Original Article

Low-Dose Dexmedetomidine (0.5 µg/Kg) With 1 MAC Sevoflurane for Attenuation of the Haemodynamic Response to Laryngoscopy and Tracheal Intubation: A Randomised, Double-Blind, Placebo-Controlled Trial

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Abstract

Background: Laryngoscopy and tracheal intubation provoke a sympathetic pressor response that can harm patients with limited cardiovascular reserve. Higher doses of dexmedetomidine attenuate this response but cause bradycardia and hypotension. We evaluated a low dose of dexmedetomidine combined with 1 MAC sevoflurane. **Material and Methods:** In this randomized, double-blind, placebo-controlled trial, 50 ASA I adults (18–60 years) scheduled for elective surgery under general Anesthesia received either dexmedetomidine 0.5 µg/kg in 100 mL normal saline (group DEX, n = 25) or 100 mL normal saline (group NS, n = 25) over 10 minutes before induction. Anesthesia was induced with morphine and thiopentone, and maintained with 2% sevoflurane (1 MAC) in 66% nitrous oxide. Heart rate (HR), systolic (SBP), diastolic (DBP) and mean arterial pressure (MAP) were recorded up to 45 minutes after intubation and for 3 hours postoperatively. **Results:** Groups were comparable at baseline. One minute after intubation, group NS showed significant rises in SBP (133.52 ± 13.54 vs 110.32 ± 17.36 mmHg), DBP (88.44 ± 13.89 vs 70.00 ± 15.64 mmHg) and MAP (104.96 ± 11.78 vs 85.08 ± 15.60 mmHg) compared with group DEX (all $p < 0.001$). HR was significantly lower in group DEX at 3 minutes (78.92 ± 14.04 vs 92.40 ± 13.09 bpm; $p < 0.001$). Bradycardia (36% vs 4%; $p = 0.005$) and hypotension (60% vs 28%; $p = 0.023$) were more frequent in group DEX; all episodes responded to standard treatment. **Conclusion:** Dexmedetomidine 0.5 µg/kg with 1 MAC sevoflurane effectively attenuates the hemodynamic response to laryngoscopy and intubation, at the cost of manageable bradycardia and hypotension.

Keywords: Dexmedetomidine, Sevoflurane, Laryngoscopy, Intubation, Intratracheal, Hemodynamics, Blood Pressure, Heart Rate.

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Introduction

Direct laryngoscopy and tracheal intubation are noxious stimuli that trigger a reflex sympathoadrenal discharge, producing tachycardia and hypertension within seconds.[1] This pressor response is accompanied by a rise in plasma noradrenaline.[2,3] Its magnitude depends on the force applied by the laryngoscope blade, the duration of laryngoscopy and the depth of Anesthesia.[3,4] Healthy patients tolerate these transient changes, but in patients with coronary artery disease, hypertension or raised intracranial pressure they may precipitate myocardial ischemia, arrhythmias, pulmonary oedema or cerebral hemorrhage.[1]

Many techniques have been used to blunt this response, including modified laryngoscope blades,[5] and drugs such as clonidine,[6] nalbuphine,[7] gabapentin,[8] lignocaine,[9] and esmolol.[10] Dexmedetomidine, a highly selective α2-adrenoceptor agonist with sympatholytic, sedative, anxiolytic and analgesic properties,[11] is an attractive option. A preinduction bolus of 2 µg/kg attenuated the intubation response but caused hypotension and bradycardia in about 20% of patients.[12] Infusions of 1 µg/kg were also effective, yet hypotension, bradycardia and postoperative sedation persisted.[13,14] Because these adverse effects are dose dependent, interest has turned to lower doses.

