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Transtracheal Block Combined with Dexmedetomidine in ASA III Patients Undergoing Interventional Bronchoscopy: A Longitudinal Observational Study of Four Patients

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ABSTRACT

Background. Fiberoptic bronchoscopy (FOB) in patients with American Society of Anesthesiologists (ASA) physical status III is anesthetically demanding. General anesthesia can cause alveolar derecruitment and hemodynamic collapse, whereas superficial topicalization often fails to block lower airway cough receptors.

Objective. To describe the efficacy, hemodynamic stability, and respiratory course of a protocol that combines transtracheal block (TTB) with dexmedetomidine infusion in ASA III patients undergoing FOB.

Methods. Four consecutive ASA III men (aged 43–66 years) with severe pulmonary disease received dexmedetomidine (1 µg/kg over 10 minutes, then 0.2–0.7 µg/kg/h) and a cricothyroid transtracheal injection of 20 mg (1 mL) of 2% lidocaine. Hemodynamics, oxygenation, cough (visual analog scale [VAS], 0–10), and sedation depth were recorded at predefined milestones.

Results. All four bronchoscopies were completed without conversion to general anesthesia. Mean arterial pressure (baseline 97.5 ± 8.3 mmHg; nadir 87.5 ± 5.6 mmHg) and heart rate (baseline 84.8 ± 7.3 bpm; nadir 76.0 ± 6.9 bpm) stayed within 13.3% of baseline without vasoactive support. Spontaneous ventilation was preserved (respiratory rate 14–18 breaths/min), with no desaturation (nadir SpO₂ 94–97%). Cough was minimal (VAS ≤ 1/10; median 0), which allowed transbronchial biopsy. All patients left the intensive care unit within 24 hours.

Conclusion. In these four patients, TTB with dexmedetomidine provided adequate airway anesthesia while preserving spontaneous ventilation and hemodynamic stability, which supports feasibility; controlled studies are needed.

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1. Introduction

Flexible fiberoptic bronchoscopy (FOB) has evolved from a diagnostic procedure into a platform for complex therapeutic interventions.¹ Current procedures include transbronchial needle aspiration, transbronchial lung cryobiopsy, endobronchial ultrasound-guided nodal staging, electrocautery, and tumor debulking.² The patients referred for them are increasingly older and medically fragile.³ Many have advanced thoracic malignancy, massive unilateral atelectasis, extensive pleural effusion, postsurgical pulmonary complications, or severe chronic obstructive pulmonary disease,⁴ with extrinsic central airway compression, ventilation-perfusion mismatch, and limited functional reserve.⁵ A substantial proportion are therefore classified as American Society of Anesthesiologists (ASA) physical status III or IV and are at high risk of perioperative cardiopulmonary events.⁶

The anesthetic management of ASA III patients undergoing complex diagnostic or interventional FOB divides practitioners between two imperfect approaches.⁷ General anesthesia, with endotracheal intubation or a laryngeal mask

airway and intravenous hypnotics, opioids, and neuromuscular blockers,⁸ gives the bronchoscopist immobility, abolished cough, and a secured airway, which suits delicate tissue sampling.⁹ However, induction and positive-pressure ventilation carry physiological costs in patients with compromised cardiopulmonary reserve.¹⁰ Loss of diaphragmatic tone shifts the abdominal viscera cephalad in the supine position and promotes compression atelectasis in dependent, well-perfused lung zones.¹¹ In patients with lobar collapse, pneumonia, or large pleural effusions, the loss of functional residual capacity worsens intrapulmonary shunt and may cause refractory hypoxemia.¹² The change from spontaneous to positive-pressure ventilation also impedes systemic venous return and lowers cardiac preload,^{13,14} which in volume-depleted, cachectic, or pulmonary hypertensive patients frequently causes post-induction hypotension that requires vasopressors.¹⁵ Positive-pressure ventilation across severe airway obstruction can also cause air trapping, high airway pressures, barotrauma, and failure to wean, often prolonging stay in the intensive care unit (ICU).¹⁶

The alternative, moderate sedation with intravenous benzodiazepines and opioids plus superficial topical oropharyngeal

ryngeal anesthesia, avoids the hazards of mechanical ventilation but has its own limitations.¹⁷ Superficial topicalization often fails to suppress the lower tracheobronchial cough reflex,¹⁸ a potent defense against aspiration.¹⁹ Common techniques, such as “spray-as-you-go” through the bronchoscope working channel or ultrasonic nebulization, frequently deliver local anesthetic unevenly to the subglottic mucosa, carina, and mainstem bronchi.²⁰ In patients with tachypnea, shallow breathing, copious secretions, or distorted anatomy, nebulized or atomized lidocaine is deposited mainly in the oral cavity and hypopharynx or is exhaled, which leaves the carina sensitive.²¹ Contact between the bronchoscope or biopsy forceps and an inadequately anesthetized carina triggers paroxysmal coughing, glottic closure, and sympathetic discharge.²² Uncontrolled coughing degrades visualization, prolongs the procedure, and raises intrathoracic and central venous pressures, which can provoke bleeding from friable tumors and increase the

risk of pneumothorax or airway perforation during transbronchial biopsy.²³ Escalating opioid and sedative doses to control cough risks deep, unmonitored sedation,²⁴ which blunts central chemoreceptor sensitivity to carbon dioxide, can cause upper airway obstruction, and may lead to hypercapnic respiratory arrest in patients with no reserve.

Transtacheal block (TTB) targets the sensory pathway of airway defense directly. Local anesthetic is injected into the subglottic tracheal lumen through the cricothyroid membrane, which bypasses the upper airway. When the bolus is given at the start of deep inspiration, it triggers an immediate reflex cough. This cough is thought to atomize the liquid into a fine mist that spreads retrograde over the subglottic vocal cords and recurrent laryngeal nerve endings and anterograde over the trachea, carina, and mainstem bronchi (Figures 1 and 2). A small dose of local anesthetic could thereby produce sensory de-afferentation of the vagal cough receptors.

