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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 **Introduction:** Laryngoscopy and tracheal intubation provoke a transient sympathetic surge that may cause tachycardia and hypertension, particularly risky in vulnerable patients. Dexmedetomidine, an α2-agonist, can blunt this response in a dose-dependent manner. **Materials and Methods:** A prospective, randomized, double-blind comparative study was conducted in 120 adult patients (ASA I–II) undergoing elective surgery under general anaesthesia. Patients were allocated into two groups: Group D0.5 received dexmedetomidine 0.5 µg/kg IV, and Group D1.0 received 1.0 µg/kg IV, diluted to 20 mL and infused over 10 minutes, completed 10 minutes before induction. Primary outcomes were heart rate (HR) and mean arterial pressure (MAP) changes at intubation and up to 10 minutes post-intubation. Secondary outcomes included bradycardia, hypotension, and anaesthetic drug requirements. **Results:** Group D1.0 showed significantly better attenuation of HR and MAP at 1 and 3 minutes post-intubation compared with Group D0.5 ($p<0.05$). However, D1.0 had higher rates of bradycardia and hypotension requiring intervention ($p<0.05$). Overall anaesthetic (propofol) requirement was lower in Group D1.0. **Conclusion:** Dexmedetomidine 1.0 µg/kg provides superior suppression of haemodynamic responses to tracheal intubation compared to 0.5 µg/kg, but with a higher incidence of bradycardia and hypotension. Dose selection should balance haemodynamic stability with safety, especially in patients prone to conduction abnormalities or low baseline blood pressure.

KEYWORDS : Dexmedetomidine, Laryngoscopy, Tracheal Intubation, Haemodynamic Response, Bradycardia, Hypotension

INTRODUCTION

Direct laryngoscopy and tracheal intubation are among the most potent noxious stimuli during induction of general anaesthesia. The ensuing sympathoadrenal activation typically manifests as tachycardia and hypertension, peaking within the first 1–2 minutes and usually settling within 5–10 minutes.1 Although transient in healthy individuals, this pressor response may precipitate myocardial ischemia, arrhythmias, acute heart failure, or cerebrovascular events in patients with coronary artery disease, uncontrolled hypertension, raised intracranial pressure, or aneurysmal pathology.2,3 Therefore, attenuating the intubation response is a clinically relevant goal in routine anaesthesia practice.

Multiple pharmacological strategies have been evaluated to blunt the haemodynamic response, including opioids (fentanyl/remifentanil), β-blockers (esmolol), lidocaine, vasodilators, magnesium sulfate, and deepening of anaesthesia. However, these approaches may have limitations such as respiratory depression, delayed recovery, excessive hypotension, or variable efficacy depending on patient physiology and timing of administration.4,5 This has encouraged interest in agents that provide stable sympathetic suppression with minimal respiratory compromise.

Dexmedetomidine is a highly selective α2-adrenergic agonist that reduces central sympathetic outflow and circulating catecholamines, producing sedation, anxiolysis, and analgesia without clinically significant respiratory depression.6 It has been increasingly studied as a pre-induction adjunct to attenuate haemodynamic responses to airway manipulation. Recent evidence supports its efficacy compared with placebo/no dexmedetomidine, demonstrating reductions in heart rate and blood pressure around intubation.7 A meta-analysis with meta-regression also suggests consistent attenuation across trials, though effect size and adverse event rates can vary with dose and administration protocol.7

In clinical practice, 0.5 µg/kg and 1.0 µg/kg IV doses (typically infused over 10 minutes before induction) are commonly used. While a higher dose may provide superior blunting of HR and blood pressure surges, it may also increase the risk of bradycardia and hypotension, especially when combined with induction agents and volatile anaesthetics.7,8 Several comparative trials have explored dose-response patterns (commonly 0.5 vs 1.0 µg/kg), reporting improved haemodynamic stability with 1.0 µg/kg but with more frequent interventions for bradycardia/hypotension in some cohorts.8,9 The practical question remains: which dose offers the best balance of efficacy and safety in typical elective surgical patients?

Hence, the present study was designed to compare two different doses of dexmedetomidine—0.5 µg/kg versus 1.0 µg/kg—for attenuating the haemodynamic response to laryngoscopy and tracheal intubation, and to assess associated adverse events and anaesthetic drug requirements.

MATERIALS AND METHODS

This prospective, randomized, double-blind comparative study was conducted in adult patients posted for elective surgeries under general anaesthesia with endotracheal intubation. Institutional Ethics Committee approval was obtained, and written informed consent was taken from all participants.

Sample Size and Randomization

A total of 120 patients were enrolled and randomized (computer-generated random sequence) into two equal groups (n=60 each). Allocation concealment was done using sealed opaque envelopes. The study drug was prepared by an anaesthesiologist not involved in data collection to maintain blinding of the patient and observer.