Volatile an aesthetics also attenuate the pressor response. Sevoflurane at 3–6% suppresses sympathetic activity but causes myocardial depression, hypotension and bradycardia at these concentrations.[15] Conversely, 1 MAC sevoflurane alone does not adequately prevent the response to intubation.[16]

In an earlier unpublished study at our centre, dexmedetomidine 0.5 µg/kg combined with 2 MAC (4%) sevoflurane controlled the intubation response but caused hypotension in 76% of patients. We therefore hypothesized that the same low dose of dexmedetomidine combined with 1 MAC (2%) sevoflurane would attenuate the pressor response with fewer hemodynamic

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adverse effects. To our knowledge, this combination after thiopentone induction has not been evaluated previously.

The primary objective was to compare systolic blood pressure (SBP) after laryngoscopy and tracheal intubation between patients receiving dexmedetomidine 0.5 µg/kg and those receiving placebo, under 1 MAC sevoflurane. Secondary objectives were to compare heart rate (HR), diastolic blood pressure (DBP), mean arterial pressure (MAP), thiopentone requirement, the incidence of hypotension and bradycardia, postoperative sedation, pain and other adverse effects.

Materials and Methods

Study design and ethics: This prospective, randomized, double-blind, placebo-controlled trial was conducted in the Department of Anesthesiology, Critical Care and Pain Medicine at a tertiary care hospital North India. The Institutional Ethics Committee – Human Research approved the protocol The trial was registered Written informed consent was obtained from all participants. The study followed the Declaration of Helsinki and is reported according to the CONSORT 2010 statement.

Participants: We enrolled adults of either sex aged 18–60 years, of American Society of Anesthesiologists (ASA) physical status I and Mallampati grade I, scheduled for elective surgery under general Anesthesia with tracheal intubation. Exclusion criteria were an anticipated difficult airway; cardiovascular, renal, hepatic, neurological or endocrine disease; pregnancy or lactation; and use of drugs affecting cardiovascular variables (e.g. calcium-channel blockers, β-blockers, α-blockers or magnesium sulphate). Patients needing more than one laryngoscopy attempt or more than 15 seconds for laryngoscopy and intubation were to be withdrawn.

Randomization and blinding: On the day of surgery, patients were allocated 1:1 to group DEX or group NS using a computer-generated random number table. An anesthesiologist not otherwise involved in the study prepared the study solution in an identical 100 mL bag. Patients, the attending anesthesiologist and the outcome assessor were unaware of group allocation.

Interventions: In the operating room, electrocardiography, non-invasive blood pressure and pulse oximetry were applied and baseline values recorded. After intravenous access, Ringer's lactate was started. Group DEX received dexmedetomidine 0.5 µg/kg diluted in 100 mL 0.9% saline, and group NS received 100 mL 0.9% saline, each infused over 10 minutes immediately before induction.

Anesthesia was induced with morphine 0.1 mg/kg and 2.5% thiopentone titrated to loss of eyelash reflex; the dose was recorded. Vecuronium 0.1 mg/kg was given, and patients were ventilated by mask with 66% nitrous oxide in oxygen and 2% sevoflurane (dial setting, approximately 1 MAC) via a Bain circuit. Once train-of-four monitoring confirmed complete neuromuscular block, an anesthesiologist with at least 3 years' experience performed laryngoscopy with an adult Macintosh blade and inserted a cuffed oral tracheal tube. The duration of laryngoscopy and intubation was recorded in seconds.

Anesthesia was maintained with 66% nitrous oxide in oxygen and sevoflurane, which was titrated to keep SBP within ±20% of baseline. Ventilation was adjusted to an end-tidal carbon dioxide of 35 ± 5 mmHg. Neuromuscular block was supplemented with vecuronium 0.025 mg/kg guided by train-of-four and reversed with neostigmine 0.05 mg/kg and glycopyrrolate 0.005 mg/kg.

Hypotension was defined as a fall in SBP of 30% or more from baseline or SBP below 90 mmHg. It was treated by increasing the fluid infusion rate and reducing sevoflurane, followed by mephentermine 6 mg IV if unresponsive. Bradycardia (HR below 60 bpm) was treated with atropine 0.3 mg IV, repeated as needed.