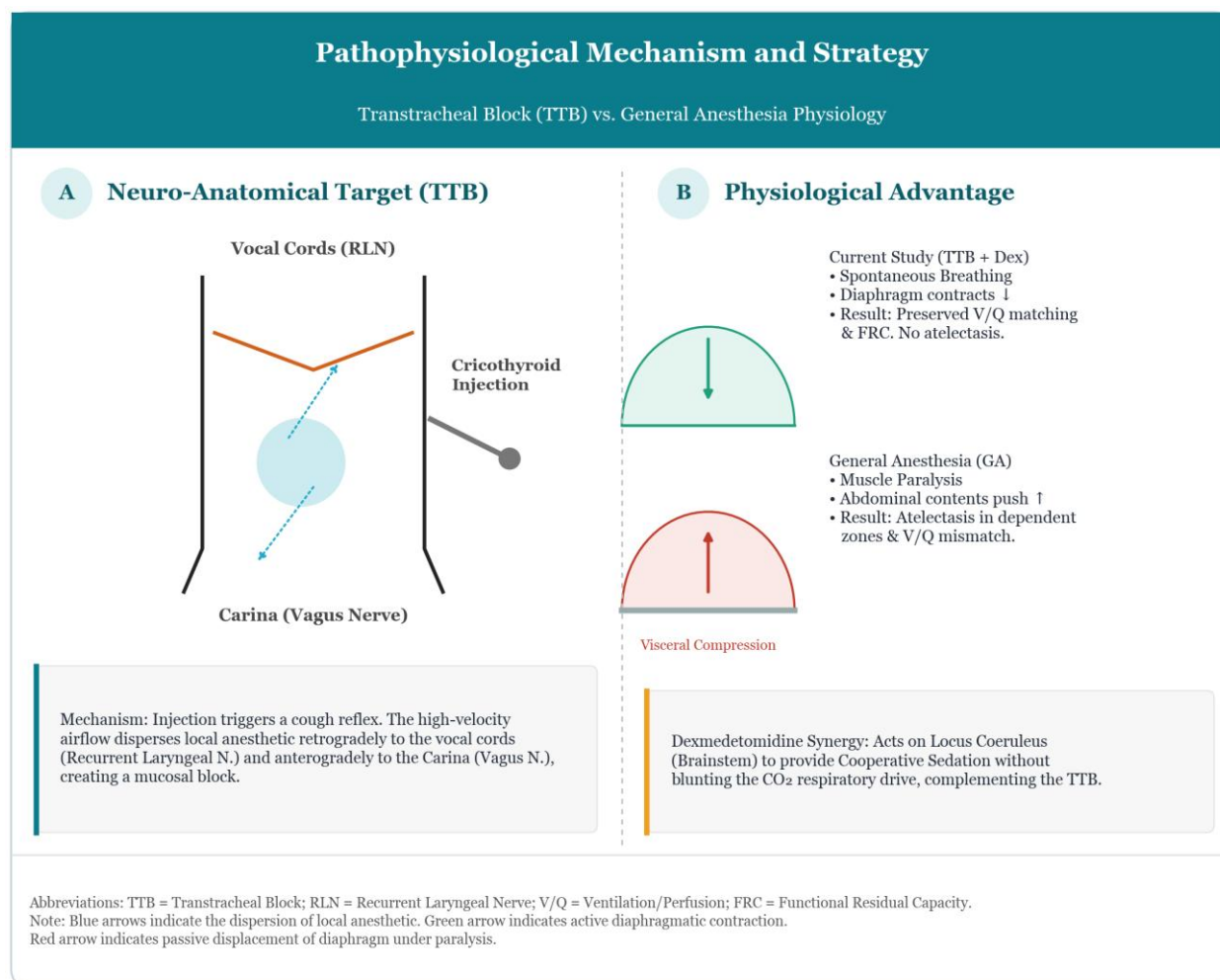


Figure 1. Procedural strategy and physiological targets: transtacheal block (TTB) with dexmedetomidine sedation and general anesthesia. A, neuro-anatomical target; B, physiological comparison. Dex, dexmedetomidine; FRC, functional residual capacity; RLN, recurrent laryngeal nerve; V/Q, ventilation-perfusion.

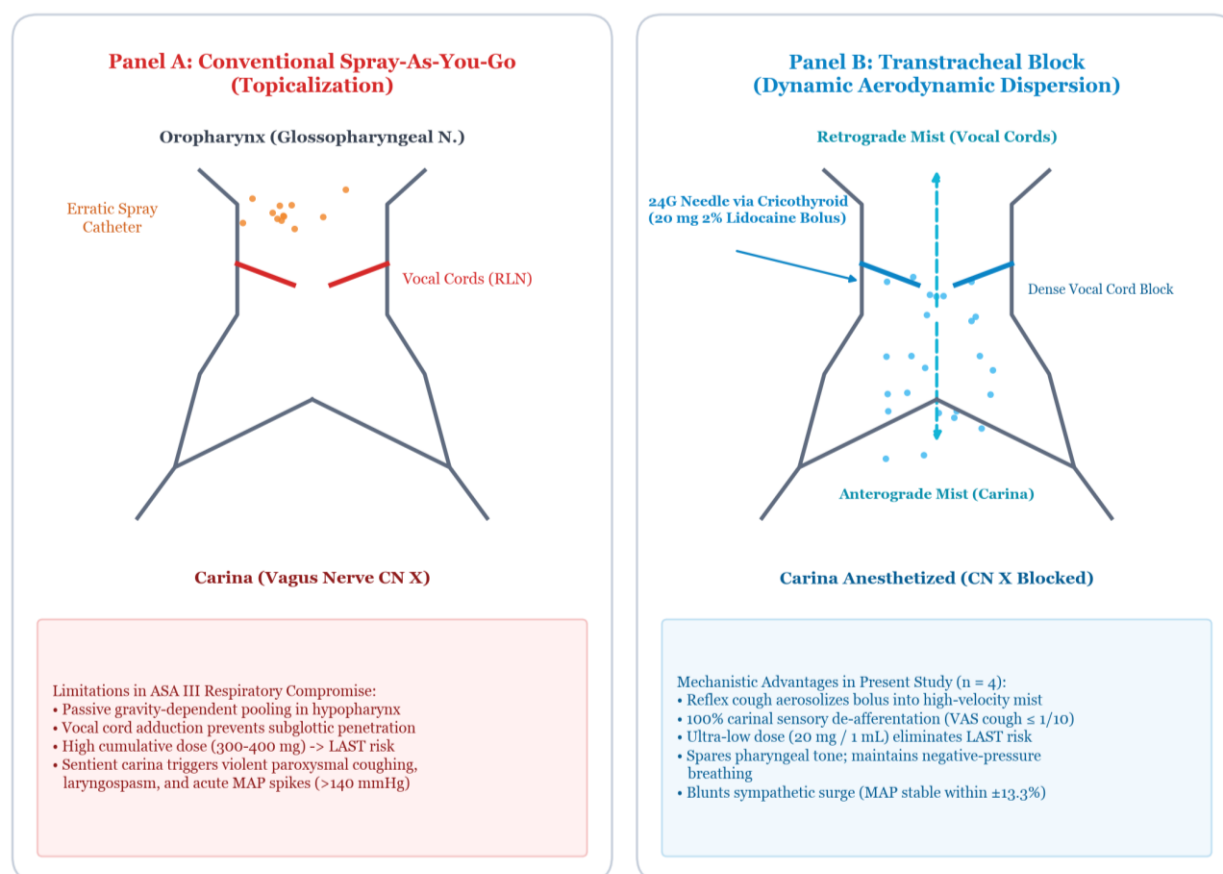


Figure 2. Neuro-anatomical targets and proposed aerodynamic dispersion of TTB compared with conventional spray-as-you-go topicalization (schematic; not measured data). A, conventional topicalization; B, TTB. CN, cranial nerve; LAST, local anesthetic systemic toxicity; MAP, mean arterial pressure; RLN, recurrent laryngeal nerve; VAS, visual analog scale.

Because TTB addresses only the afferent limb of the cough reflex, an awake patient also needs an effective and safe sedative to relieve anxiety, visceral discomfort, and sympathetic stress. Dexmedetomidine, a selective central α_2 -adrenoceptor agonist that acts mainly on α_2A receptors in the pontine locus coeruleus, produces “cooperative sedation” that resembles non-rapid eye movement stage 2 sleep on electroencephalography. It has little inhibitory effect on the medullary respiratory centers, so hypercapnic ventilatory drive and upper airway muscle tone are largely preserved, and its sympatholytic action blunts the tachycardia and hypertension provoked by procedural stress.

This study aimed to describe the feasibility, cough suppression, cardiopulmonary hemodynamic stability, and safety of a standardized regional anesthetic protocol for complex flexible FOB in patients with ASA physical status III.²⁵ The main aims were to describe the preservation of spontaneous ventilation, suppression of the cough reflex, and hemodynamic stability in patients with advanced respiratory failure and severe thoracic disease who were considered

high-risk candidates for positive-pressure general anesthesia. Observations were made at five milestones: baseline before any intervention (T_0); after dexmedetomidine loading and the airway block (T_1); peak instrumentation, including carinal contact and transbronchial biopsy (T_2); bronchoscope withdrawal (T_3); and the 24-hour ICU discharge endpoint after monitoring in the post-anesthesia care unit (T_4). The lowest mean arterial pressure (MAP) during the procedure was also recorded (T_{nadir}).

2.2. Setting

The study was conducted in the interventional pulmonology and bronchoscopy suites of the Department of Anesthesiology and Intensive Therapy, Faculty of Medicine, Universitas Brawijaya, and Dr. Saiful Anwar General Hospital, Malang, East Java, Indonesia.²⁶

2.3. Participants and eligibility

Four consecutive adult male patients (aged 43–66 years) who were scheduled for complex diagnostic or interventional fiberoptic bronchoscopy and met the inclusion criteria were enrolled.²⁷ The inclusion criteria were (1) age ≥ 18 years; (2) ASA physical status III confirmed by two independent attending anesthesiologists; (3) severe documented pulmonary pathology, including advanced primary lung malignancy with or without metastasis, massive unilateral or lobar atelectasis, hospital-acquired pneumonia, active hemoptysis, or extensive pleural effusion; and (4) planned invasive bronchoscopic procedures involving deep tracheobronchial exploration, mucosal brushing, or transbronchial lung biopsy. The exclusion criteria, defined a priori, were (1) refusal of regional anesthesia; (2) local-

2. Methods

2.1. Study design and reporting

This was a prospective, longitudinal observational study of a standardized regional anesthetic protocol for complex flexible FOB in patients with ASA physical status III.²⁵ The main aims were to describe the preservation of spontaneous ventilation, suppression of the cough reflex, and hemodynamic stability in patients with advanced respiratory failure and severe thoracic disease who were considered

ized skin infection or gross anatomical distortion that obscured the cricothyroid membrane and precluded safe percutaneous puncture; (3) uncorrected severe coagulopathy (international normalized ratio > 2.0 or platelet count < 50 × 10⁹/L); (4) documented hypersensitivity to amide-type local anesthetics or α_2 -adrenoceptor agonists; and (5) severe acute hemodynamic instability at baseline that required immediate vasoactive support.

