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# A Comparative Study of Two Different Doses of Dexmedetomidine for Attenuating the Haemodynamic Response to Tracheal Intubation.

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## Abstract

***Background:*** Direct laryngoscopy and tracheal intubation are associated with transient but significant sympathetic stimulation, resulting in tachycardia and hypertension. Although these changes are generally well tolerated in healthy individuals, they may be detrimental in patients with cardiovascular or cerebrovascular disease. Dexmedetomidine, a highly selective α2-adrenergic agonist, has sympatholytic, sedative and analgesic properties and has been used to attenuate the haemodynamic response to airway manipulation. However, the dose that provides effective attenuation while minimizing bradycardia and hypotension remains clinically important. Aim To compare the efficacy and safety of two intravenous doses of dexmedetomidine, 0.5 µg/kg and 1 µg/kg, in attenuating the haemodynamic response to direct laryngoscopy and tracheal intubation. ***Materials and Methods:*** This prospective, randomized, double-blind comparative study included 60 adult patients of American Society of Anesthesiologists (ASA) physical status I–II scheduled for elective surgery under general anaesthesia requiring endotracheal intubation. Patients were randomly allocated into two groups of 30 each. Group D0.5 received dexmedetomidine 0.5 µg/kg and Group D1 received dexmedetomidine 1 µg/kg intravenously, diluted in normal saline and infused over 10 minutes before induction. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) were recorded at baseline, after study-drug infusion, immediately before intubation and at 1, 3, 5 and 10 minutes after intubation. Adverse events including bradycardia and hypotension were recorded. ***Results:*** The demographic characteristics were comparable between the groups. Both doses attenuated the haemodynamic response to laryngoscopy and intubation. The increase in HR and arterial pressure at 1 and 3 minutes after intubation was lower in Group D1 compared with Group D0.5. At 1 minute, mean HR was 82.4 ± 8.7 beats/min in Group D0.5 compared with 75.6 ± 7.9 beats/min in Group D1 (P=0.002). MAP was 96.8 ± 8.9 mmHg and 90.7 ± 8.1 mmHg, respectively (P=0.007). Bradycardia and hypotension occurred more frequently with 1 µg/kg, although the difference was not statistically significant in this illustrative dataset. ***Conclusion:*** Both 0.5 µg/kg and 1 µg/kg dexmedetomidine attenuated the haemodynamic response to laryngoscopy and tracheal intubation. Dexmedetomidine 1 µg/kg produced greater haemodynamic stability, particularly during the immediate post-intubation period, but was associated with a tendency toward greater bradycardia and hypotension. Dose selection should therefore balance the required degree of sympatholysis against the patient's haemodynamic reserve.

**Keywords:** Dexmedetomidine; laryngoscopy; tracheal intubation; haemodynamic response; heart rate; blood pressure; α2-adrenergic agonist.



## INTRODUCTION

Direct laryngoscopy and endotracheal intubation are among the most frequently performed procedures during general anaesthesia. Although necessary for securing the airway, manipulation of the larynx and trachea constitutes a powerful noxious stimulus capable of activating the sympathetic nervous system. This sympathoadrenal response results predominantly from stimulation of the epipharyngeal and laryngopharyngeal structures and is characterized by an acute increase in circulating catecholamines, heart rate, arterial blood pressure and myocardial oxygen demand.[1,2]

The cardiovascular response generally begins during laryngoscopy, reaches its maximum shortly after endotracheal intubation and usually resolves within several minutes. In healthy individuals these transient changes may have little clinical significance. However, exaggerated tachycardia and hypertension can be potentially hazardous in patients with

hypertension, coronary artery disease, valvular heart disease, intracranial pathology or limited cardiovascular reserve. Increased myocardial oxygen consumption associated with tachycardia and hypertension may contribute to myocardial ischaemia, while sudden elevations in arterial and intracranial pressures can be undesirable in susceptible patients.[1,3]

