

**TITLE: Vocal Cord Dysfunction: Paradoxical Vocal Cord Motion - A Thorough Review**

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**Todd M. Weiss, Senior Medical Student**

**FACULTY PHYSICIAN: Francis B. Quinn, Jr., M.D.**

**SERIES EDITOR: Francis B. Quinn, Jr., MD**

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## **Introduction**

Vocal cord dysfunction (VCD), also recognized as paradoxical vocal cord motion (PVC), laryngeal dyskinesia, and vocal cord malfunction, is a condition where the larynx exhibits paradoxical vocal cord adduction during inspiration, resulting in any of a number of symptoms that would be expected from extrathoracic airway obstruction, including, wheezing, dyspnea, cough, and shortness of breath. The diagnosis of PVC is currently being made with increased frequency as physicians are becoming more aware of its prevalence (Table I)(O'Connell et al). Many patients with VCD are wrongly diagnosed with refractory asthma and suffer morbidity from unnecessary treatment such as high dose exogenous steroid and bronchodilator use. Most severely, during acute episodes patients have received intubation<sup>i</sup> and/or tracheostomy<sup>ii</sup>. Such treatment is unwarranted and can be avoided when a knowledge of and high suspicion for PVC is present within the treating physician. The following review presents what is currently known about PVC and is aimed at eliminating future misdiagnoses.

## **History**

Intermittent vocal cord dysfunction presenting as asthma was first recognized in a medical textbook in 1842 where Dunghlison<sup>iii</sup> described disorders of the laryngeal muscles brought on by "hysteria". In 1869, Mackenzie<sup>iv</sup> actually visualized the vocal cords in hysteric adults with stridor and saw them paradoxically close during inspiration. He elicited this closure as the cause of the stridor.

William Osler<sup>v</sup> later defined this condition in 1902 by describing patients with "Spasms of laryngeal muscles" occurring during inspiration and times of great distress. Osler described patients as presenting with "extraordinary inspiratory or expiratory cries", and often with cyanosis. During this period the cause for PVC was considered purely psychogenic, as it was

only described in patients presenting with hysteria or during times of great stress. Because of the lack of a known organic etiology, PVCMM was a topic confined to the psychiatric literature; literature that published virtually nothing on the topic until the 1980's when the first case reports and case series began to emerge.

Today PVCMM is a well-defined phenomenon with many known possible etiologies and hence has been classified accordingly by different authors; i.e. organic and non-organic, psychogenic and non-psychogenic. In addition to etiologic categorization, PVCMM now has a well-accepted method for diagnosis. Treatment protocols with varying success rates have been rendered based upon the specific cause of the dysfunction.

## **Prevalence**

The overall prevalence of PVCMM in the population is unknown, but a 1994 study by the National Jewish Center for Immunology and Respiratory Medicine<sup>vi</sup> evaluated patients diagnosed with refractory asthma and reported findings of PVCMM alone in up to 10% of patients, while an additional 30% of patients had both PVCMM and asthma. O'Connell et al did a retrospective chart review of 164 patients and reported that 20% of all females who underwent rhinolaryngoscopy for any indication were found to have PVCMM.

PVCMM is also very common among asthmatics. Newman et al<sup>vii</sup> later evaluated the same 95 patients with PVCMM and found 53 (56%) to have coexistent asthma. Because PVCMM is often found in asthmatics and presents with symptoms similar to those found in patients with only asthma, it is often overlooked and not included in the differential diagnosis. However, such studies have sparked a new awareness of this disorder that allow for its recognition in wrongly diagnosed patients. It is therefore no surprise that the incidence of PVCMM is on the rise.

## **Glottic Function**

The larynx is an important region that anatomically joins the extrathoracic airway with the superior alimentary tract. In doing this it serves several functions vital for life, including structural prevention of aspiration during both respiration and swallowing. The larynx also houses the vocal cords and via a complex coordination pathway allows people to phonate and subsequently communicate<sup>viii</sup>. The term glottis refers to both the true vocal cords and the opening between them, the rima glottidis. Being a mobile structure, the glottis is the primary site for laryngeal dysfunction to occur.