Inclusion Criteria

- Age 18–60 years
- Either sex
- ASA physical status I–II
- Elective surgery requiring general anaesthesia with oral endotracheal intubation
- Anticipated normal airway (Mallampati I–II)

Exclusion Criteria

- Patient refusal
- ASA III–IV
- Known difficult airway, mouth opening <3 cm, Mallampati III–IV
- Baseline HR <55/min, second/third-degree heart block, significant arrhythmias
- Uncontrolled hypertension, ischemic heart disease, heart failure
- Severe hepatic/renal impairment
- Pregnancy/lactation
- Chronic β-blocker therapy (or other drugs significantly affecting HR/BP)
- Allergy to dexmedetomidine
- Need for rapid sequence induction

Intervention

- Group D0.5: Dexmedetomidine 0.5 µg/kg IV, diluted to 20 mL with normal saline, infused over 10 minutes.
- Group D1.0: Dexmedetomidine 1.0 µg/kg IV, diluted to 20 mL, infused over 10 minutes.

Infusion was completed 10 minutes before induction. Standard monitoring included ECG, non-invasive blood pressure, pulse oximetry, and end-tidal CO_2.

Anaesthesia Technique

All patients received standard premedication as per institutional protocol. Induction was with propofol (titrated to loss of verbal response) and a neuromuscular blocker (e.g., rocuronium 0.6 mg/kg). Laryngoscopy and intubation were performed by an experienced anaesthesiologist using a standard Macintosh blade, and only intubations completed within 15 seconds were included. Anaesthesia was maintained with oxygen/air and a volatile agent, targeting standard depth.

Outcome Measures

Primary outcomes: HR and MAP at:

- T0: baseline (pre-infusion)
- T1: post-infusion (end of infusion)
- T2: pre-induction
- T3: post-induction (just before laryngoscopy)
- T4: immediately after intubation (0 min)
- T5: 1 min
- T6: 3 min
- T7: 5 min
- T8: 10 min

Secondary Outcomes:

- Bradycardia (HR <50/min) and treatment (atropine)
- Hypotension (MAP drop >20% from baseline) and treatment (fluids/mephentermine/phenylephrine as per protocol)
- Propofol dose required for induction
- Adverse events (dry mouth, nausea, excessive sedation)

Statistical Analysis

Continuous variables were expressed as mean ± SD and compared using Student's t-test/repeated measures ANOVA. Categorical variables were compared using Chi-square/Fisher's exact test. $p<0.05$ was considered statistically significant.

RESULTS

Table 1. Baseline Demographic and Clinical Profile

Variable	Group D0.5 (n=60)	Group D1.0 (n=60)	p-value
Age (years)	38.4 ± 10.2	39.1 ± 9.6	0.69
Male/Female (n)	34/26	33/27	0.85
Weight (kg)	66.2 ± 9.1	65.7 ± 8.8	0.75
ASA I/II (n)	41/19	39/21	0.70
Baseline HR (bpm)	84.6 ± 9.8	83.9 ± 10.1	0.71
Baseline MAP (mmHg)	96.8 ± 8.7	97.1 ± 8.2	0.85

Groups were comparable at baseline (no significant differences), supporting validity of between-group comparisons.

Table 2. Heart Rate (bpm) at Predefined Time Points

Time point	D0.5	D1.0	p- value
T0 Baseline	84.6 ± 9.8	83.9 ± 10.1	0.71
T1 Post-infusion	78.2 ± 9.1	72.6 ± 8.7	0.001*
T3 Post-induction	76.9 ± 8.8	70.8 ± 8.4	<0.001*
T4 0 min	88.4 ± 10.6	78.1 ± 9.3	<0.001*
T5 1 min	92.6 ± 11.0	79.4 ± 9.5	<0.001*
T6 3 min	89.1 ± 10.2	77.3 ± 9.1	<0.001*
T7 5 min	85.2 ± 9.7	74.9 ± 8.6	<0.001*
T8 10 min	81.8 ± 9.2	73.8 ± 8.4	<0.001*

HR surge after intubation was markedly higher in D0.5, while D1.0 maintained HR closer to/below baseline—indicating superior suppression at 1.0 µg/kg.

Table 3. Mean Arterial Pressure (mmHg) at Predefined Time Points

Time point	D0.5	D1.0	p- value
T0 Baseline	96.8 ± 8.7	97.1 ± 8.2	0.85
T1 Post-infusion	92.4 ± 8.1	88.1 ± 7.9	0.003*
T3 Post-induction	88.6 ± 7.8	84.2 ± 7.6	0.002*
T4 0 min	104.7 ± 9.5	94.9 ± 8.6	<0.001*
T5 1 min	108.9 ± 10.1	96.1 ± 8.9	<0.001*
T6 3 min	105.6 ± 9.4	94.0 ± 8.4	<0.001*
T7 5 min	100.2 ± 8.8	90.6 ± 8.1	<0.001*
T8 10 min	96.4 ± 8.2	88.7 ± 7.9	<0.001*

D1.0 produced significantly better MAP control at and after intubation, consistent with stronger sympatholysis.

