Section: Anaesthesiology

Original Research Article

INTRAVENOUS DEXMEDETOMIDINE VERSUS LABETALOL IN ATTENUATING HAEMODYNAMIC PRESSOR RESPONSE TO LARYNGOSCOPY AND TRACHEAL INTUBATION IN PATIENTS UNDERGOING OTORHINOLARYNGOLOGY SURGERIES UNDER GENERAL ANAESTHESIA: A PROSPECTIVE RANDOMIZED CONTROLLED TRIAL

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Received : 16/07/2026
Received in revised form : 12/08/2026
Accepted : 25/08/2026

Keywords:
Dexmedetomidine; Laryngoscopy; Labetalol; Hemodynamics; Otorhinolaryngologic Surgical Procedures.

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DOI: 10.47009/jamp.2026.8.5.178

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2026; 8 (5); 1122-1126

ABSTRACT

Background: The aim is to compare the efficacy and safety of intravenous dexmedetomidine (0.5 µg/kg) and labetalol (0.25 mg/kg) in attenuating the haemodynamic pressor response to laryngoscopy and tracheal intubation in patients undergoing elective otorhinolaryngology surgeries under general anaesthesia. **Materials and Methods:** A prospective, randomized, placebo-controlled trial enrolled 162 ASA I–II adult patients (18–60 years) undergoing elective ENT surgery. Patients were randomized equally into three groups (n = 54 each): Group A received dexmedetomidine 0.5 µg/kg IV; Group B received labetalol 0.25 mg/kg IV; and Group C received normal saline (control), each infused over 10 minutes before induction. Anaesthesia was standardized with propofol, fentanyl, and vecuronium. Heart rate (HR) and mean arterial pressure (MAP) were recorded at baseline and at 1, 3, 5, 10, 30, 60, 90, and 120 minutes post-intubation. Adverse effects were monitored throughout. **Result:** At 1 minute post-intubation, HR was 72, 81, and 94 bpm, and MAP was 87, 96, and 110 mmHg in Groups A, B, and C, respectively ($p < 0.001$ for both). Dexmedetomidine provided superior intraoperative haemodynamic stability (mean HR 72.8 ± 4.6 bpm; SBP 112.4 ± 6.9 mmHg). Bradycardia was more frequent in the labetalol group (18.5%) compared to dexmedetomidine (7.4%) and control (3.7%) ($p = 0.02$). No serious adverse events were recorded. **Conclusion:** Intravenous dexmedetomidine 0.5 µg/kg provides superior attenuation of haemodynamic pressor response with a more favourable safety profile compared to labetalol 0.25 mg/kg in patients undergoing ENT surgery under general anaesthesia.

INTRODUCTION

Laryngoscopy and tracheal intubation are among the most intense nociceptive stimuli encountered during the induction of general anaesthesia. Mechanical stimulation of the supraglottic and infraglottic structures activates a neurohormonal reflex arc via the glossopharyngeal and vagus nerves, triggering sympathoadrenal outflow with acute surges of circulating catecholamines. The resultant haemodynamic pressor response — tachycardia, systolic hypertension, and a rise in mean arterial pressure (MAP) — typically peaks within 1–2 minutes of intubation and normalizes within 5–10 minutes in physiologically intact patients.[1,2]

Although transient, this pressor response poses serious risk in patients with limited cardiovascular or cerebrovascular reserve. Acute tachycardia and hypertension increase myocardial oxygen demand, predispose to ischaemia, arrhythmia, and ventricular failure, and may worsen intracranial or intraocular hypertension.[1] Patients with coronary artery disease, hypertension, cerebrovascular pathology, head injury, intracranial tumours, or glaucoma are particularly vulnerable.

