nary area all presented as hemoptysis.\textsuperscript{3,4}

Little is known about the etiology of bronchial aneurysms. Either an increased systemic blood flow to the lungs or a mural pathology of bronchial arteries seems to be involved. Vascular diseases such as hereditary hemorrhagic telangectasia (Rendu-Osler-Weber disease), bronchiectasis, mycotic degeneration of the vessel wall, and atherosclerosis have all been recognized as predisposing factors for the development of bronchial artery aneurysms.\textsuperscript{5,6}

Both surgical intervention and percutaneous embolization as well as the combination of these two techniques have been proposed as a treatment.\textsuperscript{5,6} The physical condition of the patient can play an important role in the decision-making concerning which treatment suits the patient best. In the case reported herein, embolization seemed to be the right choice to control the bleeding and to stabilize the patient prior to a later planned thoracotomy.\textsuperscript{6}

REFERENCES


Coexistent Asthma and Functional Upper Airway Obstruction*

Case Reports and Review of the Literature

Ashraf A. Elshami, MD; and Gregory Tino, MD

Three asthmatic patients with dyspnea and episodes of apparent bronchospasm unresponsive to conventional therapy are described. During these episodes variable extrathoracic upper airway obstruction and airflow limitation typical of bronchial asthma were demonstrated by spirometry test results. In one patient, paradoxical vocal cord motion was identified by fiberoptic laryngoscopy. We believe these patients represent an unusual subgroup of asthmatic subjects who manifest laryngeal dysfunction. Recognition of this upper airway component to airflow limitation in some asthmatic patients may help physicians avoid potentially unnecessary therapy with systemic steroids and endotracheal intubation.  \textit{(CHEST} 1996; 110:1358-61\textit{)}

Key words: asthma; laryngeal dysfunction; paradoxical vocal cord motion; stridor; upper airway obstruction

A number of reports have described patients with laryngeal dysfunction and paradoxical vocal cord motion on inspiration associated with upper airway obstruction and stridor.\textsuperscript{1-5} These patients may demonstrate acute distress and require emergency endotracheal intubation or tracheostomy.\textsuperscript{1-4,6,7} Patterson et al\textsuperscript{6} named such cases in which no organic abnormality could be found as "Münchausen’s stridor." Vocal cord dysfunction may also present as wheezing which mimicks asthma.\textsuperscript{5,8} These individuals have been shown to have adduction of the vocal cords throughout the respiratory cycle with no obvious organic cause for upper airway obstruction or evidence of bronchial hyperreactivity typical of asthma.

Unlike these previous reports of laryngeal dysfunction, we describe three patients who had coexisting functional upper airway obstruction and lower airway obstruction typical of asthma. These patients presented with episodes of dyspnea which persisted despite conventional treatment for asthma and required specific intervention for laryngeal dysfunction. These observations suggest that some asthmatic subjects may have a laryngeal component to airway obstruction and that recognition of this component may have important therapeutic implications.

Case Reports

**CASE 1**

A 31-year-old non-smoking man, with steroid-treated asthma since the age of 20 years, was admitted to the hospital with a 3-day history of severe dyspnea and wheezing. The patient had a medical history of bronchospasm treated on a long-term basis with orally administered steroids; the bronchospasm was usually exacerbated by upper respiratory tract infections. He was intubated for asthma at least 13 times between the ages of 20 and 30 years. His symptoms were unrelieved at home with inhaled albuterol and orally administered prednisone, 100 mg daily for 3 days. He denied any recent upper respiratory tract or systemic symptoms.

On examination, he was in severe respiratory distress. His heart rate was 134 beats per minute with a respiratory rate of 30 breaths per minute and accessory muscle use. Diffuse inspiratory and expiratory wheezing was audible on chest auscultation. No stridor was noted.

The patient did not respond to albuterol treatments via nebulizer and was endotracheally intubated in the emergency department. Following intubation, only minimal wheezing was heard on examination; the peak inspiratory airway pressure was 25 cm H\textsubscript{2}O during the time the patient was mechanically ventilated. Arterial blood gas values with the patient receiving 100% oxygen were pH, 7.48; P\textsubscript{O}2, 435 mm Hg; and P\textsubscript{CO}2, 32 mm Hg. Steroid therapy with methylprednisolone, 60 mg administered intravenously every 6 h, was continued.

The patient was subsequently extubated within 24 h of arrival at the hospital. Spirometry tests performed (while the patient was a-
symptomatic) on day 2 revealed an FEV<sub>1</sub> of 3.30 L (88% predicted), and an FEV<sub>1</sub>/FVC ratio of 72%; maximal flow-volume loop demonstrated flattening of the inspiratory limb (Fig 1), which was consistent with variable extrathoracic upper airway obstruction. On review of prior records, there was evidence of severe airflow limitation that was consistent with a diagnosis of bronchial asthma on past occasions. Speech pathology evaluation noted intermittent inspiratory stridor. The patient was unaware of this, but the problem was corrected with trials of diaphragmatic breathing via the nares. Subsequent pulmonary function tests showed no further evidence of upper airway obstruction. Values for the FEV<sub>1</sub> and FEV<sub>1</sub>/FVC ratio remained within normal limits. Speech therapy and a tapering of the steroid dosage were planned for the patient after discharge.