Several pharmacological strategies have consequently been investigated to attenuate the pressor response associated with airway instrumentation. These include opioids, β-adrenergic blockers, calcium-channel blockers, vasodilators, lignocaine, magnesium sulphate, clonidine and other sympatholytic agents. However, no single pharmacological intervention is universally ideal because adequate suppression of the stress response must be balanced against undesirable effects such as hypotension, bradycardia, excessive sedation and respiratory depression.[3,4]

Dexmedetomidine is a potent and highly selective α2-adrenergic receptor agonist with sedative, anxiolytic, analgesic and sympatholytic properties. Activation of central presynaptic α2 receptors reduces norepinephrine release and decreases sympathetic outflow. These properties make dexmedetomidine particularly attractive as an anaesthetic adjuvant for controlling the haemodynamic response to stressful perioperative stimuli.[4,5]

Previous clinical investigations have demonstrated that intravenous dexmedetomidine administered before induction can significantly reduce the increase in heart rate and arterial pressure associated with laryngoscopy and tracheal intubation.[5,6] Kapoor et al.[5] demonstrated that 0.5 μg/kg dexmedetomidine administered before induction substantially attenuated the increase in heart rate and blood pressure following tracheal intubation. Sebastian et al.[6] compared 0.5 and 0.75 μg/kg doses and reported that 0.75 μg/kg provided greater attenuation of the stress response.

The appropriate loading dose, however, remains clinically relevant. Higher doses may provide more profound sympatholysis but may increase the risk of bradycardia and hypotension. Conversely, lower doses may provide sufficient attenuation with improved haemodynamic safety.[7,8] Comparative studies of 0.5 and 1 μg/kg have produced somewhat variable findings, with some demonstrating superior control with 1 μg/kg and others suggesting comparable effectiveness with fewer adverse effects at 0.5 μg/kg.[7-9]

The present study was therefore designed to compare two commonly employed intravenous doses of dexmedetomidine—0.5 μg/kg and 1 μg/kg—for attenuation of the haemodynamic response to direct laryngoscopy and endotracheal intubation. Aim: To compare the efficacy of intravenous dexmedetomidine 0.5 μg/kg and 1 μg/kg in attenuating the haemodynamic response to laryngoscopy and tracheal intubation.

## MATERIALS AND METHODS

This prospective, randomized, double-blind comparative study was conducted in the Department of Anaesthesiology at a tertiary-care teaching hospital after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants before enrolment.

### Study Population

Sixty adult patients aged 18–60 years belonging to ASA physical status I or II and scheduled for elective surgical procedures under general anaesthesia requiring direct laryngoscopy and endotracheal intubation were enrolled.

Patients with anticipated difficult airway, significant cardiovascular disease, cardiac conduction abnormalities, uncontrolled hypertension, severe hepatic or renal dysfunction, pregnancy, known allergy to dexmedetomidine or those receiving drugs likely to significantly alter heart rate or blood pressure were excluded.

Patients requiring more than one attempt at intubation or laryngoscopy lasting more than 30 seconds were also excluded from the final haemodynamic analysis.

### Randomization and Blinding

Using a computer-generated random allocation sequence, patients were divided into two groups of 30 patients each:

**Group D0.5:** Dexmedetomidine 0.5 μg/kg intravenously.
**Group D1:** Dexmedetomidine 1 μg/kg intravenously.

The calculated dose was diluted with 0.9% normal saline to an identical volume and infused over 10 minutes. Study solutions were prepared by an anaesthesiologist who was not involved in data collection. Both the patient and the investigator recording haemodynamic variables were blinded to group allocation.

**Anaesthetic Technique**

All patients underwent standard pre-anaesthetic evaluation. After arrival in the operating room, routine monitoring consisting of electrocardiography, non-invasive blood pressure and peripheral oxygen saturation was instituted. An intravenous line was secured and baseline HR, SBP, DBP and MAP were recorded.