The principle muscle for vocal cord abduction is the posterior cricoarytenoid (PCA) muscle. Adduction is performed primarily by the lateral cricoarytenoid muscle (LCA) that receives reinforcement from the transverse arytenoid muscles. The glottic aperture is termed the rima glottidis and its width is determined by contraction and relaxation of the PCA and LCA muscles, which is indirectly determined by the respiratory phase.

During normal inspiration, the respiratory center in the medulla stimulates the PCA muscle to contract, abducting the vocal cords via stimulation from the vagus nerve, and widening the rima glottidis. This stimulus to contract is shown to occur through a graded PCA response

secondary to varying medullary stimuli including serum carbon dioxide and oxygen levels, altered intrapulmonary pressure from thoracic airway obstruction, and various serum concentrations of anesthetic<sup>ix</sup>.

During normal expiration there is tonic contraction of the LCA muscles. However the majority of the rima glottidis narrowing comes from a decrease in the tonic activity of the PCA muscles and not an increase in the tonicity of the LCA muscles<sup>x</sup>. Newman et al estimate that the rima glottidis decreases 10% - 40% in size during expiration in normal tidal volume. By increasing and decreasing in diameter, the larynx has an active influence on the duration of inspiration and expiration and each subsequent resulting lung volume<sup>xi</sup>.

## Differential Diagnosis

When a patient presents with wheezing, stridor and/or dyspnea, a thorough list of differential diagnoses for upper airway obstruction must be suspected. This list can be guided by whether the patient is a child or adult as differences between the pediatric and adult airways account for differences in etiology.

Because the site of obstruction is more specific to the presenting symptoms than the actual cause of the obstruction, it is helpful to develop a differential diagnosis according to age group and location of obstruction (Table II). Structurally, the narrowest point in an adult airway is the glottis, while in the child it is the subglottis. Knowing that the diameter of a pediatric airway is much less than an adult's, even minimal inflammation of a child's subglottis can cause marked symptomatic airway obstruction. In adult airways the inflammation or obstruction must be much more severe before the patient will be symptomatic. This is why croup, an infection of the subglottic area, is a common cause of stridor in children, although it is not an actual laryngeal cause of dyspnea.

Anatomic locations for extrathoracic airway obstruction include the trachea, larynx, glottis, and thyroid. Endobronchial obstruction must also be suspected as a foreign body, bronchial adenoma, bronchial carcinoid, or bronchogenic carcinoma can all present with dyspnea and/or wheezing<sup>xii</sup>. A high index of suspicion derived from an awareness of the disease is necessary for making the diagnosis of PVCMM, as its presenting symptoms are very non-specific.

## Etiology

Once a diagnosis of PVCMM has been made (Table VII), the etiology of the dysfunction must be obtained. Several different causes have been described all resulting in the same presentation of VCD. The various etiologies include: cortical injury involving either upper or lower motor neurons, brainstem compression<sup>xiii</sup>, conversion disorder, malingering, and irritant induced<sup>xiv</sup>.

Cortical injury resulting in PVCMM has been noted in children with static encephalopathy<sup>xv</sup> and in adults suffering effects from a stroke or cerebral vascular accident<sup>xvi</sup>.

Lower motor neuron injury resulting in PVCMM is seen with diseases such as myasthenia gravis, medullary infarction, and amyotrophic lateral sclerosis (Maschka et al).

Brainstem compression causing PVCMM is much more common in children than in adults. Maschka et al report seeing cortical injury from brainstem compression in three children over a three-year period, two of which were caused by Arnold-Chiari malformation and the other from cerebral aqueductal stenosis. There is however only one known report of PVCMM in an adult secondary to brainstem compression, and this was attributed to a giant posterior fossa arachnoid cyst.<sup>xvii</sup>

Patients suffering from conversion or somatization disorder can unintentionally induce PVCMM. This commonly occurs in patients undergoing stressful periods of their lives or during emotionally tense circumstances. Patients suffering from conversion disorder produce the PVCMM unconsciously and without intentional gain.

In contrast to the aforementioned disorder, patients who are malingering or have factitious disorder produce the PVCMM consciously and with the intention of secondary gain from either external incentives (often monetary) or by assuming the sick role, respectively. Goldman et al<sup>xviii</sup> report how patients using forced expiration at low lung volumes when the glottic aperture is narrowest produce voluntary expiratory wheezes. Such patients often have characteristic histories and exhibit poor compliance and cooperation (Maschka et al). PVCMM and wheezing are also reproducible in patients without psychiatric disorders using this same technique<sup>xix</sup>.

