

Paradoxical Vocal Fold Movement Disorder

Laura Matrka, MD^{a,b,*}

KEYWORDS

- Vocal cord dysfunction • Paradoxical vocal fold movement disorder
- Paradoxical vocal cord movement disorder • Paradoxical vocal fold dysfunction
- Paradoxical vocal cord dysfunction • Paradoxical vocal fold motion dysfunction
- Paradoxical vocal cord motion dysfunction • Respiratory retraining therapy

KEY POINTS

- Paradoxical vocal fold movement disorder is more common than previously recognized and should be considered when dyspnea is present without pulmonary disease or out of proportion to the degree of coexistent pulmonary disease.
- Laryngeal control therapy (also called respiratory retraining therapy) with a speech language pathologist is the cornerstone of treatment of paradoxical vocal fold movement disorder.
- Flexible laryngoscopy must be performed to diagnose paradoxical vocal fold movement disorder. Laryngeal control therapy techniques should be trialed during this initial scope.
- Bilateral vocal fold paralysis, subglottic stenosis and tracheal stenosis must be ruled out, particularly when stridor is present.
- Comorbidities, such as laryngopharyngeal reflux, sinus or allergy problems, laryngeal sicca, and obstructive sleep apnea, should be identified and treated.
- Attention to controlling anxiety and stress levels is important. However, the role for counseling or psychiatric care in treating paradoxical vocal fold movement disorder may be decreasing as the contribution of medical comorbidities becomes more widely recognized.

INTRODUCTION

The first hint of paradoxical vocal fold movement disorder (PVFMD) in the medical literature came in 1842, in which a patient with “hysterical croup” was described.¹ The paradoxical movement itself was first visualized via laryngoscopy in 1869 by Mackenzie,² who

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^a Department of Otolaryngology – Head and Neck Surgery, The Ohio State University Wexner Medical Center, Eye and Ear Institute, Suite 4000, 915 Olentangy River Road, Columbus, OH 43212, USA; ^b JamesCare Voice and Swallowing Disorders Clinic, Stoneridge Medical Center, 4019 West Dublin-Granville Road, Dublin, OH 43017, USA

* Department of Otolaryngology – Head and Neck Surgery, The Ohio State University Wexner Medical Center, Eye and Ear Institute, Suite 4000, 915 Olentangy River Road, Columbus, OH 43212.

E-mail address: Laura.matrka@osumc.edu

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Abbreviations	
COPD	Chronic obstructive pulmonary disease
ILS	Irritable larynx syndrome
LCT	Laryngeal control therapy
LPR	Laryngopharyngeal reflux disease
OSA	Obstructive sleep apnea
PFT	Pulmonary function testing
PVFMD	Paradoxical vocal fold movement disorder
SLP	Speech language pathologist

visualized glottic closure in direct correlation with the patient's stridor. In current-day practice, patients with much lesser degrees of stridor and vocal fold narrowing are often evaluated, thanks in great part to the increasing recognition of this disorder by the greater medical community. However, there are clearly still gaps in recognition and understanding of PVFMD.

NATURE OF THE PROBLEM

PVFMD is a disorder in which someone with otherwise normal vocal fold motion suffers from intermittent constriction of the vocal folds during respiration, causing dyspnea and the sensation of throat tightness. The presentation often mimics asthma, although it can occur alongside asthma or other pulmonary disease. The cause of PVFMD was thought to be only psychologic for many years, with stress and anxiety as the primary triggers; the current clinical picture is evolving and may be influenced more by medical comorbidities than previously recognized.³ Husein and colleagues⁴ established in 2008 that 70% of their patients with PVFMD had a psychological profile matching, at least in part, that of a conversion disorder. However, 50% of their patients had comorbid conditions such as gastroesophageal reflux disease or asthma, and they were more likely to have these medical conditions than they were to have a psychiatric history.

