ACUTE inspiratory stridor in a patient recovering from the effects of general endotracheal anesthesia is a serious concern, because it may represent a life-threatening airway obstruction requiring immediate medical or surgical intervention. In a previously healthy preoperative patient, functional and organic anomalies of the upper airway can manifest postoperatively as acute inspiratory stridor. The differential diagnosis usually includes laryngeal stridor (from residual muscle relaxant effect), laryngeal spasm, vocal cord paralysis, intubation injury, hypocalcemic and alkalotic tetany, foreign body inhalation, angioedema, and undiagnosed tumors involving the upper airway. Abnormal vocal cord motion, although a well documented cause of stridor in other medical specialties, is usually not included in the differential diagnosis, because it is poorly documented in the anesthesia literature as a cause of recovery room stridor.1-5 This condition, therefore, may pose a significant diagnostic dilemma resulting in an inappropriate airway intervention. We present the case history of one such patient in whom timely diagnosis of this benign entity prevented an invasive airway intervention in the recovery room.

Case Report

A 49-yr-old healthy woman with a history of partial nasal obstruction presented to the ambulatory surgery center for nasal turbinatectomy and excision of a nasal polyp. She had no history of recent respiratory tract infections, allergies, asthma, or angioedema, and had not been taking any medications at the time of surgery or immediately before it. The only past medical history was that of anemia during pregnancy. The patient was 163 cm tall and weighed 85 kg. The systemic examination was unremarkable except for the airway, which was graded to be Mallampati class 2, with adequate thyromental distance.5 No abnormality was detected on a routine chemistry, hemogram, chest x-ray, and electrocardiogram.

The patient received 2 mg midazolam intravenously in the preoperative holding area. Anesthesia was induced with 100 µg fentanyl,
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80 mg lidocaine, and propofol in a 2.0-mg/kg bolus followed by a maintenance infusion of propofol ranging 100–200 μg·kg" sup"·min" sup". 

Atracurium (45 mg) was used to facilitate endotracheal intubation. Difficulty was experienced in visualizing the larynx on elective fiberoptic intubation. One attempt at direct laryngoscopy with a #3 MacIntosh laryngoscope blade (Rusch, Ontario, Canada) revealed a grade 3 visualization of the glottis: the glottis could not be expected; only corniculate cartilages could be visualized. The lungs were ventilated with 100% O₂, and fiberoptic intubation was resumed. Despite technical difficulty in visualizing the larynx, the actual insertion of the endotracheal tube over the fiberoptic bronchoscope was easy and met with no resistance. The oropharynx remained clear of blood when suctioned at the completion of intubation. The subsequent anesthetic course was uneventful. Surgery was completed over the next 30 min, the blood loss was minimal. Propofol was discontinued 5 min before expected completion of surgery, and the muscle relaxant effect was reversed with 5.5 mg neostigmine and 0.4 mg glycopyrrolate. The trachea was extubated when the patient was fully awake and when she demonstrated good muscle strength, as indicated by sustained head lift and hand grip. In the recovery room, hemoglobin oxygen saturation (SPO₂) was 98–100% while the patient breathed 40% O₂ from a face mask with no evidence of respiratory distress. Approximately 30 min after arrival to the recovery room, the patient complained of difficulty in breathing and desired to sit up. She assumed an upright sitting position with head bent forward and neck flexed. At this time, she was noted to have loud inspiratory stridor, nasal flaring, and suprasternal tug. Although appearing to be in significant respiratory distress and using accessory muscles of respiration, SPO₂ was maintained at 98–100%. When questioned, she indicated that she had experienced two similar but milder episodes of "wheezing" in the past year when she had "colds." These episodes did not require emergency medical intervention and resolved over a few hours. During conversation, it was observed that the patient did not exhibit any stridor while she spoke and could complete a sentence without evidence of respiratory distress. On physical examination, she was afibrile and normotensive. The heart rate was 76–82 beats/min, and the respiratory rate ranged between 24 and 30 breaths/min. Oropharyngeal examination revealed no evidence of injury or edema. The trachea was in the midline, and there was no evidence of swelling in the neck. Auscultation of the chest revealed good air entry with no evidence of wheezing.

