

Intravenous versus Nebulized Dexmedetomidine for Awake Fiberoptic Bronchoscopy in Head and Neck Cancer Surgery: A Randomized Double-Blind Comparative Study

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Abstract

Background: Awake fiberoptic intubation is the technique of choice when the airway is anticipated to be difficult, as it commonly is in head and neck cancer. Dexmedetomidine sedates well without depressing respiration, but the intravenous route is prone to bradycardia and hypotension. Nebulization offers topical airway anaesthesia with slower systemic uptake and may avoid these swings. We compared the two routes at an equivalent dose. **Material and Methods:** In this prospective, randomized, double-blind study, 86 adults (ASA II–III) undergoing head and neck cancer surgery with awake fiberoptic nasotracheal intubation were allocated to intravenous dexmedetomidine 1 µg/kg over 20 minutes with saline nebulization (Group I, n=43) or saline infusion with nebulized dexmedetomidine 1 µg/kg (Group N, n=43). Sedation, cough and vocal cord scores, patient tolerance, intubation time, haemodynamics, satisfaction, and adverse events were recorded. **Results:** Heart rate and mean arterial pressure were lower in Group I from 10 minutes through 60 minutes post-intubation ($p<0.05$). Sedation was deeper (median RSS 3 vs 2; $p<0.001$), cough better suppressed (no cough in 65.1% vs 27.9%; $p<0.001$), and intubation faster (52.4 ± 8.6 vs 78.9 ± 14.2 s; $p<0.001$). Excellent satisfaction was reported by 65.1% vs 32.6% ($p<0.001$). Bradycardia and hypotension were more frequent in Group I ($p=0.044$ and 0.049). Oxygenation was preserved in both. **Conclusion:** Intravenous dexmedetomidine gave better sedation, intubating conditions, comfort, and procedural speed, at the cost of more haemodynamic adverse effects. The nebulized route is a gentler alternative when haemodynamic stability is paramount.

Keywords: Dexmedetomidine; awake fiberoptic intubation; head and neck cancer; nebulization; difficult airway; sedation.

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INTRODUCTION

Securing the airway in a patient with head and neck malignancy is among the more demanding situations an anaesthesiologist meets. Tumour bulk, trismus, post-radiation fibrosis, and the scarring of previous surgery distort the normal anatomy, and conventional direct laryngoscopy is often unreliable or frankly unsafe.^[1] For these patients awake fiberoptic intubation has long been the safest road, since the airway is secured while spontaneous ventilation and protective reflexes are left intact.^[2]

The procedure is poorly tolerated, however, when sedation falls short. Coughing, gagging, and a brisk sympathetic response to instrumentation are common, and any one of them can dislodge the scope or trigger laryngospasm. The sedative therefore has to walk a narrow line: it must calm the patient and blunt airway reflexes without depressing respiration or removing cooperation.^[3]

Several classes of agent have been pressed into this role, each with a drawback that matters at the airway. Midazolam offers anxiolysis and amnesia but no analgesia, and depresses respiration as the dose climbs. Fentanyl dampens airway reflexes yet brings respiratory depression, chest-wall rigidity, and nausea. Propofol sedates beautifully but

through a narrow window, with apnoea, hypotension, and loss of reflexes never far away. Ketamine keeps reflexes and breathing intact but raises secretions and can disorientate — unwelcome traits in a compromised airway. It is against this backdrop that the alpha-2 agonists have gained ground.

Dexmedetomidine has become the favoured drug for the job. A highly selective alpha-2 agonist, it produces a sleep-like sedation from which patients are easily roused, supplies useful analgesia, and — unlike opioids or propofol — spares the respiratory drive even at higher doses.^[4,5] Its sympatholytic action additionally smooths the haemodynamic surge that accompanies intubation.^[6] Given intravenously, these effects are delivered predictably, but the route carries a familiar

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liability for bradycardia and hypotension, most of all after the loading dose.^[7]

That liability has driven interest in other routes. Nebulization is appealing because it lays the drug down across a wide field of nasal, oropharyngeal, and tracheobronchial mucosa, pairing a measure of topical airway anaesthesia with gradual systemic absorption.^[8,9] A slower rise in plasma concentration might, in principle, hold on to the sedation while sparing the patient the abrupt haemodynamic dips of an intravenous bolus.

