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# Perioperative Silent Lung Induced by Coughing During Extubation in an Elderly Male with Coronary Atherosclerosis and Suspected COPD: A Case Report

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## Abstract

Silent lung is an extreme and life-threatening manifestation of severe bronchospasm, characterized by marked attenuation or complete disappearance of bilateral breath sounds and abrupt loss of the end-tidal carbon dioxide ($EtCO_2$) waveform. This condition may rapidly progress to severe hypoxemia and cardiac arrest. Early recognition, prompt diagnosis, and standardized intervention are critical for reversing bronchospasm and preventing catastrophic perioperative complications.

Current clinical studies mainly focus on the prevention of common perioperative airway adverse events, whereas standardized and systematic management protocols specifically for extubation-induced silent lung remain lacking. Herein, we report a case of silent lung triggered by coughing during extubation in an elderly male patient following right knee arthroplasty. Timely anesthetic intervention resulted in successful resuscitation and smooth extubation. The patient was transferred uneventfully to the general ward, without subsequent pulmonary complications or prolonged hospitalization.

This case supplements current clinical experience regarding silent lung management and provides a reference for individualized anesthetic care in high-risk elderly patients with underlying cardiovascular and airway diseases.

## Keywords

Silent Lung, Bronchospasm, Extubation, Remifentanil, Ciprofol, Coronary Atherosclerosis

## Introduction

Perioperative silent lung is the most severe subtype of acute bronchospasm, predominantly triggered by asthma exacerbation, anaphylactic reactions, and perioperative mechanical airway stimulation. Typical manifestations include sharply elevated airway resistance, difficult ventilation, diminished thoracic movement, absent breath sounds on lung auscultation, disappearance of the $EtCO_2$ waveform, and progressive severe hypoxemia. If unrecognized and untreated, silent lung-induced hypoxemia may rapidly progress to cardiac arrest.

Mild-to-moderate perioperative bronchospasm can usually be relieved effectively through conventional supportive and pharmacological interventions. Existing studies mainly focus on preventive strategies, management of anaphylaxis-mediated bronchospasm, and routine bronchodilator administration [1]. However, silent lung represents a far more critical condition that may lead to irreversible catastrophic respiratory and circulatory events. To date, clinical studies on perioperative silent lung remain limited, and systematic diagnostic criteria and optimal therapeutic protocols have not yet been established.

We report the case of a 72-year-old male patient who developed typical silent lung after transient coughing during extubation following elective right knee arthroplasty. Combined administration of remifentanil and ciprofol successfully reversed severe bronchospasm without the need for epinephrine rescue therapy. The patient recovered smoothly without postoperative complications. This case analyzes the risk factors, pathogenesis, diagnosis, and individualized treatment of extubation-related silent lung, and summarizes practical anesthetic management experience for similar high-risk elderly patients.

## Case Presentation

A 72-year-old male patient weighing 74 kg was classified as American Society of Anesthesiologists (ASA) physical status II. The patient had a history of hyperthyroidism treated with radioactive iodine-131, which subsequently resulted in permanent hypothyroidism requiring long-term oral levothyroxine replacement therapy. He also had grade 2 hypertension, which was well controlled with oral amlodipine and irbesartan. Coronary computed tomography angiography revealed moderate coronary atherosclerosis, whereas routine preoperative electrocardiography, transthoracic echocardiography, and other laboratory examinations showed no obvious abnormalities.

Notably, the patient had a smoking history of more than 40 years with a cumulative smoking index of 800 pack-years and had quit smoking 4 years previously. He continued to experience chronic cough and sputum production. Preoperative arterial blood gas analysis under room air revealed a partial pressure of oxygen ($PaO_2$) of 57.9 mmHg, partial pressure of carbon dioxide ($PaCO_2$) of 38.8 mmHg, and arterial oxygen saturation ($SaO_2$) of 94.4%, consistent with type I respiratory failure. Chest computed tomography demonstrated pulmonary bullae in the left lower lobe and scattered chronic inflammatory changes in both lungs, with the thoracic anteroposterior-to-transverse diameter ratio approaching 1:1. Although the patient denied a definitive diagnosis of chronic obstructive pulmonary disease (COPD) and preoperative pulmonary function testing was not completed, comprehensive evaluation of the clinical symptoms, physical findings, and imaging features strongly suggested underlying undiagnosed COPD.