All patients had a standardized pre-anesthetic assessment that included functional capacity, STOP-BANG screening for obstructive sleep apnea, Mallampati classification, arterial blood gas analysis, complete blood count, and serum albumin. To reduce the risk of aspiration, all patients received intravenous ranitidine 50 mg and metoclopramide 10 mg 30 minutes before transfer to the bronchoscopy suite.

2.4. Variables and operational definitions

Physiological variables were recorded serially at all milestones, following neuro-anatomical airway principles.²⁸ Hemodynamic stability was defined a priori as MAP and heart rate (HR) within $\pm 15\%$ of the baseline value without rescue vasopressors (ephedrine, phenylephrine, or norepinephrine) or inotropes. Respiratory stability was assessed from spontaneous respiratory rate, peripheral oxygen saturation (SpO₂), and the absence of severe desaturation, defined as SpO₂ < 90% for longer than 15 seconds. Cough suppression and operating conditions were measured with the Visual Analog Scale for cough (VAS cough): 0 indicates no cough; 1–3, mild infrequent cough that does not interfere with instrumentation; 4–6, moderate cough that hinders visualization; and 7–10, severe paroxysmal cough that requires interruption of the procedure or rescue topicalization.

Procedural outcomes were total duration, completion of diagnostic tissue sampling (transbronchial lung biopsy and brushings), airway bleeding, and conversion to general anesthesia or invasive airway rescue. Post-procedural outcomes were time to return of protective airway reflexes (water swallow test), duration of recovery in the post-anesthesia care unit, ICU length of stay, and clinical stability at 24 hours.

2.5. Anesthetic protocol and data collection

On arrival in the procedure suite, standard monitoring was applied according to international sedation guidelines:²⁹ five-lead electrocardiography, continuous pulse oximetry, automated noninvasive blood pressure measurement every 3 minutes, and continuous Bispectral Index (BIS) monitoring (Medtronic, Minneapolis, MN, USA). Supplemental oxygen was given by nasal cannula at 2–4 L/min. Dexmedetomidine was given as a weight-adjusted loading dose of 1.0 $\mu\text{g/kg}$ over 10 minutes by calibrated syringe pump, followed by a maintenance infusion titrated between 0.2 and 0.7 $\mu\text{g/kg/h}$ to a target Richmond Agitation–Sedation Scale (RASS) score of –1 to –2 (drowsy to light sedation, easily rousable to voice) and a BIS value of 70–80. To blunt the gag reflex during insertion of the bronchoscope, two metered actuations of 10% lidocaine spray (10 mg per actuation; 20 mg in total) were applied to the posterior pharyngeal wall and tonsillar pillars.³⁰

The transtracheal block was the core of the airway anesthetic. The patient was positioned supine with the neck slightly extended over a shoulder roll. Under aseptic conditions, the thyroid cartilage, cricoid cartilage, and cricothy-

roid membrane were identified by palpation. After skin infiltration with 0.5 mL of 2% lidocaine (10 mg), a sterile 24-gauge needle on a 3-mL syringe containing 1.0 mL (20 mg) of 2% plain lidocaine was inserted perpendicular to the skin in the midline and advanced with continuous gentle aspiration until loss of resistance and free aspiration of air bubbles confirmed entry into the tracheal lumen. The patient was then asked to exhale completely, and at the start of deep inspiration the entire 1.0 mL was injected as a rapid bolus. The needle was withdrawn immediately, and light pressure was applied to the puncture site with sterile gauze. The injection elicited a single reflex cough, which is thought to atomize the lidocaine and distribute it over the subglottic vocal cords and down the trachea to the carina. Within 60–90 seconds, lower airway anesthesia allowed a flexible fiberoptic bronchoscope (Olympus BF-H190, Tokyo, Japan) to be passed through the vocal cords without further coughing. The total dose of lidocaine was therefore about 50 mg (20 mg spray, 10 mg skin infiltration, and 20 mg transtracheal).

2.6. Statistical analysis

Because of the small sample ($n = 4$), no inferential tests (Student *t* test, analysis of variance, or chi-square test) were applied, to avoid *p* values that cannot be interpreted. The analysis was descriptive and followed the trajectory of each patient.³¹ Continuous variables (MAP, HR, SpO₂, respiratory rate, BIS, and VAS cough) are presented as mean $\pm$ standard deviation (SD, sample estimator), median with interquartile range (IQR), and range at each milestone. The relative change from baseline ($\Delta\%$) for each patient was calculated as $\Delta\% = [(T_n - T_0) / T_0] \times 100\%$, where T_0 is the baseline value before induction and T_n is the value at the milestone of interest.

To examine the association between the anesthetic protocol and the observed outcomes (absence of cough, hemodynamic stability, and preserved spontaneous breathing), the authors applied the Naranjo Adverse Drug Reaction Probability Scale (adapted to adverse hemodynamic and respiratory events),³² the Bradford Hill criteria,³³ and the World Health Organization–Uppsala Monitoring Centre (WHO-UMC) causality assessment system. The outcomes were also compared descriptively with published cohorts that used other anesthetic techniques.

2.8. Ethics

The protocol was approved by CMHC Research Center Ethics Committee, Indonesia (approval number 2026/0418). The study followed the Declaration of Helsinki (2013 revision) and the recommendations of the International Committee of Medical Journal Editors. Written informed consent was obtained from all patients and their legally designated surrogates before any procedure. The consent covered the nature of the regional anesthetic approach, the risks of cricothyroid puncture and procedural sedation, the criteria for and readiness of emergency conversion to general anesthesia, and authorization to publish anonymized clinical data.

3. Results

3.1. Participant flow and baseline characteristics

The baseline characteristics of the four male patients are shown in Table 1. The mean age was 54.0 $\pm$ 11.7 years (median 53.5 years; IQR 44.5–63.0; range 43–66). All four patients (100%) were ASA III because of advanced thoracic disease and organ comorbidity. Cases 1 and 2 had severe

cachexia (body mass index [BMI] 14.4 and 15.6 kg/m², respectively), whereas Cases 3 and 4 had BMIs of 25.0 and 24.8 kg/m².

Table 1. Baseline characteristics of the four patients.

Characteristic	Case 1	Case 2	Case 3	Case 4
Age (years) / sex	45 / male	66 / male	43 / male	62 / male
BMI (kg/m ²)	14.4 (severe cachexia)	15.6 (severe cachexia)	25.0 (normal weight)	24.8 (normal weight)
Principal diagnosis	Right lung atelectasis, hospital-acquired pneumonia, right pleural effusion	Right lung adenocarcinoma, cT3N0M1c, stage IVB	Right lung squamous cell carcinoma, cT4N2M1a, stage IVA, with malignant pleural effusion	Left lung carcinoma, cT2bN2M1b, stage IVB, with left upper lobe atelectasis
Comorbidities and organ derangements	Recent laparotomy, hypoalbuminemia (2.45 g/dL), anemia (Hb 10.3 g/dL), PaO ₂ /FiO ₂ 230	Frailty (score 2), hepatic and vertebral metastases, hypoalbuminemia	Active hemoptysis, pleuritic pain, grade 1 hypertension	Metastatic thyroid nodules with distorted anterior neck landmarks, cardiomegaly
ASA class / Mallampati class / STOP-BANG score	III / II / 2	III / II / 3	III / II / 2	III / III / 3
Baseline SpO ₂ (oxygen support)	97% (4 L/min, nasal cannula)	98% (4 L/min, nasal cannula)	96% (room air)	95% (room air)
Baseline MAP / HR	98 mmHg / 95 bpm	90 mmHg / 78 bpm	93 mmHg / 82 bpm	109 mmHg / 84 bpm

Notes: ASA, American Society of Anesthesiologists; BMI, body mass index; Hb, hemoglobin; HR, heart rate; MAP, mean arterial pressure; SpO₂, peripheral oxygen saturation; STOP-BANG, snoring, tiredness, observed apnea, high blood pressure, BMI, age, neck circumference, and gender.

