Paradoxical Vocal Fold Motion: A Tutorial on a Complex Disorder and the Speech-Language Pathologist's Role

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Paradoxical vocal fold motion (PVFM) is presented as a complex, poorly understood disorder that merits our clinical and research attention. This tutorial examines PVFM characteristics, etiologies, differential diagnosis, and medical/psychological intervention. The role of the speech-language pathologist in diagnostic evaluation and intervention is delineated. An assessment protocol and specific treatment suggestions are provided. The need for future research is discussed.

Key Words: paradoxical vocal fold motion, vocal cord dysfunction, paradoxical vocal cord motion, speech-language pathology tutorial

Paradoxical vocal fold motion (PVFM) is a laryngeal disorder that has been recognized with increasing frequency over the past 15–20 years. Earlier reports of functional airway obstruction (Appelblatt & Baker, 1981) and nonorganic laryngeal obstruction (Patterson, Schatz, & Horton, 1974) suggested that this was a new syndrome. However, Dunglison (as cited in Lacy & McManis, 1993) described PVFM symptoms in 1842. Over the years, this disorder has had numerous labels. This likely has contributed to a poor understanding of PVFM; inconsistencies in reported key features, etiological assumptions, and varying intervention suggestions add to the confusion.

Speech-language pathologists (SLPs) increasingly are consulted regarding the assessment and treatment of individuals whose clinical signs and symptoms suggest PVFM. Our background in working with voice and motor speech disorder patients uniquely prepares us to detect abnormalities in laryngeal and respiratory functions and to teach laryngeal and respiratory control techniques. In the case of PVFM, our clinical effectiveness will depend on our appreciation of the disorder complexity, our ability to recognize PVFM symptoms, and our expertise in tailoring intervention to specific patient needs. The objectives of this paper are threefold: (1) to review the literature that informs our understanding of PVFM, (2) to provide the SLP with a tutorial on diagnosis and treatment of PVFM, and (3) to promote further research in this area.

Description and Patient Profile

In PVFM, the vocal folds adduct during inhalation and/or exhalation, thereby restricting the airway opening (Christopher et al., 1983; Martin, Blager, Gay, & Wood, 1987; Newman, Mason, & Schmaling, 1995). Patients typically present with episodic or recurrent symptoms of dyspnea and/or stridor. Frequently patients complain of a tightness localized to the laryngeal area; sometimes they report a sensation of being choked. These feelings lead some patients to fear that they will die during a dyspneic episode. Because of the episodic restricted airflow and respiratory sounds, PVFM symptoms may be confused with asthma, which involves bronchoconstriction of the lower airway (Christopher et al., 1983). A careful diagnosis of the true source of the airway problem includes recognition of the fact that paradoxical vocal fold movement can contribute to upper airway obstruction.

Many case reports attempt to define the PVFM patient profile. Group data depict the so-called typical adult PVFM patient as 20–40 years old, female, with at least 12 years of formal education and with some connection to working in health care (Brugman & Newman, 1993). Newman and Dubester (1994) reported that this profile only fits about 25% of the PVFM patients seen at their center. According to Kuppersmith, Rosen, and Wiatrak (1993), in 78% of the reported cases of PVFM, the patients were between 10 and 40 years old. Children and adolescents with PVFM tend to be high achievers and often participate in competitive sports (Brugman & Newman, 1993). Although PVFM tends to be more prevalent in women than in men, the gender difference is not as dramatic in children. Across studies, the female to male ratios range from 41/1 to 4/1 for adults and 3/1 to 1.3/1 for children and adolescents (Kuppersmith et al., 1993; Lacy & McManis, 1994; Newman et al., 1995).

Any straightforward description of PVFM faces the challenge of accurately representing the nature of this disorder. Some precautions and suggestions are in order. The literature is replete with descriptions of patients with conditions that may or may not be the same as PVFM. These conditions may appear under various labels. In some cases, the labels used may be indicative of author assumptions about the nature or etiology of the disorder. For example, "episodic laryngeal dyskinesis" (Nahmias, Tansey, & Karetzky, 1994) implies neuropathology, whereas "Munchausen's stridor" (Patterson et al., 1974) suggests psychopathology. Others may use the nonspecific terms of "paradoxical vocal cord movement" or "vocal cord dysfunction" in describing conditions that appear to differ significantly from what is being described in this paper (e.g., Maschka et al., 1997; Patton, DiBenedetto, Downing, Zoller, & Morgan, 1987; Shafei, El-Kholy, Azmy, Ebrahim, & Al-Ebrahim, 1997).

Throughout this paper, the term PVFM will be used to label the condition where episodic adductor spasms of the vocal folds interfere with normal breathing. Although some reports (e.g., Appelblatt & Baker, 1981; Christopher et al., 1983; Newman et al., 1995) emphasized that PVFM is a functional or nonorganic disorder, we will see that the extent to which the disorder is functional varies across patients. It is suggested that PVFM may best be viewed as a complex, heterogeneous disorder in both its etiology and expression.

Etiological Factors

A number of causes of PVFM have been proposed. These include psychological conditions, upper airway sensitivity to laryngeal irritants, and a form of laryngeal dystonia.

Psychological Conditions

Although the etiology of PVFM is poorly understood, the literature suggests that psychological factors may play a role in many cases. Patterson et al. (1974) described a case of nonorganic laryngeal obstruction with an emotional overlay and referred to the disorder as Munchausen's stridor. That diagnostic label implies that an individual has voluntary control of the symptoms and is being deliberately deceptive. This does not appear to be true for most PVFM patients. Appelblatt and Baker (1981) described three women diagnosed with "functional upper airway obstruction." In each case, psychiatric evaluation revealed emotional problems associated with family stress or a failing marriage. Consulting psychiatrists established a diagnosis of "hysterical personality" in one case and "psychoneuroticism with a hysterical convergence reaction" (p. 305) in another. Christopher et al. (1983) were the first to report on psychiatric disorders in patients whose PVFM diagnosis was based on both flow-volume studies and laryngoscopic examinations during symptomatic periods. Psychiatric evaluations of four of the five adult patients revealed disorders described as "ranging from mild stress-related exacerbation of symptoms to obsessive-compulsive disorder" (p. 1568). All patients were described as having "difficulty in directly expressing anger, sadness, or fear and…various degrees of secondary gain from respiratory symptoms" (p. 1568). Selner, Staudenmayer, Koepke, Harvey, and Christopher (1987) described the psychological characteristics of three adult patients in detail. Each case suggested a diagnosis of conversion reaction in which the apparent physical disorder was a means of diverting attention from the real internal need or conflict (primary gain). The attention, sympathy, and support surrounding PVFM were consistent with secondary gain. Other reports involving primarily adults also link PVFM to psychological factors associated with posttraumatic stress of wartime involvement (Craig, Sitz, & Squire, 1992; Heiser, Kahn, & Schmidt, 1990), a history of loss and physical abuse (Brown, Merritt, & Evans, 1988), sexual abuse (Freedman, Rosenberg, & Schmaling, 1991), polygamy and associated family stresses (Mobeireek, Alhamad, Al-Subaei, & Alzeer, 1995), and an anxious/hysterical personality with a distrust of medical personnel (Imam & Halpern, 1995).

