

Vocal cord dysfunction: an update

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Objectives: To review the clinical manifestations of vocal cord dysfunction (VCD) and to discuss new developments in the diagnosis and treatment.

Data Sources: PubMed searches were performed for articles published regarding presentation, pathogenesis, diagnosis, and treatment options of VCD using the keywords *vocal cord dysfunction*, *pathogenesis*, *clinical features*, *diagnosis*, and *management*.

Study Selection: Articles were selected based on their relevance to the topic of this review. The newest developments in VCD were defined by articles published in the past 8 years.

Results: The exact cause and pathogenesis remain unclear, although laryngeal hyperresponsiveness likely plays a role in a subset of patients. Certain findings on spirometry are often interpreted to suggest VCD, but recent studies have had varying results on how useful these are in the diagnosis of VCD. Diagnosis is made by direct visualization of the adduction of the vocal cords via rhinolaryngoscopy, but the method used to provoke symptoms and adduction varies. Other noninvasive tests have been evaluated as well.

Conclusion: The early recognition and treatment of VCD are imperative to prevent the misdiagnosis and mismanagement of asthma. In addition, VCD and asthma can occur together. The origin and pathogenesis of VCD need to be better defined. More studies comparing the provocation methods during laryngoscopy may be helpful in further standardizing a diagnostic test. Further research is needed to determine whether other noninvasive tests are as effective in diagnosing VCD as laryngoscopy.

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INTRODUCTION

Vocal cord dysfunction (VCD) is an upper airway obstruction caused by paradoxical adduction of the vocal cords primarily on inspiration.^{1,2} It has also been misleadingly referred to as Munchausen stridor, episodic laryngeal dyskinesia, psychosomatic stridor, and emotional laryngeal asthma. VCD has been used interchangeably with and is probably more accurately described by the term *paradoxical vocal fold motion*.

VCD is important for an allergist to recognize because it presents with a range of symptoms that can masquerade as various allergic disorders. VCD is most frequently misdiagnosed as asthma, but both can occur in the same patient.^{3,4} This came to the forefront in 1983, when Christopher et al⁵ described 5 patients with a history of uncontrollable asthma who actually had VCD.

The misdiagnosis of asthma or unrecognized VCD in the asthmatic patient has led to the mismanagement or overtreatment of asthma.¹ In 95 patients with a history of asthma, Newman et al⁶ showed that 42 had VCD alone and 53 had asthma and VCD. Twenty-eight percent with VCD alone were intubated and taking medications similar to patients with severe asthma. Thirty-four patients with VCD alone were receiving daily oral corticosteroids (average dose of 29.2 mg).¹ Today, this similar predicament continues to hold

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true, as illustrated by a recent study by Mikita and Parker.⁷ These authors compared 25 ambulatory patients with VCD with 25 age- and sex-matched control patients with moderate persistent asthma. Looking at the use of medical services for the year preceding the diagnosis of VCD or asthma, they found that the total number of pulmonary and subspecialty care visits were significantly greater among the VCD cohort compared with the asthmatic cohort. The groups were also found to have comparable use of prescriptions, frequency of hospitalizations, and urgent care visits. Thus, they concluded that ambulatory VCD patients used significantly more medical provider resources and similar pharmaceutical services compared with patients with moderate persistent asthma. This study is strengthened by the fact that an aggressive evaluation is performed on all patients with unexplained dyspnea, and therefore they were able to exclude asthma in 84% of the VCD cohort. This study maintains that unrecognized VCD leads to misdiagnosis of asthma and therefore mismanagement.

This article reviews the clinical manifestations of VCD and discusses new developments in the diagnosis and treatment. PubMed searches were performed for articles published regarding presentation, pathogenesis, diagnosis, and treatment options of VCD using the keywords *vocal cord dysfunction, pathogenesis, clinical features, diagnosis, and management*. Articles were selected based on their relevance to the topic of this review. New developments from recent publications are discussed to assess how our understanding of VCD has progressed. This article may stimulate new ideas for the diagnosis and treatment of VCD in the future.

