Postoperative Complications due to Paradoxical Vocal Cord Motion

GREG HAMMER, M.D.,* DEBRA SCHWINN, M.D.,* HARRY WOLLMAN, M.D.†

Acute upper airway obstruction presenting as stridor in the adult is uncommon. The differential diagnosis includes foreign body aspiration, vocal cord paralysis, infectious processes, trauma, anaphylactic reactions, and masses involving the larynx and adjacent structures. Airway obstruction in adult and adolescent patients without identifiable organic etiology has been reported.1–7 Many of these patients have paradoxical vocal cord motion (PVCN) during direct laryngoscopy, defined as adduction of the true vocal cords on inspiration with abduction on expiration.1–4 The anesthetic management of patients with a history of PVCN has not been described. We present a case of such a patient whose postoperative course was complicated by persistent stridor.

CASE REPORT

A 55-yr-old woman was scheduled for lumbar laminectomy for L4 nerve root entrapment. Her past medical history was remarkable for two childhood hospitalizations for bronchitis and a history of chronic cough, wheezing, nasal polyps, and aspirin sensitivity. Five years prior to admission, wheezing was noted during a routine physical examination at a local hospital, where she worked as a nurse in a burn unit. At that time, broncho-dilator therapy was initiated. During the next 3 yr, she had frequent episodes of wheezing which she related to "stress at work." She required occasional hospital admissions for wheezing. On one occasion, endotracheal intubation was required for 24 h. She was referred for psychiatric evaluation, as her wheezing was felt to be exacerbated by stress. One and a half years prior to admission, she developed severe stridor while hospitalized for wheezing. Fiberoptic laryngoscopy during the stridorous episode revealed PVCN. She was later discharged without residual stridor. The laryngoscopic examination was not repeated.

The patient suffered from 10–12 episodes of stridor over the next 18 months, treated only with "relaxation techniques" at home. The stridor was only occasionally associated with wheezing and upper respiratory illness. She described cyanosis during one episode which lasted 1–2 h.

She previously smoked 1–2 packs of cigarettes per day, reduced to 2–3 cigarettes per day in recent months. She continued to have frequent paroxysms of coughing at home. Her surgical history included ovarian cystectomy, lumbar laminectomy, and cholecystectomy without complications, all prior to the onset of her stridor.

On physical examination, she was 168 cm and weighed 109 kg. Her arterial blood pressure was 150/90 mmHg. Her lungs were clear. Other than mildly decreased strength in her right leg, the remainder of her physical examination was unremarkable. Pulmonary function testing revealed a forced vital capacity of 3.81 (110% of predicted), a forced expiratory volume in 1 s of 3.21 (104% of predicted), a forced expiratory volume in 1 s to forced vital capacity ratio of 84%, and a maximum mid-expiratory flow rate (FEF25–75) of 2.91/s (98% of predicted). Flow-volume loops were normal.

Preoperatively, she received her usual doses of oral theophylline and terbutaline, as well as beclomethasone and metaproterenol aerosols. She was premedicated with cimetidine, meperidine, and scopolamine im. After the iv administration of midazolam and additional meperidine, her airway was topically anesthetized with 10% lidocaine spray, and bilateral superior laryngeal nerve blocks were performed using 4% lidocaine soaked pliclets. Her trachea was easily intubated with a 7.5 mm endotracheal tube over a fiberoptic bronchoscope. Marked paradoxical coughing resolved after additional doses of lidocaine, meperidine, and midazolam, and the patient turned herself prone. Anesthesia was induced with thiopental iv and maintained with inhalation of isoflurane and oxygen. Paralysis was maintained with pancuronium iv. Her lungs remained clear throughout the procedure. Upon completion of surgery, the patient was ventilated with 100% oxygen and demonstrated responsiveness to command and good muscle strength. Her trachea was then extubated. She immediately developed marked stridor, but maintained 100% oxygen saturation as monitored with pulse oximeter, and was taken to the recovery room breathing oxygen by mask.

In the recovery room, the patient became increasingly anxious and had sustained inspiratory stridor, despite repeated treatments with aerosolized racemic epinephrine. An arterial line was inserted, after which the Pao2 was 129 mmHg, PacO2 38 mmHg, and pH 7.39 mmHg. An aminophylline drip was begun, despite the absence of audible wheezing. While breathing 100% O2 with CPAP via a mask, the Pao2 was 402 mmHg, PacO2 33.4 mmHg, and pH 7.41 mmHg. The patient, however, appeared increasingly stridorous and fatigued. A nasal endotracheal tube was inserted in the trachea in immediate resolution of her stridor. A chest roentgenogram confirmed appropriate endotracheal tube position and showed no active disease. Because the patient appeared fatigued, ventilation was controlled for a period of 4 h without significant change in arterial blood gases, after which her trachea was extubated following a satisfactory CPAP trial. After 3 h of continued observation in the recovery room, the Pao2 was 130 mmHg, PacO2 29 mmHg, and pH 7.44 mmHg while breathing 50% O2 via venamask. At that time, due to the unavailability of an ICU bed, the patient was transferred to a ward observation bed continuously monitored with a pulse oximeter. Several hours later, her stridor worsened, and she appeared markedly fatigued, requiring reintubation and transfer to the ICU. Her stridor resolved following endotracheal intubation, after which mild wheezing was noted. Her wheezing was well-controlled with an aminophylline infusion, aerosolized metaproterenol, and decadron, but she had severe paroxysms of coughing requiring large doses of iv morphine and diazepam.

