored to each individual based on patient comorbidity, desired duration of effect, and clinician tolerance for bradycardia/hypotension. For example, in our cohort of hypertensive patients, dexmedetomidine afforded a sustained sympatholytic effect throughout the critical five-minute period post-intubation, which may be advantageous for patients at risk for acute hypertensive surges [21-23]. The route and timing of administration also affect outcomes. Studies examining pre-induction infusions of dexmedetomidine via IV (like the pre-induction of dexmedetomidine in the present trial) and alternative routes, such as nebulized or intranasal dexmedetomidine, have shown consistent benefit, but onset and peak effects differ. For example, nebulized or intranasal dexmedetomidine may be useful in situations where IV access or timing of administration is a concern. However, an infusion via an IV route provides precise titration, time to onset of action before laryngoscopy, and eliminates any concern for patient compliance or patient safety. The protocol we used, which involved an infusion of dexmedetomidine over ten minutes immediately before induction of anesthesia, was similar to other protocols, and the results were consistent with several randomized trials that demonstrated attainment of optimal attenuation of response without exaggerated sedation or respiratory depression [18, 24]. Safety is an essential consideration when using α2-agonists in hypertensive patients. Although dexmedetomidine reliably reduced HR and SBP, it can also precipitate bradycardia and hypotension. In our cohort, these events were infrequent and manageable with standard interventions (atropine for bradycardia, ephedrine for hypotension). This tolerability mirrors findings from other contemporary trials that report low but non-negligible rates of bradycardia/hypotension, typically dose dependent, and reinforces the need for appropriate patient selection, monitoring, and rescue protocols when employing dexmedetomidine perioperatively [16, 25]. The findings of our study are new since earlier literature has failed to adequately represent the higher-risk group of patients with controlled hypertension. The majority of previous investigations on the effects of dexmedetomidine on hemodynamic changes during laryngoscopy and intubation have incorporated mixed or normotensive cohorts. Through observation of a cohort with hypertension, we establish that even a moderate dose of dexmedetomidine (0.5 µg/kg) given before induction can help in attenuating the increase in heart rate and systolic blood pressure during airway manipulation without significantly increasing the number of adverse events. This clinical, empirical evidence justifies the implementation of this intervention in other patients, and this offers real-life advice on the peri-intubation hemodynamic support. The increased dosage may provide stronger attenuation, but it may also raise the chances of bradycardia or hypotension, and this is why efficacy and safety balance are of great importance. Recent clinical trials and registry data have demonstrated a growing interest in the use of dexmedetomidine among hypertensive patients, yet there is still a lack of high-quality data. Our future population contributes to this information, as it proves the hemodynamic advantage and safety of a moderate dose pre-induction regimen in a controlled population with hypertension. This prospective cohort study found that one pre-induction dexmedetomidine, 0.5 µg/kg, infusion was a strong suppressor of heart rate and systolic blood pressure responses to laryngoscopy and intubation in patients with controlled hypertension with an excellent

DOI: https://doi.org/10.54393/pjhs.v7i2.3651

short-term safety profile. These results advocate moderate dose of dexmedetomidine and its application as a practical and effective peri-intubation hemodynamic adjunct in high-risk patients with hypertension and provide significant evidence to an underrepresented population in past studies.

This was a single-center prospective cohort study with a relatively small sample size and absence of randomization, which may limit causal inference and external validity. Hemodynamic outcomes were assessed only in the immediate peri-intubation period, without evaluation of longer intraoperative or postoperative cardiovascular effects. Larger randomized controlled trials comparing dexmedetomidine with alternative agents in hypertensive patients are needed to define optimal dosing and long-term safety across different surgical settings.

CONCLUSIONS

In individuals who have controlled hypertension, pre-induction intravenous infusion of dexmedetomidine safely and efficiently reduces the hemodynamic response to intubation of the endotracheal tube and laryngoscopy. The medication had very minor, tolerable side effects and significantly reduced increases in heart rate and systolic blood pressure at all post-intubation time periods as compared to a placebo. The results validate the strong sympatholytic and cardioprotective effects of dexmedetomidine and show its clinical use as an addition to anesthesia for individuals susceptible to stress-induced increases in heart rate and blood pressure.

Authors' Contribution

Conceptualization: AS
Methodology: MQA
Formal analysis: AS, SMA, MQA, SM, SK
Writing and Drafting: AS, SMA, SM, VK
Review and Editing: AS, SMA, MQA, SM, VK, SK

All authors approved the final manuscript and take responsibility for the integrity of the work.

Conflicts of Interest

All the authors declare no conflict of interest.

Source of Funding

The author received no financial support for the research, authorship and/or publication of this article.

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