



## Maintenance of Certification-CME Review

## Evaluation of paradoxical vocal fold motion

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## ARTICLE INFO

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**Release Date:** October 1, 2012**Expiration Date:** September 30, 2014**Estimated Time to Complete:** 60 minutes**Target Audience:** Physicians involved in providing patient care in the field of allergy/asthma/immunology.**Learning Objectives:** At the conclusion of this activity, participants should be able to:

- Describe the presentation of paradoxical vocal fold motion (PVFM).
- Discuss the diagnostic tests that are best used to evaluate a patient with suspected paradoxical vocal fold motion (PVFM).

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## Clinical vignette

A 15-year-old, Caucasian girl presents for evaluation of asthma after a recent emergency department visit. She was diagnosed as having asthma at the age of 7 years after presenting with intermit-

tent cough, wheeze, and shortness of breath with activity and rest. Prophylactic and rescue albuterol led to partial relief of symptoms. During the past year, she has been experiencing symptoms on a more frequent daily basis. Her physical examination findings have

previously been unremarkable, and office-based spirometry revealed no evidence of obstructive ventilator impairment or significant bronchodilator-induced reversibility. Because of increased frequency of persistent symptoms, her treatment has escalated to include a leukotriene receptor antagonist and combined inhaled corticosteroid and long-acting  $\beta$ -agonist metered dose inhaler. Despite this, her symptoms persist. She recently presented to an emergency department with acute shortness of breath without hypoxemia, which did not respond to nebulized bronchodilators or systemic corticosteroid therapy. She denies a history of esophageal reflux, rhinosinusitis with postnasal drip, or a family history of asthma.

## Introduction

Paradoxical vocal fold motion (PVFM) is a condition caused by involuntary adduction of the vocal cords during inspiration, resulting in acute upper airway obstruction. Numerous terms have described this condition, but *vocal cord dysfunction* or *PVFM* are the terms commonly used by allergists, pulmonologists, otolaryngologists, and speech pathologists.<sup>1</sup> Although PVFM is understood to be involuntary and not a manifestation of a malingering disorder, it was first described by Patterson et al<sup>2</sup> in 1974 as Munchausen stridor. In a seminal publication by Christopher et al,<sup>3</sup> the condition was reported in 5 patients with uncontrollable asthma who had normal pulmonary function and an absence of bronchial hyperreactivity. During symptomatic periods, these patients had adduction of the vocal folds with consequent limitation of inspiratory flow and compromise of the glottis due to the formation of a posterior diamond-shaped chink.

## Pathophysiology

During a normal respiratory cycle, the vocal folds widely abduct on inspiration and slightly adduct ( $<30^\circ$ ) on expiration.<sup>4</sup> Patients with PVFM have paradoxical adduction of the true vocal folds during the respiratory cycle. At the anterior commissure, the glottis angle formed by the true vocal folds can become acute, causing airflow restriction and turbulence during inspiration and/or expiration.<sup>5</sup>

Factors that lead to paradoxical movement of the vocal folds are multifactorial. Laryngeal hypersensitivity may be associated with gastroesophageal reflux (GER) or laryngopharyngeal reflux (LPR), upper respiratory tract infections, postnasal drip from rhinosinusitis, or physiologic response to exposure to irritant exposure.<sup>6</sup> Abnormal vocal fold movement can also be triggered by exercise. Numerous studies have highlighted the association of PVFM and psychological disorders.<sup>1–3</sup>

## Presentation

No unifying data support a classic profile of a patient with PVFM. Affected individuals are of all ages and of both sexes. (In my experience evaluating patients at a tertiary academic medical center, PVFM is diagnosed equally in boys and girls in childhood and adolescence and preferentially affects women in adulthood.) The true incidence of PVFM is unknown because it is often misdiagnosed as asthma. Patients with PVFM often have a history of recurrent health care use, polypharmacy, and repeated hospitalizations.<sup>7,8</sup>