It is difficult to draw any firm conclusions regarding the psychological aspects of PVFM. Despite the frequency with which PVFM is described as a factitious illness or conversion disorder, the diagnoses are unsubstantiated in most reports (Newman & Dubester, 1994; Perkner et al., 1998). Furthermore, as Lacy and McManis (1994) pointed out, most of the case reports are in the nonpsychiatric medical literature where authors have not demonstrated how the psychiatric conditions influence PVFM. Although some characteristics of PVFM patients may be consistent with conversion disorder, the diagnosis requires clear evidence of a temporal link between stress and symptoms. It also should be recognized that many experts hesitate to diagnose conversion disorder when symptoms involve the autonomic nervous system. In addition, some patients diagnosed with conversion disorder later show signs of true organic pathology (Lacy & McManis, 1994). Lacy and McManis suggested that other underlying disorders such as anxiety disorder, panic disorder, and depression warrant consideration. It also is possible that depression and anxiety may be consequences rather than causes of persistent respiratory problems (Newman & Dubester, 1994). Finally, careful assessment of PVFM patients does not always reveal an emotional conflict or psychiatric disturbance. As Newman and Dubester acknowledged, there may be a subgroup of PVFM patients with psychopathology; however, overemphasizing psychological issues in all patients may undermine effective intervention.

Upper Airway Sensitivity

It is well known that the larynx serves many functions, including that of being a protector of the lower airway. The reflexive adduction of the vocal folds has been studied in infants, in whom airway closure associated with regurgitation may lead to prolonged apneic periods (Thach, 1997). It is assumed that chemoreceptors in the laryngeal mucosa, particularly in the interarytenoid space, trigger the protective reflex in response to fluid in the upper airway. Fluids that have been implicated include both gastric reflux

(including silent reflux) and upper airway secretions. Thach (1997) suggested that abnormal airway stimuli or hyperactive laryngeal chemoreflexes may cause apneic periods in infants.

The reflexive adduction of the vocal folds also may be implicated in PVFM when gastroesophageal reflux disease (GERD) appears to contribute to the provocation of symptoms. Koufman (1994) reported that acid reflux triggered PVFM in a series of 12 patients whose gastroesophageal reflux was confirmed by 24-hour double-probe pH monitoring. Acid-suppression medication and dietary and lifestyle modifications led to resolution of the PVFM symptoms within 4 weeks. The high prevalence of GERD in PVFM patients has prompted others to suggest that GERD may be an etiological factor (Perkner et al., 1998; Powell et al., 2000). The laryngeal irritation associated with GERD may contribute to the bronchial constriction of asthma as well (Pope, 1994). Although the causal link has not been demonstrated convincingly, it is thought that bronchial constriction is a vagally mediated reflex in response to laryngeal irritation.

The postnasal drip of sinusitis is another airway irritant that has been linked with both extrathoracic airway hyperresponsiveness and bronchial hyperresponsiveness (Bucca et al., 1995). Although the cause-and-effect relationship needs to be studied further, the prevalence of rhinosinusitis in PVFM patients (73% in an irritant-associated PVFM patient group and 91% in a PVFM alone group) supports the notion that the exudate and other inflammatory processes of chronic sinusitis may play a role in PVFM (Perkner et al., 1998). Further evidence for this relationship is found in case reports in which treatment of chronic rhinitis and postnasal drip coincided with PVFM symptom relief (Lakin, Metzger, & Haughey, 1984).

Numerous other respiratory tract irritants, such as smoke, gases, vapors, dust, airborne pollutants, and odors, have been linked to PVFM onset (Brugman & Newman, 1993; Perkner et al., 1998). Although exposure to such irritants may cause vocal fold inflammation, the underlying mechanism for triggering PVFM remains unknown. Perkner et al. (1998) suggested that we use the label "irritant-associated vocal cord dysfunction" (IVCD) for those cases where the onset of respiratory symptoms is associated with exposure to occupational or environmental irritants. It is necessary to recognize both that the irritant may be contributing to the respiratory complaints and that the IVCD is responsible for the airway obstruction.

Neurologic Causes

Some authors have described a subgroup of patients whose seemingly functional PVFM disorder may be the manifestation of an underlying laryngeal dystonia. Treole, Trudeau, and Forrest (1999) reported that subtle signs of laryngeal movement disorder were found in PVFM patients who were examined between dyspneic episodes. The clinical signs included ventricular fold medialization and phonatory instability, suggesting that muscular incoordination was present during both respiration and phonation in asymptomatic patients. They also found that during vocal fold vibration, both the lateral excursion and mucosal wave patterns were reduced in almost half of the PVFM subjects. Treole et al. (1999) concluded that these abnormal patterns of laryngeal valving are likely a chronic condition, and that dyspneic episodes may be an exacerbation of a continuous laryngeal dystonia. They suggested that dyspneic episodes occur when the demands on the respiratory system exceed what the dystonic laryngeal valve will permit. These intriguing findings and conclusions warrant further exploration. For example, one must be assured that the signs of an apparent chronic and neurologically based movement disorder are not simply an examination artifact in patients who are somewhat anxious about laryngeal behavior. Furthermore, although the authors' interpretation is compelling, additional evidence is needed to support the hypothesis that there may be a neurogenic abnormality accounting for PVFM in at least a subgroup of patients.

There are other patients whose *persistent* inspiratory stridor and/or dyspnea are definitely associated with a neurogenic cause. Adductor laryngeal breathing dystonia (ALBD) was first identified by Blitzer and Brin (1991). The paradoxical movement of the vocal folds on inspiration, and associated stridor, are apparently triggered by inspiratory effort during waking states. The patient is asymptomatic during sleep. Patients have exhibited tremor or dystonic symptoms elsewhere (e.g., facial dystonia, tongue dystonia, blepharospasm), along with dysrhythmia of the respiratory muscles (Grillone, Blizer, Brin, Annino, & Saint-Hilaire, 1994). It should be noted that these patients suffer from ongoing airway compromise and inspiratory stridor that distinguish their ALBD symptoms from the episodic and paroxysmal symptoms of the PVFM patient (Gallivan, Hoffman, & Gallivan, 1996).

Other rarely occurring neurogenic causes for paradoxical vocal fold movement are discussed by Maschka et al. (1997). These etiologies include brainstem compression, cortical or upper motor neuron injury, nuclear or lower motor neuron injury, and movement disorders in addition to those mentioned above.

In summary, there are a variety of conditions that can cause PVFM. It may be linked to psychological factors. It may appear to be functional with no obvious psychological or organic etiology. Upper airway sensitivity to irritants may account for PVFM in some patients, whereas others may have an underlying neurogenic condition. It behooves speech-language pathologists to be well informed regarding these possibilities and their diagnostic and treatment implications.

Assessment and Differential Diagnosis

The differential diagnosis of PVFM should be based on clinical history, pulmonary function and lab test results, laryngoscopic findings, speech-language pathology evaluation, and psychological evaluation. The psychological evaluation is particularly important if there is evidence or suspicion of a significant psychological component. Thus, close relationships among various professional

disciplines (e.g., pulmonary medicine, allergy/immunology, otolaryngology, neurology, psychiatry/psychology, speech-language pathology) throughout the diagnostic and the intervention phases is ideal. The SLP may be involved in various ways and should be knowledgeable about the medical information provided by other specialists. The SLP evaluation protocol is presented later in this paper. The following sections consider the diagnostic findings based on clinical history, pulmonary function and lab tests, and laryngoscopic observations.