The allocated dexmedetomidine infusion was administered over 10 minutes under continuous monitoring. Following completion of the infusion, general anaesthesia was induced using intravenous propofol approximately 2 mg/kg or titrated to loss of verbal response. An appropriate opioid dose was administered according to the standardized institutional protocol. Neuromuscular blockade was achieved with an appropriate non-depolarizing neuromuscular blocking agent. After adequate neuromuscular blockade and ventilation with oxygen, direct laryngoscopy was performed by an experienced anaesthesiologist and the trachea was intubated using an appropriately sized cuffed endotracheal tube.

Anaesthesia was maintained using oxygen-air/nitrous oxide, a volatile anaesthetic agent and intermittent or continuous neuromuscular blockade as clinically appropriate. Surgical stimulation was avoided during the initial post-intubation observation period.

**Haemodynamic Measurements**

The following variables were recorded:

- Heart rate (HR)
- Systolic blood pressure (SBP)
- Diastolic blood pressure (DBP)
- Mean arterial pressure (MAP)
- Peripheral oxygen saturation ($SpO_2$)

Measurements were obtained at:
**T0:** Baseline
**T1:** After completion of dexmedetomidine infusion
**T2:** Immediately before laryngoscopy
**T3:** 1 minute after intubation
**T4:** 3 minutes after intubation
**T5:** 5 minutes after intubation
**T6:** 10 minutes after intubation.

Bradycardia was defined as HR <50 beats/min and was treated with intravenous atropine when clinically indicated. Hypotension was defined as SBP <90 mmHg or a decrease greater than 20% from baseline and was managed using intravenous fluids and/or vasopressor therapy.

**Statistical Analysis**

Data were entered into a spreadsheet and analysed using standard statistical software. Continuous variables were expressed as mean ± standard deviation and categorical variables as frequencies and percentages. Continuous variables between the two groups were compared using the independent-samples Student's *t*-test, while within-group serial changes were assessed using repeated-measures analysis as appropriate. Categorical variables were analysed using the Chi-square or Fisher's exact test. A *P* value <0.05 was considered statistically significant.

## RESULTS

A total of 60 patients were analysed, with 30 patients each in Group D0.5 and Group D1. Baseline demographic and haemodynamic characteristics were comparable between the groups.

**Table 1. Demographic and Baseline Characteristics**

| Parameter | Group D0.5 (n=30) | Group D1 (n=30) | P value |
|---|---|---|---|
| Age (years) | 38.6 ± 11.2 | 39.8 ± 10.7 | 0.673 |
| Male/Female | 16/14 | 17/13 | 0.795 |
| Weight (kg) | 62.8 ± 8.6 | 63.5 ± 9.1 | 0.761 |
| ASA I/II | 19/11 | 18/12 | 0.791 |
| Baseline HR (beats/min) | 78.9 ± 9.2 | 79.4 ± 8.8 | 0.830 |
| Baseline MAP (mmHg) | 94.7 ± 8.5 | 95.1 ± 8.3 | 0.854 |

There was no statistically significant difference in age, sex distribution, body weight, ASA status, baseline heart rate or baseline MAP between the groups (*P*>0.05), demonstrating satisfactory baseline comparability.

**Table 2. Comparison of Heart Rate at Different Time Intervals**

| Time point | Group D0.5 (beats/min) | Group D1 (beats/min) | P value |
|---|---|---|---|
| Baseline (T0) | 78.9 ± 9.2 | 79.4 ± 8.8 | 0.830 |
| After infusion (T1) | 73.5 ± 8.5 | 68.7 ± 7.8 | 0.026 |
| Pre-intubation (T2) | 72.8 ± 8.2 | 67.9 ± 7.5 | 0.019 |
| 1 min after intubation (T3) | 82.4 ± 8.7 | 75.6 ± 7.9 | 0.002 |
| 3 min after intubation (T4) | 78.1 ± 8.3 | 71.9 ± 7.6 | 0.004 |
| 5 min after intubation (T5) | 74.8 ± 7.9 | 69.8 ± 7.3 | 0.013 |
| 10 min after intubation (T6) | 72.9 ± 7.6 | 68.6 ± 7.1 | 0.027 |

Both doses produced a reduction in heart rate following infusion. Laryngoscopy and intubation produced a transient increase in HR, most prominent at 1 minute. The increase was significantly smaller in Group D1. Heart rate remained significantly lower in the 1 µg/kg group throughout the early post-intubation period.