The patient was treated with nebulized racemic epinephrine for possible airway edema. Nebulized salbutamol (Ventolin, Glaxo, England) was administered for possible bronchospasm (although no wheezing was detected clinically) with no relief in the severity of the symptoms. Thirty minutes after the onset of stridorous symptoms, the patient showed signs of fatigue, although SPO₂ remained at 98–100%. Because of progressive fatigue, an urgent otolaryngology consultation was obtained. Awake fiberoptic laryngoscopy revealed a normal nasopharynx, epiglottis, and true and false vocal cords. There was no evidence of trauma or edema. However, the true vocal cords exhibited a paradoxical motion, i.e., marked adduction during inspiration, which corrected itself when the patient was asked to cough, breath, or phonate the voice "e." Based on the findings of this direct examination, the otolaryngologists proposed a diagnosis of psychomotor (functional) laryngeal stridor. Distraction during the examination and verbal reassurance were observed to reduce the intensity of the stridor. Further verbal reassurance subsequently resulted in the spontaneous resolution of the symptoms over the following hour. The patient was admitted to the hospital for overnight observation and was discharged the following day without sequelae.

Discussion

The anesthesiologist, as an expert in airway management, must become familiar with both common and uncommon causes of airway obstruction. Acute inspiratory stridor in an otherwise normal patient emerging from the effects of general endotracheal anesthesia may be a harbinger of a life-threatening upper airway obstruction and, therefore, demands an urgent intervention. The differential diagnoses include: laryngospasm, laryngeal stridor, allergic reactions, angioedema, traumatic edema, foreign body aspiration, vocal cord paralysis, tetany, and undiagnosed tumors causing ball valve obstruction of the upper airway. Although rare, stridor due to paradoxical vocal cord motion needs to be considered in the differential diagnosis, because the awareness of this uncommon syndrome could prevent an unnecessary and potentially harmful airway intervention.

Common disorders that present as stridor in the recovery room include laryngeal spasm and laryngeal stridor. Laryngeal stimulation by blood, mucus, and/or a foreign body in a partially anesthetized patient precipitates laryngospasm, whereas depressed laryngeal activity from a residual muscle paralysis in a patient with active ventilatory effort produces laryngeal stridor. Both disorders tend to occur early in the recovery phase from residual anesthetic and muscle relaxant effects. Our patient had complete neurologic and motor recovery, indicated by the ability to sit up without support and converse without distress, before the stridorous symptoms first appeared. It is therefore unlikely that either of these laryngeal disorders were the cause of stridor in our case.

We ruled out hypocalcemic alkalotic tetany as a possible etiologic factor since serum calcium level was normal on the preoperative laboratory evaluation; and although the patient was tachypneic with a respiratory rate of 24–30 breaths/min, significant hyperventilation, which is characteristic of hypocalcemic alkalotic tetany, was absent. Also absent were other clinical signs and symptoms characteristic of tetany such as paresthesia, tingling, muscle cramps, and Chvostek's and Trousseau's signs. Furthermore, the resolution of stridor with vocalization and its immediate recurrence at the end of vocalization is not a characteristic feature of tetany.

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Diagnosis of inspiratory stridor secondary to foreign body aspiration, allergic airway edema (anaphylaxis, anaphylactoid reaction), hereditary or iatrogenic angioedema, vocal cord paralysis, and tumors of airway usually can be made through a relevant history and physical examination, i.e., clinical evidence of anaphylaxis in patients with allergic edema, dysphonia in patients with vocal cord paralysis, or facial and oropharyngeal swelling in patients with allergies and angioedema.9-10 Direct visualization of the airway would confirm the presence of abnormal pathology, such as trauma, edema, vocal cord paralysis, and supraglottic and glottic tumors.

The clinical presentation of this case was unusual in several aspects: (1) the onset of stridor was sudden and followed apparent complete neurologic and motor recovery; (2) the severity of stridor correlated poorly with the severity of respiratory distress; (3) there was no dysphonia, and the patient could complete a sentence without exhibiting respiratory distress; and (4) the patient preferred to assume a posture which compromised the upper airway, i.e., flexion of the neck. Since the patient could maintain normal oxygenation and appeared calm relative to the degree of stridor, we were hesitant to proceed with an aggressive airway intervention. Instead we chose to pursue definitive diagnostic intervention, such as upper airway endoscopy. Upon direct laryngeal examination, we did not detect any structural abnormality of the upper airway and found a functional abnormality in the vocal cord motion. Based on the findings of the upper airway endoscopy and after the exclusion of other organic and nonorganic causes of stridor, we established the diagnosis of functional stridor secondary to paradoxical vocal cord motion.