Stress and anxiety are still recognized as significant triggers for many patients, but anything that irritates the vocal folds can make paradoxical movement more likely.^{3,5-7} There is a well-established link to laryngopharyngeal reflux (LPR), although evidence on whether its treatment leads to resolution of PVFMD is contradictory.⁸⁻¹⁴ Laryngeal edema (associated with reflux complaints in 90%) was found in 72% of patients diagnosed with PVFMD in a recent prospective study.³ This reflux and edema can trigger mild PVFMD in some and full-blown laryngospasm in others.¹⁰

Other factors that lead to laryngeal mucosal irritation, such as tobacco abuse, allergic laryngitis, viral illness, and untreated sleep apnea, may trigger episodes of PVFMD and make it more difficult to treat.^{5,6} Rhinosinusitis and the resulting postnasal drip can directly cause irritation of the vocal folds; however, inflammation may also result indirectly from the release of inflammatory mediators, as described in the "One Airway" theory.¹⁵ Other respiratory tract irritants such as inhaled chemicals, smoke, or gases have long been recognized as prominent triggers in PVFMD as well.^{12,16-18} Understanding of the irritable larynx syndrome (ILS), as described by Morrison and colleagues⁷ in 1999, is crucial to a full understanding of PVFMD. PVFMD may, in fact, represent a subset of ILS in many cases.^{3,5,6}

At the more severe end of this spectrum of vocal fold irritability is laryngeal sensory neuropathy, in which a generalized laryngeal hyperresponsiveness develops after an initial inflammatory insult (such as a viral illness, trauma, or surgery in the neck). Even after controlling for factors such as reflux or allergic inflammation, the patient

remains hypersensitive; chronic cough is often present as well.⁵ The overlap between PVFMD and laryngeal sensory neuropathy should be recognized, particularly when someone is not responding well to laryngeal control therapy (LCT) alone.

Distinction between PVFMD and the spectrum of vocal cord paresis, paralysis, or synkinesis is crucial. Patients with PVFMD do not have any deficit in vocal fold mobility; full abduction must be seen at some point during the examination to differentiate it from bilateral vocal fold paralysis or paresis. Failure to do so could lead to respiratory embarrassment. Another crucial responsibility of the otolaryngologist, particularly when stridor is present, is ruling out subglottic or tracheal stenosis as the cause of dyspnea.

TERMINOLOGY

The nomenclature associated with PVFMD is confusing; it is also generally unimportant. A shift away from the term “Vocal Cord Dysfunction” has occurred primarily because of confusion with pathologic abnormalities causing dysphonia rather than dyspnea. Laryngologists may receive referrals for “vocal cord dysfunction” when a patient has a lesion of the vocal fold or a vocal fold paralysis, a very different entity from “Vocal Cord Dysfunction,” in which there is typically no dysphonia and necessarily no impairment of nerve function or vocal fold mobility. (It should be noted that some authors would disagree that dysphonia is not part of the clinical picture of PVFMD.^{19,20})

There is some argument that because a small amount of adduction normally occurs with expiration, the term “paradoxical” is not appropriate when constriction is limited to expiration.^{21,22} However, it would be argued that a constriction greater than 50% on either inspiration or expiration is not normal and is paradoxical to the open airway configuration needed for comfortable respiration.

EPIDEMIOLOGY

PVFMD is likely widely underdiagnosed, particularly in rural primary care settings in which asthma not responding to typical treatments is simply assumed to be poorly-controlled. A study of 52 school-age children with suspected poorly controlled asthma found that only 15% actually met criteria for asthma, but 27% were found to have PVFMD.²³ PVFMD has been found to be present at rates of up to 40% among patients with refractory or exercise-induced dyspnea.^{19,23–25} Studies focusing specifically on elite athletes and military personnel have been carried out, making it clear that these populations are very much prone to PVFMD as well.^{26–28} Another study showed that 20% of female patients undergoing flexible laryngoscopy for any reason were found to have some degree of PVFMD.²⁹

There is also the question of whether PVFMD is actually overdiagnosed in some centers. As practitioners in a high-volume laryngology practice often referred to as the “VCD Clinic” within our medical center, we have raised this question ourselves. We now receive referrals for PVFMD evaluations on patients with significant underlying pulmonary disease, such as moderate to severe chronic obstructive pulmonary disease (COPD), but with dyspnea out of proportion to the results of their pulmonary testing. We at first questioned the utility of testing in these patients; glottic constriction on exhalation has been shown in multiple studies to be part of the clinical picture of obstructive pulmonary disease.^{25,30–33} However, we are finding that if we see clear improvement of vocal fold abduction with use of the breathing techniques, LCT can help some of these patients decrease the frequency or severity of their dyspnea and medication use. Studies are desperately needed on efficacy of therapy in this group and are underway at the author’s institution.