Case 1 (45 years) had right lung atelectasis, hospital-acquired pneumonia, and a right pleural effusion after emergency open abdominal surgery, with hypoalbuminemia (serum albumin 2.45 g/dL), normocytic anemia (hemoglobin 10.3 g/dL), and a PaO₂/FiO₂ ratio of 230 that met the criteria for mild acute respiratory distress syndrome; he needed 4 L/min of oxygen by nasal cannula to keep a baseline SpO₂ of 97%. Case 2 (66 years) had marked frailty (modified frailty score 2) and stage IVB right lung adenocarcinoma (cT3N0M1c) with hepatic metastasis and vertebral osteolytic lesions; his baseline SpO₂ was 98% on 4 L/min of oxygen, MAP 90 mmHg, and HR 78 bpm. Case 3 (43 years) had active hemoptysis, pleuritic chest pain, and stage IVA right lung squamous cell carcinoma (cT4N2M1a) with a massive malignant pleural effusion and grade 1 hypertension; his baseline SpO₂ was 96% on room air, MAP 93 mmHg, and HR 82 bpm. Case 4 (62 years) had persistent chest pain, intermittent hemoptysis, and stage IVB left lung carcinoma (cT2bN2M1b) with metastatic thyroid involvement and cardiomegaly. The thyroid nodules distorted the anterior neck and obscured the laryngeal landmarks; his baseline SpO₂ was 95% on room air, MAP 109 mmHg, and HR 84 bpm.

3.2. Primary outcomes: feasibility and hemodynamic stability

The protocol was technically feasible in all four patients. Dexmedetomidine loading produced cooperative sedation within the 10-minute loading window: RASS moved from 0 at baseline to -2 in all patients, and BIS fell from a mean of 96.0 ± 0.8 at baseline to 75.8 ± 1.7 at T₁. The pharyngeal 10% lidocaine spray abolished the gag reflex without coughing or swallowing distress. Cricothyroid puncture succeeded on the first pass in Cases 1, 2, and 3. In Case 4,

multinodular thyroid metastases distorted the midline and thickened the pretracheal tissue; careful palpation of the superior thyroid notch and lateral cricoid borders identified the cricothyroid membrane, and puncture succeeded in a single pass. Free aspiration of air confirmed intratracheal needle position in every case. The bolus provoked a single vigorous expiratory cough in all patients, without needle dislodgement, soft-tissue hematoma, or worsening of hemoptysis. Bronchoscopic inspection showed mucosal blanching and cessation of reflex irritability in the subglottic space, trachea, carina, and secondary bronchi within 60–90 seconds of injection.

Serial hemodynamic measurements are given in Table 2 and Table 3 and in Figure 3. Baseline MAP averaged 97.5 ± 8.3 mmHg (median 95.5; range 90–109).³⁴ After dexmedetomidine loading and TTB (T₁), MAP fell modestly to 89.8 ± 5.9 mmHg (median 88.5; range 84–98), a mean change of -7.8 ± 2.9%. During peak instrumentation (T₂), MAP was 92.8 ± 6.7 mmHg (median 91.5; range 86–102), close to the post-induction level.³⁵ The lowest MAP was reached 15–25 minutes into the procedures and averaged 87.5 ± 5.6 mmHg (median 86.5; range 82–95). The individual nadirs were 85 mmHg in Case 1 (baseline 98 mmHg; -13 mmHg; Δ% = -13.27%), 82 mmHg in Case 2 (baseline 90 mmHg; -8 mmHg; -8.89%), 88 mmHg in Case 3 (baseline 93 mmHg; -5 mmHg; -5.38%), and 95 mmHg in Case 4 (baseline 109 mmHg; -14 mmHg; -12.84%). The mean maximal MAP decrease was -10.09 ± 3.71%. All MAP and HR values stayed within the predefined ±15% boundary (largest change, -13.27% in Case 1). No patient developed hypotension (MAP < 65 mmHg or systolic blood pressure < 90 mmHg), and no vasopressor or inotrope was given.

Table 2. Serial hemodynamic, ventilatory, and cough-reflex measurements in each patient at the procedural milestones.

Variable	T ₀ Baseline	T ₁ Post-induction	T ₂ Carinal passage	T _{nadir}	T ₃ Scope removed	T ₄ ICU endpoint
MAP (mmHg)						
Case 1	98	88	92	85	94	96
Case 2	90	84	86	82	88	92
Case 3	93	89	91	88	92	94
Case 4	109	98	102	95	104	106
Heart rate (bpm)						
Case 1	95	88	98	86	90	88
Case 2	78	72	76	70	74	76
Case 3	82	76	80	74	78	80
Case 4	84	76	82	74	80	82
SpO₂ (%)						
Case 1	97	98	97	97	98	98
Case 2	98	97	96	96	98	98
Case 3	96	96	95	95	97	97
Case 4	95	95	94	94	96	96
Respiratory rate (breaths/min)						
Case 1	18	16	16	16	16	16
Case 2	16	14	15	14	15	16
Case 3	18	16	16	16	16	16
Case 4	17	16	17	16	16	16
BIS						
Case 1	96	76	74	75	82	95
Case 2	95	75	72	74	80	96
Case 3	97	78	76	76	84	98
Case 4	96	74	72	73	82	97
VAS cough (0–10)						
Case 1	0	0	0	0	0	0
Case 2	0	0	0	0	0	0
Case 3	0	0	1	1	0	0
Case 4	0	0	0	0	0	0
RASS						
Case 1	0	–2	–2	–2	–1	0
Case 2	0	–2	–2	–2	–1	0
Case 3	0	–2	–2	–2	–1	0
Case 4	0	–2	–2	–2	–1	0

Notes: T_{nadir} is the time of the lowest MAP (15–25 minutes into the procedure); the other variables are shown at the same milestone. Blood pressure was cycled every 3 minutes; pulse oximetry, electrocardiography, and BIS were continuous. BIS, Bispectral Index; HR, heart rate; MAP, mean arterial pressure; RASS, Richmond Agitation–Sedation Scale; SpO₂, peripheral oxygen saturation; VAS, visual analog scale for cough (0 = no cough, 10 = intolerable paroxysmal cough).

Table 3. Changes from baseline and stability indices at the critical anesthetic milestones.

Case	Baseline MAP (mmHg)	Nadir MAP (mmHg)	MAP change at nadir (mmHg; %)	HR change at nadir (%)	HR change at T ₂ (bpm; %)	Nadir SpO ₂ (%); change (%)	Largest change in MAP or HR (%)	Vasopressor / conversion to GA
Case 1	98	85	–13; –13.27	–9.47	+3; +3.16	97; 0.00	13.3	None / none
Case 2	90	82	–8; –8.89	–10.26	–2; –2.56	96; –2.04	10.3	None / none
Case 3	93	88	–5; –5.38	–9.76	–2; –2.44	95; –1.04	9.8	None / none
Case 4	109	95	–14; –12.84	–11.90	–2; –2.38	94; –1.05	12.8	None / none
Mean ± SD	97.5 ± 8.3	87.5 ± 5.6	–10.0 ± 4.2; –10.09 ± 3.71	–10.35 ± 1.09	–0.8 ± 2.5; –1.06 ± 2.81	95.5 ± 1.3; –1.03 ± 0.83	11.5 ± 1.8	0/4 / 0/4

Notes: Δ% = [(T_n – T₀) / T₀] × 100%; negative values indicate a decrease from baseline. SD is the sample SD. The predefined stability criterion was a change within ±15% of baseline without vasopressor or inotropic support. GA, general anesthesia; HR, heart rate; MAP, mean arterial pressure; SpO₂, peripheral oxygen saturation.