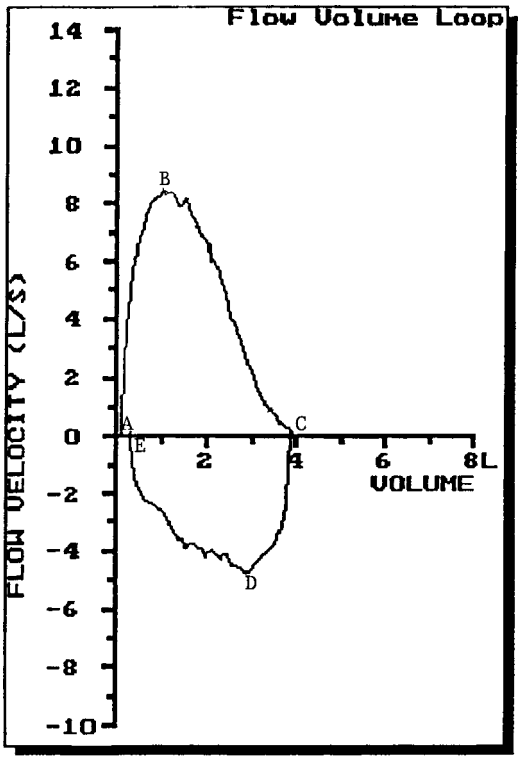
Clinical History

The case history provides critical clues to making the PVFM diagnosis (Brugman & Newman, 1993; Martin et al., 1987). It is important first to focus on the patient's description of the problem. Patients usually point to the laryngeal area when asked where the breathing obstruction occurs. Initially, some patients may focus on the chest, but they readily will appreciate the laryngeal region involvement when asked directly about "throat area" airflow restriction. Occasionally, patients also report dysphonia and dysphagia. Although the pattern and duration of breathing obstruction vary across patients, many individuals with PVFM report sudden onset and cessation of symptoms (Brugman & Newman, 1993). Patients may recall that the disorder began subsequent to an upper respiratory infection or similar illness (Rogers & Stell, 1978). Adults are often aware of conditions or substances that trigger the symptoms. In one study of a group of adolescents and adults, 45% readily identified a psychological trigger (O'Connell, Sklarew, & Goodman, 1995). Other precipitators frequently mentioned by adults include exercise; exertion; hyperventilation; laughing, very hot or cold air; and airborne irritants such as dust, smoke, odors, gases/fumes, and chemicals (Brugman & Newman, 1993; Kivity et al., 1986; Perkner et al., 1998). Children and adolescents tend to experience PVFM attacks during heightened physical activity (e.g., Kayani & Shannon, 1998; Landwehr, Wood, Blager, & Milgrom, 1996; McFadden & Zawadski, 1996) and in association with psychosocial stressors (e.g., Alpert, Dearborn, & Kercsmar, 1991; Dinulos, Karas, Carey, & Del Beccaro, 1997; Kuppersmith et al., 1993).

Pulmonary Function Tests

A number of different tests are used to determine the nature and cause of airway obstruction. Although a full explanation is beyond the scope of this paper, several key tests will be described. The flow-volume loop (see Figure 1) maneuver is the most commonly cited test of pulmonary mechanics that provides some insight into the nature of airway obstruction (Tenholder, Moser, Koval, Bennett, & Rajagopal, 1987). The curve represents airflow during a forced vital capacity maneuver, where maximally forced expiration and maximally forced inspiration are displayed on the same figure. Airflow rate, plotted on the ordinate, is recorded in liters per second, whereas volume, plotted on the abscissa, is recorded in liters. The flow-volume maneuver entails having the subject inhale completely (to total lung capacity), then exhale as completely and quickly as possible, and then inhale maximally. In Figure 1, ABC is the expiratory portion of the loop; CDE is the inspiratory portion of the loop. Maximal expiratory flow at 50% of the vital capacity (MEF50) and maximal inspiratory flow at 50% of the vital capacity (MIF50) are also reference points that are mentioned in the battery of pulmonary function tests. The flow-volume loop and MEF50/MIF50 ratios provide important diagnostic clues, since restrictive lung disease and various sources of airway obstruction result in characteristic flow-volume loops and ratios (Talavera, Kumar, & Casaburi, 1998; Tenholder et al., 1987).

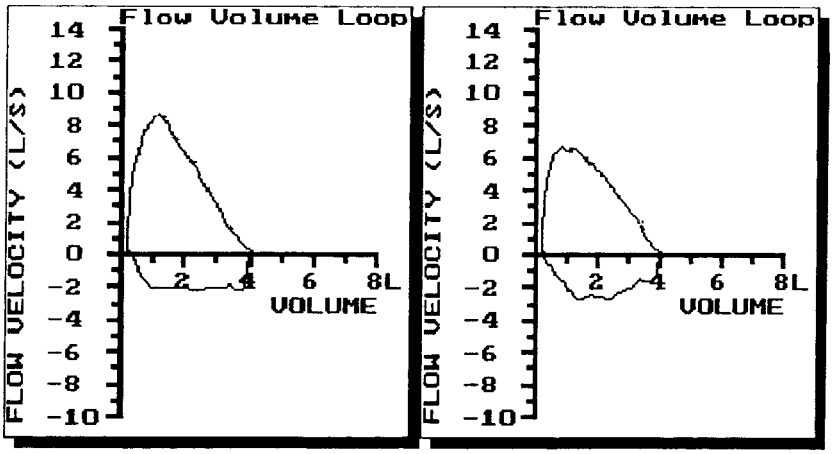
FIGURE 1. Normal flow-volume curve of a patient with vocal cord dysfunction during an asymptomatic period. ABC is the expiratory portion of the loop; CDE is the inspiratory portion of the loop.



When there is a variable extrathoracic[1] upper airway obstruction, the flow-volume loop shows an inspiratory cut-off, or flattening of the inspiratory limb (see Figure 2). This abnormal flow-volume loop is seen in PVFM patients *when they are symptomatic* (Brugman & Newman, 1993; Christopher et al., 1983; Selner et al., 1987). Some PVFM patients will show a reduction of the expiratory limb in addition to the inspiratory loop cut-off (Christopher et al., 1983; McFadden, 1987); in such cases, a cough may elicit

[1] The upper airway extends from the mouth to the level where the most inferior tracheal cartilage bifurcates to form main stem bronchi. The extrathoracic portion of the upper airway is located superior to the sternal notch (Littner, 1994).

FIGURE 2. Abnormal flow-volume curves on repeated trials during a symptomatic period.



vocal fold abduction on exhalation, with subsequent increased flow on the expiratory loop (Martin et al., 1987). Although the flow-volume loop tends to be normal when subjects are asymptomatic, Newman et al. (1995) reported that 25% of their asymptomatic PVFM patients showed the characteristic inspiratory cut-off. Extrathoracic airway obstruction also is suggested by abnormal flow ratios, with the MEF50/MIF50 exceeding 1.5 in symptomatic patients (Brugman & Newman, 1993). These ratios usually will be normal when the PVFM patients are asymptomatic (Christopher et al., 1983), although elevated MEF50/MIF50 ratios have been reported in asymptomatic PVFM patients (Newman et al., 1995).

Referral information on PVFM patients may include results of other tests related to pulmonary function (e.g., McFadden, 1987). Pulse oximetry is used to measure the amount of oxygen carried by arterial blood, with results reported as percentage of oxygen saturation. In this noninvasive procedure, a probe sensor is placed around a finger (Arvedson & Rogers, 1998; Rushworth, 1999). In the individual with PVFM symptoms, pulse oximetry and arterial-blood-gas analyses usually will be normal to mildly abnormal, and show patterns that suggest glottic dysfunction rather than any type of obstructive lung disease. For example, both arterial O_2 and CO_2 will be normal to slightly reduced during an acute attack of PVFM. Alveolar-arterial gradients for oxygen will be normal, and may even narrow (McFadden, 1987; Newman & Dubester, 1994). The patient's response to airway reactivity testing, or "bronchial provocation," also is often reported. The procedure involves completing standard pulmonary function tests before (baseline) and after the patient inhales methacholine or histamine. If the forced expiratory volume at one second (FEV_1) is reduced 20% or more when compared to baseline, the test indicates bronchial hyperreactivity characteristic of asthma (Christopher et al., 1983; Corren & Newman, 1992). Hence, a negative histamine/methacholine challenge test, suggesting normal lower airway reactivity, helps to rule out asthma. In patients with combined PVFM and asthma, pulmonary function test results will be the same as those of asthma-only patients (Newman et al., 1995).