Undiagnosed PVFMD has been shown to lead to immense health care costs.^{19,34,35} There are multiple reports of unnecessary intubations and even tracheostomies performed in cases of undiagnosed PVFMD.^{36,37} A retrospective case-control study showed that, before their diagnosis, patients with PVFMD had higher utilization of health care than those with moderate persistent asthma.³⁵ Research is currently underway at the author's institution to study whether asthma medication use decreases after the diagnosis of PVFMD is made.

DIAGNOSIS AND CLINICAL FINDINGS: HISTORY

There are several symptoms and elements of the history that are characteristic of PVFMD (**Box 1**). These elements include a feeling of tightness in the neck or throat, more difficulty getting air in than out, inconsistent or failed response to inhalers, and symptoms that are precipitated by anxiety, strong emotion, odors, changes in humidity or temperature, and exposure to chemicals.^{3,7} Dyspnea tends to come on more quickly with PVFMD than with asthma. It also tends to resolve more quickly with rest, rather than becoming most severe after cessation of activity, as in exercise-induced bronchoconstriction.^{38,39} In elite athletes particularly, the dyspnea may be provoked only by high-intensity exercise rather than with long, lower-intensity workouts. Patients will often note "wheezing," but on detailed questioning actually describe noisy breathing more on inspiration than expiration. A choking sensation has been found to be more predictive of PVFMD than of exercise-induced bronchoconstriction.⁴⁰ It is not uncommon for patients to have breathing "tricks" that they have tried on their own before presentation, often with some success.

In patients who ultimately are diagnosed with both asthma and PVFMD, there is often a distinction between episodes of dyspnea provoked by asthma and those related to PVFMD. Keeping in mind how commonly the two exist together, it is important not to discount the possibility of PVFMD when the patient reports some episodes that respond quickly to albuterol use. These same patients, on detailed questioning, will often say that they can tell the difference between the two even before they unsuccessfully try their inhaler.

DIAGNOSIS AND CLINICAL FINDINGS: PHYSICAL EXAMINATION

Physical examination findings, apart from those found on laryngoscopy (**Box 2**), are nonspecific in PVFMD. The absence of a true end-expiratory wheeze supports the

Box 1

History associated with PVFMD

- Tightness in neck rather than chest
- More difficulty getting air in than out
- Symptoms brought on by exertion
- Events associated with stress or strong emotions
- Events triggered by strong odors, perfumes, or chemicals
- Rapid onset of dyspnea
- Noisy breathing (usually on inhalation)
- Poor or inconsistent response to inhalers
- History of negative asthma workup

Box 2**Laryngoscopic findings that support PVFMD**

- Severe vocal cord constriction during respiration that corresponds temporally to audible stridor
- Posterior glottic chink during respiration (severe vocal cord adduction with only a small opening posteriorly)
- Greater than 50% narrowing of the glottis occurring at least twice following each of these tasks: breath-holding, counting out loud to 10 on one breath, counting out loud as high as possible during one breath, sustaining an “ee” as long as possible on one breath
- Narrowing of the glottis of greater than 50% occurring during “easy breathing”
- Increased narrowing of the glottis after presentation of strong odors or exertion
- Improvement in vocal fold abduction with use of LCT techniques

Laryngoscopic findings that do not support PVFMD

- Audible stridor heard during full vocal fold abduction
- Constant narrowing on exhalation that worsens with LCT techniques (often seen in COPD patients)
- Failure to fully abduct vocal folds at any point during examination (could indicate a bilateral vocal fold paresis or paralysis)

diagnosis, as does the presence of stridor loudest at the neck in the absence of subglottic or tracheal stenosis.