Outcomes and measurements

The primary outcome was SBP after laryngoscopy and intubation. Secondary outcomes were HR, DBP and MAP; thiopentone dose; delivered sevoflurane concentration; the incidence of hypotension and bradycardia; postoperative sedation (Ramsay score),[17] pain on a 0–10 visual analogue scale (VAS); and adverse effects including postoperative nausea and vomiting (PONV), dry mouth and shivering.

HR, SBP, DBP, MAP and SpO_2 were recorded before the study infusion (baseline), at 5 and 10 minutes of infusion, before induction, immediately before intubation, at 1, 3, 5, 7, 10 and 15 minutes after intubation, and every 15 minutes thereafter. As surgical duration varied, intraoperative data were analyzed up to 45 minutes after intubation. Postoperatively, hemodynamic variables, Ramsay score and VAS were recorded hourly for 3 hours. A VAS of 3 or more was treated with diclofenac 75 mg IV and PONV with ondansetron 4 mg IV.

Sample size: Based on an SD of SBP of 9.98 and 11.19 mmHg at 3 minutes after intubation in our earlier unpublished study, 25 patients per group were needed to detect a 10 mmHg difference in SBP with a two-sided α of 0.05 and 90% power.

Statistical analysis: Continuous data are presented as mean ± standard deviation (SD) and categorical data as number (%). Between-group comparisons used the unpaired Student's t-test. Within-group changes over time were analyzed by repeated-measures analysis of variance with Tukey's post hoc test, against baseline and pre-induction values. Categorical variables were compared with the χ^2 test. A two-sided $p < 0.05$ was considered significant. For between-group comparisons of hemodynamic variables across the 17 time points, a conservative threshold of $p < 0.001$ was applied to limit type I error from multiple testing.

Results

Fifty patients were randomized, 25 to each group; all received the allocated intervention and were analyzed. No patient required more than one intubation attempt or more than 15 seconds for intubation, and none was withdrawn.

Baseline and perioperative characteristics: The groups were comparable in age, weight, body mass index, duration of surgery, thiopentone dose, duration of laryngoscopy and intravenous fluid volume (Table 1). Dexmedetomidine 0.5 µg/kg did not reduce the thiopentone dose needed for loss of eyelash reflex (4.97 ± 1.04 vs 4.71 ± 0.54 mg/kg; $p = 0.280$).

Table 1: Baseline and perioperative characteristics

Characteristic	Group NS (n = 25)	Group DEX (n = 25)	p-value
Age (years)	32.36 ± 10.55	32.88 ± 10.01	0.859
Male : female ratio	1 : 5	1 : 2	–
Body weight (kg)	51.92 ± 8.48	49.92 ± 9.10	0.426
BMI (kg/m²)	22.44 ± 2.36	20.64 ± 4.25	0.070
Duration of surgery (min)	103.80 ± 42.41	115.20 ± 32.16	0.290
Thiopentone dose (mg)	243.00 ± 38.54	243.00 ± 52.30	1.000
Thiopentone dose (mg/kg)	4.71 ± 0.54	4.97 ± 1.04	0.280
Duration of laryngoscopy and intubation (s)	8.24 ± 1.98	8.60 ± 1.53	0.476
Intravenous fluids (mL)	1536.00 ± 495.72	1614.00 ± 631.72	0.629

Values are mean ± SD or ratio. NS, normal saline; DEX, dexmedetomidine; BMI, body mass index.

Hemodynamic response: Baseline HR, SBP, DBP and MAP did not differ between groups (Table 2). During the study infusion, HR was unchanged in both groups. SBP fell significantly from baseline at 10 minutes of infusion and before induction in both groups, and DBP fell significantly in group DEX only; between-group differences were not significant. After induction, all variables decreased significantly from pre-induction values in both groups, without differences between-group.

