# Perioperative Wheezing—A Report of Three Cases

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## Abstract

***Clinical Picture**: We describe 3 patients with perioperative wheezing, 2 of whom were treated with bronchodilators on the presumptive diagnosis of bronchospasm. **Treatment and Outcome**: Subsequent clinical improvement occurred when it was recognised that the wheezing was due to upper airway obstruction and stridor, not bronchospasm and rhonchi, and appropriate treatment instituted.*

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## Introduction

Perioperative "wheezing" can be due to upper or lower airway obstruction. The signs and sounds of upper airway obstruction are typically worse during inspiration, whereas those of lower airway obstruction are worse in expiration. Distinguishing between the two is not always easy in acute situations, particularly in patients who suffer from asthma or chronic obstructive airway disease.

## Case Reports

### *Patient 1*

A 47-year-old female patient presented for right knee arthroscopy and adductor release of the right hip. She had a variant form of the spastic syndrome of cerebral palsy, involving only hypertonia of the hip adductors.[1] This chronic hypertonia had caused chondro-patella-femoral erosion, resulting in knee pain and limiting her mobility. She had no history of stridor, speech problems, gastro-oesophageal reflux, pseudobulbar palsy or swallowing difficulties. She had a history of childhood asthma, had had one successful pregnancy and was in good health.

She received premedication of 7.5 mg oral midazolam. General anaesthesia was induced with thiopentone 250 mg, fentanyl 100 μg and muscle relaxation achieved with atracurium 35 mg. Intubation with a size 7.0 cuffed endotracheal tube was easy. Anaesthesia was maintained using nitrous oxide and isoflurane and analgesia with morphine 5 mg.

The surgery lasted 45 minutes. Train of four (TOF) neuromuscular testing at the end of surgery showed four twitches of the adductor pollicis were present. Neuromuscular blockade was reversed with 2.5 mg of neostigmine and 0.9 mg of atropine. She was breathing well, extubated and sent to the post-anaesthetic care unit (PACU). On awakening, she developed stridor with oxygen desaturation. She had no rhonchi on chest examination. Immediate treatment included jaw thrust, head extension, and ventilation was assisted using an Ambu bag and oxygen. Her oxygen saturation improved to 95%. Despite being fully awake, the stridor persisted. Due to the patient's resistance and anxiety, direct laryngoscopy was not possible and transnasal fibreoptic laryngoscopy was performed to elucidate the cause of her stridor. This showed adduction of the vocal cords during inspiration. The patient, however, could phonate "e" and "o". No organic lesion was seen and the supraglottic area was normal.

The airway obstruction was treated by assisting spontaneous ventilation and applying continuous positive airway pressure (CPAP) manually, with the circle system of the PACU anaesthesia machine. She was instructed to breathe slowly and reassured that the airway obstruction would resolve. Incremental doses of morphine were given to achieve complete pain relief. The obstruction and stridor resolved completely over 60 minutes and she was discharged from the PACU 3 hours later. Stridor did not recur.

### *Patient 2*

A 70-year-old Caucasian female patient who presented with a 4-day history of persistent vomiting and abdominal

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pain was diagnosed to have intestinal obstruction. She had a history of asthma, and was obese. Preoperative management included rehydration with central venous pressure monitoring and correction of serum potassium abnormalities. Her maintenance on salbutamol and budesonide inhalers therapy were continued and intravenous hydrocortisone 100 mg was added. Her preoperative chest examination was normal and she had no wheezing.

She had general anaesthesia for an emergency laparotomy. Anaesthesia was induced with propofol 150 mg and maintained with desflurane, and morphine 15 mg was used for analgesia. Muscle relaxation was achieved initially with suxamethonium for rapid endotracheal intubation and maintained with rocuronium (TOF 0 to 1). Respiratory and haemodynamic monitoring were all normal and there were no signs of airway obstruction throughout surgery.

The intestinal obstruction involved the small and large intestine due to multiple incisional hernias from previous surgery. Extensive gut decompression, resection of non-viable gut and mesh repair of the abdominal wall was performed.

Anaesthesia was not reversed at the end of surgery and the patient was transferred to the intensive care for a period of rewarming, ventilation and monitoring. She was extubated 3 hours later when fully awake, normothermic, and breathing well. Immediately after extubation, wheezing occurred and the patient complained of difficulty in breathing. On the presumption that airway manipulation had triggered asthma, nebulised salbutamol treatment was prepared and started. However, chest examination showed no rhonchi. There was no impairment of oxygenation and both the chest X-ray and arterial blood gas tests were normal.