DIAGNOSIS AND CLINICAL FINDINGS: DIAGNOSTIC MODALITIES

Diagnosis of PVFMD requires a flexible laryngoscopic examination, during which the movement of the vocal folds is observed during quiet breathing, after provocative techniques, and again after performance of laryngeal control techniques. This flexible laryngoscopic examination must be performed to confirm the presence of PVFMD, as well as to assess which breathing techniques are most beneficial. It is also absolutely crucial in ruling out actual paralysis, in addition to excluding other pathologic abnormality. The rate of coexistent laryngeal lesions in PVFMD patients has been shown to be as high as 33%.^{11,26} Subglottic or tracheal stenosis must also be ruled out. In patients with marked stridor or risk factors for stenosis, lidocaine is often dripped onto the vocal folds and office tracheobronchoscopy performed at the time of their initial evaluation, if imaging of the trachea has not been previously obtained.

Topical decongestant and lidocaine are applied to the nasal cavity before the examination, because it has been shown not to affect vocal fold movement and prevents some of the false positives resulting from poor scope tolerance.⁴¹ Vocal fold movement during respiration in patients without PVFMD typically shows wide abduction with inspiration and slight narrowing with exhalation. Patients with PVFMD show narrowing on inspiration and/or a more marked narrowing on exhalation.^{36,39,42–48} The latter is more often seen when the patient is not acutely symptomatic. In some cases the patient will suspend breathing between inhalation and exhalation, with a narrowed glottis. In severe cases of inspiratory narrowing, the “posterior glottic chink” described by Christopher and colleagues³⁶ in 1983 is seen. Most patients with PVFMD will demonstrate a degree of narrowing even at rest, particularly following the provocative exercises (breath-holding with strong Valsalva, counting out loud as long as possible

on a single breath).^{13,19,49} If they do not, various strong odors are presented during the laryngoscopic examination (choosing those that are most bothersome to the patient) or the patient is taken to an exertion room where they run on a treadmill as their heart rate and oxygen saturation are monitored. The flexible laryngoscopy is then repeated immediately after the patient reaches the point of feeling dyspneic (**Box 3**). It is rare to get absolutely no narrowing after provocative exercises and attempts to replicate the patient's particular triggers.⁴⁹ However, in certain high-risk groups such as young athletes or adults with absolutely no comorbid pulmonary disease, the diagnosis is still suspected even after laryngoscopy does not demonstrate narrowing.²⁷ It is one of the challenges of diagnosing this disorder that the situation or sport that induces the problem (ie, skiing or swimming) cannot always be replicated. In this setting, a trial of LCT is performed and the flexible laryngoscopy is repeated during therapy (particularly if biofeedback is used as a therapeutic technique) to see if this represents a sampling error. If the patient thinks the breathing techniques are helping him or her, a full course of therapy is continued.

There are numerous diagnostic adjuncts for evaluating PVFMD described in the literature. Besides the laryngoscopic examination described above, the most useful testing is that done by the referring pulmonologist or primary care practitioner. Normal

Box 3

Ohio State University protocol for initial laryngoscopic examination in diagnostic evaluation of PVFMD

- Topical lidocaine/Afrin mix is applied to nasal cavities before examination
- Flexible scope is passed via the nasal cavity to observe respiration at rest
- Patient directed to breathe normally so that their typical breathing pattern may be observed
- Patient directed to hold his or her breath for 5 seconds then "let it go"
- Patient directed to count 1 to 10 on one breath
- Patient directed to hold an "ee" or to count out loud as long as he or she can on one breath
- If constriction is observed during normal respiration or following any of the tasks above, the SLP will direct the patient in various therapeutic breathing techniques while the scope is still in place to discover what achieves full abduction. If the LCT techniques worked, they will be enrolled in LCT. Their examination will end here.
- If constriction is not noted and odors are a reported trigger, the patient will be instructed to breathe in strong odors from containers of scents held in front of the patient. These scents include cleaning agents, perfumes, and potpourri. If a reaction is noted, the SLP will direct the patient in various therapeutic breathing techniques while the scope is still in place to discover what achieves full abduction. If the LCT techniques worked, they will be enrolled in LCT. Their examination will end here.
- When a patient does not exhibit constriction with strong odors or during normal respiration, the scope will be removed but the evaluation will not end. The patient will be directed to participate in exertional activities until symptomatic or until heart rate is significantly elevated (treadmill, riding a stationary bike, climbing stairs, jumping jacks). The laryngoscopic examination will then be repeated immediately to observe for glottic narrowing during the presence of symptoms. The SLP will direct the patient in various therapeutic breathing techniques while the scope is still in place to discover what achieves full abduction. If the breathing techniques worked, they will be enrolled in LCT. Their examination will end here.
- If narrowing has still not been observed by this point, the patient's symptoms, history, and overall clinical picture are assessed to determine whether a trial of LCT is appropriate.