Irritant-induced PVCMM is a newly documented phenomenon. Perkner et al conducted a case-control study and reported eleven cases of PVCMM in which there was a temporal relationship between the onset of the PVCMM and environmental or occupational irritant exposure. Such an association was previously unreported in the literature. Irritants such as ammonia, cleaning chemicals, organic solvents, flux flames, and smoke were linked to the onset of PVCMM. Since then, Bhargava et al<sup>xx</sup> reported an additional case of chlorine induced PVCMM in a 17-year-old male associated with multiple episodes of swimming. This recently documented etiology of PVCMM supports evidence that symptomatic patients should be evaluated for VCD after reported irritant exposures.

Because each etiology of PVCMM carries unique therapeutic measures, Maschka et al have proposed a classification scheme that separates PVCMM according to its cause and subsequently its treatment protocol (Table III). Not only does such a classification simplify management options, but also it allows practitioners to employ a well-directed evaluation aimed at maximizing cost efficiency.

## **Demographics**

### *Juveniles*

Studies have recently been done on the pediatric age group to examine the demographics and presentation of juveniles with PVCMM separate from those of adults<sup>xxi, xxii</sup> (Table IV). With all patients under the age of 18, Powell et al reported the average age of presentation to be 14.5

years with 82% of patients being female. Landwehr et al reported an average age of 14.7 years with 86% females. Similarities among the patients were participation in organized sports, social stresses, and exercise inducible symptoms. These are similar to those found in adults.

Powell et al found a strong association between PVCMM positive juvenile patients and laryngeal signs of chronic gastroesophageal reflux. They propose that PVCMM may be a low-grade form of laryngospasm in response to the irritation from reflux. GERD is present in many patients with PVCMM, although a cause-effect relationship is yet to be established (Maschka et al). Powell's findings therefore confirmed that PVCMM could be linked to physiologic disease and is not always secondary to psychological factors as once believed.

### *All Age Groups*

Most of the published literature does not separate juveniles from adults, but instead combines the two age groups in their results (Table V). PVCMM is found predominantly in women in their twenties and thirties and is associated with both physiologic and psychologic risk factors. Documented psychologic risk factors include employment in a medical profession, being overweight, stress, anxiety, and a history of childhood abuse or psychiatric disturbance<sup>xxiii</sup> (Patterson et al, Newman et al). Physiologic risk factors include asthma, brainstem abnormalities, cystic fibrosis, and GERD (Powell et al).

The psychopathology of patients with PVCMM varies (Ramirez et al), however the prevalence of psychiatric disorders in patients with PVCMM does not differ greatly than that among confirmed asthmatics (Christopher et al). A recent prospective study of adolescents with PVCMM found no change in their level of family functioning when compared to asthmatics with the exception of an increased incidence of anxiety disorders<sup>xxiv</sup>.

Among the previously mentioned risks for PVCMM, there is also association between PVCMM and wartime<sup>xxv</sup>. This should not be surprising as the frequency of conversion reaction rises during stressful periods such as wars. Several studies have been done on military bases and clinics (Morris et al, O'Connell et al). These areas serve many recruits and officers in training who are continually exposed to physical and mental stresses, which may account for the high prevalence of this disorder. Nonetheless, it reemphasizes the important risk of stress and anxiety and its relation to PVCMM.

### **Clinical Presentation**

Patients with PVCMM exhibit a wide variety of symptoms ranging from cough, inspiratory/expiratory wheeze, dyspnea with/without exertion, stridor, hoarseness, throat tightness, and reflux (Newman et al, Wood II et al) to no symptoms at all<sup>xxvi</sup>. Their histories are often significant for frequent, episodic attacks of such symptoms leading to shortness of breath. Diamond et al<sup>xxvii</sup> evaluated 90 patients with PVCMM and found cough to be the most common pre-evaluation diagnosis with it being reported in 77% of patients. Juveniles with PVCMM have shown an increased incidence in anxiety disorders when compared to asthmatics, however, anxiety attacks were found to precede the respiratory symptoms in patients with PVCMM while anxiety often follows respiratory symptoms in patients with asthma (Gavin et al).