Head-to-head comparisons of the two routes in head and neck cancer — the setting where the airway is most hostile — remain few.^[10] We therefore set out to compare intravenous and nebulized dexmedetomidine, given at the same dose, during awake fiberoptic bronchoscopy in this population.

MATERIALS AND METHODS

This was a hospital-based, prospective, randomized, double-blind comparative study carried out in the Department of Anaesthesiology, Mahatma Gandhi Medical College, Jaipur, between April 2024 and September 2025. The Institutional Ethics Committee approved the protocol, and every participant gave written informed consent after the nature and purpose of the study had been explained in a language they understood.

We enrolled 86 adults aged 18 to 65 years, of ASA physical status II or III and with a body mass index below 35 kg/m², who were scheduled for head and neck cancer surgery requiring awake fiberoptic nasotracheal intubation. Patients who declined the technique were excluded, as were those with bleeding disorders or on anticoagulants, nasal pathology, known drug allergy, uncontrolled hypertension, bradyarrhythmia, or psychiatric illness, and pregnant or lactating women. The sample size was based on the intubation-time difference reported by Srinivas et al,^[11] at 95% confidence and 80% power, 78 patients were required, and the figure was raised to 86 to allow for dropouts, giving 43 per group.

A computer-generated random number table allocated patients equally to the two groups, and allocation was concealed so that neither the patient nor the assessing anaesthesiologist knew the assignment. Group I received intravenous dexmedetomidine 1 µg/kg over 20 minutes together with nebulization of 5 mL normal saline. Group N received an intravenous infusion of normal saline over 20 minutes followed by nebulized dexmedetomidine 1 µg/kg diluted to 5 mL. Both preparations were drawn up identically by an anaesthesiologist not otherwise involved in the study, preserving the blind.

On arrival in theatre, intravenous access was secured and baseline heart rate, blood pressure, electrocardiogram, and oxygen saturation were recorded. Oxygen was delivered at 4 L/min by nasal prongs. Every patient received glycopyrrolate 0.2 mg and ondansetron 4 mg intravenously, with xylometazoline 0.1% drops in both nostrils. Airway anaesthesia was completed by bilateral superior laryngeal nerve blocks (2 mL of 2% lignocaine each side) and a

transtracheal injection of 2 mL of 4% lignocaine through the cricothyroid membrane. Once the Ramsay Sedation Score reached 2 or more, awake fiberoptic nasotracheal intubation was performed; propofol was titrated only where sedation proved insufficient. After tube position was confirmed by fiberoptic view and capnography, general anaesthesia was induced and maintained in the usual way.

Intubating conditions were graded by cough score, vocal cord movement, and patient tolerance during and immediately after intubation. Sedation (Ramsay Sedation Score), intubation time, and adverse events were recorded, and patient satisfaction was assessed 24 hours later. Hypotension, bradycardia, and desaturation were managed by protocol with fluids and mephentermine, atropine, and supplemental oxygen respectively. Data were analysed in SPSS version 25 using the Student t-test, Mann–Whitney U test, and Fisher's exact test, with a two-sided $p < 0.05$ taken as significant.

RESULTS

The two groups were closely matched at baseline. Mean age was 48.6 ± 10.2 years in Group I and 46.9 ± 9.9 years in Group N ($p = 0.445$), and men predominated in both (81.4% and 86.0%; $p = 0.558$), in keeping with the male preponderance of head and neck cancer in this region. Weight, height, BMI, ASA distribution, and the spread of tumour types were all comparable, oral cavity carcinoma being the commonest malignancy in each group. Airway indices — Mallampati grade, mouth opening, thyromental and sternomental distances, and neck movement — did not differ, nor did baseline haemodynamic values (all $p > 0.05$). This even starting point lends weight to the comparisons that follow.