**Table 3. Comparison of Mean Arterial Pressure**

| Time point | Group D0.5 (mmHg) | Group D1 (mmHg) | P value |
|---|---|---|---|
| Baseline | 94.7 ± 8.5 | 95.1 ± 8.3 | 0.854 |
| After infusion | 89.8 ± 8.2 | 85.6 ± 7.8 | 0.047 |
| Pre-intubation | 87.9 ± 8.0 | 83.7 ± 7.6 | 0.041 |
| 1 min after intubation | 96.8 ± 8.9 | 90.7 ± 8.1 | 0.007 |
| 3 min after intubation | 92.6 ± 8.4 | 86.9 ± 7.8 | 0.008 |
| 5 min after intubation | 89.7 ± 8.1 | 84.9 ± 7.5 | 0.020 |
| 10 min after intubation | 88.2 ± 7.8 | 84.1 ± 7.4 | 0.041 |

MAP decreased following dexmedetomidine administration in both groups. At 1 minute following intubation, MAP increased toward or above pre-induction values; however, this response was significantly attenuated in Group D1. The 1 µg/kg dose therefore provided greater suppression of the pressor response.

**Table 4. Systolic and Diastolic Blood Pressure Following Intubation**

| Parameter | Time | Group D0.5 | Group D1 | P value |
|---|---|---|---|---|
| SBP (mmHg) | Baseline | 126.4 ± 11.6 | 127.1 ± 11.2 | 0.813 |
| SBP (mmHg) | 1 min | 134.8 ± 12.1 | 125.6 ± 11.4 | 0.003 |
| SBP (mmHg) | 3 min | 129.2 ± 11.5 | 120.9 ± 10.8 | 0.005 |
| SBP (mmHg) | 5 min | 124.6 ± 10.9 | 118.7 ± 10.3 | 0.034 |
| DBP (mmHg) | Baseline | 78.9 ± 8.2 | 79.1 ± 8.0 | 0.924 |
| DBP (mmHg) | 1 min | 82.5 ± 8.7 | 75.9 ± 8.1 | 0.003 |
| DBP (mmHg) | 3 min | 78.7 ± 8.3 | 72.8 ± 7.8 | 0.006 |
| DBP (mmHg) | 5 min | 75.9 ± 7.9 | 71.6 ± 7.5 | 0.035 |

The 1 µg/kg group demonstrated significantly lower systolic and diastolic blood pressure during the first 5 minutes following intubation, indicating stronger attenuation of the acute pressor response.

**Table 5. Adverse Haemodynamic Events**

| Adverse event | Group D0.5 (n=30) | Group D1 (n=30) | P value |
|---|---|---|---|
| Bradycardia | 1 (3.3%) | 4 (13.3%) | 0.353 |
| Hypotension | 1 (3.3%) | 3 (10.0%) | 0.612 |
| Hypertension after intubation | 6 (20.0%) | 1 (3.3%) | 0.103 |
| Desaturation | 0 | 0 | — |

Bradycardia and hypotension tended to occur more frequently with dexmedetomidine 1 µg/kg, whereas post-intubation hypertension was more frequent with 0.5 µg/kg. These differences did not reach statistical significance in this illustrative sample.

## DISCUSSION

The present comparative study evaluated the haemodynamic effects of two intravenous doses of dexmedetomidine administered before induction of general anaesthesia. Both 0.5 µg/kg and 1 µg/kg attenuated the cardiovascular response to direct laryngoscopy and endotracheal intubation, but the higher dose produced greater suppression of tachycardia and the pressor response during the immediate post-intubation period.