Otorhinolaryngology (ENT) surgeries present additional haemodynamic challenges. Operating in

ISSN (O): 2687-5365; ISSN (P): 2753-6556

highly vascular territories — nasal mucosa, nasopharynx, oropharynx — even modest hypertension amplifies intraoperative bleeding, compromises surgical field visibility, and prolongs operative time. Controlled hypotension is, therefore, a central anaesthetic goal in ENT practice, rendering precise control of the intubation pressor response especially important.[3-9]

Various pharmacological strategies have been employed, including opioids, beta-blockers, alpha-2 adrenergic agonists, calcium channel blockers, vasodilators, and lignocaine, each with specific limitations.[10-16] Dexmedetomidine — a highly selective α_2-adrenergic receptor agonist (α_2:α_1 selectivity ≈ 1600:1) — exerts central sympatholysis with concomitant sedation, analgesia, and anxiolysis and with minimal respiratory depression, making it pharmacologically well suited for attenuating haemodynamic stress responses.[12] Labetalol, a combined α_1- and non-selective β-adrenergic antagonist, reduces systemic vascular resistance and controls heart rate without significant reflex tachycardia, and has been used effectively in perioperative hypertensive crises.[3]

Despite individual evidence supporting each agent, direct comparative trials at the lower, safer doses advocated for ENT procedures — dexmedetomidine 0.5 µg/kg and labetalol 0.25 mg/kg — remain scarce. This trial was designed to evaluate and compare the efficacy and safety of these two low-dose regimens in attenuating the haemodynamic pressor response to laryngoscopy and tracheal intubation in patients scheduled for elective otorhinolaryngology surgery under general anaesthesia.

MATERIALS AND METHODS

Study design and setting: A prospective, randomized, double-blind, placebo-controlled, parallel-group trial was conducted in the Department of Anaesthesiology, Aarupadai Veedu Medical College and Hospital (AVMCH), Puducherry, India, over 18 months. The study was approved by the Institutional Human Ethics Committee (AV/IHEC/01/2024/019) and was registered with the Clinical Trials Registry – India (CTRI/2025/04/085181). All participants provided written informed consent. The study was conducted in accordance with the Declaration of Helsinki (2013 revision).

Participants: Adult patients aged 18–60 years with ASA Physical Status I or II, Modified Mallampati Grade I or II, and BMI ≤ 30 kg/m², scheduled for elective ENT surgery (anticipated duration 120–180 minutes) under general anaesthesia, were eligible for inclusion.

Exclusion criteria were: documented allergy to dexmedetomidine or labetalol; pre-existing hypertension, cardiac arrhythmia, conduction abnormalities, or severe systemic comorbidities; anticipated difficult airway; concurrent psychiatric or cardiovascular pharmacotherapy; pregnancy; and refusal of consent.

Randomization and blinding: Patients were randomized in a 1:1:1 ratio using computer-generated block randomization (block size 6). Allocation was concealed in sequentially numbered, sealed, opaque envelopes opened by an independent nurse immediately before drug preparation. Study drugs and saline were prepared in identical 20 mL syringes. The attending anaesthesiologist and outcome assessor were blinded to group allocation throughout the study period.

Interventions: All patients received standardized premedication: glycopyrrolate 0.2 mg IV, midazolam 2 mg IV, and ondansetron 4 mg IV. Pre-oxygenation with 100% O_2 was administered for 3 minutes. Commencing 10 minutes before induction, the allocated study drug was infused via a syringe pump:

- Group A (Dexmedetomidine): 0.5 µg/kg IV in 20 mL normal saline over 10 minutes
- Group B (Labetalol): 0.25 mg/kg IV in 20 mL normal saline over 10 minutes
- Group C (Control): 20 mL normal saline over 10 minutes

Anaesthesia was induced with propofol 2 mg/kg IV and fentanyl 2 µg/kg IV, followed by vecuronium 0.1 mg/kg IV. Direct laryngoscopy and orotracheal intubation were performed by a single senior anaesthesiologist with more than five years of experience. Anaesthesia was maintained with isoflurane 1% in 33% O_2 / 66% N_2O via volume-controlled ventilation (tidal volume 6–8 mL/kg; respiratory rate 14–18 breaths/minute). Incremental vecuronium 0.02 mg/kg IV was used for muscle relaxation. Reversal was achieved with neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg IV at the end of surgery.

Outcome measures: The primary outcome was attenuation of the haemodynamic pressor response, defined as HR and MAP at 1 minute post-intubation. Secondary outcomes included intraoperative haemodynamic stability (mean HR, SBP, and MAP averaged over the surgical period), provision of controlled hypotension, and the incidence of adverse effects — bradycardia (HR < 50 bpm), hypotension (MAP < 60 mmHg or > 20% reduction from baseline), and requirement for rescue medications.