Heart rate and mean arterial pressure told the clearest story. From 10 minutes after the drug was started, through intubation, and up to 60 minutes afterwards, both were significantly lower in Group I than in Group N ($p < 0.05$ at each of these time points). The gap was widest around intubation itself and had closed by 90 minutes and 2 hours. Oxygen saturation, in contrast, stayed steady and essentially identical in the two groups throughout the procedure ($p > 0.05$).

Sedation was deeper with the intravenous route. Most Group I patients reached a Ramsay score of 3 (60.5%) or 4 (20.9%), whereas lighter sedation predominated in Group N; the median score was 3 (IQR 2–3) against 2 (IQR 2–3) ($p < 0.001$). Airway reflexes were better controlled in Group I as well — 65.1% did not cough at all during bronchoscopy compared with 27.9% in Group N ($p < 0.001$), and open vocal cords were seen in 55.8% versus 32.6% ($p = 0.003$).

Patients in Group I were also more comfortable. Over half (51.2%) showed no reflex response to passage of the tube, against 18.6% in Group N ($p < 0.001$), and 83.7% were calm and cooperative immediately afterwards compared with 55.8% ($p = 0.002$). Intubation was quicker too, at a mean of 52.4 ± 8.6 seconds in Group I versus 78.9 ± 14.2 seconds in Group N ($p < 0.001$). Only four Group I patients (9.3%) needed supplementary propofol, whereas every patient in Group N did so, and at a higher mean dose ($p < 0.001$). At 24 hours, 65.1% of Group I rated their experience excellent against 32.6% of Group N ($p < 0.001$).

The advantage came at a price. Bradycardia ($p=0.044$) and hypotension ($p=0.049$) were both more frequent in Group I, and atropine and mephentermine were needed correspondingly more often. Desaturation, nausea or

vomiting, and epistaxis occurred at similar rates in the two groups. Total fentanyl consumption, on the other hand, was lower in Group I ($p=0.032$).

The principal results are summarized in [Table 1 to 5].

Table 1: Baseline demographic, clinical and airway characteristics

Characteristic	Group I (n=43)	Group N (n=43)	p-value
Age (years)	48.56 $\pm$ 10.24	46.91 $\pm$ 9.87	0.445
Male, n (%)	35 (81.4)	37 (86.0)	0.558
Weight (kg)	62.84 $\pm$ 8.12	64.21 $\pm$ 7.89	0.425
BMI (kg/m ²)	23.78 $\pm$ 2.54	23.92 $\pm$ 2.41	0.792
ASA II / III, n	28 / 15	31 / 12	0.489
Mallampati III / IV, n	16 / 27	14 / 29	0.650
Mouth opening (cm)	2.12 $\pm$ 0.48	2.08 $\pm$ 0.52	0.709
Thyromental distance (cm)	5.42 $\pm$ 0.58	5.56 $\pm$ 0.51	0.233

Values are mean $\pm$ SD or n (%). No baseline parameter differed significantly ($p>0.05$).

Table 2: Heart rate (bpm) at different time points

Time point	Group I (n=43)	Group N (n=43)	p-value
Baseline	84.26 $\pm$ 12.48	82.91 $\pm$ 11.86	0.605
10 min after drug	72.84 $\pm$ 10.26	80.12 $\pm$ 11.42	0.002*
20 min after drug	68.42 $\pm$ 9.84	76.56 $\pm$ 10.68	<0.001*
At intubation	74.56 $\pm$ 11.24	86.42 $\pm$ 12.86	<0.001*
10 min after intubation	72.18 $\pm$ 10.12	82.24 $\pm$ 11.48	<0.001*
30 min	70.84 $\pm$ 9.56	78.42 $\pm$ 10.24	<0.001*
60 min	72.26 $\pm$ 8.92	76.84 $\pm$ 9.86	0.027*
90 min	74.12 $\pm$ 9.24	78.26 $\pm$ 10.12	0.050
2 hr	75.84 $\pm$ 8.86	78.92 $\pm$ 9.48	0.120

Values are mean $\pm$ SD. * $p<0.05$ statistically significant.