Patients present to a spectrum of different care facilities such as ENT clinics, Family practitioners, medical wards, and ER departments during acute exacerbations. They present to physicians with a variety of previous diagnoses including asthma, exercise-induced bronchospasm, COPD, and anaphylaxis (O'Connell et al). Often their "asthma" is refractory to treatment. In patients with psychogenic or non-organic PVCMM, symptoms usually resolve when the patient is distracted or with anesthesia/sleep<sup>xxviii</sup>. This is not always the case, as there have been reports of PVCMM in patients who experience nocturnal waking, making this a less reliable distinguishing feature<sup>xxix</sup>.

## Physical Exam

During PVCMM there is adduction of the vocal cords and narrowing of the rima glottidis during inspiration causing a functional extrathoracic airway obstruction. A classic finding of the paradoxical adduction on laryngoscopic examination is an inspiratory anterior vocal cord closure with posterior chinking, or diamond-shaped margination between the posterior vocal cords and the corniculate tubercles<sup>xxx, xxxi</sup> (Figure 1). This obstruction decreases laminar airflow through the glottis and produces an inspiratory wheeze or stridorous sound similar to that heard in asthmatics, making the two entities difficult to differentiate symptomatically. Physical exam, however, reveals that patients with only asthma do not exhibit posterior chinking<sup>xxxii</sup>.

Patients with asthma alone can also exhibit adduction of the vocal cords during inspiration. Higenbottam<sup>xxxiii</sup> experimentally induced bronchoconstriction in study patients and revealed a decrease in the glottic aperture during quiet respiration. This glottic narrowing allows for a certain amount of positive end expiratory pressure (PEEP) to maintain airway patency preventing alveolar collapse from the bronchoconstriction. Such a physiologic response is suspected to be therapeutic, not pathologic<sup>xxxiv, xxxv</sup> (Ramirez et al) as pursed-lip breathing almost always relieves asthmatic wheezing.

In patients with PVCMM the wheezing is usually greatest over the larynx (Craig et al) and is less evident over the rest of the lung fields. There can be constant flexion of the anterior neck muscles and tachypnea. Their lungs reveal auscultatory absence of hyperinflation presuming COPD and/or asthma is not also present (Goldman et al). Patients with or without concurrent asthma are often hypoxic and therefore may appear cyanotic during exacerbations. Laryngoscopy will show evidence of vocal cord adduction during inspiration, expiration, or both.

## Laboratory Studies

Many patients with PVCMM have been previously diagnosed with asthma although different laboratory tests can be performed to distinguish the two. There is much controversy as to whether or not patients with only PVCMM actually undergo respiratory compromise during acute exacerbations. The literature does show evidence of patients exhibiting no respiratory distress and presenting without hypoxemia, though such a presentation seems less common<sup>xxxvi</sup>. Typically patients have arterial blood gas values with normal Aa gradients and no evidence of hypercapnia or acidosis, but do show evidence of hypoxemia during exacerbations<sup>xxxvii</sup>.

(Christopher et al, Appelblatt et al, Murray et al) (Table VI). Normal alveolar-arterial oxygen-tension gradients are not expected in asthmatics during an acute attack<sup>xxxviii</sup>.

During periods when patients are asymptomatic they have a tendency to show normal laboratory values. Physicians have therefore begun to stress patients during testing, exposing them to the same elements which typically initiate attacks. Attacks are commonly exercise induced or irritant induced, and both methods are used for provocative testing. Because as many as 50% of patients with PVCMM also have asthma, methacholine challenges are used to detect bronchial hyperresponsiveness when pulmonary function tests (PFT's) are inconclusive. This test can determine the patient's asthma status and is useful for guiding treatment regimens, but alone cannot diagnose PVCMM.

PFT's with flow-volume loops have also been used to support the diagnosis of PVCMM in symptomatic patients. Flow-volume loops of patients with PVCMM often show flattening of the inspiratory curve, or a decrease in maximal inspiratory flow during acute attacks, and are normal while asymptomatic (Figure 2)(Pierce et al, Ramirez et al). Although inspiratory blunting is common in patients with PVCMM, it is not specific for VCD and may be produced by most types of extrathoracic airway obstruction. Parker et al<sup>xxxix</sup> evaluated 26 patients with PVCMM and found exercise tidal volume loops to indicate the upper airway as a cause for symptoms in 74% of patients while showing inspiratory flow limitation in 62% of patients. Although the flow-volume loop is not specific in diagnosing patients with PVCMM, it can be a useful tool in eliminating asthma from the differential diagnosis.