Patients with PVFM present with symptoms often indistinguishable from asthma, such as dyspnea, stridor, wheeze, chest tightness, and cough. Symptoms may occur only on inspiration or during both phases of the respiratory cycle. In addition, however, patients may experience a sensation of choking, tightening of the throat, dysphonia, or aphonia with episodes. Symptoms often occur suddenly, are short-lived, and remit without intervention. A review of 288 PVFM-focused articles representing 1020 patients revealed

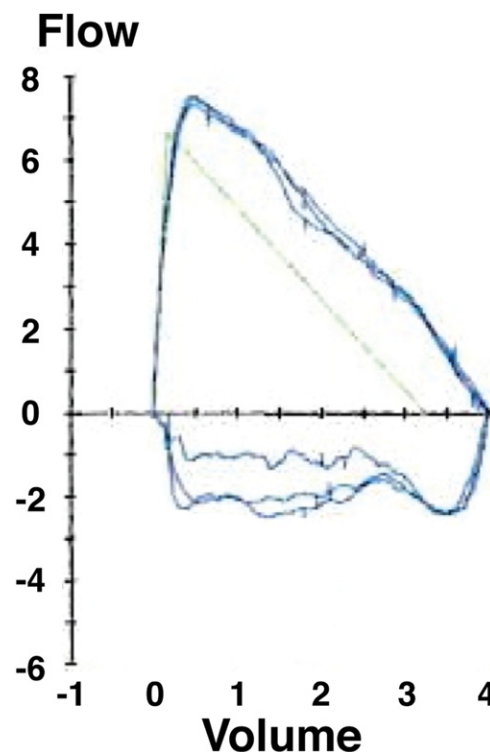


**Fig. 1.** Inspiratory curve of flow volume loop consistent with variable extrathoracic obstruction.

predominant symptoms of dyspnea (73%), wheeze (36%), perceivable stridor (28%), cough (25%), chest tightness (25%), throat tightness (22%), and changes in voice (12%).<sup>1</sup>

The difficulty in establishing a diagnosis of PVFM is partly attributable to the fact that many patients have both PVFM and asthma. In PVFM, however, the pattern of symptoms may be different. As noted, symptoms may occur on inspiration and can be associated with voice changes. A helpful way to differentiate an acute episode of PVFM from an asthma exacerbation is to measure oxygen saturation because patients with acute PVFM will have a normal oxygen saturation during attacks as noted in patients aged 12 to 21 years who presented to an emergency department with acute respiratory symptoms.<sup>9</sup> At one institution, 10% of all patients referred for inpatient evaluation of refractory asthma had PVFM, and 30% had diagnoses of PVFM and asthma.<sup>10</sup>

In patients with PVFM, physical examination findings are typically normal during asymptomatic periods. During acute episodes, tracheal localization of wheezing or stridor may occur. Audible peripheral airway expiratory wheezing is typically absent during acute PVFM, which is also a finding of severe acute asthma.<sup>1</sup>

## Diagnosis

History, spirometry, and direct visualization of aberrant vocal fold movement are key components to explore for the diagnosis of PVFM. Psychological assessment should be considered in every patient, evaluating for current stressors or at the onset of symptoms. Objective evaluation of patients with suspected PVFM often begins with spirometry. Physicians should routinely check the appearance of the flow volume loops and the ratio of forced inspiratory-expiratory flow at 50% of forced vital capacity. In PVFM, spirometry is typically normal without evidence of obstructive ventilatory impairment. Variable extrathoracic obstruction, which represents dynamic narrowing of the extrathoracic airway during inspiration, can be seen on flow volume loops or expiratory-inspiratory flow volume tracings (Fig 1), and the forced inspiratory-