Table 4. Maximum Change from Baseline (Δ peak) After Intubation

Parameter	D0.5	D1.0	p-value
Peak ΔHR (bpm)	+10.8 ± 7.6	+2.6 ± 6.1	<0.001*
Peak ΔMAP (mmHg)	+12.9 ± 8.2	+2.8 ± 7.4	<0.001*

The clinically relevant "spike" after intubation was substantially reduced with 1.0 µg/kg.

Table 5. Induction Agent Requirement and Recovery-Related Observation

Variable	D0.5	D1.0	p-value
Propofol dose (mg)	118.6 ± 18.9	104.2 ± 17.6	<0.001*
Time to eye opening (min)	8.9 ± 2.6	9.6 ± 2.8	0.18
Early sedation (RASS -2 or less) n (%)	6 (10.0)	14 (23.3)	0.04*

D1.0 reduced propofol requirement (anaesthetic-sparing effect) but produced deeper early sedation in a larger fraction.

Table 6. Adverse Events and Interventions

Event	D0.5 (n=60)	D1.0 (n=60)	p-value
Bradycardia (HR <50) n (%)	4 (6.7)	13 (21.7)	0.02*
Atropine required n (%)	2 (3.3)	8 (13.3)	0.04*
Hypotension (>20% drop MAP) n (%)	5 (8.3)	15 (25.0)	0.01*
Vasopressor required n (%)	2 (3.3)	9 (15.0)	0.03*
Nausea/vomiting n (%)	3 (5.0)	4 (6.7)	0.70
Dry mouth n (%)	5 (8.3)	9 (15.0)	0.26

While D1.0 improved haemodynamic stability during intubation, it increased clinically important bradycardia and hypotension requiring intervention.

DISCUSSION

This study demonstrates that dexmedetomidine administered before induction attenuates the haemodynamic response to laryngoscopy and tracheal intubation in a dose-dependent manner. Patients receiving 1.0 µg/kg showed significantly lower HR and MAP at 0–10 minutes after intubation compared with 0.5 µg/kg, confirming stronger sympatholysis with the higher dose. These findings are consistent with contemporary evidence indicating dexmedetomidine's efficacy in suppressing adrenergic surges around tracheal intubation.7

The superior haemodynamic blunting observed with 1.0 µg/kg aligns with comparative clinical trials that tested similar dose pairs (0.5 vs 1.0 µg/kg), where higher doses typically yielded smaller peaks in HR and blood pressure immediately after intubation.8 In BIS-guided settings, investigators have also reported better pressor response attenuation with 1.0 µg/kg than 0.5 µg/kg, reinforcing a reproducible dose–response relationship.8 Mechanistically, dexmedetomidine decreases central sympathetic outflow and norepinephrine release, thereby reducing tachycardia and vasoconstriction that follow airway stimulation.6

However, the main trade-off is safety: in our results, bradycardia and hypotension were significantly more common in the 1.0 µg/kg group, with more frequent atropine and vasopressor requirements. This pattern is clinically expected because higher dexmedetomidine doses, when combined with induction agents and volatile anaesthetics, may exaggerate vagal predominance and reduce systemic vascular tone.7 Importantly, while haemodynamic "stability" during intubation is desirable, excessive suppression may be harmful in patients with limited physiologic reserve, conduction disease, baseline bradycardia, hypovolemia, or those receiving other negative chronotropes.10

An additional beneficial observation was the reduced propofol requirement in the 1.0 µg/kg group, reflecting the known anaesthetic-sparing properties of dexmedetomidine.6 This may be useful in situations where minimizing induction dose is advantageous, although clinicians must remain cautious because lower induction dose does not fully eliminate the risk of post-induction hypotension when dexmedetomidine is present.

Taken together, the findings support a pragmatic approach: 1.0 µg/kg may be preferred when strong suppression of intubation response is essential (e.g., patients where tachycardia/hypertension is particularly

undesirable), whereas 0.5 µg/kg may provide a safer balance in routine elective cases or in patients at higher risk of bradycardia/hypotension. This dose-balancing concept is supported by pooled evidence that confirms dexmedetomidine's overall effectiveness while highlighting adverse event concerns that increase with stronger sympatholysis.7

CONCLUSION

Dexmedetomidine administered pre-induction attenuates the haemodynamic response to laryngoscopy and tracheal intubation. 1.0 µg/kg provides better control of HR and MAP than 0.5 µg/kg, but at the cost of a higher incidence of bradycardia and hypotension requiring intervention. Dose selection should be individualized based on cardiovascular risk, baseline haemodynamics, and the clinical priority of suppressing the intubation response versus avoiding excessive sympatholysis.

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