Pulmonary function testing in patients with only PVCMM reveals normal total lung capacity without evidence of lung hyperinflation during acute attacks (Patterson et al, Morris et al). Chest x-rays also show no evidence of lung hyperinflation (Powell et al). This is in contrast to asthmatics. Other PFT parameters including FEF50/FIF50, FEV1/FVC, and SRaw (specific airway resistance) are shown to have a high sensitivity and specificity for detecting extrathoracic airway obstruction but again are not specific for PVCMM (Patterson et al). Nonetheless, PFT's should be performed on all patients in whom PVCMM is suspected (Morris et al) as they are useful in distinguishing PVCMM from its most common masquerader, asthma.

## Diagnosis

The diagnosis of PVCMM is difficult due to its episodic nature and presentation. Some authors have considered PVCMM a diagnosis of exclusion (Fields et al). This observation is turning out not to be the case, as making the diagnosis is currently an active process involving clearly defined criteria (Table VII). While other types of vocal cord disease must still be excluded, PVCMM itself has specific distinguishing physical characteristics.

To make the diagnosis, direct visualization of the cords must be obtained while patients are experiencing their symptoms. Because their symptoms are often unpredictable, bronchoprovocative, irritant associated, and exercise stress tests (Morris et al, Wood II et al, Perkner et al) have been used to induce symptoms. Hypnotic suggestion has also been used to induce symptoms in patients<sup>xl</sup>. Laryngoscopy performed with a flexible fiberoptic scope can be

performed in the office and has yielded good results with few complications and minimal patient discomfort (Wood II et al).

Current criteria for diagnosis in adults includes direct visualization of vocal cord adduction with posterior “chinking” during either inspiration, early expiration, or both inspiration and expiration while the patient is experiencing symptoms (Perkner et al, O’Hollaren, Wood II et al, Newman et al). Powell et al have shown that a majority of juveniles with VCD exhibit PVCMM during normal quiet respiration and that they do not require provocative testing. However, if the patient is asymptomatic, negative laryngoscopic findings do not exclude the diagnosis.

Vocal cord adduction occurring during only the last half of expiration is not pathologic and has been shown to be a normal variant (Wood II et al). While inspiratory vocal cord adduction is always pathologic, vocal cord adduction during only expiration is a normal variant in asthmatics and is an adaptation to their obstructive disease (Collett et al). This should not be confused with PVCMM.

Care must also be made to not confuse PVCMM with vocal cord motion produced by a laryngoscope induced gag reflex. This is reportedly prevented by exercising good technique (Wood II et al). Nasal endoscopy has produced more accurate results than oral endoscopy, as the latter technique can distort the appearance of the epiglottic position and may interfere with the interpretation of the endoscopic data (Powell et al).

## **Treatment**

Appropriate treatment of PVCMM begins with an accurate diagnosis adhering to the previous mentioned criteria. Many patients have undergone unnecessary intubation and tracheostomy procedures secondary to misdiagnoses. Arriving at a correct diagnosis also allows cessation of all unnecessary medications. Once the correct diagnosis is established, treatment can be divided into acute and chronic management (Table VIII).

### *Acute Management*

The cause of the PVCMM must first be elicited. With PVCMM secondary to preexisting organic disease states such as brainstem compression, encephalopathy, stroke, ALS, myasthenia gravis, GERD, etc. the underlying disorder should be treated appropriately. A history of previous exposure to irritants should also be obtained. With no obvious source of causative organic disease, acute treatment is henceforth symptomatic.

Heliox therapy is recommended for immediate relief of respiratory distress in most patients with PVCMM<sup>xli</sup>(Murray et al). Heliox is a gaseous mixture of oxygen and helium often found in ratios of 20/80 and 30/70 respectively. Because this mixture is less dense than air, inhalation reduces turbulence in the airway and eliminates respiratory noise. This reduces anxiety that is often the predisposing factor to many attacks (Gavin et al). It provides short-term relief of dyspnea to patients with both PVCMM and other forms of upper airway obstructive disease. Heliox treatment is however not effective for relief of symptoms due to asthma or other lower airway disease (Weir et al).