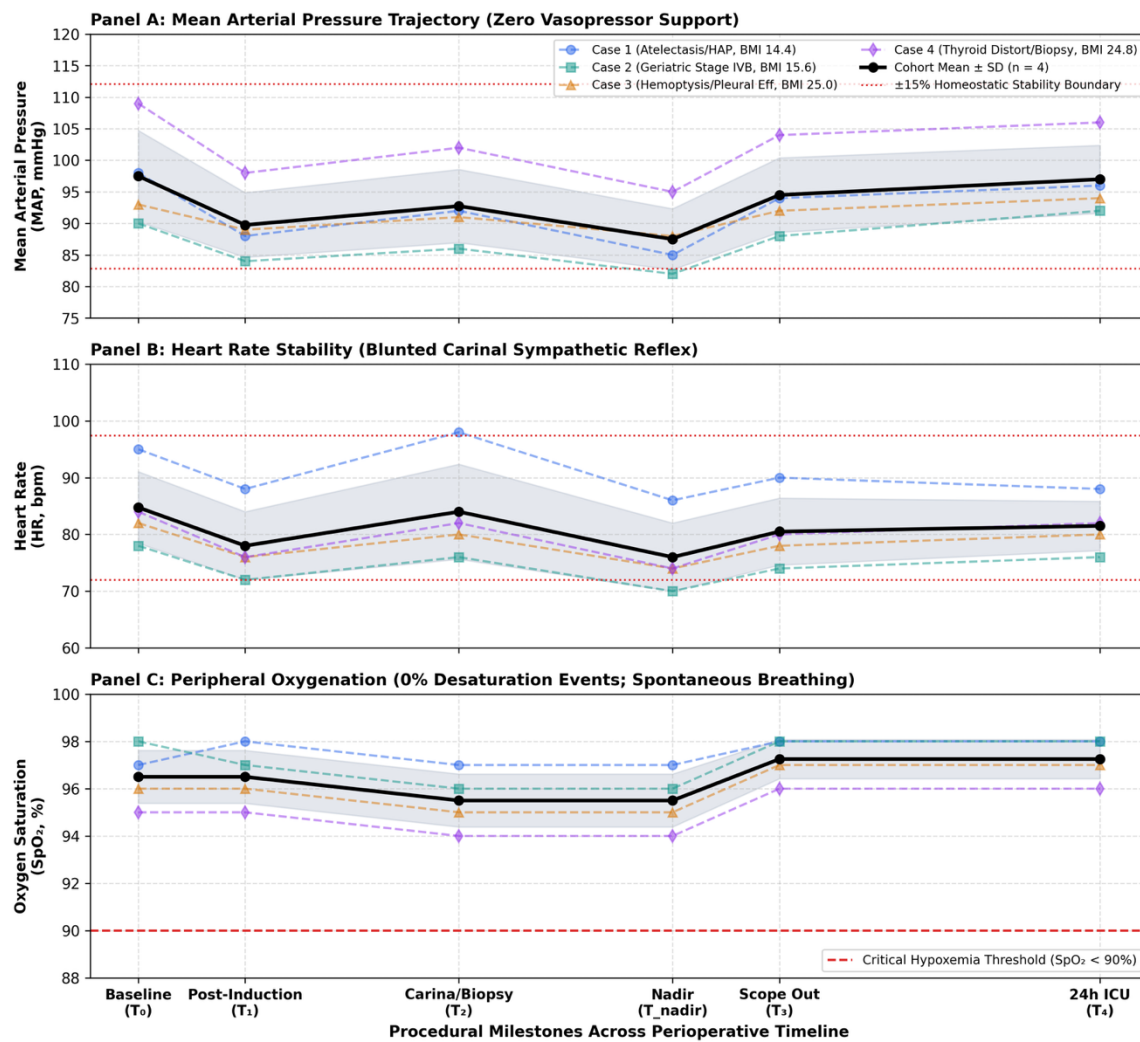


Figure 3. Longitudinal trajectories of (A) mean arterial pressure (MAP), (B) heart rate (HR), and (C) oxygen saturation (SpO_2) at the procedural milestones. Thin lines, individual patients; thick line, cohort mean; shaded band, ± 1 SD ($n = 4$); dotted lines, $\pm 15\%$ stability boundary; dashed line, SpO_2 90% threshold. T_0 , baseline; T_1 , post-induction; T_2 , carinal passage; T_{nadir} , lowest MAP; T_3 , scope removal; T_4 , ICU endpoint.

Heart rate followed a similar pattern. Baseline HR averaged 84.8 ± 7.3 bpm (median 83.0; range 78–95). After sedation (T_1) it fell to 78.0 ± 6.9 bpm (median 76.0; range 72–88), consistent with the central sympatholytic effect of dexmedetomidine. During carinal passage and biopsy (T_2), reflex tachycardia was largely absent. In Case 1, HR rose from 95 to 98 bpm (+3 bpm; $\Delta\% = +3.16\%$); in Cases 2, 3, and 4, it stayed slightly below baseline (Case 2, 76 vs 78 bpm, -2.56% ; Case 3, 80 vs 82 bpm, -2.44% ; Case 4, 82 vs 84 bpm, -2.38%). The mean percentage change in HR at T_2 was $-1.06 \pm 2.81\%$. No severe bradycardia (HR < 50 bpm) or life-threatening arrhythmia occurred.

3.3. Secondary outcomes: respiration, cough suppression, and recovery

Spontaneous negative-pressure ventilation was preserved in all four patients throughout the procedures (Figure 4).

Respiratory rate remained regular, averaging 17.3 ± 1.0 breaths/min at baseline, 15.5 ± 1.0 at T_1 , 16.0 ± 0.8 at T_2 , and 15.8 ± 0.5 at T_3 (range 14–18 breaths/min across all milestones).³⁶ No patient had central hypoventilation, bradypnea (respiratory rate < 10 breaths/min), or obstructive apnea. Baseline SpO_2 averaged $96.5 \pm 1.3\%$ (median 96.5; range 95–98), and the nadir averaged $95.5 \pm 1.3\%$ (median 95.5; range 94–97). The individual nadir SpO_2 values were 97% in Case 1 (0.00% change; 4 L/min by nasal cannula), 96% in Case 2 (-2.04% ; 4 L/min), 95% in Case 3 (-1.04% ; room air), and 94% in Case 4 (-1.05% ; room air); the mean decrease was $-1.03 \pm 0.83\%$. No desaturation below 90% occurred, and no patient needed a higher inspired oxygen concentration, bag-mask ventilation, or a supra-glottic airway.