Haemodynamic parameters (HR, SBP, DBP, MAP) were recorded at baseline, and at 1, 3, 5, 10, 30, 60, 90, and 120 minutes post-intubation using a calibrated multiparameter monitor (Philips IntelliVue MX800).

Sample size: Based on data from Bhatt et al.,4 assuming a mean difference in MAP of 8 mmHg between groups (SD = 10 mmHg), at $\alpha = 0.05$ (two-tailed) and 80% power, 48 patients per group were required. Accounting for a projected 12% attrition rate, 54 patients per group (162 total) were enrolled.

Statistical analysis: Continuous data are presented as mean ± standard deviation (SD). Intergroup differences were assessed by one-way ANOVA for normally distributed variables and the Kruskal–

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Wallis test for non-parametric data; post-hoc pairwise comparisons were performed with Tukey's HSD test. Categorical variables were analysed by the chi-square or Fisher's exact test as appropriate. Statistical significance was defined as $p < 0.05$. All analyses were conducted with SPSS version 29.0 (IBM Corp., Armonk, NY, USA).

RESULTS

Participant characteristics

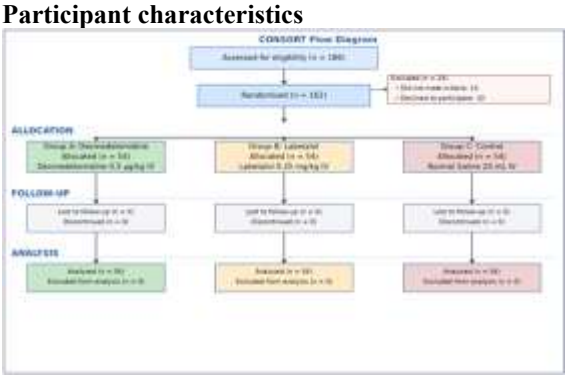


Figure 1: CONSORT flow diagram showing participant enrolment, allocation, follow-up, and analysis across the three study groups.

A total of 162 patients were enrolled and randomized (54 per group). All patients completed the study protocol; no withdrawals, protocol deviations, or intraoperative adverse events necessitating exclusion occurred. The three groups were comparable in baseline demographic and clinical characteristics, with no statistically significant differences (Table 1).

Primary outcome — Pressor response at 1 minute post-intubation

At 1 minute post-intubation, the dexmedetomidine group demonstrated the smallest increase in HR (76 → 72 bpm; net change −4 bpm) compared to the labetalol group (77 → 81 bpm; +4 bpm) and the control group (78 → 94 bpm; +16 bpm) ($p < 0.001$). MAP was best preserved with dexmedetomidine (94 → 87 mmHg; −7 mmHg) versus labetalol (95 → 96 mmHg; +1 mmHg) and control (96 → 110 mmHg; +14 mmHg) ($p < 0.001$). Intergroup differences remained statistically significant at 3 and 5 minutes post-intubation ($p < 0.001$) and at 10 minutes ($p = 0.03–0.04$), normalizing by 30 minutes in all groups. Full haemodynamic profiles are presented in [Table 2].

Table 1: Baseline Demographic And Clinical Characteristics Of Study Participants

Variable	Group A: Dexmedetomidine (n=54)	Group B: Labetalol (n=54)	Group C: Control (n=54)
Age (years), mean ± SD	38.6 ± 9.2	39.1 ± 8.7	37.9 ± 9.5
Weight (kg), mean ± SD	62.4 ± 7.8	63.1 ± 8.1	61.8 ± 7.9
Height (cm), mean ± SD	163.2 ± 7.1	164.0 ± 6.8	162.8 ± 7.4
BMI (kg/m²), mean ± SD	23.4 ± 2.6	23.7 ± 2.4	23.2 ± 2.8
Sex (Male/Female), n	27 / 27	28 / 26	28 / 26
ASA I / II, n (%)	38 (70%) / 16 (30%)	37 (68%) / 17 (32%)	39 (72%) / 15 (28%)
Mallampati Grade I / II, n	44 (81%) / 10 (19%)	43 (80%) / 11 (20%)	45 (83%) / 9 (17%)
Baseline HR (bpm)	76 ± 3.1	77 ± 2.9	78 ± 3.4
Baseline MAP (mmHg)	94 ± 4.2	95 ± 4.6	96 ± 4.3
p-value (ANOVA)	> 0.05 (NS)	> 0.05 (NS)	> 0.05 (NS)

Note. NS = not significant. HR = heart rate; MAP = mean arterial pressure; SD = standard deviation; BMI = body mass index; ASA = American Society of Anesthesiologists physical status classification.