Laryngoscopic Observations

Christopher et al. (1983) were the first to report the classic laryngeal examination findings in the PVFM patient, and described them in the following way. When the patient is not experiencing PVFM symptoms, the laryngeal structure and function appear to be normal. When the patient is symptomatic, however, the true vocal folds adduct almost completely during tidal breathing, with a small diamond-shaped glottal chink posteriorly. There also may be a medialization of the ventricular folds. When the PVFM patient is asked to voluntarily produce the dyspnea symptoms, vocal fold behavior is similar to that of control and asthmatic subjects during attempted simulation; that is, the true vocal folds adduct but with a bowing configuration. The posterior glottal chink and false vocal fold medialization are not observed when patients attempt to simulate the PVFM symptoms. Others have confirmed and expanded Christopher's observations, noting that adduction of the anterior two-thirds of the vocal folds and posterior chinking typically occur during inspiration but may be present during both inspiration and expiration or during expiration only (Brugman & Newman, 1993; Martin et al., 1987; Newman & Dubester, 1994). Newman et al. (1995) reported laryngoscopic findings diagnostic of PVFM in 100% of their symptomatic patients, and *60% of their asymptomatic patients*. Similarly, Powell et al. (2000) identified true vocal fold adduction during quiet respiration in 55% of their asymptomatic juvenile patients. If the patient is asymptomatic at the time of the laryngoscopic examination, symptoms may sometimes be elicited by having the patient pant, breathe deeply, and phonate; exercise and bronchial challenge also may induce PVFM symptoms (Brugman & Newman, 1993; Martin et al., 1987; Newman et al., 1995). Idiosyncratic triggers reported by the patient very likely will provoke symptoms as well (Selner et al., 1987). Despite the reported definitive nature

of the laryngoscopic exam, it must be noted that in some cases the patient remains asymptomatic throughout the exam; therefore, a normal laryngeal exam in an asymptomatic patient does not preclude PVFM diagnosis (Landwehr et al., 1996; Newman et al., 1995).

Airway Fluoroscopic Evaluation

Although laryngoscopic examination of the symptomatic patient is considered the gold standard of diagnosing PVFM, in some cases it may not be possible to do the examination—perhaps because of concern about triggering a bronchospasm or because it is difficult to complete the invasive procedure while the patient is symptomatic. Several medical teams have used airway radiographs and videofluoroscopy to evaluate the laryngeal structures during respiration (Nastasi, Howard, Raby, Lew, & Blaiss, 1997; Shao et al., 1995). Soft tissue radiography provides a means of evaluating airway mass or narrowing. When the patient is symptomatic, both the frontal view airway videofluoroscopy and radiography reveal inspiratory vocal fold medialization. Both Nastasi et al. (1997) and Shao et al. (1995) acknowledged that the noninvasive radiographic and fluoroscopic examinations must be supplemented by a laryngoscopic examination to rule out other possible laryngeal abnormalities.

Differential Diagnosis

The diagnosis of PVFM is often one of exclusion. These patients are most commonly misdiagnosed as having asthma or exercise-induced bronchospasm/asthma (EIB/EIA) (Christopher et al., 1983; O'Connell et al., 1995). Those characteristics that distinguish PVFM from asthma are presented in Table 1.

Both the PVFM and asthma patient will complain of dyspnea that is precipitated by triggers that might include exercise, extreme temperatures, airway irritants, or emotional stressors (Brugman & Newman, 1993; Interiano & Guntupalli, 1996; Perkner et al., 1998). Whereas PVFM usually is elicited by only one trigger, asthma commonly is aggravated by multiple triggers (McFadden & Gilbert, 1994). Unlike the PVFM patient, asthma patients may report sputum production upon coughing and a sensation of increased chest pressure during an attack (Newman et al., 1995). Although the frequencies of asthmatic wheezing and extrathoracic stridor are similar, the location and timing of the breathing noises differ (Baughman & Loudon, 1989). PVFM stridor is heard on inspiration, with the sound being prominent in the neck area. Wheezing, on the other hand, is heard on expiration and is more prominent over the chest area. Following an acute asthma attack, pulmonary function tests may find signs of airway obstruction for several weeks, suggesting a longer recovery period for asthma compared to PVFM (Corren & Newman, 1992). In addition, the presence of nocturnal awakening with symptoms frequently is cited as being characteristic of asthma and not PVFM (e.g., McFadden, 1987). The asthmatic individual also demonstrates respiratory symptom relief in response to either bronchodilators or more aggressive therapy involving systemic corticosteroids (Corren & Newman, 1992; Landwehr et al., 1996; Newman et al., 1995). This is not the case with the PVFM patient.

TABLE 1. Some differential diagnostic features of PVFM and asthma.

Diagnostic Features	PVFM	Asthma
Precipitators (triggers)	Exercise, extreme temperatures, airway irritants, emotional stressors	Exercise, extreme temperatures, airway irritants, allergens, emotional stressors
Number of triggers	Usually one (e.g., exercise)	Usually multiple triggers
Breathing obstruction location	Laryngeal area	Chest area
Timing of breathing noises	Stridor on inspiration	Wheezing on exhalation
Pattern of dyspneic event	Sudden onset and relatively rapid cessation	More gradual onset and longer recovery period
Nocturnal awakening with symptoms	Rarely	Almost always
Response to bronchodilators and/or systemic corticosteroids	No response	Good response
Flow-volume loop	Inspiratory cut-off; perhaps some expiratory limb reduction[a]	Reduced expiratory limb only
MEF50/MIF50	Abnormal (>1.5)[a]	Abnormally small ratio
Arterial-blood-gas analyses	O_2: normal to slightly hypoxemic CO_2: normal to slightly reduced	O_2: arterial hypoxemia
Bronchial provocation test	Negative	Positive
Laryngoscopic observations	Inspiratory adduction of anterior 2/3 of vocal folds; posterior diamond-shaped chink; perhaps a medialization of ventricular folds Inspiratory adduction may carry over to expiration[a]	Vocal folds may adduct during exhalation

Note. See text for detailed discussion and exceptions to these distinguishing features.

[a]When patient is symptomatic.

The asthma patient will respond differently to pulmonary function and lab tests as well. Typically, asthma is indicated by a reduced expiratory limb on the flow-volume loop and reduced MEF50 to MIF50 ratios (Brugman & Newman, 1993). Blood gas analyses will indicate arterial hypoxemia (deficient oxygenation of the blood) and enlarged alveolar-arterial gradients for oxygen (Christopher et al., 1983; McFadden, 1987). In addition, patients with asthma typically show airway hyperreactivity to bronchial provocation studies (e.g., a methacholine challenge).

As mentioned above, the definitive diagnosis of PVFM relies on the laryngoscopic finding of inspiratory/expiratory adduction of the anterior two-thirds of the vocal folds and posterior diamond-shaped chinking in the symptomatic patient. It should be noted that it is normal for the vocal folds to adduct slightly during exhalation in healthy adults. The slowing of the exhalatory airflow as a result of both increased laryngeal resistance and inspiratory muscle contraction maintains an ideal rate of respiratory system collapse (Kuna, Insalaco, & Woodson, 1988). In patients with active asthma, glottic narrowing during exhalation is substantially increased, thereby stabilizing the airways and chest wall and providing more time for alveolar ventilation and gas exchange (Collett, Brancatisano, & Engel, 1983; Imam & Halpern, 1995). Using the relatively small laryngeal muscles to regulate ventilation, rather than contracting the larger abdominal and thoracic muscles, is metabolically cost-effective and reduces the likelihood of respiratory fatigue in asthma patients (McFadden, 1987). Thus, in the patient with chronic airflow obstruction, increased vocal fold adduction during exhalation should be viewed as normal, particularly during exercise and active disease. This should be kept in mind when making a diagnosis of PVFM based on seemingly abnormal laryngeal configuration changes that only take place during exhalation (Brugman & Newman, 1993).