After entering the operating room, standard vital sign monitoring was established. Baseline vital signs were as follows: heart rate 72 beats/min, blood pressure 156/90 mmHg, and pulse oxygen saturation ($SpO_2$) 96% under room air. Lung auscultation revealed distant bilateral breath sounds without moist rales or wheezing.

Anesthesia induction was performed using intranasal dexmedetomidine spray 25 μg, sufentanil 20 μg, ciprofol 25 mg, and rocuronium bromide 70 mg. Tracheal intubation was completed uneventfully, and correct endotracheal tube placement was confirmed by lung auscultation and a stable $EtCO_2$ waveform. Volume-controlled ventilation (VCV) was initiated with the following parameters: fraction of inspired oxygen ($FiO_2$) 50%, tidal volume (TV) 500 mL, inspiratory-to-expiratory ratio 1:2, respiratory rate 12 breaths/min, and baseline peak airway pressure maintained at approximately 15 $cmH_2O$. Anesthesia was maintained with continuous intravenous remifentanil infusion and inhaled sevoflurane. The total operative duration was 50 minutes, and the intraoperative course remained stable.

Before the end of surgery, oropharyngeal and tracheal suctioning were performed under adequate anesthetic depth, yielding only a small amount of thin airway secretions. At the end of surgery, sevoflurane inhalation and remifentanil infusion were discontinued immediately. After placement of a dental pad and oral cavity suctioning, 50 mg of 2% lidocaine was

administered intravenously, combined with 50 mg intratracheal instillation, to prevent extubation-related coughing. Ten minutes after completion of surgery, sugammadex 200 mg was administered for reversal of neuromuscular blockade.

Two minutes later, the patient developed transient violent coughing, followed by a sharp increase in peak airway pressure to 35-40 $cmH_2O$, while tidal volume decreased to less than 100 mL. Considering that the patient had not yet fully regained consciousness or spontaneous respiration, ciprofol 7.5 mg was administered intravenously immediately, and the ventilation mode was converted to manual bag ventilation with $FiO_2$ adjusted to 100%, APL valve pressure limited to 35 $cmH_2O$, and oxygen flow increased to 8 L/min. However, ventilation remained unsatisfactory, with persistent tidal volume below 100 mL, and the $EtCO_2$ waveform became flat and nearly disappeared.

At that time, cyanosis of the lips and oral mucosa was observed. Bilateral breath sounds were completely absent on auscultation, accompanied by mild elevations in heart rate and blood pressure, while $SpO_2$ rapidly declined from 97% to 66%. No cutaneous rash, angioedema, or other systemic allergic manifestations were observed. Considering severe reflex bronchospasm leading to silent lung, together with relatively stable circulatory status and no indication for emergency epinephrine administration, additional ciprofol showed limited efficacy. Therefore, remifentanil 100 μg was administered intravenously immediately.

Shortly after administration, ventilation improved significantly. Tidal volume increased from less than 100 mL to more than 200 mL, the $EtCO_2$ waveform gradually recovered, and $SpO_2$ rose to 85%. The APL valve pressure was then reduced from 35 $cmH_2O$ to 20 $cmH_2O$. Under the condition of maintaining tidal volume above 300 mL, ventilation was switched to pressure-controlled ventilation (PCV) with a pressure limit of 20 $cmH_2O$, respiratory rate 16 breaths/min, inspiratory-to-expiratory ratio 1:2, and minute ventilation stabilized above 4 L/min. During this period, heart rate and blood pressure gradually returned to stable levels, $SpO_2$ steadily increased to 95%, and $EtCO_2$ recovered to 45 mmHg.

The patient remained unconscious at this stage, and approximately 2 mL of viscous sputum was aspirated through the endotracheal tube. Repeat auscultation revealed distant bilateral breath sounds consistent with the preoperative status, without obvious wheezing rales. Five minutes after remifentanil administration, the patient coughed again and responded to verbal stimulation by opening his eyes. Manual ventilation with pressure limited to 20 $cmH_2O$ achieved tidal volume greater than 300 mL without recurrent elevation of airway pressure.

To avoid recurrent bronchospasm induced by secondary coughing, tracheal extubation was performed immediately with standby mask positive-pressure ventilation prepared. After extubation, the patient remained somnolent with regular thoracic movement and no upper airway obstruction. Vital signs remained stable, and continuous oxygen inhalation via face mask at 4 L/min was maintained until full recovery of consciousness. The patient was transferred to the general ward after recovery from anesthesia, achieved an uneventful postoperative course without secondary respiratory failure or obvious pulmonary complications, and was discharged on postoperative day 3.