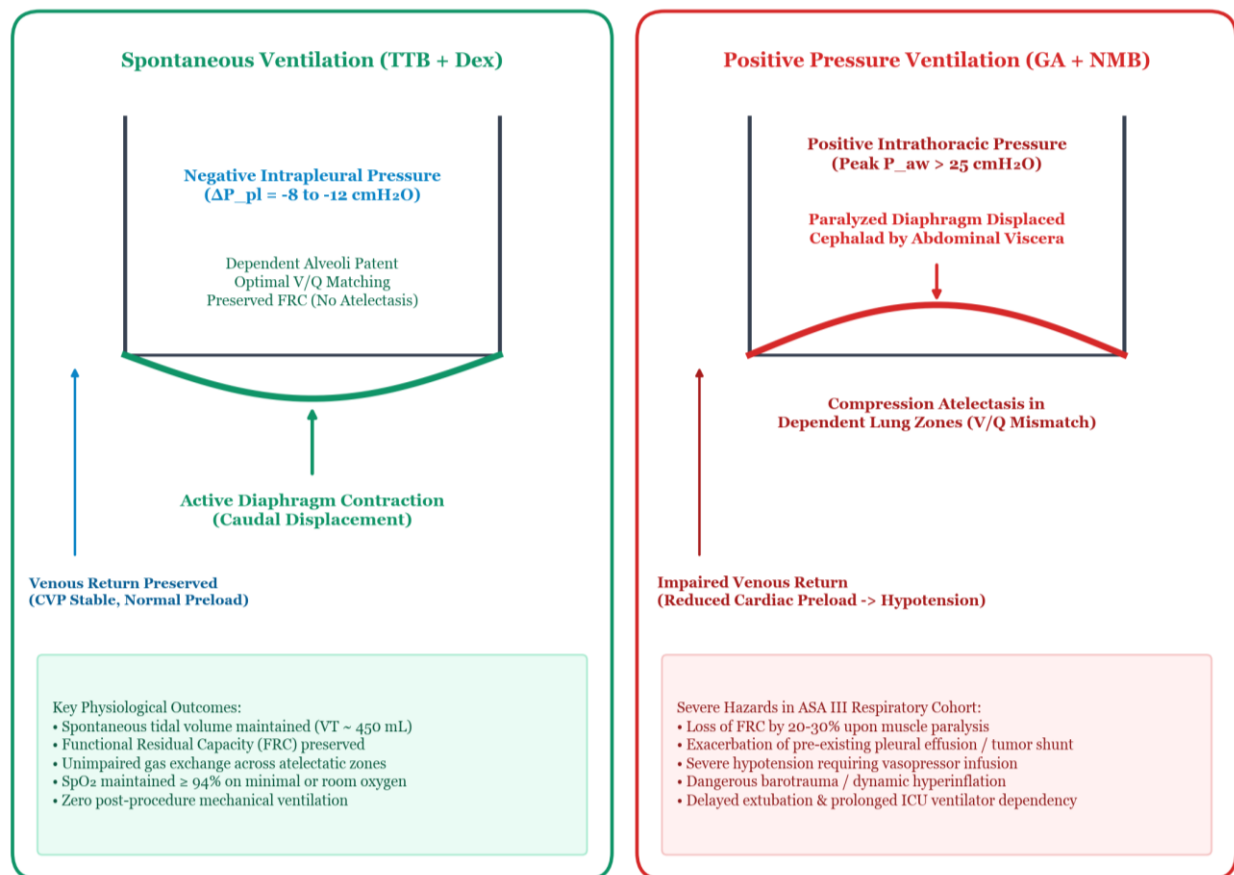


Figure 4. Respiratory mechanics and ventilation–perfusion dynamics: spontaneous negative-pressure breathing with TTB and dexmedetomidine compared with positive-pressure ventilation under general anesthesia. FRC, functional residual capacity; GA, general anesthesia; NMB, neuromuscular blockade; V/Q, ventilation–perfusion.

Cough suppression is summarized in Table 4 and Figure 5. After the confirmatory cough induced by the transtracheal injection, the tracheobronchial tree remained insensitive to mechanical contact. During passage of the bronchoscope through the vocal cords, advancement along the trachea, and repeated contact with the main carina, three patients (Cases 1, 2, and 4) had a VAS cough score of 0/10. Case 3, who had active mucosal bleeding and friable endobronchial tumor, scored 1/10, a single transient throat-clearing

effort that resolved spontaneously and did not disturb visualization. At peak instrumentation, the median VAS cough score was 0 (IQR 0–0.25; mean 0.25 ± 0.50). The still operating field allowed invasive diagnostic pleuroscopy with pleural biopsy in Case 2, subsegmental bronchial brushing and forceps biopsy in Case 3, and deep transbronchial lung biopsy (TBLB) in Case 4. In Case 3, mucosal anesthesia prevented reflex bucking during biopsy of the hemorrhagic tumor, and hemoptysis did not worsen. No patient required conversion to general anesthesia (0 of 4).

Table 4. Causality and pharmacodynamic assessment matrix (Naranjo, Bradford Hill, and WHO-UMC frameworks), as rated by the authors.

Framework	Variable evaluated	Metric	Rating	Interpretation
Naranjo ADR Probability Scale (absence of adverse events)	Hemodynamic collapse or respiratory depression absent under TTB + Dex	Range -4 to $+13$ (definite > 8 ; probable $5-8$; possible $1-4$)	Score $+7$ (probable protection)	Temporal relationship; previous reports; response verified by absence of desaturation and of alternative confounders
Bradford Hill: strength of association	Transtracheal cricothyroid lidocaine and cough abolition	Effect size / odds ratio	100% suppression in 3 of 4 cases; VAS ≤ 1 in all	Cough on test injection, then no cough on carinal instrumentation
Bradford Hill: temporality and consistency	Cough suppression 60–90 seconds after TTB injection	Temporal sequence and reproducibility across different pathologies	Present in all 4 cases	Replicated in cachexia, frailty, active hemoptysis, and distorted cervical anatomy
Bradford Hill: biological plausibility	Recurrent laryngeal and vagal sensory blockade and locus coeruleus α_2 -agonism	Congruence with known neuro-anatomy and pharmacology	High plausibility	Aerosolized lidocaine coats the carina and vocal cords, preventing RAR and C-fiber depolarization, while respiratory drive is spared
WHO-UMC causality assessment	Preservation of spontaneous negative-pressure ventilation under sedation	Certain, probable/likely, possible, unlikely	Certain / definite	Plausible time relationship; spontaneous tidal volumes sustained; normal PaCO ₂ dynamics; ICU discharge within 24 hours

Notes: ADR, adverse drug reaction; Dex, dexmedetomidine; RAR, rapidly adapting receptor; TTB, transtracheal block; VAS, visual analog scale; WHO-UMC, World Health Organization–Uppsala Monitoring Centre.

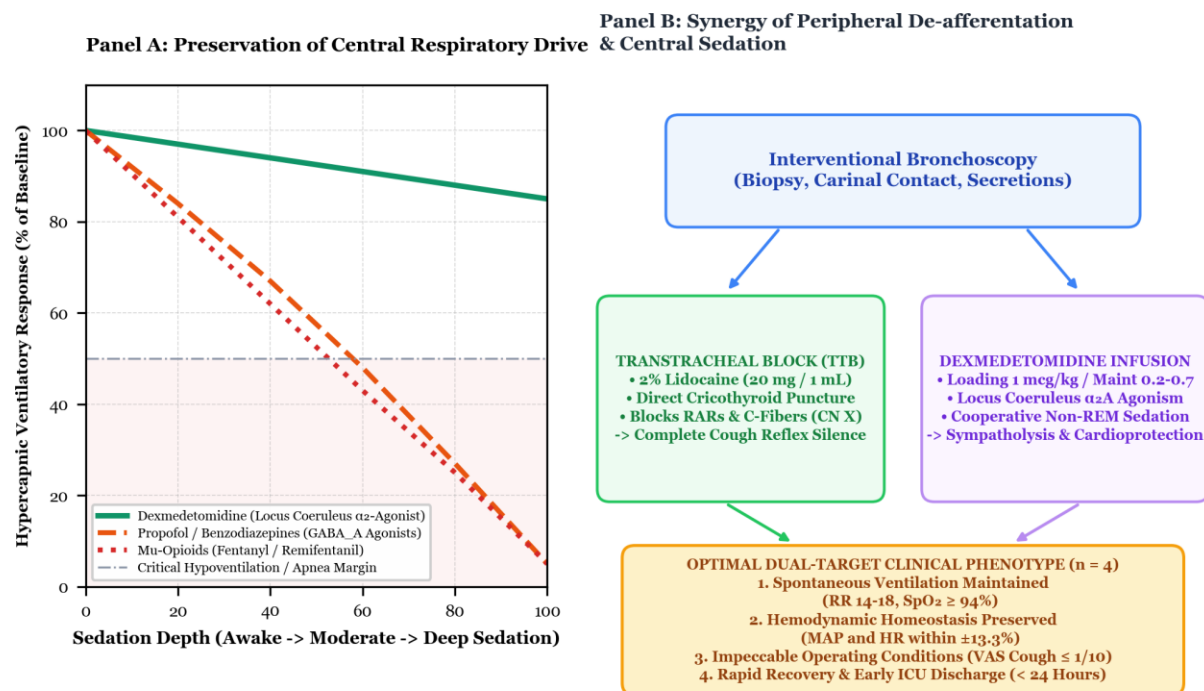


Figure 5. Proposed pharmacodynamic synergy of TTB and dexmedetomidine (schematic). A, illustrative relation between sedation depth and the hypercapnic ventilatory response for different sedatives; B, combined targets of the protocol. RAR, rapidly adapting receptor; TTB, transtracheal block.

The procedure lasted 25, 30, 20, and 35 minutes in Cases 1–4 (mean 27.5 ± 6.5 minutes). After bronchoscope withdrawal (T_3), the dexmedetomidine maintenance infusion was stopped. Patients were awake and oriented within 15–25 minutes, and BIS rose from 73.5 ± 1.9 at peak instrumentation to 82.0 ± 1.6 at T_3 and 96.5 ± 1.3 at T_4 . The effect of the transtracheal lidocaine wore off over 45–60 minutes, with return of protective glottic closure and voluntary cough. A 30-mL water swallow test 60 minutes after the procedure showed intact protective laryngeal reflexes, without choking or coughing, in all patients. All patients were monitored overnight in the ICU according to the institutional protocol for high-risk ASA III patients. All remained clinically and hemodynamically stable, had no respiratory complication (no postprocedural pneumonia, no progression of atelectasis, and no pneumothorax), and were discharged from the ICU to the general ward within 24 hours.