Other acute measures include IPPV (intermittent positive pressure ventilation) and CPAP (continuous positive airway pressure) which widen the rima glottidis and also reduce turbulence (Christopher et al). Panting brings about acute relief by physiologically increasing the glottic aperture (Patterson et al). General anesthetic induction with small doses of propofol can relieve acute attacks<sup>xliii</sup> (Murray et al). Benzodiazepines and reassurance both reduce anxiety and therefore have been shown effective (Ramirez et al, Patterson et al). Conversely, asthma therapy with bronchodilators, oxygen, and corticosteroids has been shown ineffective for relief of symptoms in patients with PVCMM (Wood II et al).

A more invasive approach involves intralaryngeal injection of botulinum toxin type A<sup>xliiii</sup> (Maillard et al). This when combined with sedation was shown to be effective at relieving symptoms and avoided more invasive intervention. It is recommended that this treatment be considered in severe cases of PVCMM.

### *Long-term Management*

This requires a multidisciplinary approach involving speech therapy, psychiatric support, and physician education regarding the syndrome. Speech therapy techniques are aimed at focusing attention on expiration and abdominal breathing rather than on inspiration and laryngeal breathing (Goldman et al). Therapy with early recognition of symptoms allows relaxation of neck, shoulder and chest muscles and promotes normal laryngeal breathing.

Psychotherapy should be initiated in patients if there has been insufficient improvement with speech therapy alone, if there is psychological tumult in the patient's life, or if the patient requests it<sup>xliv</sup>. Psychotherapy allows the patient to explore for potential causes of the disorder and trains the patient with relaxation techniques. Because many patients with PVCMM are considered to have a conversion disorder, psychotherapy is often utilized.

Educating the patient about the condition is another method useful for reducing stress. Biofeedback training and panting exercises have been used as long-term treatment approaches, however they are not considered primary agents (Patterson et al).

A new device has been suggested for both the acute and long-term management of inspiratory PVCMM<sup>xlv</sup>. It consists of a facemask that provides airway resistance during inspiration but not expiration. This slows airflow during inspiration and has shown effective in reducing inspiratory stridor, giving the device both a physiological and psychological mechanism of action.

### **Prognosis**

The long-term outcome of patients with PVCMM is unknown as most of the literature consists of case reports and retrospective studies. One study followed three patients over a 10-year period, all of who showed continued symptomatic PVCMM at follow-up<sup>xlvi</sup>. More trials need to be conducted with long-term outcomes before conclusions about management efficacy can be drawn.

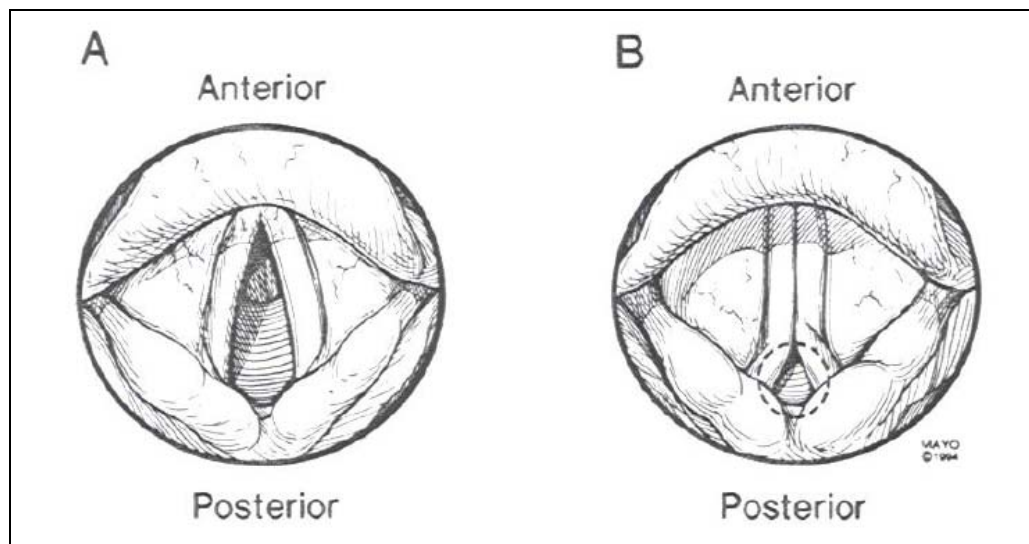
In contrast to long-term outcomes, the initial response to standard treatment such as speech therapy and psychotherapy is reported to be good (Mobeireek et al). Link et al<sup>xlvii</sup> recently conducted a telephone interview with 15 patients all diagnosed with PVCM who had received prior therapy. The interview took place an average of 20 months (range 11-62) after initial diagnosis of the disorder. The results showed that most responded well with improved functioning and fewer symptoms after intervention (Table IX). Others studies indicate that a good prognosis relies upon a positive reaction to the initial explanation of the diagnosis (Goldman et al), placing a heavier emphasis on patient education as a means to therapy.