One minute after intubation, SBP rose significantly above its pre-induction value in group NS (133.52 ± 13.54 mmHg), whereas in group DEX it stayed below that value (110.32 ± 17.36 mmHg; $p < 0.001$ between groups) (Figure 1). At 3 minutes, SBP remained elevated in group NS (121.92 ± 16.14 mmHg) and fell significantly below pre-induction values in group DEX (98.00 ± 11.61 mmHg; $p < 0.001$). DBP and MAP followed the same pattern, with significant between-group differences at 1 and 3 minutes (all $p < 0.001$). HR rose significantly 1 minute after intubation in group NS (97.04 ± 11.66 bpm) but not in group DEX (85.40 ± 14.46 bpm; $p = 0.003$). At 3 minutes, HR was significantly lower in group DEX (78.92 ± 14.04 vs 92.40 ± 13.09 bpm; $p < 0.001$) (Figure 2). In group DEX, HR stayed below pre-induction values throughout the 45-minute intraoperative period, with significant reductions at 3–15 minutes. From 5 minutes after intubation onwards, no between-group difference reached the $p < 0.001$ threshold for any variable, either intraoperatively or during the 3 postoperative hours.

Table 2: Hemodynamic variables at key time points

Variable	Time point	Group NS (n = 25)	Group DEX (n = 25)	p-value
HR (bpm)	Baseline	85.40 ± 15.25	91.36 ± 16.19	0.187
	Before induction	86.12 ± 13.47	88.24 ± 17.08	0.628
	Before intubation	79.32 ± 10.51†	80.80 ± 15.15†	0.690
	1 min after intubation	97.04 ± 11.66†	85.40 ± 14.46	0.003
	3 min after intubation	92.40 ± 13.09	78.92 ± 14.04†	<0.001*
	5 min after intubation	86.48 ± 14.16	78.60 ± 13.82†	0.052
SBP (mmHg)	Baseline	123.32 ± 12.92	124.32 ± 17.00	0.816
	Before induction	115.68 ± 13.15	115.08 ± 14.55	0.879
	Before intubation	97.32 ± 11.19†	92.12 ± 10.97†	0.104
	1 min after intubation	133.52 ± 13.54†	110.32 ± 17.36	<0.001*
	3 min after intubation	121.92 ± 16.14	98.00 ± 11.61†	<0.001*
	5 min after intubation	110.88 ± 14.13	99.48 ± 10.96†	0.003
DBP (mmHg)	Baseline	77.32 ± 12.61	77.04 ± 12.14	0.937
	Before induction	76.96 ± 12.90	70.28 ± 9.28	0.041
	Before intubation	62.56 ± 9.98†	56.48 ± 9.35†	0.031
	1 min after intubation	88.44 ± 13.89	70.00 ± 15.64	<0.001*
	3 min after intubation	75.60 ± 13.57†	61.20 ± 9.73†	<0.001*
	5 min after intubation	69.72 ± 13.31†	62.20 ± 9.59†	0.027
MAP (mmHg)	Baseline	92.84 ± 11.58	92.56 ± 12.84	0.936
	Before induction	90.52 ± 11.45	86.72 ± 10.02	0.218
	Before intubation	75.00 ± 9.92†	69.80 ± 9.78†	0.068
	1 min after intubation	104.96 ± 11.78†	85.08 ± 15.60	<0.001*
	3 min after intubation	93.24 ± 12.83	75.48 ± 10.25†	<0.001*
	5 min after intubation	80.44 ± 19.96†	76.54 ± 9.93†	0.384

Values are mean ± SD. HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure. †$p < 0.05$ versus the pre-induction value within the group (repeated-measures ANOVA with Tukey's test). *Significant difference between groups ($p < 0.001$, threshold adjusted for multiple comparisons).

Sevoflurane requirement: The mean delivered sevoflurane concentration was slightly lower in group DEX at every time point from 1 to 30 minutes after intubation (range 1.85–1.94% vs 1.98–2.00%), but no difference was statistically significant ($p = 0.070–0.193$). SpO_2 remained normal and normocapnia was maintained in all patients.