EPIDEMIOLOGY

The prevalence of VCD is unknown. In the general population, VCD is believed to be relatively uncommon. Past studies looking at different subgroups of the population report various prevalences. An article on adult patients with difficult to control asthma reports that 10% have VCD and 30% have VCD and asthma.⁸ The methods of diagnosis were not elaborated on. Another study on severe asthmatic children found 14% to have VCD by laryngoscopy.⁴ However, Jain et al⁹ found an only 2% incidence of VCD by laryngoscopy in patients admitted for an acute asthma exacerbation. Studies evaluating patients with exertional dyspnea also report varying prevalences of VCD. One study using laryngoscopy before and after exercise found 15% to have VCD in an adult military cohort.¹⁰ Another study of schoolchildren diagnosed 26.9% as having VCD based on clinical symptoms, flow-volume loops (FVLs), and a negative exercise treadmill test result.³ Among the highly athletic population, it is suspected that VCD occurs in approximately 3% of the intercollegiate athletes with exercise-induced asthma.³

A recent study evaluated patients with exertional dyspnea. In 2004, Abu-Hasan et al¹¹ found that 9% (13/142) of children and adolescents with exercise-induced dyspnea and no response to inhaled β_2 -agonists had VCD. Nine percent appears low, but the authors could be missing a cohort of

patients who have both asthma and VCD. Also, laryngoscopy was only performed on patients with stridor or flattening of the inspiratory loop. VCD can present with different symptoms, and a change in the FVL is not always apparent. Therefore, they might be missing the diagnosis of VCD in other patients who present with different symptoms or do not have a change in their FVL. In 2003, Rundell and Spiering¹² estimated the prevalence of inspiratory stridor (IS) in elite athletes to be 5.1%. These results on exertional dyspnea corroborate past studies estimating the prevalence of VCD in this subgroup of patients. More studies are needed to accurately assess the true incidence and prevalence of VCD in the general population.

Initial reports describe the typical patient with VCD as a young, white female with a psychiatric disorder. VCD seems to be more common in females with a 2:1 to 3:1 predominance.^{3,4} A recent review of the literature found 71% of patients were adults and the remaining 29% of patients were younger than 18 years.¹³ The average age of diagnosis has been reported to be 14.5 years for children and 33 years for young adults.³ Newman et al¹⁶ reported a previous psychiatric diagnosis in 73% of patients with VCD.

A few recent reviews and studies deviate in some aspects of the characteristic patient with VCD. First, one comprehensive review failed to document a greater incidence of psychologic dysfunction or medical background in VCD than in the general population.⁴ Second, VCD is more common in adults but has been described in a few infants in the past. Omland and Brøndbo¹⁴ described a case series of 4 infants with stridor since birth (age of 2 weeks to 14 months) in 2008. Flexible laryngoscopy showed active adductory movement of the vocal cords during inspiration in these 4 infants. Diagnosing VCD in infants is challenging because they may have laryngomalacia, which is a different entity. VCD should be a diagnosis of exclusion in this cohort.

SYMPTOMS

Symptoms include a patient's description of "difficulty in breathing-in" or "fighting for breath" or dyspnea on inspiration. There are also descriptions of wheezing, throat, or chest tightness. Patients have described choking or stridor, and the stridorous sounds are loudest above the throat and less audible throughout the chest wall. Some patients (typically elite athletes) will experience IS, which is mistaken as wheezing, with exercise that spontaneously resolves on activity cessation.^{12,15,16} Dysphonia or aphonia have also been described between attacks of respiratory distress. Most patients will also experience a sense of panic and agitation. There is a history of multiple emergency department visits, even multiple intubations. Some patients describe intractable asthma, and medications do not seem to help their symptoms.¹⁷

Generally, patients with asthma will complain of wheezing on expiration, a cough that may cause nocturnal awakenings, and cough or wheezing with exercise or activity. Symptoms of asthma usually improve with albuterol use.

Like patients with asthma, patients with VCD describe wheezing and throat or chest tightness. Both VCD and severe asthma symptoms will include shortness of breath, respiratory distress, and panic. Thus, it is important to try to discern the correct diagnosis to provide the patient with the correct medical management.