Table 2: Comparison Of Heart Rate (bpm) And Mean Arterial Pressure (mmHg) At Key Time Points

Time Interval	HR: Group A	HR: Group B	HR: Group C	MAP: Group A	MAP: Group B	MAP: Group C
Baseline	76	77	78	94	95	96
1 min†	72	81	94	87	96	110
3 min†	73	79	89	89	97	105
5 min†	75	77	85	91	96	101
10 min*	76	76	81	93	95	98
30 min	74	76	80	92	94	96
60 min	75	75	78	93	94	95

Note. † $p < 0.001$; * $p < 0.05$ — intergroup comparison (one-way ANOVA, Tukey post-hoc). HR = heart rate; MAP = mean arterial pressure. Group A = dexmedetomidine; Group B = labetalol; Group C = control (normal saline).

Secondary outcomes — Intraoperative haemodynamic stability

Throughout the intraoperative period, dexmedetomidine maintained significantly lower mean HR, SBP, and MAP compared to labetalol and control groups ($p < 0.001$ for HR and SBP; $p = 0.01$ for MAP). Labetalol demonstrated moderate haemodynamic stability but with greater fluctuation, particularly in HR. These data are summarized in [Table 3].

Table 3: Mean Intraoperative Haemodynamic Parameters Across Study Groups

Parameter (mean ± SD)	Group A: Dexmedetomidine	Group B: Labetalol	Group C: Control	p-value
Heart Rate (bpm)	72.8 ± 4.6	78.9 ± 5.8	86.7 ± 7.4	< 0.001
Systolic Blood Pressure (mmHg)	112.4 ± 6.9	118.6 ± 8.1	126.8 ± 9.5	< 0.001

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Mean Arterial Pressure (mmHg)	88.6 ± 4.2	93.8 ± 6.1	99.4 ± 8.3	0.01

Note. SD = standard deviation.

Adverse effects: The incidence of adverse effects is shown in Table 4. Bradycardia occurred significantly more frequently in the labetalol group (18.5%) compared with dexmedetomidine (7.4%) and control (3.7%) (p = 0.02). Hypotension was also more common in the labetalol group (14.8%) than in the dexmedetomidine (9.2%) and control (5.5%) groups. All adverse events in the dexmedetomidine group were transient and self-limiting; no patient in this group required vasopressor administration. No life-threatening complications or deaths occurred in any group.

Table 4: Incidence Of Adverse Effects Among Study Groups

Adverse Effect	Group A: Dexmedetomidine n (%)	Group B: Labetalol n (%)	Group C: Control n (%)	p-value
Bradycardia (HR < 50 bpm)	4 (7.4%)	10 (18.5%)	2 (3.7%)	0.02
Hypotension (MAP fall > 20%)	5 (9.2%)	8 (14.8%)	3 (5.5%)	0.08
Atropine required	3 (5.6%)	1 (1.9%)	2 (3.7%)	0.54
Vasopressor (mephentermine) required	0 (0.0%)	5 (9.3%)	2 (3.7%)	0.02

Note. Values are expressed as n (%). Statistical tests: chi-square / Fisher's exact test as appropriate. HR = heart rate; MAP = mean arterial pressure.

DISCUSSION

This prospective randomized controlled trial compared low-dose intravenous dexmedetomidine (0.5 µg/kg) and labetalol (0.25 mg/kg) against placebo in 162 patients undergoing elective ENT surgery under general anaesthesia. The principal finding is that dexmedetomidine provides significantly superior attenuation of the haemodynamic pressor response at the critical 1-minute post-intubation interval, with a more favourable adverse-effect profile, when compared to both labetalol and control.