Table 3: Mean arterial pressure (mmHg) at different time points

Time point	Group I (n=43)	Group N (n=43)	p-value
Baseline	95.18 $\pm$ 10.12	93.89 $\pm$ 9.78	0.546
10 min after drug	86.42 $\pm$ 9.24	91.84 $\pm$ 10.12	0.011*
20 min after drug	82.56 $\pm$ 8.86	89.42 $\pm$ 9.56	<0.001*
At intubation	88.24 $\pm$ 10.48	98.56 $\pm$ 11.86	<0.001*
10 min after intubation	84.86 $\pm$ 9.12	94.24 $\pm$ 10.42	<0.001*
30 min	82.42 $\pm$ 8.56	90.18 $\pm$ 9.84	<0.001*
60 min	84.18 $\pm$ 8.24	88.56 $\pm$ 9.12	0.022*
90 min	86.24 $\pm$ 8.48	89.42 $\pm$ 9.24	0.099
2 hr	88.12 $\pm$ 8.12	90.24 $\pm$ 8.86	0.251

Values are mean $\pm$ SD. * $p<0.05$ statistically significant. SpO₂ remained comparable at all time points ($p>0.05$).

Table 4: Sedation, intubating conditions and procedural outcomes

Outcome	Group I (n=43)	Group N (n=43)	p-value
Ramsay Sedation Score, median (IQR)	3 (2–3)	2 (2–3)	<0.001*
No cough during AFOI, n (%)	28 (65.1)	12 (27.9)	<0.001*
Coughing score, median (IQR)	1 (1–2)	2 (1–3)	<0.001*
Vocal cord movement score, median (IQR)	1 (1–2)	2 (1–2)	0.003*
Tolerance during intubation, median (IQR)	1 (1–2)	2 (2–3)	<0.001*
Tolerance after intubation, median (IQR)	1 (1–1)	1 (1–2)	0.002*
Time for intubation (s), mean $\pm$ SD	52.42 $\pm$ 8.56	78.86 $\pm$ 14.24	<0.001*
Propofol required, n (%)	4 (9.3)	43 (100.0)	<0.001*
Excellent satisfaction at 24 h, n (%)	28 (65.1)	14 (32.6)	<0.001*

AFOI, awake fiberoptic intubation; IQR, interquartile range. * $p<0.05$ statistically significant.

Table 5: Adverse events and rescue medication

Variable	Group I (n=43)	Group N (n=43)	p-value
Bradycardia (HR <60 bpm), n (%)	8 (18.6)	2 (4.7)	0.044*
Hypotension (MAP $\downarrow$ >20%), n (%)	6 (14.0)	1 (2.3)	0.049*
Desaturation (SpO ₂ <95%), n (%)	2 (4.7)	4 (9.3)	0.397
Nausea / vomiting, n (%)	1 (2.3)	2 (4.7)	0.556
Epistaxis, n (%)	3 (7.0)	4 (9.3)	0.693
Atropine required, n (%)	8 (18.6)	2 (4.7)	0.044*
Mephentermine required, n (%)	6 (14.0)	1 (2.3)	0.049*
Total fentanyl (μ g), mean $\pm$ SD	142.56 $\pm$ 28.42	156.84 $\pm$ 32.18	0.032*

HR, heart rate; MAP, mean arterial pressure. * $p<0.05$ statistically significant.

DISCUSSION

The principal finding of this trial is straightforward. At an identical dose of 1 µg/kg, intravenous dexmedetomidine produced better sedation, smoother intubating conditions, greater patient comfort, and faster intubation than the nebulized route during awake fiberoptic bronchoscopy — but it did so at the cost of more bradycardia and hypotension. Both routes maintained oxygenation without incident, which reaffirms the respiratory safety that makes dexmedetomidine attractive for awake airway work in the first place.