pulmonary function testing (PFT), inspiratory limb flattening on flow volume loop, or increased symptoms after methacholine challenge (without good response to bronchodilator) all support the diagnosis of PVFMD.^{19,25,28,40,44,50–53}

Pulmonary testing is typically done prior to referral for PVFMD. Pulmonary testing is important, because the presence of PVFMD does not exclude coexistent pulmonary disease and should not lead to complacency on the part of the physician working up the patient's dyspnea. Conversely, the presence of abnormal pulmonary testing also does not rule out PVFMD. It is crucial to understand that, in our experience, even patients with moderately severe underlying pulmonary disease can occasionally benefit from LCT, if constriction is present and if the laryngeal control techniques are shown to improve vocal fold abduction during the initial diagnostic laryngoscopy. Occasionally (although rarely) the breathing techniques can actually make the sensation of dyspnea worse; this seems most common in COPD patients and can be predicted when a pattern of consistent (rather than intermittent) glottic narrowing on exhalation is seen.^{25,30–33} As mentioned above, however, the use of LCT for patients with moderately severe underlying lung disease has not been well studied to this point.

MANAGEMENT GOALS

The foremost goal of management is subjective improvement in dyspnea. Associated goals include resumption of athletic or social events that were previously avoided, means of managing future exacerbations brought on by illness or stress, cessation of unnecessary asthma medication use, and avoidance of Emergency Department visits or missed work or school.

TREATMENT: LCT WITH A SPEECH LANGUAGE PATHOLOGIST

There is no standard pharmacologic management of PVFMD besides that used to control comorbid conditions. The cornerstone of treatment of PVFMD is LCT, also called respiratory retraining therapy, performed by a licensed speech language pathologist (SLP). It has been shown to be effective in up to 95% of patients.^{26,54} A lengthy description of specific therapy techniques is beyond the scope of the article; **Box 4** describes basic principles. In treating patients who have failed LCT performed elsewhere, the addition of desensitization and biofeedback has been found to be helpful if not previously done; old therapy records can be reviewed to help decide between a repeat course of therapy versus a different diagnostic path altogether.⁵⁵ It is also

Box 4

Brief instruction in LCT techniques

- Low abdominal breathing with pressurized breaths
- Breath should be pressurized through a point of constriction at the nose or lips
- Lips should be rounded and the breath noisy
- Envision “shooting a column of air out” through the lips
- A straw cut in thirds can serve as a prop to teach the proper technique initially, with the patient exhaling through it and blowing a cool, forceful stream of air toward their hand held out in front of them
- Breaths should be performed slowly so as not to hyperventilate
- Patients are instructed to practice techniques a total of 10 minutes a day to reinforce muscle memory and achieve a more abducted vocal fold position over time

important to ensure that the SLP who is treating the patient is comfortable with providing LCT. SLPs are becoming more specialized, just as physicians are, and LCT is not always part of the repertoire of, for example, a hospital-based SLP who typically works to rehabilitate stroke patients.

TREATMENT: ADDRESSING UNDERLYING PSYCHIATRIC ISSUES

The psychologic underpinnings of PVFMD cannot be overlooked. Besides the studies discussed above that link PVFMD to conversion disorder, there is also literature demonstrating a high rate of psychiatric diagnoses and history of sexual abuse in these patients.^{19,25,56,57} Although these studies may not be representative of the population at a high-volume PVFMD clinic within a medical system that widely recognizes its prevalence, they do point out how important it is to screen these patients for undiagnosed underlying psychiatric issues or possible abuse, particularly when not responding well to therapy or control of medical comorbidities. Counseling and even pharmacologic management of anxiety can be an important adjunct in these cases.