There are limitations and exceptions to these differential diagnostic guidelines. The presence or absence of stridor is not a reliable indicator of vocal fold function: Stridor may be perceived even when the vocal folds function normally, and it may be absent when vocal folds function abnormally (Martin et al., 1987). Furthermore, since the large airways are excellent sound conductors, it may be difficult to determine if the sound is originating in the neck or chest. Hence, auscultation may be an unreliable physical examination for differentiating laryngeal from lower airway sounds (Newman & Dubester, 1994). The differential diagnostic utility of nocturnal awakening with symptoms also has been questioned, given the number of PVFM cases who report this feature (Reisner & Nelson, 1997). However, the *absence* of nocturnal awakening is suggestive of a nonasthmatic condition, as almost all asthmatic patients experience nighttime symptoms (McFadden & Zawadski, 1996). There are exceptions to the distinguishing features of the pulmonary function tests as well. Niven and Pickering (1991) cited numerous examples of PVFM patients with abnormal blood gases. Similarly, Hayes, Nolan, Brennan, and FitzGerald (1993) reported marked hypoxemia in a young woman with PVFM. McFadden (1987) emphasized that given the dynamic nature of PVFM, and the fact that various abnormalities can occur over time in the same patient, the flow-volume loop is not a definitive diagnostic measure. He also noted that lack of airway reactivity does not always characterize the PVFM patient. Both McFadden (1987) and Selner et al. (1987) observed exacerbation of PVFM symptoms in response to bronchoprovocation.

The differential diagnosis is further complicated by the fact that the spectrum of PVFM includes cases where it co-occurs with asthma or exercise-induced bronchospasms (McFadden, 1987; O'Connell et al., 1995). These patients show signs of asthma but experience intermittent airway obstruction symptoms that are not adequately responsive to the usual regimen of bronchodilators and steroids (Elshami & Tino, 1996; Imam & Halpern, 1995; Meltzer et al., 1991). The laryngoscopic finding of vocal fold adduction on inspiration is an important diagnostic clue, because it always is considered to be abnormal and may be indicative of PVFM (Newman, Mason, & Schmaling, 1995). Accurate diagnosis of coexisting asthma and PVFM leads to appropriate treatment, which includes pharmacologic treatment for the asthma in addition to behavioral intervention for PVFM.

The consequences of treating unrecognized PVFM as asthma have been presented in numerous case reports. In many instances, failing to recognize PVFM has led to unnecessary drug use, hospitalization, intubation, and even tracheostomy in both children and adults (e.g., Appelblatt & Baker, 1981; Dinulos et al., 1997; George, O'Connell, & Batch, 1991; Landwehr et al., 1996; Logvinoff, Lau, Weinstein, & Chandra, 1990; Pacht & St. John, 1995).

The differential diagnosis of PVFM requires recognition of other conditions that might produce symptoms of dyspnea with or without stridor. These include infection, anaphylaxis, trauma, tumor, and structural abnormalities throughout the airway; in addition, congestive heart failure and chronic obstructive pulmonary disease (COPD or emphysema) must be considered (Fields, Roy, & Ossorio, 1992; Heiser et al., 1990; Mullinax & Kuhn, 1996; O'Connell et al., 1995). When the upper airway is the clear site of obstruction, several conditions other than PVFM must be ruled out. Examples include tracheal stenosis; thyroid enlargement; laryngeal tumor, edema, or abscess; foreign body aspiration; laryngeal trauma; and vocal fold paralysis or other neurologically based dyskinesia (Fields et al., 1992; O'Hollaren, 1990). Finally, PVFM may coexist not only with asthma, but also with COPD—with laryngeal behavior ranging from a compensatory respiratory braking mechanism during exhalation to a noncompensatory (i.e., pathologic) vocal fold adduction during inspiration (Wood, Jafek, & Cherniack, 1986). It is important for the speech-language pathologist to be aware of these many alternative diagnostic considerations. When symptoms do not support a diagnosis of PVFM, patients should be referred back to their physicians for consideration of other possible diagnoses.

Childhood and Adolescent PVFM

As in adult cases, children with PVFM may be misdiagnosed by their pediatricians or emergency room physicians

and may experience months, or even years, of misdirected intervention while being treated for refractory asthma (e.g., Logvinoff et al., 1990; Tan, Eng, & Ong, 1997). PVFM has been reported in children and adolescents without coexisting respiratory disease (Dinulos et al., 1997; Kivity et al., 1986; Logvinoff et al., 1990), in children with cystic fibrosis (Crowley & Bush, 1995; Shiels, Hayes, & Fitzgerald, 1995), and in children with underlying asthma (Caraon & O'Toole, 1991; Dinulos et al., 1997; Meltzer et al., 1991; Sette, Pajno-Ferrara, Mocella, Portuese, & Boner, 1993; Warburton, Niven, Higgins, & Pickering, 1996).

Among children and adolescents, exercise-induced PVFM is becoming increasingly recognized in athletes, who frequently have been misdiagnosed with refractory exercise-induced asthma (EIA). Sports competition, practice, or simply strenuous activity can trigger the PVFM attacks (Brugman & Newman, 1993; McFadden & Zawadski, 1996). In some cases transference occurs, and PVFM symptoms will be elicited in other stressful situations, such as when taking exams at school (Brugman & Newman, 1993). It should be noted that in exercise-induced PVFM the patients frequently report only one trigger, whereas in the asthmatic patient additional precipitants will emerge over time (McFadden & Gilbert, 1994). Exercise-induced PVFM also is suspected when there is no response to the antiasthma medications. Typically, EIA is prevented by administering antiasthma medications before the activity. In the case of an EIA attack, control is established with the subsequent use of medications (McFadden & Zawadski, 1996). Symptom onset with PVFM is sudden and occurs within a few minutes of the increased level of physical activity; symptom resolution usually is rapid as well (Landwehr et al., 1996). In EIA, on the other hand, symptoms develop after 5 to 10 minutes of intense exercise, and there often is a relatively long recovery period ranging from 15 to 30 minutes (Interiano & Guntupalli, 1996; Storms, 1999).

Most reports of exercise-induced PVFM include descriptions of emotional issues that may be playing a role, such as "immature personality in conflict with his family" (Sette et al., 1993, p. 410), anxiety disorder, depressive disorder, and both anxiety and depressive disorder (Landwehr et al., 1996). Many reports acknowledge the highly competitive personality of the exercise-induced PVFM patient population, including adults. They typically show little tolerance for failure, may have advanced recently to a higher level of competition, have high academic and performance standards, and are under parental pressure to succeed (Brugman & Newman, 1993; McFadden & Zawadski, 1996; Morris, Deal, Bean, Grbach, & Morgan, 1999).

Once again, one must be careful regarding the assumptions that are made about psychopathology. According to Brugman and Newman (1993), psychopathology is not typically seen in children with PVFM, although stress may play a role. However, in several pediatric PVFM case reports, the authors present evidence of clearly established coexisting psychiatric disorder—such as posttraumatic stress associated with sexual abuse (Tajchman & Gitterman, 1996), a history of anxiety and depression (Mullinax & Kuhn, 1996), or family and child investment in the illness (Meltzer et al., 1991). In others, psychosocial stressors such as parental divorce, parent-child conflict, or parental anxiety about academic performance may be associated with PVFM (Alpert et al., 1991). A retrospective study by Powell et al. (2000) revealed that 55% of their juvenile patients had social stresses, including involvement in extracurricular activities and competitive sports. However, as in many other case reports and retrospective studies, the researchers did not compare this finding to a healthy control group, nor did they demonstrate a cause and effect link between these stresses and PVFM. Only one small prospective study has demonstrated that adolescents with PVFM are more likely to experience anxiety disorders, with anxiety preceding dyspneic episodes, than are matched control subjects with asthma alone (Gavin, Wamboldt, Brugman, Roesler, & Wamboldt, 1998).