Adverse events and postoperative outcomes

Bradycardia occurred in 9 patients (36%) in group DEX and 1 patient (4%) in group NS ($p = 0.005$); all responded to atropine [Table 3]. Hypotension occurred in 15 patients (60%) in group DEX and 7 (28%) in group NS ($p = 0.023$). Most episodes responded to fluids and reduced sevoflurane;

mephentermine was needed in 5 patients (20%) in group DEX and 1 (4%) in group NS (p = 0.082).

Postoperatively, most patients in both groups (16 each, 64%) had a Ramsay score of 2 at 0 hours, and sedation scores were similar thereafter. Rescue analgesia, PONV, dry mouth and shivering did not differ significantly between groups.

Table 3: Intraoperative and postoperative adverse events

Event	Group NS (n = 25)	Group DEX (n = 25)	p-value
Bradycardia (HR < 60 bpm)	1 (4)	9 (36)	0.005
Hypotension	7 (28)	15 (60)	0.023
Mephentermine required	1 (4)	5 (20)	0.082
Postoperative pain (VAS ≥ 3) requiring analgesia	23 (92)	21 (84)	0.384
PONV	22 (88)	20 (80)	0.440
Dry mouth	2 (8)	6 (24)	0.123
Shivering	4 (16)	5 (20)	0.713

Values are number (%). Comparisons by χ^2 test. VAS, visual analogue scale; PONV, postoperative nausea and vomiting.

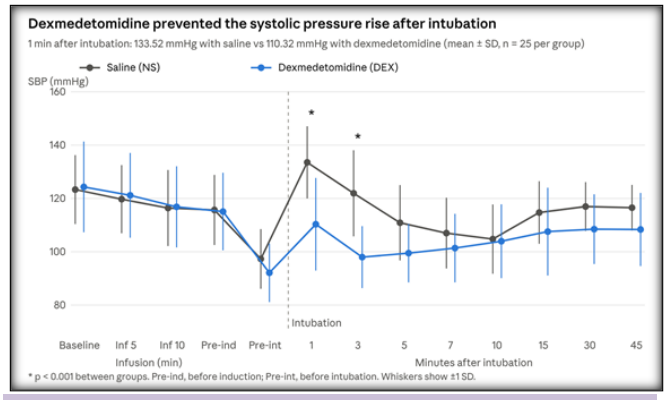


Figure 1: Systolic blood pressure (mean ± SD) from baseline to 45 minutes after intubation in the saline (NS) and dexmedetomidine (DEX) groups. The dashed line marks laryngoscopy and intubation. *p < 0.001 between groups.

[Figure 1] trial data, [Table 2] and full intraoperative series · n = 25 per group

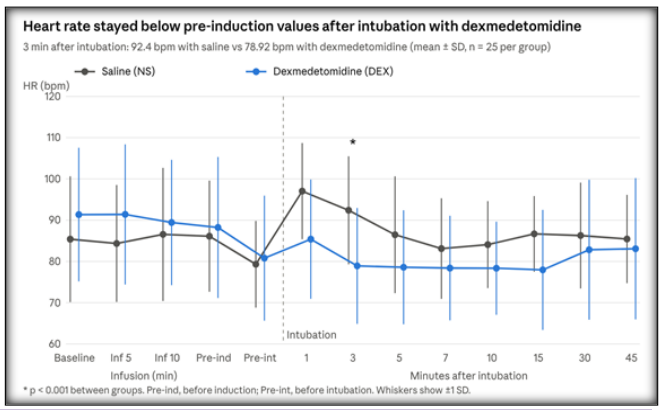


Figure 2: Heart rate (mean ± SD) from baseline to 45 minutes after intubation in the saline (NS) and dexmedetomidine (DEX) groups. The dashed line marks laryngoscopy and intubation. *p < 0.001 between groups.

[Figure 2] trial data, full intraoperative series n = 25 per group

Discussion

In this trial, dexmedetomidine 0.5 µg/kg infused over 10 minutes before thiopentone induction, combined with 1 MAC sevoflurane, abolished the rise in blood pressure and heart rate after laryngoscopy and intubation. One minute after intubation, SBP was 23 mmHg and MAP 20 mmHg lower than with placebo, and both stayed below pre-induction values. The cost was more bradycardia (36% vs 4%) and hypotension (60% vs 28%), almost all of which responded to simple measures.