4. Discussion

4.1. Principal findings

In four consecutive ASA III patients with severe pulmonary disease, cricothyroid TTB combined with dexmedetomidine sedation allowed complex bronchoscopy, including transbronchial biopsy, to be completed without conversion to general anesthesia, vasopressor support, or desaturation.³⁷ Spontaneous ventilation was preserved, and cough was minimal (VAS $\leq 1/10$). These results describe feasibility and tolerability in a small, uncontrolled series. They

suggest that targeted airway de-afferentation with preserved spontaneous ventilation is a workable option for patients in whom general anesthesia and heavier sedation are hazardous.

4.2. Comparison with the published literature

The outcomes of other studies are summarized in Table 5. They differ in design, population, and definitions, so formal comparison is not possible. In patients having brachial plexus surgery under general anesthesia, dexmedetomidine as an adjunct to TTB gave hemodynamics comparable to those with propofol and a shorter time to extubation.¹ In painless FOB under deep sedation with spontaneous breathing, a reinforced nasal tube placed in front of the glottis was associated with less transient hypoxia, lower cough scores at passage through the glottis, and better hemodynamic stability than mask oxygenation.⁷ In flexible FOB, remimazolam with alfentanil reduced severe hypoxemia (3.4% vs 15.8%), hypotension, and cough grade compared with propofol with alfentanil.¹⁴ The statement that general anesthesia cohorts show post-induction hypotension that requires vasopressors in 25–30% of patients and more postprocedural atelectasis, and that conventional sedation cohorts show persistent coughing that requires interruption in 15–35%, is not supported by the cited review.³⁸ None of these studies used TTB for bronchoscopy in ASA III patients, so the present results cannot be compared directly. In the present series, there was no hemodynamic collapse, desaturation, or conversion to general anesthesia, and all patients left the ICU within 24 hours.

Table 5. Selected published studies of airway anesthesia or sedation for airway procedures, compared with the present series.

Study	Design and sample	Technique	Cough and operating conditions	Hemodynamics	Hypoxia or desaturation	Recovery and conclusion
Present series	ASA III, severe lung disease, n = 4	TTB with 2% lidocaine 20 mg plus dexmedetomidine 0.2–0.7 µg/kg/h	VAS ≤ 1/10 (0 in 3 of 4; 1 in 1 of 4)	MAP and HR within 13.3% of baseline; no vasopressors	None (lowest SpO ₂ 94%); spontaneous ventilation maintained	ICU discharge within 24 hours in 4 of 4; no conversion to GA
Rajan and Tosh 2019 ¹	Randomized trial, n = 40, brachial plexus surgery under general anesthesia	TTB before induction; dexmedetomidine 0.5 µg/kg then 0.5 µg/kg/h, or propofol 1 mg/kg/h	Propofol boluses for movement comparable between groups	Comparable at all time points	Not reported	Time to extubation 9.6 ± 11.5 vs 16.1 ± 5.8 min, shorter with dexmedetomidine
Zhang 2025 ⁷	Randomized trial, n = 64, painless FOB under deep sedation, spontaneous breathing	Nasal reinforced 5.0 tube to the front of the glottis, or oxygen mask with nasal cannula	Lower cough scores at passage through the glottis with the tube	Lower blood pressure from glottis passage to end of FOB and lower HR at glottis passage with the tube	Less transient hypoxia with the tube (P < 0.05)	Shorter induction time and fewer remedies with the tube; adverse events similar
Lv 2025 ¹⁴	Randomized single-blind trial, n = 118 (115 analyzed), flexible FOB	Alfentanil 10 µg/kg with remimazolam 0.2 mg/kg or propofol 2 mg/kg	Lower cough grade with remimazolam (P < 0.05)	Less hypotension with remimazolam	Severe hypoxemia 3.4% vs 15.8% (P = 0.029)	Shorter recovery and higher comfort scores with remimazolam

Notes: Values for the published studies are taken from the abstracts of the cited articles. ASA, American Society of Anesthesiologists; FOB, fiberoptic bronchoscopy; GA, general anesthesia; HR, heart rate; MAP, mean arterial pressure; TTB, transtracheal block; VAS, visual analog scale.

The authors rated the association between the protocol and the observed stability with the Naranjo scale (adapted to adverse hemodynamic and respiratory events; score +7, “probable”), the Bradford Hill criteria, and the WHO-UMC system (Table 4). Because the series has no comparator, these ratings describe the consistency of the observations with the proposed mechanism and should not be read as proof of causation.

4.3. Interpretation and possible mechanisms

The mechanisms discussed below are proposed from the literature and from the observed clinical response; none was measured in this study (Figure 6). Sensory innervation of the airway is compartmentalized.³⁹ The glossopharyngeal nerve supplies the posterior tongue, palatine tonsils, vallecula, and upper epiglottis and mediates the gag reflex.

The internal branch of the superior laryngeal nerve supplies the base of the tongue, epiglottis, and laryngeal mucosa down to the true vocal cords. Below the glottis, sensation is carried by the recurrent laryngeal nerve and the vagus nerve. The subglottic mucosa, tracheal rings, carina, and proximal mainstem bronchi contain myelinated rapidly adapting stretch receptors (RARs) and unmyelinated nociceptive C-fibers, which are densest at the carina. Mechanical contact with an unanesthetized carina produces an afferent discharge through the vagus nerve to the nucleus tractus solitarius and a reflex efferent response through the recurrent laryngeal and phrenic nerves, with glottic closure, forceful diaphragmatic and abdominal contraction, and paroxysmal coughing.

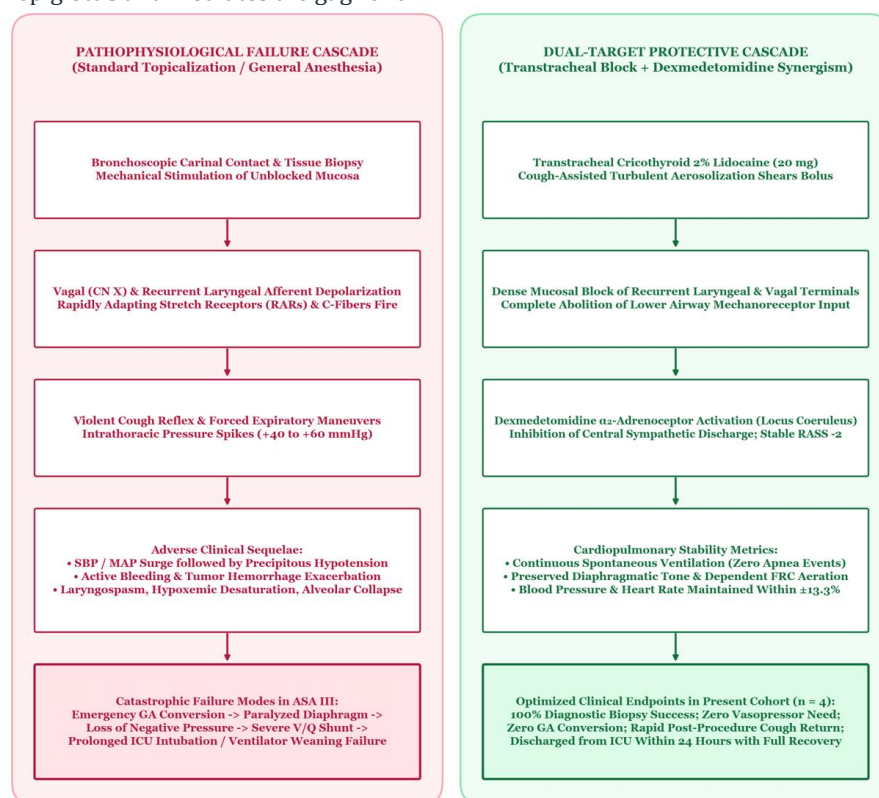


Figure 6. Proposed mechanistic cascade of cough-induced intrathoracic pressure surges and the protective effect of TTB with dexmedetomidine (schematic; not measured data). ASA, American Society of Anesthesiologists; FRC, functional residual capacity; GA, general anesthesia; RAR, rapidly adapting receptor; RASS, Richmond Agitation–Sedation Scale; SBP, systolic blood pressure.