## **Conclusion**

PVCM is an under recognized disorder effecting more people than previously thought with the majority of patients being young to middle-aged females. To make the diagnosis of PVCM, the clinician must have a high suspicion for it and be knowledgeable about its presentation. Many people every year are misdiagnosed and wrongly treated for refractory asthma and anaphylaxis. Inappropriate hospitalization, high-doses of unnecessary corticosteroids, intubation, and tracheostomy add to the morbidity of wrongly diagnosed patients. Distinct history and physical exam findings should raise the suspicion for PVCM in the treating physician, while the diagnosis is based on laryngoscopic evidence of inspiratory vocal cord adduction. Certain laboratory findings are also characteristic of the disorder yet alone are not diagnostic.

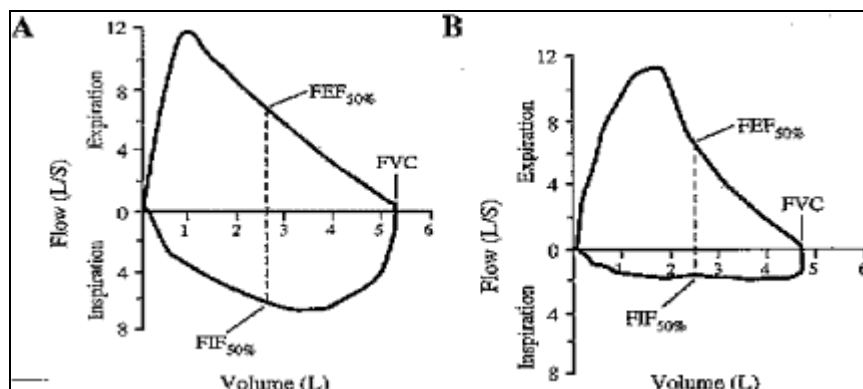
There is a strong association between people with PVCM and those with asthma. The presentation of both patient groups can be identical, yet the finding of one in a patient does not rule out the presence of the other. In fact it seems to make it more likely. It is important to differentiate between the two diseases in symptomatic patients because each carries its own unique treatment, with asthma therapy being ineffective against symptoms of PVCM and vice-versa. There is little data available about the long-term effects of therapy, but short-term studies have revealed promising results. As more clinicians become aware about the spectrum of presentation seen with PVCM, fewer misdiagnoses will be made. Additionally, as more is learned about the pathophysiology of this disorder more effective treatment protocols will be developed.

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**Figure 1**

Illustration of vocal cords during inspiration in both the normal patient (A) and the patient with VCD (B). Note the characteristic posterior thickening in the vocal cords of patient B. Illustration taken from Patterson et al.



**Figure 2**

Flow-volume loop in a normal patient (A) and in a patient with extrathoracic airway obstruction (B). Note the blunting of the inspiratory portion in the patient with extrathoracic airway obstruction. Illustration taken from Perkner et al.

**Table I**

Prevalence of VCD with or without the presence of asthma among patients being evaluated for symptoms of upper airway obstruction.

Study Group	Number of patients	Presenting symptoms	VCD alone	VCD + Asthma
Newman et al		Refractory asthma	10%	30%
Jain et al <sup>xlviii</sup>	50	Acute asthma, wheeze,	10%	12%

		Shortness of breath		
Morris et al <sup>xlix</sup>	40	Exertional dyspnea	15%	*60%

\*Indicates percent of patients with abnormal flow-volume loops after methacholine challenge.