* Resident in Anesthesiology, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania
† Professor and Chairman, Department of Anesthesiology, Professor of Pharmacology, University of Pennsylvania, Philadelphia, Pennsylvania
Received from the Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania. Accepted for publication December 2, 1986.
Address reprint requests to Dr. Hamner: Department of Anesthesiology, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania 19104.

Key words: Airway: obstruction, vocal cords.

686
With a $FiO_2$ of 0.6 and assist-control ventilation, the $PaO_2$ was 154 mmHg, $PaCO_2$ 28 mmHg, and $PbH_2$ 7.45 mmHg. The following day, bronchoscopy was performed, revealing a normal-appearing trachea. The trachea was extubated at which time laryngoscopy revealed complete paradoxical motion of the true vocal cords. She required reintubation after failing to improve with iv diazepam and reassurance. A tracheostomy was performed with a $No. 6$ Shiley tracheostomy tube was performed in the operating room on the same day.

Over the next 24–48 h, she remained wheeze-free with bronchodilator therapy, but had recurrent paroxysms of coughing, despite large doses of iv narcotics and diazepam, superior laryngeal nerve blocks, and intratracheal and iv lidocaine. Four days later, the coughing episodes diminished, and a tracheostomy collar was placed. With a 50% $O_2$ mist collar, the $PaO_2$ was 88 mmHg. Direct laryngoscopy at that time revealed completely normal vocal cord motion. Her tracheostomy was decannulated on postoperative day number 16, but, the following day, stridor returned, and a $No. 4$ Shiley tracheostomy tube was inserted, as the stoma had diminished in size. Fiberoptic examination showed normal vocal cord movement, widely patent glottic chink, and the absence of laryngeal masses. She continued to be free of wheezing. She was later transferred to the rehabilitation ward due to persistent right lower extremity monoparesis, and was transferred to a local rehabilitation facility with a tracheostomy tube in place on the 28th postoperative day. There, her tracheostomy was again decannulated on the 58th postoperative day, at which time she was treated with a $He-O_2$ mixture for recurrent stridor, which resolved after 24 hours. She was eventually discharged home 6 weeks later, with her usual bronchodilator regimen, without further episodes of stridor.

**DISCUSSION**

Functional airway obstruction has been previously described in patients presenting with acute dyspnea and stridor in the absence of diagnostic findings on extensive physical examination. In many of these patients, laryngoscopy demonstrated PVCM. In a few cases, upper airway obstruction was severe enough to warrant tracheostomy. Additional cases of functional upper airway obstruction have been reported in patients with normal laryngoscopic findings; none of these patients required endotracheal intubation. In addition, five patients were identified in whom PVCM presented as paroxysms of wheezing. All of these patients had been previously given the diagnosis of asthma, although aggressive bronchodilator therapy had been ineffective.

These previously reported cases of functional disorders of the larynx have certain features in common. Nearly all patients were female. The majority of patients had histories of a variety of airway complaints, including “asthma,” hay fever, and allergic rhinitis. Many of these patients had recent upper respiratory illnesses. In those cases in which flow-volume studies were performed, the maximal expiratory and inspiratory flow-volume relationships were consistent with variable extrathoracic obstruction. In several reports, psychiatric evaluation demonstrated functional disorders to which the authors attribute the acute airway obstruction.

We have presented a patient sharing many clinical features with these previously described cases, including her history of chronic respiratory complaints, gender, and reported stress-related psychiatric history. She had a history of the triad of wheezing, nasal polyps, and aspirin sensitivity, which may be seen in approximately 10% of asthmatic patients. However, pulmonary function studies, including flow-volume loops, were done while the patient was wheeze-free and were normal. The etiology of her wheezing, therefore, remains unclear. This case presents the additional problem of anesthetic management. Our patient underwent general endotracheal anesthesia for lumbar laminectomy, developed severe stridor in the postoperative period, and required prolonged intensive care. Her stridor and respiratory distress persisted, despite pharmacologic therapy and supportive care, thereby necessitating tracheostomy. PVCM was repeatedly demonstrated by direct laryngoscopy during periods of stridor, while the patient had normal vocal cord movement when she was asymptomatic.

Patients with a history of functional upper airway obstruction, including PVCM, present a unique problem regarding anesthetic management. Particular attention to preoperative evaluation might include psychiatric consultation. Regional anesthetic techniques in which intubation is avoided should be strongly considered in cases in which access to the airway is not compromised. Although it is unknown whether mechanical stimulation of the airway may induce PVCM, endotracheal intubation and extubation while the patient is in a surgical plane of anesthesia may be desirable. When general endotracheal anesthesia is necessary, these patients require especially careful observation and monitoring in the postoperative period following extubation. Should stridor occur, laryngoscopy should be performed to confirm PVCM and rule out a separate etiogy. A trial of a $He-O_2$ mixture may prove beneficial. This gas mixture, with a density of roughly 1/3 that of $O_2$ or air, should reduce resistance to turbulent gas flow at the vocal cords. Persistent stridor may require tracheostomy when conservative measures have proven unsuccessful.

**REFERENCES**