These features may explain why conventional topicalization often fails in high-risk patients.⁴⁰ Spray-as-you-go topicalization relies on passive gravitational flow and unassisted inspiratory airflow to carry droplets across the glottis; in tachypneic patients with small tidal volumes, inspiratory flow is erratic. Droplets in the supraglottic larynx can also trigger vocal cord adduction that blocks entry into the distal trachea, leaving the lower tracheal rings and carina unanesthetized. TTB bypasses the glottis by puncture of the cricothyroid membrane. The proposed mechanism is that a 1.0-mL bolus injected at the start of deep inspiration provokes a single reflex cough whose expiratory airflow (stated in the submitted text to exceed 20–25 m/s) fragments the liquid into droplets of 10–50 μm that spread retrograde over the subglottic mucosa and the underside of the vocal cords and anterograde along the trachea to the carina and mainstem bronchi. In this series, cough was abolished or minimal ($\text{VAS} \leq 1/10$ in all four patients; 0 in three).

Nebulization typically requires 300–400 mg of lidocaine (7.5–10 mL of a 4% solution) because 70–80% of the output is lost to room air or tubing or is swallowed.⁴¹ In this series, the transtracheal dose was 20 mg (1.0 mL of 2% lidocaine), and the total dose, including pharyngeal spray and skin infiltration, was about 50 mg, roughly 6- to 8-fold lower than the nebulization dose quoted above. A lower dose may reduce the risk of local anesthetic systemic toxicity, which matters in frail patients with reduced hepatic clearance or cardiac dysfunction, such as Case 2 (BMI 15.6 kg/m^2 ; hepatic metastases), in whom impaired hepatic enzyme activity and reduced protein binding could favor drug accumulation. Plasma concentrations were not measured.

The physiological rationale for preserving spontaneous ventilation is as follows.⁴² In a spontaneously breathing supine patient, diaphragmatic contraction generates negative pleural pressure that is distributed preferentially to the dependent lung zones, where perfusion is also greatest, which supports ventilation–perfusion (V/Q) matching. Under general anesthesia with neuromuscular blockade, loss of diaphragmatic tone allows the abdominal viscera to move cephalad (2–4 cm in the submitted text) and compress dependent lung, which reduces functional residual capacity (FRC) by 20–30% within minutes of induction. In patients who already have lung collapse or pneumonia (Case 1) or left upper lobe collapse (Case 4), a further loss of FRC could cause marked right-to-left shunting and refractory hypoxemia. Because neuromuscular blockade was avoided and spontaneous breathing was maintained, diaphragmatic activity was presumably preserved. This is consistent with $\text{SpO}_2 \geq 94\%$ in all patients without positive-pressure support (mean nadir $95.5 \pm 1.3\%$), although FRC, shunt, and V/Q matching were not measured.

Avoiding positive-pressure ventilation also has hemodynamic implications.⁴³ Positive airway pressure raises mean intrathoracic pressure and right atrial pressure, narrows the gradient for venous return, and lowers right ventricular preload. This may cause marked hypotension in patients with large pleural effusions (Cases 1 and 3) or central mediastinal tumor burden. Positive-pressure ventilation also carries a risk of barotrauma and air trapping behind endobronchial tumor stenosis through a ball-valve mechanism. With continuous spontaneous breathing, negative intrapleural pressure swings during inspiration support venous return and preload. In this series, MAP stayed within 13.3% of baseline without vasopressors (Table 3), but hemodynamics under positive-pressure ventilation were not measured in these patients.

TTB does not provide sedation, so an awake patient needs a sedative adjunct to relieve anxiety and visceral discomfort.⁴⁴ Midazolam and short-acting opioids (fentanyl, remifentanyl, or alfentanil) give reliable anxiolysis but depress ventilatory drive, blunt the hypercapnic ventilatory response, and relax the pharyngeal dilator muscles, so even modest doses may cause hypoventilation or upper airway obstruction in ASA III patients with respiratory failure. Dexmedetomidine is a highly selective α_2 -adrenoceptor agonist ($\alpha_2:\alpha_1$ selectivity ratio 1620:1) that acts on $\alpha_2\text{A}$ receptors in the locus coeruleus, reduces central noradrenergic transmission, and produces a state resembling non-REM stage 2 sleep. Patients remain calm but rousable and able to follow instructions, such as taking a deep breath, and the hypercapnic ventilatory response is thought to be preserved (Figure 5). Its sympatholytic action blunts tachycardia and hypertensive responses to stress, which may be relevant in the older patients (Cases 2 and 4) and in the patient with hypertension (Case 3), in whom a sympathetic surge could provoke myocardial ischemia, left ventricular failure, or worsening hemoptysis. In this series, HR did not rise at carinal contact (mean change $-1.06 \pm 2.81\%$).

4.4. Implications for practice

Technical execution of TTB requires careful anatomical assessment, particularly when the neck is distorted.⁴⁵ Localization of the cricothyroid membrane was straightforward in Cases 1, 2, and 3. In Case 4, thyroid metastases distorted the midline and obscured the surface landmarks; dynamic palpation of the superior thyroid notch and the inferior cricoid ring, with continuous aspiration during needle advancement, allowed safe single-pass endotracheal access without vascular puncture or hematoma. Landmark-guided cricothyroid puncture is reported to fail or cause complications in up to 10–15% of patients with morbid obesity, large goiter, or previous cervical radiotherapy. Point-of-care ultrasound could be used to visualize the cricothyroid membrane, measure the skin-to-membrane depth, and identify overlying vessels, which may extend the use of TTB to patients with distorted neck anatomy; it was not used in this series. In patients in whom general anesthesia and heavier sedation carry a high risk, TTB with dexmedetomidine may be considered within institutional protocols, pending controlled evidence.

4.5. Strengths

The strengths of this study are consecutive enrollment, predefined hemodynamic and desaturation thresholds, serial per-patient measurements at standardized milestones, a detailed description of the block technique, and the inclusion of a patient with distorted cervical anatomy.

4.6. Limitations

This study has several limitations. First, it includes only four consecutive patients, so the findings are descriptive and provide a physiological proof of concept; they cannot replace randomized controlled trials for establishing population-level efficacy, and the absence of events in four patients cannot exclude a clinically important event rate. Second, there was no comparison group that received positive-pressure general anesthesia, so comparisons of operating-room efficiency, biomarker release, and long-term pulmonary outcomes are not possible, and the benchmarks in Table 5 are indirect. Third, the patients were all men treated at one center, and it is not stated who recorded the VAS cough scores or whether the assessor was independent of the anesthesiologist who performed the block.

4.7. Recommendations for future research

Multicenter randomized controlled trials should compare TTb with dexmedetomidine against total intravenous anesthesia with propofol and remifentanyl in high-risk thoracic patients, with hard clinical endpoints such as postoperative pulmonary complications, hospital length of stay, and health-care costs.

5. Conclusion

In four consecutive ASA III patients with severe pulmonary disease, cricothyroid transtracheal block combined with titrated dexmedetomidine sedation allowed complex fiberoptic bronchoscopy, including transbronchial biopsy, to be completed without conversion to general anesthesia, vasopressor support, or desaturation. Spontaneous ventilation was preserved, cough was minimal ($VAS \leq 1/10$), MAP and HR stayed within 13.3% of baseline, and all patients were discharged from the ICU within 24 hours. These findings support the feasibility of the protocol in high-risk patients, particularly when general anesthesia or deeper sedation is hazardous. Because the series is small, uncontrolled, and from a single center, the efficacy and safety of the protocol relative to general anesthesia or conventional topicalization should be tested in controlled studies.

6. Declarations

Ethical approval and consent to participate. The study followed the Declaration of Helsinki (2013 revision) and was approved by the CMHC Research Center Ethics Committee, Indonesia (approval number 2026/0418). Written informed consent was obtained from all participants and their legally authorized representatives before the procedures.

Consent for publication. Written informed consent for the publication of anonymized medical history, clinical parameters, intraoperative physiological trajectories, and associated diagnostic data was obtained from all patients and their legal guardians.

Availability of data and materials. The de-identified datasets, monitoring recordings, and calculation sheets that support the conclusions of this article are available from the corresponding author on reasonable request for academic and clinical research purposes.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tool was used in the preparation of this manuscript.

Author contribution statement. Y.I.P.: Conceptualization, Investigation, Data curation, Writing – original draft. R.H.: Methodology, Supervision, Writing – review & editing. M.F.Y.: Investigation, Data curation, Formal analysis, Writing – review & editing. All authors approved the final version of the manuscript.

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