Finally, it is interesting to note that Mrazek (1992) estimated that 25% of asthmatic children have some type of psychopathology and that antiasthmatic medications may contribute to emotional disorders such as depression or anxiety via a pharmacologic effect. This should be carefully considered in PVFM cases where there is coexisting asthma or long-term drug treatment of PVFM that had been misdiagnosed as asthma.

McQuaid, Spieth, and Spirito (1997) provided a helpful perspective on the role of psychological factors in pediatric PVFM cases. When a child experiences difficulty breathing for which there is no clear medical explanation and for which the treatment being provided is not effective, it stands to reason that the parent and child will become anxious and frustrated. The child and parent may become caught in a cycle wherein the emotional reaction to the disorder exacerbates the disorder. According to McQuaid et al. (1997), it is both adaptive and appropriate for a child in this situation to seek comfort from a parent. Furthermore, the emotional issues warrant supportive intervention, even though they are not necessarily causal. It is best left to a qualified mental health professional to determine to what extent the child and parent are reacting reasonably to a frustrating situation, and to what extent there may be an element of secondary gain. In addition, it is important for all involved to appreciate that even when it is clearly established that psychological factors trigger PVFM symptoms, these symptoms are not under voluntary control.

Treatment of PVFM

In each case of PVFM, the differential diagnosis will determine appropriate treatment. Numerous disciplines likely will be involved in the intervention process.

Medical Intervention

Medical intervention for PVFM typically involves patient education, termination of unnecessary medications, and prescription of medications to treat contributory conditions and PVFM. A physician, usually the otolaryngologist or pulmonologist, reviews the diagnosis with the

patient and family members. Martin et al. (1987) recommended reviewing both the results of the tests and the videotape of the laryngeal exam to help the patient understand the nature of PVFM. Termination of unnecessary medical treatment is attempted early in the intervention process. For patients who were misdiagnosed as asthmatic or overmedicated for a coexisting asthma, physicians will taper the use of drugs, particularly corticosteroids where long-term-use side effects include growth retardation in children (Brugman & Newman, 1993; Christopher et al., 1983; Newman et al., 1995). Similarly, when a tracheotomized patient achieves control of breathing, decannulation will be completed (Martin et al., 1987).

When airway irritants such as GERD, rhinosinusitis, or allergens are identified as likely PVFM triggers, pharmacological therapy and lifestyle modifications are warranted. Some patients benefit from medical intervention to alleviate the dyspnea symptoms. For example, many patients experience at least temporary relief from breathing heliox, a 70% helium and 30% oxygen mixture that is easily inhaled because of its low density (Brugman & Newman, 1993; Christopher et al., 1983; Martin et al., 1987). Other physicians have found this ineffective, at least in some cases (Logvinoff et al., 1990). Another short-term treatment approach, that of administering continuous positive airway pressure (CPAP), also has been reported. Goldman and Muers (1991) suggested that this is particularly effective when there is vocal fold adduction during exhalation. They assumed that CPAP relieves dyspnea symptoms by slowing the expiratory flow, thereby increasing lung volume, which in turn results in a more open glottis (Goldman & Muers, 1991). CPAP also is administered for the more typical PVFM where there is glottic closure on inhalation; in this case, CPAP effectiveness is attributed to reducing the effort needed for inspiration by establishing a favorable pressure gradient for inhalation (Heiser et al., 1990). Although they are not usually indicated, sedative or antianxiety drugs may be used temporarily when the patient's anxiety exacerbates PVFM. Once techniques are learned to control the symptoms, the drug is discontinued (e.g., Brown et al., 1988; Heiser et al., 1990; Mullinax & Kuhn, 1996). The use of sedatives in treating acute PVFM also has been reported in several cases when PVFM occurred postoperatively following endotracheal tube removal (Arndt & Voth, 1996; Michelsen & Vanderspek, 1988; Snyder & Weiss, 1989). A prospective study has suggested that electroencephalographic (EEG) neurofeedback can be used to treat paradoxical vocal fold motion (Nahmias et al., 1994). In severe cases of PVFM when the patient is unresponsive to other types of intervention, tracheostomy may be warranted as a treatment of last resort (Newman & Dubester, 1994). Other isolated medical treatment approaches include botulinum toxin injection and laryngeal nerve sectioning (Newman & Dubester, 1994). Although these techniques have been used successfully in treating laryngeal dystonia, there currently is little justification for such extreme treatment measures in PVFM. Furthermore, unilateral laryngeal nerve sectioning places the patient at risk for severe dysphonia should the contralateral nerve be injured.

Psychotherapy

Psychological intervention may be indicated in some PVFM cases and may range from helping patients learn to express the real source of stress to dealing with serious psychopathology. Martin et al. (1987), who viewed PVFM as a type of conversion disorder, recommended that the immediate goal of psychotherapy should be to reduce the need for PVFM symptoms, rather than to focus on resolving more serious underlying psychopathology. Therapy is supportive—providing a context for patients to express fears, discuss concerns about their care, and find support for the positive changes they make. Patients will first learn to recognize emotional stresses that may trigger symptoms, and then to express emotions in increasingly difficult situations. The focus is on gaining control of situations that trigger PVFM rather than dealing with psychopathology. McQuaid et al. (1997) recommended traditional intervention, which might include the following: relaxation therapy to alleviate distress associated with symptoms, identification of stressors that contribute to PVFM, and development of new coping strategies for dealing with those stressors. Family therapy may be indicated as well, because a child's illness may serve a negative function in the family and may provide the mechanism for thwarting a child's emancipation from the family (Martin et al., 1987). Longer-term psychotherapy is recommended when serious underlying issues need to be addressed more fully (Newman & Dubester, 1994). Care must be taken to recognize when psychiatric evaluation is indicated (e.g., Heiser et al., 1990).

Psychotherapeutic intervention in PVFM has not been studied systematically, although case reports provide some insight. Child and adolescent PVFM case reports range from a 12-year-old boy responding positively to 1 week of psychotherapy (Meltzer et al., 1991) to a 16-year-old boy requiring 9 months of psychological intervention, along with speech therapy, to achieve full recovery (Sette et al., 1993). Family counseling revealed patient anxiety regarding parental expectations in a patient described by Alpert et al. (1991). Hypnotherapy also has been reported as a successful intervention modality. Caraon and O'Toole (1991) reported success using short-term hypnotherapy with a 14-year-old boy with both PVFM and asthma.

There is a spectrum of PVFM patient characteristics. Some patients will accept the notion that psychological factors may play a role in their disorder and pursue recommended psychiatric/psychological treatment, whereas others cannot accept that notion and fail to follow such recommendations (Freedman et al., 1991; Kivity et al., 1986; McFadden & Zawadski, 1996; Selner et al., 1987). Many patients, however, will respond to the behavioral intervention that is provided by the speech-language pathologist (Christopher et al., 1983; Martin et al., 1987; Newman et al., 1995). In fact, Brugman and Newman (1993, p. 3) stated, "Speech therapy is the first line of treatment for [PVFM] and by itself often is sufficient to correct the disorder."

Speech-Language Pathology

The medical literature suggests that the SLP's primary role is to teach the PVFM patient how to control the laryngeal area and maintain an adequately open airway during respiration (e.g., Brugman & Newman, 1993; Kuppersmith et al., 1993; Lacy & McManis, 1994; Martin et al., 1987; Mullinax & Kuhn, 1996). Though this may be true, I believe that the SLP plays a broader role in the management of patients with PVFM. That role extends to assessment, diagnostic input, patient education, supportive counseling, PVFM symptom management, and possible voice therapy.