## Discussion

### *Risk Factors for Perioperative Silent Lung in This Patient*:

This patient possessed multiple independent high-risk factors for silent lung. Chronic smoking, unresolved pulmonary inflammation, and suspected latent COPD collectively resulted in heightened airway hyperresponsiveness. Coexisting hypertension and coronary atherosclerosis impaired coronary reserve and reduced myocardial tolerance to physiological stress. Shallow anesthetic depth during extubation, combined with coughing, served as direct triggers for vagally mediated reflex bronchospasm. Prophylactic lidocaine failed to adequately suppress this airway reflex in the present high-risk patient.

### *Diagnosis and Differential Diagnosis of Silent Lung*:

The acute increase in airway pressure after coughing,

absent breath sounds, disappearance of the $EtCO_2$ waveform, and rapid onset of hypoxemia were consistent with the typical diagnostic manifestations of silent lung. This episode represented vagal reflex-mediated bronchospasm triggered by mechanical airway stimulation rather than allergic anaphylaxis. No systemic allergic manifestations were observed throughout the perioperative period. The underlying mechanism involved exaggerated vagal reflex activity causing transient bronchial spasm without inflammatory mediator release, which fundamentally distinguishes this condition from anaphylaxis-induced bronchospasm.

*Rationality of the Individualized Therapeutic Strategy*:

Epinephrine is considered the first-line treatment for life-threatening perioperative bronchospasm. However, it significantly increases myocardial oxygen consumption. In this elderly patient with coronary atherosclerosis and hypertension, the cardiovascular risks associated with epinephrine administration were considerable, making routine first-line use inappropriate [2].

Unlike anaphylactic bronchospasm, which requires epinephrine to inhibit mast cell degranulation and reduce mucosal edema, this reflex-induced silent lung involved neither allergic mediator release nor airway edema. Therefore, epinephrine was reserved solely as a rescue option rather than a primary therapeutic strategy.

The combination of remifentanil and ciprofol constituted an optimal individualized treatment regimen. Remifentanil has rapid onset and stable metabolism, allowing effective suppression of vagal airway reflexes and relaxation of bronchial smooth muscle. Ciprofol provides stable sedation with only mild hemodynamic suppression, thereby deepening anesthetic depth and reducing extubation-related stress. This regimen successfully reversed silent lung with only slight and tolerable hemodynamic fluctuations while avoiding the cardiovascular risks associated with epinephrine in high-risk patients.

*Limitations and Clinical Experience Summary*:

Extubation-related bronchospasm caused by shallow anesthetic depth is frequently underestimated compared with airway complications occurring during intubation. In this patient, prophylactic lidocaine demonstrated insufficient protective efficacy [3], mainly because of unrecognized severe airway hyperresponsiveness secondary to latent COPD.

The absence of preoperative pulmonary function testing represented a major clinical limitation and hindered accurate perioperative risk stratification. Standardized preoperative pulmonary assessment and targeted interventions may reduce airway hyperreactivity and help prevent extubation-related severe bronchospasm and silent lung [4].

Timely intervention and maintenance of stable circulatory function during acute hypoxemia prevented fatal cardiopulmonary arrest in this case. For elderly patients with combined pulmonary and cardiovascular comorbidities, comprehensive standardized preoperative risk assessment is essential. Enhanced extubation-stage anesthetic management and airway protection strategies may effectively reduce the incidence of silent lung in high-risk populations.

## Conclusions

Extubation-induced silent lung is a fatal perioperative emergency that can be easily triggered by coughing in elderly patients with airway hyperresponsiveness and coronary atherosclerosis. Secondary hypoxemia and sympathetic stress may lead to severe cardiovascular complications, including myocardial ischemia and malignant arrhythmias.

For stable reflex-mediated silent lung without evidence of anaphylaxis, remifentanil combined with ciprofol may serve as a safe and effective first-line therapeutic strategy while avoiding the cardiac risks associated with epinephrine administration. Epinephrine should be reserved as rescue therapy for cases with progressive hypoxemia or circulatory instability. Standardized preoperative risk stratification and rigorous anesthetic management during extubation are essential to reduce the incidence of silent lung in high-risk elderly patients.

## Conflict of Interest

The author has read and approved the final version of the manuscript and declares no conflicts of interest.

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