Case 2

A 53-year-old woman with a medical history of asthma since childhood developed a productive cough and dyspnea for which she was treated with orally administered erythromycin. Over the ensuing 3 days, however, she had progressive dyspnea. Her last exacerbation of asthma had occurred 5 years earlier and was associated with a viral upper respiratory tract infection. She smoked a pack of cigarettes per day for 10 years. When she came to the emergency department, she had respiratory distress, decreased breath sounds bilaterally, and minimal wheezes that were heard diffusely over the entire chest. Arterial blood gas values with the patient breathing 100% from a nonrebreather mask were pH, 7.44; P<sub>O</sub>2, 349 mm Hg, and P<sub>O</sub>2, 41 mm Hg. Clinically, there was improvement in her symptoms after therapy with nebulized albuterol. She was admitted to the hospital, and therapy with intravenously administered methylprednisolone, 60 mg every 6 h, was begun. Several hours after admission, she had an acute worsening of dyspnea, accompanied by scattered wheezes and minimal air movement, which were detected by chest auscultation. On further observation, the wheezing appeared to be loudest over the neck. She was treated with 0.3 mg of epinephrine subcutaneously with some relief of her symptoms. Fiberoptic laryngoscopy revealed impressive paradoxical adduction of the true vocal cords on inspiration with marked narrowing of the airway. The cords moved normally with phonation. Pulmonary function testing not only confirmed the presence of a variable extrathoracic obstruction, but also revealed a component of expiratory flow limitation with an FEV<sub>1</sub> value of 1.64 L (56% predicted), an FVC of 2.42 L (72% predicted), an FEV<sub>1</sub>/FVC ratio of 68%, and a reduced specific conductance of 0.08 cm H<sub>2</sub>O/s (normal range = 0.13 to 0.35) as measured by body plethysmography. There was no improvement with the use of bronchodilators (Fig 2).

Her symptoms were felt to be primarily due to vocal cord dysfunction. The patient received speech therapy and steroid dosage was tapered over a 2-week period.

Case 3

A 24-year-old non-smoking medical student, developed upper respiratory tract symptoms marked by paroxysmal coughing 2 months prior to examination. After these episodes, she would develop inspiratory stridor which would resolve spontaneously within several minutes. She had a history of seasonal allergies, but no history of asthma. At the time of examination, she had wheezing that was heard over the entire chest. Pulmonary function testing revealed expiratory flow limitation with an FEV<sub>1</sub> of 1.72 L (46% predicted), an FVC of 3.73 L (81% predicted), and an FEV<sub>1</sub>/FVC ratio of 46%, which improved with nebulized albuterol therapy to 60%. The flow-volume relationship showed a flattening of the inspiratory limb, which was consistent with a variable extrathoracic obstruction (Fig 3).

She was treated with a course of systemic steroids and further spirometry showed improvement in the FEV<sub>1</sub> value to 3.6 L (97% predicted) and the FEV<sub>1</sub>/FVC ratio to 91%. The inspiratory airflow also improved but still showed persistent flow limitation with the ratio of the maximal inspiratory flow rate at 50% vital capacity (VC) to the maximal inspiratory flow rate at 50% VC to be 1.3. She continued to have episodes of inspiratory stridor one to two times daily. These symptoms also occurred with exercise. Fiberoptic laryngoscopy showed no structural or functional abnormalities during an asymptomatic period. Maintenance therapy with inhaled steroids and inhaled albuterol was prescribed.
in asthma and COPD. Lisboa et al. studied pressure flow curves in a group of asthmatic patients and found inspiratory resistance exceeded expiratory resistance during panting maneuvers. Later Collett et al. found significant expiratory glottic constriction in asthmatic subjects during histamine-induced bronchospasm. They suspected a glottic source of upper airway obstruction that may have a pathophysiologic role in asthma, analogous to pursed-lip breathing. The mechanism for this finding remains unclear, but there is evidence that mechanoreceptors in the chest wall and lower airways may affect the laryngeal aperture. We postulate that a subgroup of asthmatic patients may have a loss of regulation of the normal reflexes controlling the laryngeal muscles, and this may result in overrecruitment of glottic constrictor muscles. Excessive glottic narrowing may occur during inspiration, expiration, or throughout the respiratory cycle, resulting in increased airway resistance. A better understanding of the mechanism responsible for motor control of the larynx and its contribution to airway resistance are needed to determine the role of glottic narrowing in asthma.

Distinguishing variable extrathoracic obstruction from intrathoracic lower airway obstruction characteristic of asthma by clinical examination can be difficult. The value of laryngoscopy in this group of disorders is also variable. During symptomatic periods, adduction of the vocal cords on inspiration and/or on expiration may be present. In addition, laryngoscopy can be normal during asymptomatic periods as observed in patient 3. This syndrome also has been confused with unilateral and bilateral vocal cord paralysis on laryngoscopy.