Functional airway obstruction has been described under several terminologies, e.g., hysterical stridor, psychogenic stridor, functional stridor, Munchausen stridor, and spasmodic croup.1,2,11,12 In a review by Skinner and Bradley, inspiratory stridor was the presenting feature in 54% of the cases, expiratory stridor in 10% of the cases, and biphasic stridor in 32% of the cases.13 Excessive adduction of vocal cords during expiration may present as wheezing, resulting in the diagnosis of bronchial asthma in several cases.14 Variability in pathophysiology and clinical presentation apparently explains the multiple terminologies used to describe the syndrome. The syndrome has been reported by several medical specialties, including as emergency medicine, allergy, pediatrics, otolaryngology, and pulmonary medicine.1-4,9,12 To our knowledge, only three case reports related to this disorder have been published in the anesthesiology literature.5,15,16 Of these case reports, in one the diagnosis had been established preoperatively,15 and in another the patients had stridor when they presented for anesthesia.16 There is only one case report in which the postoperative stridor in a previously healthy patient was attributed to the paradoxical vocal cord, and because the authors were not aware of the benign nature of the stridor, the patient was reintubated in the recovery room.5

The underlying mechanisms that precipitate the functional stridor (paradoxical vocal cord motion) are unclear and the patients themselves may be unaware of their role in producing the obstruction. Ward et al. proposed a central neurogenic etiology and suggested sedation as a treatment modality.17 Kellman and Leopold suggested that upper respiratory infection or mechanical stimulation of the airway, such as endotracheal intubation, may direct patients attention to the larynx and precipitate stridor.4 Psychologic evaluation may reveal underlying anxiety, depression, emotional disturbance, or stress-related disorders in 85% of the patients.13

To conclude, functional stridor from paradoxical vocal cord motion or laryngeal dysfunction should be included in the differential diagnosis of acute inspiratory stridor occurring in the postanesthesia recovery room. The diagnosis of this rare entity in a postoperative patient should be made by exclusion, after consideration of both common and uncommon disorders affecting the patency of the upper airway. A high index of suspicion, a proper history, and direct visualization of the upper airway anatomy are essential to the diagnosis. A conservative approach consisting of reassurance, neck extension (sniffing position), coughing, mouth breathing, sedation, and continuous positive airway pressure may terminate an episode effectively. The patient may require psychologic evaluation to rule out underlying anxiety, depression, or psychosomatic disorder, such as “conversion reaction.” The importance of prompt diagnosis cannot be overemphasized; a timely recognition of this benign disorder could prevent potentially harmful airway intervention, such as endotracheal intubation and tracheostomy.

References

SEVERE bronchospassm during anesthesia and surgery can be life-threatening, and, fortunately, the incidence is low.\textsuperscript{1} The few reported cases of bronchospassm during cardiac surgery have been noted to occur at termination of cardiopulmonary bypass (CPB), after patient rewarming. We report the case of a patient with asthma who developed severe bronchospassm associated with patient cooling during CPB, and we suggest an additional therapeutic modality in the treatment of intraoperative bronchospassm.

\textbf{Case Report}

A 64-yr-old, 82-kg woman presented with a 6-month history of worsening exertional angina and dyspnea on exertion. There was no history of orthopnea, paroxysmal nocturnal dyspnea, ankle edema, or myocardial infarction. Other medical history was significant for well controlled hypertension, noninsulin-dependent diabetes, retinal hemorrhage, and a 30-yr history of asthma. Because her asthma was worsened by cold weather, she regularly spent the winter in the warmer climates. She had been treated with $\beta$-agonist inhalers for many years and had received an aminophylline preparation, but not for the last year. Her last asthma attack was 8 months before admission and was relieved by the use of a $\beta$-agonist inhaler. She never required tracheal intubation to treat her asthma and did not report recent upper or lower respiratory infections. She was allergic to penicillin and iodine, with an allergic manifestation to both of a rash, and not bronchospassm. Her preoperative medication included albuterol and beclomethasone inhalers, glipizide, diltiazem, enalapril and a nitrroglycerin patch. Although she had never received systemic steroids...