Laryngoscopy and tracheal intubation activate the sympathoadrenal system and may produce substantial increases in heart rate and arterial pressure.[1,2] The magnitude of this response depends on several factors, including the duration and

difficulty of laryngoscopy, depth of anaesthesia and underlying cardiovascular status. Prevention of excessive haemodynamic fluctuations is therefore an important objective of anaesthetic management, particularly in high-risk patients.

Dexmedetomidine decreases central sympathetic activity through highly selective α2-adrenoceptor agonism. The resulting reduction in norepinephrine release provides a physiological explanation for the lower heart rate and arterial pressure observed after dexmedetomidine administration.[4,5]

Kapoor et al.[5] evaluated a pre-induction dose of 0.5 µg/kg and demonstrated significant attenuation of post-intubation increases in HR, SBP, DBP and MAP compared with placebo. Their findings establish that even the lower dose used in the present study can provide clinically meaningful sympatholysis.

Sebastian et al.[6] compared dexmedetomidine 0.5 and 0.75 µg/kg with saline and observed significant attenuation of HR and arterial pressure responses with dexmedetomidine, with 0.75 µg/kg providing the greatest effect. They concluded that 0.75 µg/kg represented an effective dose for attenuation of the stress response.

The current findings are also consistent with the randomized double-blind study by Sulaiman et al.[7], who compared 0.5 and 1 µg/kg dexmedetomidine in patients undergoing elective cardiac surgery. They observed a greater incidence of post-intubation hypertension in the lower-dose group and concluded that 1 µg/kg was more effective in controlling the haemodynamic stress response. Importantly, clinically significant hypotension and bradycardia were not observed with the higher dose in that population.

However, not all investigations have demonstrated a clinically meaningful advantage with 1 µg/kg. Gunalan et al.[8] compared 0.5 and 1 µg/kg loading doses and reported that both doses effectively attenuated the haemodynamic response and improved intubating conditions. They noted a lower incidence of hypotension and bradycardia with 0.5 µg/kg and suggested that the lower dose may offer an advantageous efficacy-safety balance.

Similarly, comparative investigations involving α2 agonists have shown that although 1 µg/kg dexmedetomidine can produce profound suppression of the intubation response, the haemodynamic effect may be accompanied by an increased tendency toward bradycardia and hypotension.[9] Thus, greater sympatholysis does not automatically indicate that the higher dose is optimal for every patient.

In the present study, the greatest difference between the groups was observed during the first few minutes following intubation, when the sympathetic response would be expected to be maximal. Group D1 demonstrated significantly lower HR, SBP, DBP and MAP at 1 and 3 minutes. These observations support a dose-dependent sympatholytic action of dexmedetomidine.

Nevertheless, the higher dose also produced a greater numerical incidence of bradycardia and hypotension. This finding highlights the importance of individualized dose selection. In young, haemodynamically stable patients where strong suppression of the intubation response is desirable, 1 µg/kg may be advantageous. In elderly patients, hypovolaemic patients, individuals with conduction abnormalities or those receiving other negative chronotropic drugs, a lower dose may provide a better safety margin.

## CONCLUSION

Intravenous dexmedetomidine in doses of both 0.5 µg/kg and 1 µg/kg administered before induction of general anaesthesia is effective in attenuating the haemodynamic response associated with direct laryngoscopy and endotracheal intubation. Dexmedetomidine 1 µg/kg provides greater attenuation of the increases in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure, particularly during the first few minutes after tracheal intubation. However, the higher dose may be associated with a greater tendency toward bradycardia and hypotension. Therefore, 1 µg/kg may be preferred when maximal attenuation of the intubation response is required in haemodynamically stable patients, whereas 0.5 µg/kg may offer an appropriate efficacy-safety balance in patients susceptible to bradycardia or hypotension.

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