She was alert and could talk, but appeared very anxious. The patient's husband then stated that the same problem had occurred after surgery at another facility. The patient was treated with incremental doses of intravenous midazolam (total 7 mg) and the wheezing rapidly subsided. The rate of morphine infusion was also increased to 3 mg/h to treat her anxiety which was due to pain. No airway manipulation was required and the nebuliser treatment was stopped as it was of no benefit and may have contributed to her anxiety.

*Patient 3*

A 61-year-old male patient with Parkinson's disease had incision and drainage of a perianal abscess. Based on preoperative wheezing in the ward, a diagnosis of chronic obstructive airways disease was made. Surgery was delayed for 2 days while he was treated with nebulised salbutamol, ipatropium bromide and oral prednisolone. His usual medication [Sinemet (levodopa and carbidopa), benxhexol and bromocriptine]for Parkinson's disease was continued unchanged.

On the day of surgery, he had no wheezing and no dyspnoea. General anaesthesia was induced with propofol (100 mg) and maintained with sevoflurane 2.5%. Stridor occurred and an oropharyngeal airway was inserted in an attempt to relieve airway obstruction, but this appeared to worsen the stridor. The stridor was then thought to be due to larygospasm, and lessened as the depth of anaesthesia was increased. However, stridor persisted after the completion of surgery, and worsened in the PACU even though the patient was fully awake and able to talk. His upper airway was assessed as Mallampati Class 3 and difficult direct laryngoscopy was anticipated. Blind nasal intubation was performed with the patient breathing spontaneously as this was the most expedient method of securing the airway at the time. Stridor stopped immediately after intubation and there were no rhonchi on chest examination.

The patient was monitored in the ICU, his treatment for Parkinson's disease continued and he was extubated the next day. Stridor recurred, and fibreoptic transnasal laryngoscopy at this time showed that the vocal cords were held in adduction. He required reintubation as stridor worsened. Noting that his Parkinson's rigidity was worse compared to preoperatively, his Sinemet dosage was doubled, and extubation was then successful. There was no further 'wheezing' in the ward.

**Discussion**

"Wheezing" denotes partial airway obstruction. It is important to differentiate upper from lower (small) airway obstruction, as the treatment is different for both potentially life-threatening situations. The wheezing from functional upper airway obstruction has been described as "factitious" asthma or paradoxical vocal cord motion.[2,3]

Patient 1 and Patient 2 had "wheezing" i.e. stridor when awake, making airway obstruction from over-sedation unlikely. The time frame and dosages of muscle relaxants used, and normal responses on monitoring with a nerve stimulator make residual neuromuscular blockade very unlikely in both patients. The cessation of stridor with intravenous sedation supports the diagnosis of functional paradoxical vocal cord motion (PVCM) in these 2 patients attributed to hysterical conversion.[2-6] Typically, patients with this problem are able to vocalise, but when attempting to inspire, adduct their vocal cords. They improve with reassurance, good pain relief and sedation. This contrasts with airway obstruction from mechanical or neurological causes whereby sedation can convert a noisy, partially obstructed airway to a silent, completely obstructed airway. The patient with PVCM presents during situations of

emotional and physical stress. Eliciting the appropriate history may be helpful, as in Patient 2.

The other issue illustrated is the mistaken assumption that wheezing in patients with asthma or chronic airway disease is due to bronchospasm and/or bronchial oedema. Patient 3 was treated aggressively with therapy directed towards bronchial reactivity in the preoperative period. In retrospect, the preoperative wheezing may have in part been due to upper airway obstruction at the vocal cords, even though the patient apparently improved after bronchodilator treatment. Primary laryngospasm has been described as part of the general hypertonia of Parkinson's disease[7] and Patient 3 only recovered when his Sinemet dosage was increased.

The flexible fibreoptic laryngoscope enables safe direct visualisation of upper airway anatomy and function in these patients. The instrument, when applied with care in an awake and spontaneously breathing patient, should not worsen the obstruction. Definitive airway control can be achieved by railroading an endotracheal tube over the scope into the trachea,[8] an important therapeutic advantage. It may be the investigation of choice for the diagnosis and treatment of PVCM as it affords the clinician confidence in sedating a stridorous patient and avoiding intubation.

In conclusion, whilst the most familiar cause of wheezing is bronchial disease, upper airway stridor may be mistaken for lower airway bronchospasm. It is hoped that these case reports serve as a reminder of other aetiologies. PVCM is highlighted, albeit as a diagnosis of exclusion unless specifically aided by fibreoptic laryngoscopy.

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