**Table II**

Differential Diagnosis of Airway Obstruction by Anatomic Location in Both Pediatric and Adult Age Groups

<b>Adult</b>		<b>Pediatric</b>	
<b>Location</b>	<b>Cause</b>	<b>Location</b>	<b>Cause</b>
Oral Cavity	Trauma	Oral Cavity	Macroglossia, angioedema
	Neoplasm		Dermoid cyst, Robin's Syndrome
	Angioedema		Ludwig's angina,
Pharynx	Neoplasm, Trauma	Pharynx	Lingual thyroid,
	Ludwig's Angina, Abscess		Peritonsillar abscess
	Post-operative, goiter, angioedema		Tonsillar hypertrophy, Thyroglossal duct cyst Branchial cleft cyst
Larynx	Vocal cord dysfunction	Larynx	Vocal cord dysfunction
	Papillomatosis, Ludwig's Angina		Epiglottitis, vocal cord web
	Laryngospasm, angioedema		Bilateral vocal cord paralysis
	Bilateral vocal cord paralysis, myasthenia gravis, Bulbar Palsy		Laryngeal stenosis Laryngomalacia, cyst
	Prolonged intubation, anaphylaxis		Hemangioma, croup
	Cricothyroid arthritis, foreign body		Prolonged intubation, Papillomatosis
Trachea	Blunt trauma, Neoplasm	Trachea	Web, stenosis, foreign body
	Vascular anomalies, tracheitis		Cyst, thyroid tumor, Vascular anomalies, tracheomalacia
Endobronchial	Asthma, COPD, carcinoid syndrome	Endobronchial	Asthma
	Congestive heart failure		Tracheobronchitis

**Table III**

Proposed Classification for Paradoxical Vocal Cord Motion from Maschka et al

<b>Organic Causes</b>	Brainstem Compression
	Cortical or upper motor neuron injury
	Nuclear or lower motor neuron injury
	Movement disorders
	Gastroesophageal reflux
<b>Nonorganic Causes</b>	Factitious or malingering disorder
	Somatization/conversion disorder

**Table IV**

Demographics of Juvenile VCD Positive Patients

	<b>Number of Patients</b>	<b>Number/Percent Female</b>	<b>Average Age at Diagnosis</b>
Powell et al	22	18/82	14.5 years
Landwehr et al	7	6/86	14.7 years

**Table V**

Demographics of Patients Diagnosed with PVCM (all age groups)

	<b>Number of Patients</b>	<b>Number/Percent Female</b>	<b>Average Age at Diagnosis</b>
O'Connell et al	20	16/80	33 years
Morris et al	10	7/70	22.5 years
Newman et al	42*	41/98	34.3

\*Value for patients with VCD only

**Table VI**

ABG values for patients with VCD during symptomatic episodes

	<b>PO2</b>	<b>PCO2</b>	<b>pH</b>	<b>SpO2</b>
Appelblatt et al	50	38	7.41	87
	52	45	7.41	85

	44	36	7.46	80
				<b>P(A-a)O<sub>2</sub></b>
Christopher et al	75	35	7.47	3
	89	39	7.40	11
	64	34	7.48	15
	75	37	7.43	1
	68	36	7.40	9

**Table VII**  
Criteria for the Diagnosis of VCD

<b>1</b>	Presence of symptoms including wheeze, cough, dyspnea, stridor, hoarseness
<b>2</b>	Laryngoscopic confirmed adduction of vocal cords during inspiration, early expiration, or both inspiration and expiration
<b>3</b>	Absence of gagging or coughing during laryngoscopy
<b>4</b>	Presence of a posterior glottic chink
<b>5</b>	Exclusion of alternative vocal cord disease

**Table VIII**  
Treatment For Patients with VCD

<b>Acute Therapy</b>	<b>Long-term Therapy</b>
Heliox Therapy	Speech Therapy
IPPV, CPAP	Psychotherapy
Panting	Patient Education
Anxiolytics	
Anesthesia	
Botulinum Toxin Injection	
Reassurance	

**Table IX**  
Results of therapy for patients with VCD\*

	Symptoms		Activity Level
<b>A Lot Better</b>	54%	<b>More Active</b>	60%
<b>A Little Better</b>	13%	<b>No Change</b>	33%
<b>Unchanged</b>	33%	<b>Less Active</b>	7%

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