SLP Assessment. Assessment includes conducting a patient interview, determining laryngeal valving performance, measuring respiratory driving pressure for speech, assessing musculoskeletal tension in the laryngeal area via laryngeal palpation, and completing a structural/functional examination of the speech structures. A history and assessment form and protocol can be found in the Appendix. Although the referring physician often provides some information, the SLP should take a thorough and complete history. During that history-taking, information not previously obtained may emerge, inconsistent or discrepant information can be pursued, the interaction between a parent and child can be observed, and the impact of the problem can be gauged.

In the majority of patients, laryngeal and respiratory behaviors are found to be unimpaired. This information is useful in the patient education stage of treatment, as it supports the concept of the inherent "normalcy" of the structures and functions involved. However, when a problem in a given area is uncovered, it is important that further exploration of it be considered. For example, if the patient is noted to have a voice abnormality, a complete voice examination may be indicated (Colton & Casper, 1996; Morrison & Rammage, 1994). The result of this assessment should be an affirmation of the suspected PVFM and the initiation of treatment, or the recommendation that further examination be pursued, be it psychiatric, neurological, or other.

SLP Treatment. A treatment protocol is outlined, with the caveat that treatment must be tailored to the specific needs of the patient. Furthermore, each aspect of this approach warrants investigation in controlled studies in order to determine research-based clinical efficacy.

Patient education. Review the assessment results, demonstrating for the patient (and family members) all that is normal about the patient's respiratory and laryngeal control. This is done to reassure the patient, in a manner that does not diminish the reality of the patient's symptoms and concerns. Briefly educate the patient about respiratory and laryngeal structures, the active and passive forces associated with respiration, and the respiratory and phonatory functions of the larynx. This is accomplished, of course, at a comprehension and interest level appropriate for the patient. Providing the patient with a basic understanding of respiration seems to facilitate both learning to trust the process and learning to control it. Following the discussion of normal processes, review the PVFM symptoms in light of what the patient has just learned. If possible, review the patient's laryngeal exam videotape.

Supportive counseling. At this point, it is extremely important to acknowledge the patient's fears and the feeling that the respiratory symptoms cannot be controlled during an acute attack. The feeling of being unable to breathe is terrifying and understandably leads to a sense of panic and a tendency to struggle. It may be appropriate to reflect what the patient already has shared about related emotional issues and possible triggers for the PVFM. Discuss how the patient will learn techniques to control breathing, gradually working toward using the techniques in situations similar to those where the PVFM is most likely to occur.

Instruction in tension identification and control. These skills can be taught according to a modified progressive relaxation approach. In Skill Level 1, guide the patient through steps of tightening *maximally*, and then letting go of tension, in relevant muscle groups, as follows: Forehead—"Lift the eyebrows, tightening the muscles in the forehead as much as you can. Hold it. Notice what that feels like when you are tightening the muscles. Let go. Notice the difference." Provide similar instructions as the patient is guided through other muscle groups (eye area—squint and furrow the brow; jaw area—bite down; lips—purse/press together; tongue—pull tongue back/press tongue tip up to alveolar ridge; neck—pull the head back and down slightly; shoulders—lift the shoulders; laryngeal area—take a breath, hold it, and bear down as though lifting something heavy; inhalatory muscles—inhale maximally and hold it; abdominal area—move the abdominal wall inward by tightening the abdominal muscles). For each muscle group, the patient is told to maximally contract the muscles; hold the posture, noting what it feels like when tightening muscles excessively; let go; and notice the difference.

After the patient completes this series of tasks that involve discriminating and controlling excessive tension and reduced tension, move to the next skill levels. In Skill Level 2, instruct the patient to go through the muscle groups as indicated above, tightening *moderately*, rather than maximally. In Skill Level 3, instruct the patient to simply tune into the various muscle groups (noting to what extent the patient is tightening the muscles) and then let go to achieve a reduced tension level. As each skill level is mastered with clinician guidance, the patient is encouraged to practice frequently in settings outside the clinic. The rationale for these exercises is that the patient will develop an enhanced awareness of even slight tendencies to tighten muscles. More important, the patient will recognize that one can "let go" of this tension. The extent to which these steps are practiced in nonrespiratory muscle groups depends on each patient's tendency both to tighten excessively throughout the body and to generalize that increased tension to the respiratory and laryngeal area muscles. Ultimately, the emphasis should be on readily identifying and reducing excessive tension associated with respiration, during a variety of activities and in a variety of settings. By learning to detect increased tension, the patient can implement easier breathing behaviors before an acute PVFM episode occurs.

Instruction in "relaxed-throat breathing" (Brugman & Newman, 1993; Martin et al., 1987). The relaxed-throat breathing technique consists of a few simple steps that focus attention away from the larynx and emphasize control of diaphragmatic breathing—or lower chest wall expansion—during inhalation. After reminding the patient to release tensions throughout the jaw, lips, tongue, and shoulder area, instruct the patient as follows:

> *Inhalation*—"Breathe in through the nose, with the tongue resting forward on the floor of the mouth and the lips gently together. Allow expansion in the lower rib cage and abdominal area." Highlight the expansion of inhalation by having the patient place a hand inferior to the sternum, or by having the patient place the hands on either side of the lower rib cage with the thumbs pointing toward the back.
>
> *Exhalation*—"Let the air out with a hissing sound or gently prolonged 's'." Emphasize the passive aspect of exhalation. Martin et al. (1987) suggest that the patient should be taught to focus on exhalation, thereby lessening the patient's tendency to hold his or her breath and then struggle to inhale. Instruct the patient to count silently while exhaling, gradually increasing the count to facilitate exhaling to a lung volume around resting exhalatory level. Exhaling to an appropriate lung volume establishes pressure-volume relationships that are conducive to optimal respiratory patterns.

The relaxed-throat breathing technique is mastered first in the comfortable, quiet clinic setting during an asymptomatic period. Always emphasize allowing the body to work the way it wants to, permitting expansion of the lower rib cage and abdominal area during inhalation. This focuses the patient on an adaptive response, rather than a maladaptive struggle behavior. Guide the patient in shifting in and out of relaxed-throat breathing during nonchallenging situations. Then practice in increasingly difficult situations, often progressing to the trigger challenges, which may include exposure to laryngeal irritants, increased level of physical activity, or discussion of emotional topics. Outside practice is assigned according to the progression of activities and skills that are mastered in the clinic setting. It may be worthwhile to encourage some of the exercise-induced PVFM patients, particularly teenagers, to elicit the help of coaches or physical education teachers in planning a program for improving physical conditioning. This can be done concurrently with practicing relaxed-throat breathing in increasingly demanding physical activities. This seems to help educate the coaches/physical education teachers about PVFM and allows the young athlete to make changes in a supportive environment.

Clinical Examples

Although it is critical to note again that carefully controlled research is needed to determine the clinical efficacy of any PVFM treatment, the following abbreviated case descriptions provide examples of intervention with patients who had different types of PVFM. The first case is a patient with exercise-induced PVFM who seemed to respond well to the behavioral intervention provided by the SLP. The second case is a patient with irritant-associated PVFM who responded minimally to SLP intervention.