The haemodynamic pattern we observed fits the pharmacokinetics of the two routes. A controlled intravenous infusion reaches predictable plasma concentrations and occupies central alpha-2 receptors before the intubation stimulus arrives, so sympatholysis is reliable and, at intubation, pronounced. Transmucosal absorption after nebulization is slower and more variable, peak levels are lower, and the haemodynamic effect — though clinically adequate — is gentler. Singh et al described exactly this contrast, with deeper intra-procedural suppression from the intravenous route but steadier intraoperative haemodynamics and fewer adverse effects from nebulization.^[12] Srinivas et al, using a dexmedetomidine-ketamine combination, likewise found tighter control with the intravenous route,^[11] and Ankita et al showed that even a modest intravenous dose matched the intranasal route in blunting the pressor response to laryngoscopy.^[13]

Sedation followed the same logic. Four of every five Group I patients reached the Ramsay 3–4 range generally considered ideal for awake intubation, a depth the nebulized group attained less often, and Srinivas et al reported the same direction of effect.^[11] The trade-off is worth keeping in view, though — deeper sedation shades into over-sedation, airway compromise, and the very haemodynamic depression that turned up more often in our intravenous arm.

Our cough and vocal cord data deserve a closer reading, because here the literature is not unanimous. We found markedly better cough suppression and more favourable cord positions with the intravenous route, and Srinivas et al agree.^[11] Yet Bhalotra et al, comparing the same two routes for awake fiberoptic intubation in ENT patients, found the opposite — lower cough scores with nebulization — which they attributed to the topical airway anaesthesia that nebulized drug provides over and above its systemic effect.^[14] Zhao et al similarly reported less coughing with a dexmedetomidine nasal spray during flexible bronchoscopy.^[15] The discrepancy is probably real rather than artefactual: it most likely reflects differences in the patient population (distorted cancer anatomy in our series against a more typical ENT airway), in the topical anaesthesia protocol, and in the dose actually deposited on the mucosa. Centrally mediated reflexes such as vocal cord adduction are, to our reading, more dependably attenuated by a systemic agent, which is consistent with what we saw. The shorter intubation time in Group I — roughly half a

minute faster — was a direct consequence of better cords, deeper sedation, and a calmer patient, and the figures track closely with Srinivas et al.^[11] The propofol data make the same point from another angle: every nebulized-group patient needed rescue propofol whereas only a handful of intravenous-group patients did, and the lower fentanyl use in Group I points to the analgesic contribution of adequately dosed dexmedetomidine.

Oxygen saturation stayed within a narrow, comparable band in both groups. That is an unremarkable result, but a reassuring one. Preserved spontaneous ventilation is the whole premise of an awake technique, and the fact that neither route disturbed it confirms that dexmedetomidine, however delivered, does not carry the respiratory burden of opioids or benzodiazepines. Bhalotra et al, Rajan et al, and Liu et al all reported similarly untroubled oxygenation and recovery with nebulized dexmedetomidine across different surgical settings.^[14,16,17]

Greater comfort and a quicker, smoother procedure plausibly explain the higher 24-hour satisfaction in Group I. The countervailing finding — more bradycardia and hypotension, and a greater call for atropine and vasopressor — matters most in exactly this population. Patients with head and neck cancer often carry cardiovascular comorbidity or autonomic impairment from earlier chemoradiation, and several authors have argued that nebulization is the safer choice when haemodynamic instability is a genuine concern.^[12,16] Bhalotra et al and Rajan et al both found nebulized dexmedetomidine free of clinically important adverse events, which supports that position.^[14,16]

This study has limitations. It was conducted at a single centre, follow-up extended only to 24 hours, and subjective scores carry an inherent risk of observer bias despite blinding of group allocation. We did not test dexmedetomidine against other sedative regimens, and individual variation in response to the drug was not formally accounted for. Multicentre trials with longer follow-up, and direct comparison against alternative agents, would help place these findings in a wider frame.

CONCLUSION

In patients undergoing awake fiberoptic bronchoscopy for head and neck cancer surgery, intravenous dexmedetomidine provided deeper sedation, better intubating conditions, greater patient comfort, and shorter intubation times than nebulized dexmedetomidine at the same dose. These gains were offset by more frequent bradycardia and hypotension and a greater need for rescue medication. Intravenous dexmedetomidine remains the better choice where optimal procedural conditions are the priority, while the nebulized route is a reasonable and gentler alternative for patients in whom haemodynamic stability is the overriding concern.

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Conflicts of interest

There are no conflicts of interest.

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