PATHOPHYSIOLOGY

The exact pathophysiology of VCD is unclear. It is not factitious because patients cannot consciously control their symptoms. There is likely no single cause. Nonorganic and organic causes have been reported in the literature, with nonorganic referring to psychological factors.⁴ Conversion disorder, major depression, obsessive-compulsive disorder, and anxiety are psychiatric disorders that have been associated with VCD.⁴ A prior history of sexual abuse may be an underlying cause as well.¹⁸ In addition, stress appears to be an important trigger for VCD in some patients. One of the most common psychological stresses reported is that associated with competitive sports.

In regard to an organic cause of VCD, noncompetitive exercise is a commonly recognized precipitant, although the exact mechanism has not been determined.¹⁹ Postoperative neurologic injury may also be a cause.^{17,20} Otherwise, it has been hypothesized that an inflammatory or irritant insult causes laryngeal hyperresponsiveness, resulting in airway narrowing at the glottic level. This results in an altered autonomic balance, which can become persistent with repeated stimuli.^{3,21} Also, it has been suggested that persistent stimulation of laryngeal sensory afferent nerves by inhaled chemicals may result in a hyperintense laryngeal adduction reflex that may be inappropriately activated during inspiration.²² Laryngeal hyperresponsiveness has also been referred to as *irritable larynx syndrome*. Gurevich-Uvena et al²⁰ conducted a large, well-controlled study involving adult patients in the military, evaluating gastroesophageal reflux disease (GERD) or laryngopharyngeal reflux disease causing laryngeal hyperresponsiveness. Other inflammatory insults include postnasal drip secondary to allergic and nonallergic rhinitis or viral upper respiratory tract infection (URI), toxic inhalation exposure, occupational irritant inhalants, cold and dry air, and episodic croup. These inflammatory insults have been well described in children and adolescents in review articles by Hicks et al⁴ and Tilles.²³

Little information is available on the relationship between GERD and VCD. Several studies have noted GERD as a common underlying disorder in VCD patients. Powell et al²⁴ evaluated videolaryngoscopic features in a large series of juveniles with VCD. They commonly found anatomical laryngeal changes typically associated with GERD. In a canine model, there is evidence of vagally mediated chemoreceptor-induced laryngospasm when the larynx is exposed to acidic levels of pH 2.5 or less. There was a significant increase in the degree of subglottic stenosis in canines after long-term application of gastric contents to subglottic mucosal tissues.² In 2006, Suttithawil et al²⁵ published the first human case

report to confirm the animal data showing that an infusion of hydrochloric acid into a human esophagus can provoke acute VCD that resolves when the pH is raised by isotonic sodium chloride infusion. On the basis of the current information and in our opinion, screening patients with VCD for GERD should be considered. Questions regarding any classic symptoms of GERD, such as heartburn, acid regurgitation, or dysphagia, should be asked. Less common symptoms of GERD may include chest pain, water brash, odynophagia, and nausea. If the clinical symptoms are consistent with GERD, then an empiric trial of a proton pump inhibitor (PPI) can be instituted. If the empiric trial of a PPI fails or the clinical symptoms are not clearly GERD, then diagnostic tests such as an endoscopy and ambulatory pH monitoring can be performed to confirm the diagnosis.²⁶ Currently, there are no definitive studies confirming that treatment of GERD improves VCD.

URI as a trigger is not well defined. It is thought that postnasal drip may irritate the upper airways and cause VCD. A recent report provides new evidence that viral URI can trigger VCD.²⁷ The authors report 2 cases of viral infections causing transient VCD and 1 case of a viral infection leading to a severe exacerbation of preexisting VCD. They used validated polymerase chain reaction techniques to detect the presence of picornavirus, influenza A, or human metapneumovirus, providing the first documented cases of postviral VCD. The identification of human metapneumovirus infection as a trigger for VCD has not been previously described. The authors suggest that viral infections cause epithelial damage, subsequent neural activation, and inflammation in the larynx and extrathoracic airways. They demonstrate that VCD can be completely reversed after recovery from an acute viral infection.

