ACUTE upper airway obstruction is a serious medical condition that requires prompt diagnosis and treatment. Usually, there is an organic cause for obstruction, but an increasing number of reports have documented cases without a definite organic etiology. Paradoxic vocal cord motion (PVCM) is a well-known cause of nonorganic upper airway obstruction and stridor in adults and children and has been reported frequently in the nonanesthesiology literature.1-3 There are only few reported cases occurring after general endotracheal anesthesia.4-9 In some of these cases, functional disorder of vocal cords was diagnosed by laryngoscopy only after unsuccessful medical therapy, reintubation, or tracheotomy.9,7,10 It is important to recognize this functional laryngeal disorder to avoid unnecessary diagnostic procedures and possibly harmful therapies.

We present a case report of a patient with stridor after general endotracheal anesthesia in whom PVCM was diagnosed by nasal fiberoptic laryngoscopy. The patient was treated with intravenously administered midazolam with complete cessation of stridor and return of normal vocal cord motion.

Case Report

A 24-yr-old woman presented for repair of arteriovenous malformation of the right thigh. Her medical history was remarkable for intermittent stridor initially diagnosed as exercise-induced asthma. When her symptoms persisted despite therapy, an indirect laryngoscopic examination revealed PVCM. The patient was treated with steroids but continued to have episodes of stridor.

On presentation for surgery, the patient was without dyspnea, stridor, or wheezing. The only medication she was taking was omeprazole (20 mg orally) for gastroesophageal reflux. The patient reported urticarial reactions to morphine and codeine. The patient had three previous episodes of general anesthesia without complication. Physical examination did not reveal stridor or wheezing. Oropharynx was Mallampati class I. Hemoglobin oxygen saturation was 100% on room air.

The patient was premedicated with 2 mg intravenously administered midazolam. Anesthesia was induced with 250 μg fentanyl and 2.5 mg/kg propofol. Rocuronium (40 mg) was used to facilitate tracheal intubation. Her trachea was easily intubated with a 7-mm endotracheal tube. Anesthesia was maintained with 50% nitrous oxide in oxygen and isoflurane 0.5-0.8%. An orogastric tube was placed without difficulty, the stomach was suctioned, and the tube was removed. Anesthesia was discontinued, and the neuromuscular blockade was reversed with 2 mg neostigmine and 0.4 mg glycopyrrolate given intravenously. Before extubation, the patient received 80 mg xylazine intravenously to attenuate laryngeal reflex irritability. Her trachea was extubated when she was fully awake, and adequate tidal volume, sustained head lift, and hand grip had returned. The patient was transferred to the recovery room with no evidence of respiratory distress. Hemoglobin oxygen saturation was 100% on room air.

Approximately 30 min after extubation, the patient developed inspiratory stridor. The patient, who was responding appropriately to verbal commands, appeared in moderate to severe respiratory distress, with upper airway obstruction and loud stridor. Auscultation of the chest demonstrated reasonable alveolar air entry without wheezing, and auscultation of the larynx revealed loud inspiratory stridor. Hemoglobin oxygen saturation remained at 98-100%. A differential diagnosis including soft-tissue upper airway obstruction, airway edema, laryngospasm, foreign body aspiration, and PVCM was entertained. Because hemoglobin oxygen saturation was well maintained and auscultation revealed reasonable air entry, bag and mask ventilation and muscle relaxants were not used. The patient was treated with 100 mg hydrocortisone given intravenously, 10 mg dexamethasone given intravenously, and racemic epinephrine by nebulizer with no improvement. An otolaryngologic consultation was obtained, and fiberoptic nasal laryngoscopy revealed PVCM with addition of the vocal cords with inspiration and abduction on expiration. No laryngeal edema, foreign bodies, or lesions were found. Assuming a functional airway obstruction, the patient was treated with 1.0 mg midazolam given intravenously with almost immediate resolution of her symptoms. Repeated laryngoscopy showed normal vocal cord motion. The patient was transferred to the ward from the recovery room after observation for 2 h without recurrent episodes and was discharged from the hospital the subsequent day without further sequelae.
Discussion

Functional upper airway obstruction with stridor has been described previously with differing nomenclature as psychogenic stridor,1 functional stridor,2,10 hysterical stridor,3 emotional laryngeal wheezing,11 functional upper airway obstruction,12 and Munchausen stridor13 by practitioners of various medical specialties.