The three study groups were demographically comparable at baseline, validating the randomization process and ensuring that observed haemodynamic differences are attributable to the pharmacological interventions rather than to confounding patient-related variables. This aligns with the methodological approach of Bhatt et al,[4] whose study also demonstrated comparable baseline characteristics and served as the reference for our sample size calculation.

The superior HR attenuation with dexmedetomidine is mechanistically explicable through its central action at α_2-adrenoreceptors in the locus coeruleus and brainstem autonomic centres, where it inhibits norepinephrine release and attenuates efferent sympathetic outflow — upstream suppression at the neuroendocrine source.[12] This central mechanism produces consistent, sustained haemodynamic blunting across all post-intubation time points. In contrast, labetalol acts peripherally through α_1- and β-receptor blockade, reducing vascular resistance and cardiac output but leaving the central catecholamine surge unmodulated. The labetalol group, therefore, exhibited a higher residual HR response. These findings are consistent with reports by Bhatt et al,[4] El-Shmaa et al,[17] Reddy and Madhu,[15] and the multicentre comparative trial by Kunisawa et al.[19]

Dexmedetomidine also maintained MAP values closest to baseline across all post-intubation time points. This is attributable to the same central sympatholytic mechanism supplemented by peripheral vasomodulation. Superior MAP control with dexmedetomidine has been demonstrated in earlier studies including those by Talke et al,[10] and Bajwa et al,[12] the latter specifically conducted in ENT populations — the same setting as the present trial. In ENT surgery, MAP control in the range of 65–90 mmHg is associated with reduced mucosal bleeding, improved endoscopic visibility, and shorter operative time,[9] the mean intraoperative MAP of 88.6 ± 4.2 mmHg in the dexmedetomidine group is well aligned with these clinical targets.

The safety profile further distinguishes dexmedetomidine from labetalol. The higher incidence of bradycardia (18.5% vs. 7.4%; p = 0.02) and vasopressor requirement (9.3% vs. 0%; p = 0.02) in the labetalol group reflects the combined negative chronotropic effects of β_1-blockade superimposed on vagal rebound during laryngoscopy, an effect amplified when the reflex tachycardia that normally buffers α_1-mediated vasodilation is suppressed by concurrent β_2-blockade. Singla et al,[6] and Kunisawa et al,[19] reported similar trends. Dexmedetomidine-related bradycardia was mild, transient, and self-limiting, consistent with the expected physiological response to reduced sympathetic tone rather than direct sinoatrial node depression — a distinction validated by Santhosh et al,[8] who confirmed the superior safety profile of the 0.5 µg/kg dose versus higher loading doses.

Labetalol retains clinical value in specific perioperative scenarios — particularly where rapid blood pressure reduction via peripheral adrenergic blockade is advantageous, in patients in whom α_2-agonists are contraindicated, or where cost constrains drug selection. However, for the ENT setting, its higher rate of clinically significant bradycardia and vasopressor requirement represents a meaningful safety disadvantage when compared to low-dose dexmedetomidine.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Limitations: This study was conducted at a single tertiary-care centre, limiting external generalizability. The study population was restricted to ASA I–II patients, excluding those with the highest cardiovascular risk. Biochemical markers of sympathoadrenal activation — plasma catecholamines, cortisol — were not assayed, precluding objective correlation between haemodynamic observations and neuroendocrine responses. Only fixed doses of each drug were examined, so dose–response relationships cannot be inferred. Postoperative outcomes including analgesia requirements, sedation scores, and time to readiness for discharge were not assessed.

CONCLUSION

Intravenous dexmedetomidine 0.5 µg/kg, administered as a 10-minute infusion before anaesthetic induction, provides significantly superior and safer attenuation of the haemodynamic pressor response to laryngoscopy and tracheal intubation compared to labetalol 0.25 mg/kg in patients undergoing elective otorhinolaryngology surgery under general anaesthesia. Its central sympatholytic mechanism, combined with analgesic and sedative properties and a favourable adverse-effect profile, supports its use as the preferred pharmacological agent for haemodynamic management in ENT surgery. Multicentre trials evaluating biochemical stress markers, dose–response relationships, and postoperative recovery profiles are warranted to further consolidate these findings.

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ISSN (O): 2687-5365; ISSN (P): 2753-6556