Another recent report, by de la Hoz et al,²⁸ underscores the importance of irritant exposure and psychological illness in VCD. This study describes patients who were workers in the rescue, recovery, and cleanup of the World Trade Center disaster site. They developed symptoms of dyspnea and were subsequently diagnosed as having VCD by laryngoscopy. These workers were exposed to a large amount of cement dust and considerable psychological stressors, and it was thought that their VCD was a consequence of their exposure to the disaster site.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes uncontrolled or poorly controlled asthma and anatomical defects, including laryngomalacia, subglottic stenosis, tracheal masses, and vocal cord polyps. Laryngeal edema may also cause similar symptoms. Neurologic problems, including injury to the vagus or recurrent laryngeal nerve, may mimic the symptoms. Paroxysmal laryngospasm may also cause a similar presentation of symptoms.²⁹ Patients with amyotrophic lateral sclerosis have also been described with symptoms of VCD and glottic narrowing, which may cause sudden death.³⁰

DIAGNOSIS

Differentiating asthma from VCD based on clinical symptoms can be difficult. The more common diagnostic studies currently used to evaluate respiratory symptoms that could represent asthma or VCD include rhinolaryngoscopy, spirometry, chest radiography, and arterial blood gas measurement. Differences in these studies, especially when the patient is symptomatic, can help distinguish between asthma and VCD. Rhinolaryngoscopy is normal in an asthmatic patient but shows inspiratory vocal cord adduction in a patient with VCD. Spirometry may show a scooped expiratory loop in asthma. In VCD, spirometry may show a flattened or truncated inspiratory loop and an increased ratio between the forced expiratory flow and forced inspiratory flow at 50% of vital capacity (FEF_{50}/FIF_{50}). In VCD, the results of chest radiography are normal, whereas hyperinflation and peribronchial thickening can be seen in asthma. Lastly, arterial blood gas measurement showing hypoxemia and hypercapnia is consistent with a severe asthma exacerbation. Although there have been a few reports, hypoxemia is extremely uncommon in VCD.³¹ A more detailed discussion on laryngoscopy and spirometry follows.

RHINOLARYNGOSCOPY

The criterion standard for diagnosing VCD is to directly visualize adduction of the vocal cords during inspiration while the patient is having symptoms. This is done by flexible fiberoptic rhinolaryngoscopy. Seeing complete adduction of the vocal cords on inspiration with the formation of a small, posterior, diamond-shaped glottic chink is pathognomonic but seen in only 6% of cases⁴ (Fig 1).

Laryngoscopic evidence of VCD with or without a glottic chink has been reported in up to 60% of patients during quiet respiration.³ In asymptomatic patients whose vocal cords

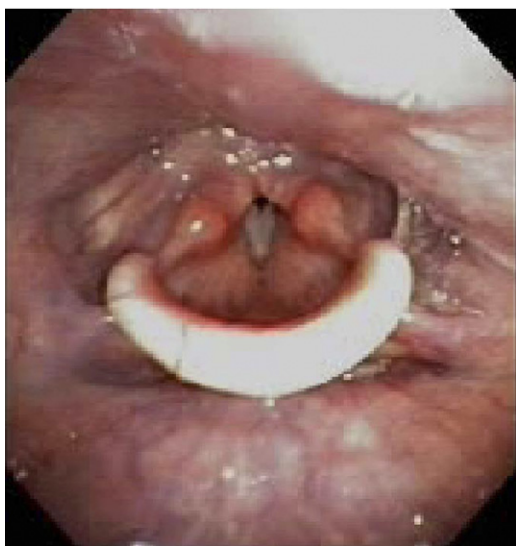


Figure 1. The pathognomonic, posterior, diamond-shaped glottis chink.

appear normal during laryngoscopy, several methods have been used to provoke symptoms. These methods include methacholine, histamine, exercise, breathing maneuvers (pant, breathe deeply, phonate, cough, sniffing), and specific irritants that have clinically induced symptoms. One case report successfully used hypnosis to provoke symptoms and demonstrated adduction of the vocal cords on inspiration during laryngoscopy.³²