Treatment for functional upper airway obstruction has met with varied success. A range of psychological therapies have been anecdotally reported to be effective. Speech therapy, directed at relaxation techniques and training of vocal cords, has been used successfully. For more resistant cases, psychiatric or psychological support, as well as hypnotherapy, have been added. Two patients in this report required speech therapy intervention in addition to specific therapy for asthma.

In summary, we report three cases of asthma associated with functional upper airway obstruction contributing to airflow limitation. The importance of recognizing this component in asthmatic patients is severalfold, including (1) avoidance of unnecessary medications; (2) prevention of emergency intubation or tracheostomy; (3) the need for psychiatric and speech therapy intervention. These observations also suggest that a more extensive study of functional upper airway obstruction in asthmatic subjects is needed.

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![Flow-volume relationship for patient in case 3](http://journal.publications.chestnet.org/pdfaccess.ashx?url=data/journals/chest/21739/)
Oxygenation Improvement With Nitric Oxide in Right-to-Left Shunt Without Significant Effects on Pulmonary Arterial Pressure*

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Following surgical closure of an interventricular communication complicating an anterior myocardial infarction, a 74-year-old woman developed severe right ventricular failure and hypoxemia due to the opening of a patent foramen ovale (PFO). Mean pulmonary artery pressure was 24 mm Hg. Treatment with inhaled nitric oxide (5 ppm) increased PaO₂ from 47 to 90 mm Hg (FIO₂ 1). The present observation points out that nitric oxide inhalation could be useful in the management of severe hypoxemia from a right-to-left shunt due to a PFO even when there is no significant pulmonary hypertension present.

(CHEST 1996; 110:1361-63)

Key words: cardiac surgery; hypoxemia; myocardial infarction; nitric oxide; patent foramen ovale; postoperative care; respiratory failure; right ventricular failure

Abbreviations: MPAP=mean pulmonary artery pressure; PFO=patent foramen ovale

Inhaled nitric oxide has been used for the treatment of pulmonary hypertension either idiopathic (primary) or secondary to various diseases such as ARDS, congenital heart disease, or persistent pulmonary hypertension in the newborn.1-4

One may postulate that inhaled nitric oxide may reduce pulmonary vascular resistance even when there is no significant pulmonary hypertension and thereby decrease the severity of an intracardiac right-to-left shunt. In a patient with severe hypoxemia associated with the opening of a patent foramen ovale (PFO), we observed that therapy with inhaled nitric oxide improved gas exchange without significant effect on the pulmonary artery pressures.

CASE REPORT

A 74-year-old woman was referred to Erasme University Hospital from another hospital for surgical correction of a ventricular defect complicating an anterior myocardial infarction. The initial course of the myocardial infarction was uncomplicated until day 9 when a new murmur was heard. A transthoracic echocardiogram demonstrated extensive anterior wall hypokinesia, an apical aneurysm associated with a small thrombus, and a ventricular septal defect associated with significant left-to-right shunt.

Her past medical history included hemorrhagic ulcerative colitis treated with prednisolone, 4 mg/d, and mesalazine (Mesalazine), 250 mg/d. A left colectomy was performed 10 years before for iatrogenic perforation after colonoscopy.

On admission to Erasme University Hospital, the patient was hemodynamically stable. Routine biochemistry tests were normal except for a BUN value of 28 mg/dL and a creatinine level of 1.5 mg/dL. A second transthoracic echocardiographic study was performed and was similar to the previous one; no atrial abnormality was noted. Catheterization of the right side of the heart confirmed the severe left-to-right shunt with a Qp/Qs value of 2.7. The coronary angiogram was normal. On day 12, an aneurysmectomy was performed with closure of the interventricular communication. Weaning from cardiopulmonary bypass required the administration of dobutamine, 20 μg/kg/min; dopamine, 20 μg/kg/min; and epinephrine, 3 mg/kg/min; ventricular pacing for a complete atrioventricular block also was required. Her hemodynamic condition worsened with the development of severe hypoxemia (PaO₂/FIO₂ ratio of 80 mm Hg with a positive end-expiratory pressure level of 5 cm H₂O) despite a normal chest radiograph. The pulmonary artery pressures were 31/19 mm Hg, the pulmonary artery balloon-occluded pressure was 17 mm Hg, and the right atrial pressure was 19 mm Hg. A contrast transesophageal echographic study showed a right-to-left shunt through the reopening of a PFO, but the Doppler signal was inadequate to quantify the degree of intracardiac shunt. Left ventricular ejection fraction was estimated at 45% with a relatively small left ventricle. Right ventricular function was normal, and the atria were dilated. No valvular dysfunction was observed. After increasing the pacing rate and fluid administration, dopamine and epinephrine infusions could be discontinued, but dobutamine infusion was continued at 10 μg/kg/min. On day 14,

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