Psychiatric evaluation of patients who present with the symptom complex has revealed emotional disturbances, anxiety, conversion disorders, hysterical traits, difficulty coping with stress, and emotional immaturity.3,4,10,12 Pulmonary function testing during stridor revealed extrathoracic airway obstruction12,14 or normal function.6,11

We are aware of six cases of PVCM previously reported in the anesthesiology literature.6-10 In the first of these case reports, a patient with a long history of respiratory symptoms and previously documented episodes of PVCM developed persistent severe stridor after general anesthesia and extubation. The diagnosis of PVCM was confirmed by laryngoscopy. Placement of a tracheostomy tube and prolonged intensive care was required after initial unsuccessful therapy with reassurance and parenteral treatment with narcotic agents and diazepam.8 In the second report, a previously healthy patient was reintubated in the recovery room after awakening from general endotracheal anesthesia because PVCM was not recognized as a functional and benign complication.7 In the third report, two patients who had previously required multiple intubations or tracheostomy were treated successfully in subsequent stridorous episodes with benzodiazepine agents (diazepam, lorazepam, midazolam).10 In the fourth case report, the diagnosis was established by laryngoscopy, and therapy with reassurance improved stridor and prevented an invasive airway intervention.8 In the fifth report, a patient developed stridor postoperatively that resolved after intravenous administration of hydromorphone for analgesia and verbal reassurance.9

In our case, inspiratory stridor developed in the fully awake patient with complete recovery from neuromuscular blockade, 30 min after extubation. After elimination of consideration of residual effects of anesthesia and neuromuscular blockade, the patient was initially treated for possible airway edema without improvement. Nasal fiberoptic laryngoscopy revealed normal airway anatomy without the presence of blood, mucus, foreign bodies, edema, or trauma. Functional upper airway obstruction was suspected, and PVCM was diagnosed after laryngoscopic evidence of adduction of vocal cords during inspiration and abduction during expiration. The patient was treated successfully with 1.0 mg midazolam given intravenously. The same therapeutic effect was reproduced with this patient in one later acute episode when the patient came to the emergency room with stridor. Tousignant and Kleinman reported a similar effect on PVCM with intravenously administered midazolam, which obviated the need for tracheostomy in their preoperative patient.10

A psychological component is usually proposed as part of the underlying mechanism of PVCM, and sedation with various medications has been used commonly. Benzodiazepine agents have been shown to have effect on peripheral and central receptors and are a logical choice for the therapy for PVCM. The Kratschmer reflex is attenuated by benzodiazepine agents.15,16 This protective airway reflex is evoked by mechanical or chemical stimulation of receptors located in the larynx and hypopharynx. Afferent conduction is by parasympathetic and sympathetic pathways, and the effector response results in temporary closure of the vocal cords. Central benzodiazepine receptor agonism results in release of the inhibitory neurotransmitter γ-aminobutyric acid,16 which may have a role not only in the central sedative effects of the benzodiazepine agents but also in their attenuation of this laryngeal reflex.

Paradoxical vocal cord motion should be considered in the differential diagnoses of postoperative stridor and upper respiratory obstruction. An increased awareness of this disorder will aid timely diagnosis. The diagnosis of a functional upper airway disorder should be made only after other more serious causes of airway obstruction have been ruled out and the characteristic findings have been identified by fiberoptic laryngoscopy. The differential diagnosis of postoperative stridor and upper airway obstruction must first focus on the elimination of life-threatening conditions requiring immediate therapy. The first step is to establish the adequacy of air entry and oxygenation. If these are inadequate, then continuous positive airway pressure, bag and mask ventilation, direct laryngoscopy, intubation of the trachea, or tracheostomy must proceed immediately as required. While establishing the adequacy of air entry, auscultation of the chest and larynx should be performed. The finding of loud laryngeal stridor and little wheezing helps to eliminate bronchospasm, pulmonary edema, or postobstructive pulmonary edema from consideration. If air entry and oxygenation are adequate, then fiberoptic laryngoscopy and, if necessary, bronchos-
case eliminate airway edema and foreign body aspiration from consideration and establish the diagnoses of PVCM. Once PVCM is diagnosed, reassurance and sedation with 0.5–1.0 mg midazolam given intravenously is recommended in the acute phase as a temporary measure for this benign condition, instead of aggressive airway intervention. All patients should be referred for psychiatric evaluation, anxiolytic therapy, speech therapy, and otolaryngologic follow-up.

References

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Perforation of the Right Ventricle with a Coronary Sinus Catheter during Preparation for Minimally Invasive Cardiac Surgery

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PERFORATION of the right atrium or ventricle with vascular catheters or wires has been reported many times. With the advent of minimally invasive cardiac surgical procedures, new technologies in monitoring and catheter placement are evolving. One such system of monitoring catheters has been developed by Heartport, Inc. (Redwood City, CA). This case report illustrates inadvertent right ventricular perforation with the proprietary endocoronary sinus catheter inserted via the internal jugular vein.

Case Report
A 64-yr-old, 1.65-m tall, 57-kg woman presented for minimally invasive coronary revascularization surgery. Preoperative history and physical examination were unremarkable except for an allergic re-