A study by Perkins and Morris³³ in 2002 tried to determine whether methacholine challenge testing (MCT) provokes VCD. Four of 10 patients with documented evidence of VCD had vocal cord adduction visualized by laryngoscopy after MCT; however, 2 of these patients had vocal cord adduction at baseline. None of the healthy patients or those with exercise-induced asthma developed vocal cord adduction after MCT. Their findings suggest that methacholine may be a direct airway irritant and caused an acute episode of vocal cord adduction. Their study also suggests that MCT is not sensitive but is specific for VCD. A subsequent study by Guss and Mirza²² confirmed that VCD can be induced by inhaled methacholine and demonstrated that flexible fiberoptic laryngoscopy may be a useful adjunct to pulmonary function tests during MCT. These were small studies, and larger studies are needed to better define the incidence of MCT reactivity in VCD patients.

Another phenotype of VCD occurs only during exercise has been termed *exercise-induced VCD* (EIVCD). This is commonly seen in adolescent athletes during competitive aerobic activities.³⁴ An exercise challenge using treadmill running or bicycle ergometry monitored by spirometry and followed by rhinolaryngoscopy can be done. However, symptoms of EIVCD can appear suddenly during hard exercise and resolve within seconds or minutes of slowing down. Thus, a negative provocation test result may not rule out the diagnosis.

More recent studies have combined continuous laryngoscopy with exercise to overcome this pitfall. Heimdal et al³⁵ used an exercise treadmill, and Tervonen et al³⁶ used a bicycle ergometry test. Heimdal et al³⁵ studied 12 nonsymptomatic controls and 4 patients with documented dyspnea and noisy breathing during exercise. All individuals exercised to exhaustion on a treadmill while attached to a fully equipped ergo-spirometry unit and a fiberoptic laryngoscope linked to a video camera and sound recorder. The 4 symptomatic patients and none of the controls demonstrated exercise-induced IS and vocal cord adduction during the test.³⁵ Tervonen et al³⁶ studied 27 patients with symptoms suggestive of EIVCD. They were able to induce dyspnea in more than half of the patients, and the most frequent diagnosis was EIVCD. These studies describe a new method that may be better at evaluating patients with suspected EIVCD. However, the type of exercise that will elicit VCD may vary for each patient; therefore, standardizing a test that would be the most sensitive and specific for EIVCD may be difficult. In these academic settings, continuous laryngoscopy was used to study VCD per their protocol. At this time, for the routine

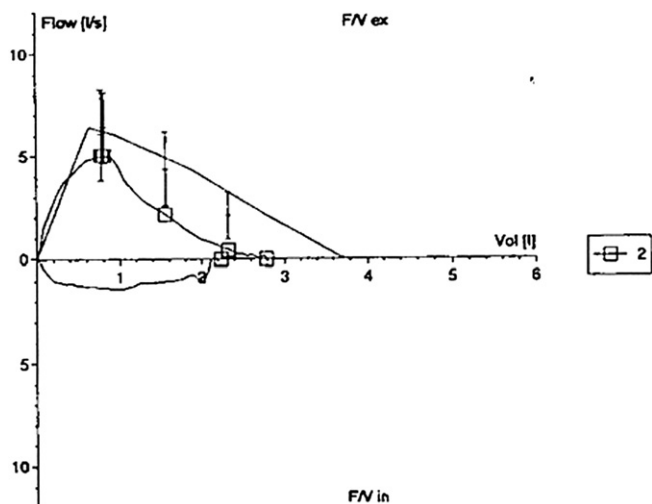


Figure 2. Flattening of the inspiratory loop.

diagnosis of VCD, continuous laryngoscopy is not available in many institutions.