TREATMENT: ELITE ATHLETES

Elite athletes with PVFMD have different characteristics and may require a different treatment approach than the general population with PVFMD. There is a lower rate of psychiatric pathology in elite athletes, as demonstrated by the recent review by Chiang and colleagues.²⁶ They also have lower rates of coexistent reflux. They tend to have primarily exertion-induced symptoms and may require therapy sessions performed “on site” (ie, on the football field, tennis court, or track) to identify subtle provoking factors and to achieve the necessary degree of exertion needed to carry out adequate desensitization.

TREATMENT: INVESTIGATIONAL THERAPIES

Inhaled anticholinergic was shown to prevent PVFMD triggered by exertion when used before onset of activity in a very small retrospective study.⁵⁸ Continuous positive airway pressure has been shown to relieve expiratory constriction in case reports,³¹ and an investigational mask that functions as a one-way “inspiratory valve” was developed to decrease the rate of inspiratory airflow, reducing stridor and perhaps breaking the spiral of anxiety and distress that patients feel when they hear their own noisy breathing.⁵⁹ For acute or severe cases, benzodiazepines, heliox, and even laryngeal botox have been used with some benefit.^{51,60,61}

TREATMENT: COMORBID CONDITIONS

Managing reflux, allergies, sinus disease, and extreme dryness is crucial and often leads to the prescription of medications during the evaluation of PVFMD. Untreated obstructive sleep apnea (OSA) also seems to contribute to the development of PVFMD, although more research must be done in this area. Asthma often coexists with PVFMD, but PVFMD can also be misdiagnosed as asthma. In the latter situation, patients are often on unnecessary inhalers that can actually worsen the PVFMD. If coexistent asthma has been ruled out, the importance of stopping steroid and β -agonist inhalers is strongly emphasized; this eliminates the risk for fungal laryngitis, decreases dryness, and generally reduces the degree of laryngeal irritation, making the PVFMD easier to treat.

TREATMENT: SELF-MANAGEMENT STRATEGIES

Some insurance providers do not cover LCT or place limits on the amount of therapy; in these situations it is helpful to show patients some simple techniques they can try on their own (see [Box 4](#)). In all cases patients are shown the video of their laryngoscopy, which is used to point out the vocal fold narrowing and help them better understand the condition. They are also educated in general vocal hygiene and in identifying and avoiding triggers and common vocal fold irritants. The particular breathing techniques that were most helpful to them during the laryngoscopic exam are redemonstrated. Many patients seem to benefit from simply understanding the cause of their dyspneic episodes, even if they are not able to go through a full course of treatment.

TREATMENT: RECURRENCE

Patients should be warned that recurrence is frequent at times of illness or flare in allergy or reflux symptoms. To minimize this, patients are encouraged to practice the breathing techniques even when asymptomatic to develop the “muscle memory” that will keep the glottis more widely abducted during respiration. In addition to control of comorbid conditions, a “refresher session” of LCT may be helpful, along with desensitization and biofeedback if not previously done. However, if symptoms that were previously controlled do not improve with therapy, focus should then shift to look for other underlying factors such as new or worsened pulmonary disease, cardiac problems, or airway stenosis.

SUMMARY

PVFMD is typically a very treatable cause of dyspnea, so long as it is identified. However, it is also a multifactorial disease, and treatment must be tailored to the individual patient and his or her particular comorbid conditions. Failure to recognize PVFMD can lead to immense health care costs. Important challenges include (1) ensuring PVFMD is considered when a pulmonary workup is negative or does not fully explain the dyspnea, and (2) identifying SLPs who are experienced in the treatment of PVFMD. Treating comorbidities such as reflux disease, allergies, asthma, and psychiatric illness remain important, and ruling out underlying vocal fold paralysis or airway stenosis is crucial.

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