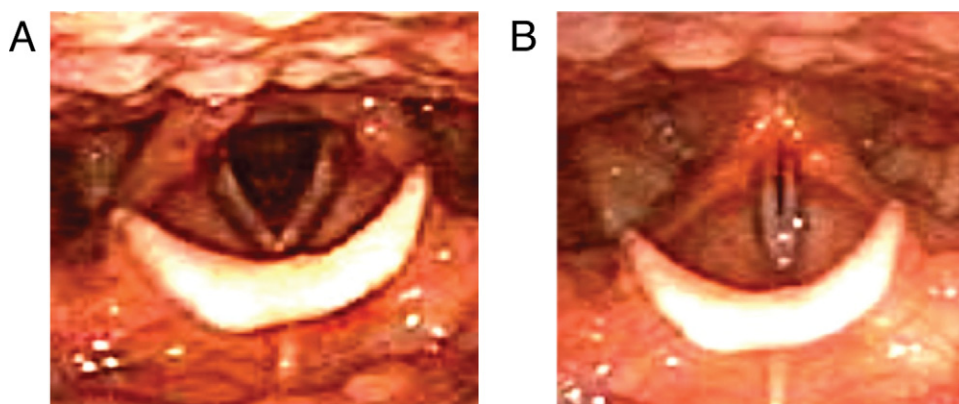


**Fig. 2.** Abduction of the vocal cords during expiration (A) and adduction of the vocal cords during inspiration (B) consistent with paradoxical vocal fold motion.

expiratory flow at 50% of forced vital capacity is often increased.<sup>8</sup> However, flow volume loops and expiratory-inspiratory flow volume curves are insensitive for the diagnosis of upper airway obstruction, and when looking at a specific variable, spirometry is not predictive of PVFM.<sup>1,11,12</sup> Inspiratory curve abnormalities are, at best, merely suggestive of PVFM rather than diagnostic of it. Up to 25% of patients with PVFM exhibit abnormal flow volume curves, which may be due to evaluation when the patient is asymptomatic.<sup>1</sup> As noted earlier, during acute episodes of respiratory distress, pulse oximetry is often helpful for differentiating PVFM from asthma in adolescents and has yet to be seen if this is applicable to an older population.<sup>9</sup>

The criterion standard for the diagnosis of PVFM is direct visualization of the vocal folds when the patient is symptomatic.<sup>1</sup> As originally reported, classic PVFM is visualized as adduction of the anterior two-thirds of the vocal folds and the presence of a posterior diamond-shaped chink in the folds during inspiration.<sup>3</sup> More recent criteria suggest that PVFM can be confirmed when a specific degree of inappropriate adduction of the true vocal folds is seen; during inspiration, patients have more than 50% medicalization but less than full closure (Fig 2).<sup>13</sup> Normal laryngeal function should be observed in patients with PVFM when they are asymptomatic and when the patient is not subject to alternative conditions that elicit symptoms.<sup>8,14</sup> However, during conditions of high airflow stress, such as when an asymptomatic patient is voluntarily asked to reproduce wheeze, pant, or phonate, classic findings of PVFM may be produced.<sup>3</sup>

Provocation of PVFM can occur with exercise, irritant exposure, or patient-specific stressors, which are identified by history. Bronchoprovocation testing with methacholine can cause an acute episode of vocal cord dysfunction (as seen in less than half the patients studied who presented with PVFM symptoms), and when used with concomitant flexible laryngoscopy, it is an excellent adjunctive tool for the evaluation of asthma and PVFM.<sup>15,16</sup> Laryngeal videography is beneficial in diagnosing exercise-induced PVFM when used in the field, outside the hospital setting.<sup>17</sup>

Radiologic evaluations, such as chest radiography, in patients with PVFM without asthma are typically of minimal utility. However, a new imaging tool using computed tomography may help in the evaluation of laryngeal function in patients with and without asthma. Low et al<sup>18</sup> used dynamic, high-speed computed tomography to obtain 320 sections of the larynx to quantify normal vocal fold function by the ratio of vocal fold diameter to tracheal diameter. This technology may become more widely used in the assessment of PVFM.