SPIROMETRY

Spirometry is usually normal if the patient is asymptomatic. There are certain findings on spirometry that have been used to support the diagnosis or initiate further investigation. One finding is an FEF_{50}/FIF_{50} ratio greater than or equal to 1. Typically, this ratio is less than or equal to 1.⁴ Another finding is cutoff or flattening of the inspiratory FVL (Fig 2). The sensitivity of the FVL is low in the absence of acute symptoms. Bronchoprovocation testing with methacholine, histamine, exercise, and specific irritants have been used to induce VCD and demonstrate changes in the FVL. Morris et al¹⁰ reported that 60% of VCD-positive patients exhibited inspiratory limb truncation in the FVL after methacholine (20% had this truncation at baseline), which was significantly increased compared with controls and patients with other causes of exertional dyspnea. These patients' conditions were diagnosed by laryngoscopy after exercise. Therefore, it is not confirmed that the changes on FVL after methacholine correlate with inspiratory adduction of the vocal cords. A subsequent study by Perkins and Morris³³ was performed to further investigate changes in spirometry with VCD. They wanted to determine whether MCT not only provokes VCD but also whether spirometry and FVL demonstrate changes suggestive of VCD. As previously discussed, MCT was not sensitive but specific for provoking VCD. Contrary to their earlier study, they found that the FVL had limited utility. Two patients with evidence of VCD by laryngoscopy had normal FVLs before MCT. The 1 patient with definite FVL truncation at baseline (before MCT) had normal vocal cord findings. Five of 10 patients with documented evidence of VCD had definitive FVL changes from baseline after MCT. However, these findings correlated with laryngoscopy findings in only 2 of the 5 patients for a 60%

false-positive rate. The authors concluded that a flattening or truncation of the inspiratory FVL after MCT is not diagnostic of inspiratory vocal cord adduction. Again, larger studies are needed to determine the significance of spirometric changes after MCT in VCD.

In one study, spirometric changes were evaluated after exercise in a cohort of elite athletes with IS.¹² Three hundred seventy athletes underwent spirometry before and after an exercise challenge (actual or stimulated competition or a 7- to 8-minute free run). Athletes were monitored for IS during exercise. In addition, a postexercise decrease in forced expiratory volume in 1 second greater than 10% was used to identify athletes with exercise-induced bronchospasm (EIB). They observed IS in 19 (5.1%) and identified EIB in 111 (30%). Ten IS-positive athletes also had EIB. Spirometry data from 19 randomly selected sex-matched and EIB-matched controls were compared with the 19 IS-positive athletes. They found none of the resting pulmonary function test measurements distinguished IS-positive athletes from IS-negative controls athletes. The postexercise FEF_{50}/FIF_{50} ratio was not different between IS-positive and IS-negative athletes. However, when grouped according to the EIB status, a postexercise FEF_{50}/FIF_{50} ratio greater than 1.5 was significantly more frequent in IS-positive and EIB-positive athletes vs IS-negative and EIB-positive athletes. They did not observe truncation of the inspiratory loop in any of the athletes. Further studies to confirm VCD via rhinolaryngoscopy are needed to positively correlate their finding of an increased FEF_{50}/FIF_{50} ratio with VCD.

More recent studies looking at the utility of spirometry have had varying results. Sterner et al³⁷ performed a retrospective review of 2,662 spirometries during a 12-month period to determine how frequently evaluations are performed for abnormal inspiratory curves. Further evaluation of the upper airway was undertaken in 17% of all patients. A specific cause was found in 52%, and VCD was the most frequent diagnosis. This study was limited in that most patients with an abnormal inspiratory curve did not undergo an upper airway evaluation, which may have skewed the results. In addition, this was a retrospective review, and a prospective study on inspiratory curves would be needed to determine its clinical importance.

Watson et al³⁸ looked at whether pulmonologists could predict VCD based solely on examination of FVL. Two hundred twenty-six patients with symptoms suggestive of VCD underwent laryngoscopy. Three masked pulmonologists were able to agree on which inspiratory curves predicted VCD, but those predictions were not correct. None of the physicians correctly differentiated FVLs of patients with VCD from those without VCD. There were 37 patients (of 226) with FVLs rated normal by all 3 pulmonologists, 14 of whom were diagnosed as having VCD. Of the 18 patients with FVLs graded highly suggestive of VCD, only 5 were diagnosed as having VCD. The patients in this study presented to their specialty clinic for existing symptoms and likely had prior workup to rule out disease. Therefore, the

results of this study are subject to selection bias, which may limit its generalization to other settings.