The placebo group confirms that 1 MAC sevoflurane alone is insufficient. SBP rose by about 18 mmHg and HR by 11 bpm above pre-induction values at 1 minute, consistent with Hoda and Khan, who found that 1 MAC sevoflurane needed fentanyl to control the response.[16] Higher concentrations (3–6%) blunt the response but depress the myocardium,[15] and our earlier experience with 2 MAC sevoflurane plus dexmedetomidine produced hypotension in 76% of patients. Adding low-dose dexmedetomidine to 1 MAC sevoflurane therefore offers a middle path.

Our findings agree with dose-finding work on dexmedetomidine. Sağıroğlu et al. found that 0.5 µg/kg attenuated the intubation response, although 1 µg/kg was more effective.[18] Saraf et al. and Jaakola et al. reported effective suppression with 0.6 µg/kg.[19,20] Higher doses of 1–2 µg/kg are effective too,[10,12–14,21] but with more hypotension, bradycardia and sedation. In our study, the additional effect of sevoflurane appears to compensate for the lower dexmedetomidine dose.

Unlike studies with 1–2 µg/kg, we found no fall in HR during the infusion itself.[12,21] The low dose also did not reduce the thiopentone requirement, whereas 1 µg/kg reduced it in the studies of Bajwa et al. and Yildiz et al.[13,14] Similarly, the slightly lower sevoflurane concentration in group DEX was not significant. Scheinin et al. also found no reduction in volatile requirement with 0.6 µg/kg,[22] whereas higher doses reduce it.[12,14] Low-dose dexmedetomidine thus appears to act mainly by sympatholysis rather than by deepening Anesthesia.

Bradycardia was more frequent than in studies of 0.6 µg/kg dexmedetomidine without volatile agents.[20,22] Those studies, however, defined bradycardia as HR below 50 or 45 bpm, against our threshold of 60 bpm. All episodes in our study responded to a single dose of atropine.

The 60% incidence of hypotension in group DEX was higher than the 6–20% reported with 0.6 µg/kg,[19,20] and the absence of hypotension with 1 µg/kg reported elsewhere.[10,13] None of those studies used a volatile agent during induction. In our study most hypotensive episodes occurred in the period between induction and intubation, when sevoflurane and dexmedetomidine act together on vascular tone. They were usually mild: only 20% of

group DEX needed a vasopressor, not significantly more than the 4% in group NS. This suggests that the sevoflurane concentration during induction, rather than the dexmedetomidine dose, could be reduced further, a hypothesis that needs testing.

Postoperative sedation was similar in both groups, with most patients cooperative and oriented (Ramsay 2), in line with Saraf et al.[19] Slightly fewer patients in group DEX needed rescue analgesia or antiemetics, but the differences were small and not significant; the trial was not powered for these outcomes.

Limitations: This study has several limitations. First, it was single-centre and small, powered only for the primary outcome; the secondary and adverse-event comparisons should be seen as exploratory. Second, only young ASA I patients were enrolled, while the patients who would benefit most have cardiovascular disease. Third, sevoflurane was given by dial setting rather than end-tidal concentration, and depth of Anesthesia was not monitored. Fourth, blood pressure was measured non-invasively, so short peaks may have been missed. Finally, we did not compare dexmedetomidine doses, so the optimal dose with 1 MAC sevoflurane remains unknown.

Conclusion

In ASA I adults, dexmedetomidine 0.5 µg/kg infused over 10 minutes before induction, combined with 1 MAC sevoflurane, effectively attenuates the hemodynamic response to laryngoscopy and tracheal intubation without increasing postoperative sedation. Bradycardia and hypotension are more frequent but respond readily to standard treatment. Studies in patients with cardiovascular disease, with end-tidal sevoflurane control and invasive monitoring, are needed before the regimen can be recommended for high-risk patients.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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