### Differential diagnosis

The differential diagnosis of PVFM includes asthma and numerous conditions that affect the larynx, including GER or LPR disease,

rhinosinusitis, or oropharyngeal thrush. In addition, aberrant arytenoid closure, laryngomalacia, laryngospasm due to prior airway instrumentation, unilateral or bilateral vocal fold paralysis, or mass effect from adjacent structures can be implicated.<sup>5</sup>

### Treatment

Treatment of patients with acute PVFM is focused on symptom control. Acute severe episodes have been treated with heliox, a combination of helium (80%) and oxygen (20%), which decreases airflow turbulence and resistance by promoting laminar flow<sup>10</sup> and by continuous positive airway pressure.<sup>19</sup> Long-term therapy focuses on identification and treatment of associated conditions. Avoidance of identifiable environmental irritants and treatment of associated conditions, such as GER or LPR, rhinosinusitis, anxiety, or depression, should be pursued. For example, patients with symptomatic GER or LPR should undergo a time-limited trial of antireflux therapy.

Laryngeal control therapy as taught by speech therapy and otolaryngology is the mainstay of therapy. Abdominally driven respiration decreases throat tension, resulting in passive inspiration. The patient is coached to relax the upper body, especially the neck, and use vocal hygiene measures.

Behavioral modification strategies, such as biofeedback, respiratory training, and laryngeal relaxation, are most efficacious when paired with patient reassurance, education, stress management, and other psychiatric approaches as indicated.<sup>20</sup> More recently, in a large retrospective review of pediatric patients with PVFM, those patients with symptoms at rest were more likely to benefit from psychiatric management rather than speech therapy.<sup>14</sup>

Finally, ipratropium dosed before exercise has been found to be beneficial in patients with exercise-induced symptoms.<sup>21</sup> A possible explanation for the role of an anticholinergic agent in the treatment of PVFM is understood by the notion that postinflammatory and irritant stressors promote neurologic hypersensitivity mediated through the vagus, which can lead to transient laryngeal narrowing.

### The case revisited

The patient's pattern of symptoms and lack of response to standard asthma therapy prompted referral to our institution for evaluation of suspected PVFM. She underwent methacholine bronchoprovocation testing and tolerated the maximal dose concentration without evidence of a 20% decrease in her forced expiratory volume in 1 second. The testing, however, provoked symptoms of inspiratory dyspnea localized to her throat. Peripheral oxygenation was normal, and she had inspiratory wheeze on auscultation localized to her trachea. Flexible laryngoscopy was immediately performed and revealed PVFM during multiple respiratory cycles. A postprocedural dose of inhaled ipratropium was associated with subjective improvement of

symptoms and resolution of previously noted auscultation findings.

The patient was referred to otolaryngology and speech therapy, where individualized facilitative breathing exercises were taught. The patient was able to perform the recommended facilitative breathing exercises and noted almost complete resolution of symptoms without a need for additional rescue medications. Subsequently, her asthma medications were successfully weaned. With understanding of her diagnosis and early initiation of facilitative breathing exercises, she no longer reported symptoms and now leads an active life. She was subsequently asked to seek evaluation if symptoms returned.

## Conclusion

PVFM is a multifactorial condition that is often underdiagnosed because of similarities with other common disorders. Clinicians should have a high index of suspicion for PVFM, especially in patients who report a classic pattern of symptoms, fail to respond to standard asthma therapies, and have normal physical examination and spirometric evaluation findings. Fiberoptic laryngoscopy, the criterion standard for diagnosis of PVFM, may fail to detect the characteristic vocal fold features when the patient is asymptomatic or in the absence of specific symptom triggers. Collaboration with health care professionals in otolaryngology, speech therapy, and mental health is beneficial in the evaluation and treatment of patients with PVFM.

This case highlights several key points. First, the patient reported symptoms that were inconsistent with her physical examination findings. Second, she began escalating asthma therapy despite a lack of clinical response to albuterol, normal baseline spirometry results, and a lack of bronchodilator-induced reversibility. Third, facilitative breathing exercises may allow patients with PVFM to decrease asthma medication use. Although PVFM and asthma can coexist, it is likely that our patient did not have asthma or exercise-induced bronchospasm. Patients should be monitored after the diagnosis of PVFM to discern between those with or without concomitant asthma.

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