OTHER NONINVASIVE TECHNIQUES

With the limitations and invasiveness of rhinolaryngoscopy, recent studies have looked at noninvasive techniques. Vlahakis et al²¹ reported a case of VCD where whole-body plethysmography was helpful in confirming the diagnosis. They graphed airway flow vs plethysmographic box pressure, which showed a pronounced flattening in the inspiratory portion, suggesting more significant flow limitation on inspiration compared with expiration. They note that this finding is not commonly found in cases of extrathoracic airway obstruction. The patient had laryngoscopy that confirmed the diagnosis of VCD. Larger case controlled studies would need to be performed to evaluate the reliability of this finding.

Rigau et al³⁹ used a mechanical model of the airways to determine whether oscillatory resistance measured by the forced oscillation technique is potentially useful in noninvasively estimating the area reduction in an orifice under conditions mimicking those in VCD. They concluded that the forced oscillation technique could be a useful tool for noninvasively assessing glottic closure in VCD diagnosis, obviating the need for other invasive techniques. No human studies using this technique have been reported.

A case report by Davis et al⁴⁰ describes the use of videography in the diagnosis of exercise-induced VCD. Their patient is a competitive swimmer with “difficulty breathing and wheezing” unresponsive to traditional asthma therapy, and all commonly used diagnostic evaluations failed to prove she had VCD. A videotape with audio of her swimming competitively revealed what appeared to be IS. She underwent speech therapy, and after 2 visits her symptoms resolved. Although her clinical course is highly suggestive of VCD, laryngoscopy after competitive swimming while symptomatic would confirm her diagnosis.

TREATMENT

Short-term control of symptoms include controlling asthma with appropriate asthma medications according to the National Heart, Lung, and Blood Institute guidelines (if the patient is also diagnosed as having coexisting asthma). Relaxation techniques, such as pursing lips, panting, and relaxing shoulders, should also be implemented. Continuous positive airway pressure and a heliox (helium-oxygen) mixture have also been tried in a study with 20 patients with severe symptoms and was found to be effective in approximately 4% of patients.¹³ If the patients have severe anxiety attacks, anxiolytics have been found to be helpful as well. In a study by Morris et al,¹³ intubation has also been performed in 62 patients with severe symptoms of respiratory distress and was effective in 12.4% of patients.

Long-term therapy includes speech and behavioral treatment, which would typically involve progressive muscle relaxation techniques or controlled breathing exercises. In a study performed by Doshi and Weinberger,⁴¹ 2 phenotypes of

VCD are apparent: EIVCD and spontaneously occurring VCD. Speech therapy was shown to provide relief of symptoms for spontaneously occurring VCD. An anticholinergic inhaler was shown to be effective in the prevention of EIVCD in 6 patients, suggesting that a controlled clinical trial may be warranted. Some patients have tried anticholinergic medications before exercise. Psychological counseling and psychopharmacologic management are also important to resolve anxiety issues. Hypnotic suggestion has also been described as a diagnostic and therapeutic tool to help control symptoms.⁴² In 1 patient, electromyographic feedback was found to be an effective means of gaining control over muscle tension near the vocal cords, and overall baseline tension levels were reduced by more than 60%. The patient reported improvement in school attendance and overall adaptive functioning.⁴³ Long-term treatment of comorbid conditions (eg, proton pump inhibitors for GERD and laryngopharyngeal reflux) is also important.