Case 1

K., a 15-year-old girl with a 1-year history of breathing problems associated exclusively with playing soccer, was referred for evaluation and treatment of PVFM. Description of symptoms included chest tightness after brief sprinting, followed quickly by laryngeal tightness and inability to breathe, to move her hands, or to speak. All symptoms resolved with 5 minutes of rest. Despite use of a bronchodilator and a nasal steroid, dyspneic episodes occurred throughout the soccer season. Pulmonary function tests revealed an inspiratory cut-off on the flow-volume loop. Classic paradoxical vocal fold posture was visualized endoscopically after induction of the symptom through exercise. SLP assessment revealed normal respiratory and laryngeal functions. Excessive laryngeal musculoskeletal tension was noted both during the symptomatic and asymptomatic periods. Both K. and her mother denied stress as a contributory factor and were unaware of airway irritant triggers. Treatment began with patient education and muscle tension reduction techniques, and then incorporated the relaxed-throat breathing technique. K. practiced maintaining appropriate tension levels during increasing levels of physical activity. She was guided in using relaxed-throat breathing in the clinic room, while running up and down stairs, and while jogging on a nearby track. K. mastered shifting into relaxed-throat breathing as soon as she sensed any increase in laryngeal area tension. Her independent practice included using relaxed-throat breathing in increasingly demanding physical activities, in addition to initiating a swimming workout to improve physical conditioning. K.'s treatment was terminated when she experienced no PVFM symptoms while sprinting during clinical sessions and two indoor soccer games. At 1-year follow-up, no further episodes of PVFM had been experienced.

Case 2

C., a 50-year-old physician, reported a history of allergies, acute rhinosinusitis, and a 4-year history of chronic, nonproductive cough that varied in severity and frequency of occurrence. PVFM had been diagnosed recently, with triggers for this symptom being respiratory tract infections, aeroallergens, and airborne irritant exposures. Pulmonary and immunology examinations had resulted in diagnoses of allergies (grass pollen and dust mites) and minimal bronchospastic tendency, and a finding of inspiratory cut-off on the flow-volume loop. The forced vital capacity efforts induced the cough and PVFM symptoms, including inspiratory and expiratory stridor identified at the laryngeal level. The therapy process included patient education and instruction in relaxed-throat breathing. The technique alleviated the intensity and duration of the laryngospasms, but the chronic cough and occasional PVFM episodes persisted. C. was referred back

for further assessment of the apparent airway hyperreactivity to irritants.

Conclusions

The PVFM literature abounds with case reports of intervention provided by speech-language pathologists resulting in resolution of PVFM in children and adolescents (e.g., Alpert et al., 1991; Mullinax & Kuhn, 1996), adolescents with exercise-induced PVFM (e.g., Kayani & Shannon, 1998; Landwehr et al., 1996), and adults with or without coexisting asthma (e.g., Elshami & Tino, 1996; Tan et al., 1997). As we become more involved in working with this patient population, we will have opportunities to contribute to understanding the disorder, its etiological factors, and effective intervention. For example, we do not know why exhaling with a gentle "s" seems to play a role in controlling the PVFM symptoms. Although some experts see this as a technique that helps to relax the throat (e.g., Brugman & Newman, 1993), it could be that the "s" constriction simply moves the airway resistance location away from the larynx to the oral cavity, functioning much like "pursed-lip" breathing for the asthmatic patient. This might alleviate some of the adductor activity during exhalation and carry over into reduced adductor activity during inhalation. When the benefits of this aspect of treatment are better understood, intervention could be tailored according to patient needs.

There is also much to be learned about abnormal respiratory patterns in the symptomatic PVFM patient (Lim, 1991). Inductance plethysmography could yield useful information about how the rib cage and abdominal wall movement patterns vary across symptomatic and asymptomatic PVFM periods. Increased understanding of this aspect of PVFM would have important treatment implications. It may be that the current intervention technique of having a patient focus on lower chest wall expansion during inhalation is effective because it facilitates more normal respiratory patterns, in addition to helping reduce laryngeal area tension, as has been suggested (Martin et al., 1987). If abnormal respiratory patterns contribute to PVFM symptoms, at least in some individuals, then inductance plethysmography instrumentation might be used in providing biofeedback during PVFM intervention.

As one considers the literature review, it also is apparent that much remains to be known about other aspects of PVFM. We need to know more about upper and lower airway sensitivity, the protective function of the larynx, and whether some seemingly functional PVFM may be at least partially attributable to a reaction to irritants. If this is so, then intervention must focus not only on environmental modifications and patient desensitization, but also on reduction of airway sensitivity. We also need to understand more about respiratory behaviors during different levels of activity. This may be particularly relevant to athletes who experience exercise-induced PVFM. The connection between psychological factors and airway obstruction needs further exploration as well. Ultimately, to fully understand PVFM, we need to investigate how each of these factors (abnormal respiration, excessive tension, airway irritants, activity level, and psychological factors) triggers abnormal adduction of the vocal folds.

Prospective studies, rather than reviews of case reports, will provide the information base for making optimal intervention decisions. These studies need to be conducted with careful attention to subject selection criteria and, in many cases, with comparisons of PVFM subject behavior to that of healthy control subjects. Finally, we need to systematically determine which intervention components are essential to patient improvement and why. Given the typical history of misdiagnosis and prolonged unnecessary treatment, the PVFM patient group deserves both our research efforts and our well-informed clinical intervention.

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Appendix

SLP Protocol for Assessment of PVFM

Patient name: _____ Evaluation date: _____

DOB: _____ Age: _____

Referral source: _____ Medical diagnosis: _____

Patient History

Patient PVFM Symptom History

Patient complaint

Description of symptoms

Disorder onset

Short- and long-term variability

Dyspneic episode duration and frequency

Precipitants (triggers) of symptoms

Patient response to symptoms (coping behaviors)

Medical intervention and response to that intervention

Impact of symptoms on psychosocial well-being

Parent's, spouse's, significant other's description of PVFM symptoms and impact

Pertinent Medical History

Present health (GERD or other gastrointestinal problems, allergies, asthma or other respiratory conditions, neurological status, psychological status, voice disorder, dysphagia, other chronic conditions)

Past health conditions (as above)

Disease/surgery/trauma (that may have affected laryngeal/respiratory structures and function)

Medications

Exposure to environmental irritants (e.g., smoke, chemicals, allergens)

Substance use (caffeine, alcohol, tobacco, drugs)

Social History

Family constellation/marital status

Academic level (for juveniles)

Occupation/parents' occupations

Athletic activities

Interests

Emotionally upsetting or stressful events (presently or in the recent past)

Evaluation

Laryngeal Valving Efficiency and Control/ Respiratory Support and Control

(Rationale: To demonstrate for the patient that the laryngeal and respiratory systems function normally under a number of conditions. To identify abnormalities that warrant further attention.)

Note respiratory patterns, ranges of respiratory and phonatory performance, steadiness of sound production during the following tasks:

- Physiological fundamental frequency range
- Maximally sustained phonemes /a/, /s/, /z/
- s/z ratio (see Boone & McFarlane, 2000)
- Loudness variation of sustained /a/ (normal, soft, normal, twice as loud)
- Phonatory onset/offset during sentence-length utterances that contain a variety of voiced and voiceless phonemes (e.g., "We climbed to the top of Mt. Shuksan.")
- Vocal quality during the above tasks and throughout the interview

Respiratory Driving Pressure Control

(Rationale: To demonstrate normal respiratory behaviors, or to identify abnormalities that warrant further attention.)

Determine if the patient can sustain a steady respiratory driving pressure of 5 cm H_2O for 5 seconds (Netsell & Hixon, 1978).

Laryngeal Musculoskeletal Tension

(Rationale: To determine if the patient tends to tighten excessively during speech and nonspeech. To estimate baseline levels of laryngeal area tension and tension reduction control.)

Palpate the laryngeal area to assess musculoskeletal tension during quiet breathing and phonation samples (Aronson, 1990; Roy, Ford, & Bless, 1996).

Structural/Functional Integrity of the Speech Structures

(Rationale: To determine normalcy of structure and function throughout the motor speech processes. To identify abnormalities that warrant further attention.)

Assess the size, shape, and symmetry of the structures. During nonspeech and speech tasks, determine the adequacy of speech structure function (muscular contraction strength, movement speed, range of motion, movement accuracy, motor steadiness, ability to vary muscular tension, coordination, tone) (see Duffy, 1995).