

# Severe Bronchospasm Following Bilateral T2-T5 Sympathectomy

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## Citation

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## Abstract

We describe a case report of a 32-year-old woman who suffered severe bronchospasm following a bilateral T2-T5 video assisted thoracoscopic sympathetic ganglion ablation. The patient required overnight sedation, mechanical ventilation, nebulized albuterol and a prednisone taper prior to extubation. The combination of the patient's asthma, and the bilateral sympathectomy, enhanced the risk and severity of this complication. A heightened awareness for bronchospasm should be considered when planning surgical therapy and anesthesia in patients being treated for hyperhidrosis, and special consideration be given to the risk benefit ratio of performing bilateral versus staged surgery in patients with reactive airway disease.

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## INTRODUCTION

Palmar hyperhidrosis is a condition associated with increased eccrine sweating. Treatment options include surgical ablation of the T2, T3 and T4 sympathetic ganglion. Video Assisted Thoracic Surgery (VATS) offers decreased morbidity and mortality, and has largely replaced an open approach to surgical sympathectomy. Risks of surgical sympathectomy include Horner's syndrome, pneumothorax, and compensatory hyperhidrosis.<sup>1,2,3,4,5,6,7,8</sup> We describe a case of a bilateral T2-T5 VATS sympathetic ganglion ablation that was complicated by severe bronchospasm.

## CASE REPORT

A 32-year-old woman was admitted to same day surgery for an elective bilateral T2-T5 VATS sympathetic ganglion ablation for palmar hyperhidrosis. Past medical history was significant for remote mild exercise-induced asthma.

After placement of standard ASA monitors, induction of anesthesia was accomplished with 100mg lidocaine, 250 mcg fentanyl, 200 mg propofol, and 80 mg rocuronium. To facilitate single lung ventilation, the airway was secured on the second laryngoscopy with a 35F left-sided double lumen endotracheal tube (DLT). Arterial blood pressure monitoring

was initiated post-induction, and anesthesia was maintained with sevoflurane, fentanyl, 100% oxygen, and rocuronium.

The patient was placed in left lateral decubitus position and the right lung was deflated. Shortly after T2-T5 sympathetic ganglion ablation, the delivered tidal volume decreased by 60 ml. Arterial blood gas revealed a pH 7.47, PaCO<sub>2</sub> 36 mmHg, PaO<sub>2</sub> of 87mmHg, Base excess 3. The patient's position was changed to right lateral decubitus and a left T2-T5 sympathetic ganglion ablation proceeded. Upon completion of the left sympathetic ganglion ablation, the patient was placed in the supine position for emergence from general anesthesia. Neuromuscular blockade was reversed with neostigmine 4mg and glycopyrrolate 0.8mg, and confirmed by five second sustained tetanus.

During emergence under volume control ventilation, the peak inspiratory pressure increased from 30 to 40cm H<sub>2</sub>O. In addition, an up-sloping end tidal CO<sub>2</sub> capnogram was noted. Decreased compliance with hand ventilation, and bilateral wheezing were suggestive of bronchospasm. Increased depth of anesthesia with sevoflurane and boluses of propofol resulted in improved compliance to hand ventilation, and resolution of the wheezing. The patient was further treated with multiple doses of albuterol MDI, and intravenous hydrocortisone. Symptoms of bronchospasm resumed during several attempts at emergence. A low dose propofol infusion was started, and fiberoptic bronchoscopy revealed erythema and mild edema in the conducting

airways. A chest x-ray was obtained and was normal. The DLT was replaced with a single lumen endotracheal tube, and the patient was transferred to the ICU on propofol sedation.

The patient was sedated and mechanically ventilated overnight, and received nebulized albuterol and a prednisone taper during post-operative day one (POD #1). On POD #2, the patient's peak inspiratory pressures and oxygenation had normalized, and repeat bronchoscopy showed significant improvement. The patient was extubated late on POD #2, and discharged from the hospital on POD #5 without further complication.

### DISCUSSION

Patients at risk for intraoperative bronchospasm include those with a history of asthma, COPD, or recent upper respiratory infection. Typical situations causing bronchospasm include mechanical or chemical irritation of the airways, pulmonary aspiration, pulmonary embolus, and administration of medications with beta-antagonist, anticholinesterase, or histamine releasing properties.<sup>9</sup>

Studies investigating the effects of thoracic sympathectomy on bronchial tone and pulmonary function have generated conflicting results. A prospective study of 47 patients undergoing bilateral thoroscopic sympathicotomy disclosed a minimal but permanent decrease in forced expiratory flow, and suggested that airway bronchomotor tone is influenced by sympathetic innervation in patients with hyperhidrosis.<sup>10</sup> A later study, including 10 patients who underwent bilateral thoroscopic sympathectomies, revealed no statistically significant loss of lung function or pulmonary complications.<sup>11</sup> Mohlo et al., studying 24 patients undergoing open thoracic sympathectomies, reported pneumoconstriction and small airway bronchoconstriction after upper dorsal sympathectomy.<sup>12</sup>

In our case, a patient with a remote history of asthma suffered severe bronchospasm following a bilateral T2-T5 VATS sympathetic ganglion ablation. An initial decrease in pulmonary compliance was demonstrated following ablation of the right T2-T5 sympathetic ganglion, and severe bronchospasm refractory to medical management occurred during several attempts at emergence. Although aspiration can lead to bronchospasm and fiberoptic findings similar to those seen in our patient, we believe the temporal relationship to the surgical sympathectomies, normal white blood count, normal serial chest x-rays, and lack of fever or post-operative pneumonia rules out this etiology. We believe

the combination of the patient's medical history for asthma, and the bilateral sympathectomy, was the major factor for the incident and severity of bronchospasm. We suggest that a heightened awareness for this complication be considered when planning surgery and anesthesia in patients with hyperhidrosis and reactive airway disease. We routinely use  $\beta_2$ -agonist prophylaxis in our patients undergoing bilateral VATS sympathectomy for palmar hyperhidrosis. We recommend further study on the physiologic effects of bilateral VATS on bronchial tone, and that special consideration be given to the risk benefit ratio of performing bilateral versus staged VATS in patients with reactive airway disease.

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