Obtaining a careful history and determining triggers are important in deciding what direction to take in terms of proper therapy. Speech therapy and relaxation techniques have been used in most cases, in addition to treatment of other underlying conditions, with much improvement of symptoms. Sullivan et al¹⁶ studied 20 adolescent female athletes with EIVCD. Ninety-five percent were able to control their symptoms up to 6 months after speech therapy. Nine (45%) indicated no longer needing breathing techniques during exercise. However, this was a retrospective, nonrandomized study and the diagnosis of EIVCD was based on clinical symptoms without mention of confirmation by rhinolaryngoscopy. More recent studies looking at long-term follow-up after speech therapy have not been published to our knowledge. At this point, it has been observed that with proper treatment and speech therapy, the long-term outcome of this disorder is resolution of symptoms. Without treatment, symptoms persist and patients tend to accommodate symptoms with lifestyle changes.³⁴

CONCLUSION

It is well established that the early recognition and treatment of VCD are imperative to prevent the misdiagnosis and mismanagement of asthma. Figure 3 outlines an algorithm for evaluating VCD. Numerous studies and case reports have been published to date to attempt to better define VCD. VCD was initially thought to inflict mainly young women with a psychiatric disorder. Newer information shows that VCD can affect a broader age span. Although stridor and difficulty with inspiration would be highly indicative of VCD, they may not always be present. Therefore, there should be a high index of suspicion for VCD in any patient with asthma that is difficult to control based on the National Heart, Lung, and Blood Institute guidelines. Recent studies suggest a variety of underlying causes and triggers can be involved in the pathogenesis. A detailed history eliciting GERD, a recent viral URI, allergic rhinitis, and any extreme inhaled irritant exposure

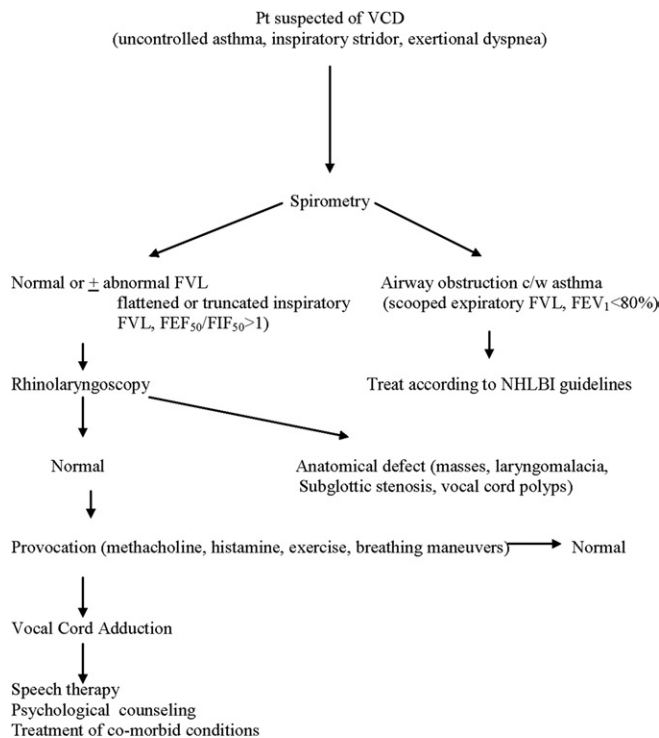


Figure 3. Algorithm of vocal cord dysfunction (VCD) evaluation. FEF₅₀ indicates forced expiratory flow at 50% of vital capacity; FEV₁, forced expiratory volume in 1 second; FIF₅₀, forced inspiratory flow at 50% of vital capacity; and NHLBI, National Heart, Lung, and Blood Institute.

should be sought. The development of a hyperresponsive larynx is likely an important factor in some patients. Confirming the diagnosis of VCD can be difficult. The studies so far indicate that changes on spirometry are not always a reliable marker of VCD. Larger studies are needed to better delineate the role of spirometric changes in the evaluation of VCD. Despite newer studies using other noninvasive techniques to diagnose VCD, there is not enough evidence to recommend its use as a routine diagnostic tool. At this time, direct visualization of the vocal cords adducting via rhinolaryngoscopy continues to be the criterion standard. The methods used to provoke symptoms vary, and further research may be helpful to further standardize this diagnostic test. There is promising evidence that continuous laryngoscopy with exercise will improve the diagnosis of EIVCD, but currently the availability is limited to study protocols. Fortunately, once the diagnosis of VCD is made, proper treatment appears to be highly successful. In conclusion, although VCD is not common, it is an important condition to recognize for anyone who cares for patients with asthma.

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