

Gasping for a Diagnosis: Pediatric Vocal Cord Dysfunction

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ABSTRACT

Vocal cord dysfunction is an obstruction of the upper airway, primarily on inspiration, due to the paradoxical adduction of the vocal cords. Vocal cord dysfunction continues to be underdiagnosed as its own entity. The lack of diagnosis can be attributed to the overlap of symptoms between asthma and exercise-induced bronchospasm. It is possible for patients diagnosed with asthma and/or exercise-induced bronchospasm to have underlying vocal cord dysfunction, which needs to be considered when prescribing asthma medications. This article will review the history of vocal cord dysfunction, the differential diagnosis, diagnostic testing, and the role of the nurse practitioner in caring for these patients. J Pediatr Health Care. (2018) ■■, ■■-■■.

KEY WORDS

Vocal cord dysfunction, paradoxical vocal cord dysfunction, paradoxical vocal cord motion, asthma, exercise induced bronchospasm

CLINICAL VIGNETTE

An 18-year-old male presented to the Pulmonary Clinic for an exercise stress test with spirometry to rule out exercise-induced bronchospasm (EIB) for complaints of occasional severe shortness of breath with exercise. When asked about the nature of his recent shortness of breath, the patient stated, "I sometimes have problems taking deep breaths in when I exercise." Past medical history included treatment for asthma and anxiety. Previously he had used various brands of short-acting beta

agonists (SABAs) and dual therapy inhaled corticosteroids/long-acting beta agonists, with little relief.

OVERVIEW

Vocal cord dysfunction (VCD), also referred to as *paradoxical vocal fold dysfunction* or *motion*, is characterized by spasms of the true vocal cords resulting in adduction of the cords and glottic obstruction during inspiration. Adduction of the cords results in symptoms that include dyspnea and noisy breathing (Morris & Christopher, 2010). The exact cause of VCD is unknown, and there is likely no single cause. Organic and nonorganic or psychological factors have been reported in the literature (Gimenez & Zafra, 2011).

History of VCD

VCD was first defined by Dunglison in 1842 (Truong & Truong, 2011). Adduction of the vocal cords upon inspiration, confirming VCD as its own entity, was first imaged by laryngoscopy in 1869 by Mackenzie (Morris, Allan, & Perkins, 2006). The true diagnosis of VCD came to the forefront when Christopher et al. (1983) described five patients with uncontrolled asthma. VCD has become a collective title for a variety of clinical presentations due to the lack of provider competence in the diagnosis of VCD. Other terms historically used to describe symptoms of VCD have included *spasmodic croup*, *Munchausen's stridor*, *functional laryngeal stridor*, *factitious asthma*, and *episodic laryngeal dyskinesia*. The term *paradoxical vocal fold dysfunction* was first introduced in the literature by Nalin J. Patel in 2004 (Truong & Truong, 2011). Although VCD is said to be uncommon, it is likely more common than the published literature suggests because of misdiagnosis (Gimenez & Zafra, 2011).

Epidemiology

Morris et al. (2006) reviewed retrospective studies showing that there is a 2:1 female predominance of VCD, with females making up 63.6% of patients and

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males 36.4%. Adults (over 18 years old) made up 71% of patients, and 29% of patients were adolescents (Morris et al., 2006). VCD has been reported in neonates due to gastroesophageal reflux disease (GERD; Morris et al., 2006). The average age of diagnosis of VCD is 14.5 years for children and 33 years for young adults (Ibrahim, Gheriani, Almohamed, & Raza, 2007). Ethnicity has not been acknowledged in the literature.

Pathophysiology

During normal respiration, the vocal cords remain open; they should close with swallowing, coughing, phonation, or breath holding (Denipah et al., 2016). VCD involves an abnormal closure or adduction of the vocal cords, resulting in airflow obstruction that can lead to functional laryngeal obstruction (Palla & Friedman, 2016).

CLINICAL PRESENTATION

History

Because VCD can present at all ages, inquiring about a patient's perinatal and neonatal history is paramount, because prenatal and postnatal factors can contribute to the development of airway diseases. The hallmark presentation of VCD is inspiratory stridor. Additionally, obstruction of inspiratory airflow can result in changes in quality of voice, chest tightness, shortness of breath, and cough (Landwehr, Wood, Blager, & Milgrom, 1996). Many patients report symptoms of dyspnea as "difficult to breathe in" or "fighting to catch my breath." Patients have also described a choking sensation (being unable to clear the throat), with no history of foreign body inhalation or clinical signs of chemical or allergic reaction (Denipah et al., 2016). It is important to illicit where the origin of symptoms present. Providers should ask if the patient's symptoms are confined to the throat or upper chest. Symptoms can occur at rest (even during sleep) or may be associated with exercise. VCD attacks can be self-limiting and subside spontaneously.

The hallmark presentation of VCD is inspiratory stridor.

Physical Examination

Physical examination findings of VCD include high-pitched inspiratory stridor that is heard over the upper portion of the trachea with auscultation, related to the adduction of the vocal cords during inspiratory breaths and less distinct when listening to the chest wall (Gimenez & Zafra, 2011; Truong & Truong, 2011). More commonly, the inspiratory stridor can be audible without the use of a stethoscope. There is a lack of upper airway swelling, with clear lung fields, which may result in transmitted stridor from the larynx. A defining feature of VCD is that the patient's lungs are clear to auscul-

tation. Patients with VCD have normal to near normal oxygenation (Denipah et al., 2016).

Differential Diagnosis

Symptoms of VCD can be mistaken for various upper airway diseases and disorders. It is of extreme importance to take a careful history and physical examination to prevent misdiagnosis and mismanagement in patients with VCD. The recognition of VCD as a diagnosis may also be impeded by lack of consensus surrounding typical clinical presentation and lack of diagnostic criteria. The misdiagnosis of VCD has resulted in patients requiring emergency department visits and/or hospitalization with inappropriate treatment of high-dose corticosteroids, intubation, or tracheostomy (Zelcer, Henri, Twefik, & Mazer, 2002). VCD can present independently or coexist with other pulmonary conditions. Most commonly, VCD is misdiagnosed or presents concurrently with asthma, EIB laryngomalacia (LM) and exercise-induced laryngomalacia (EIL; Hartley, Petty, Johnson, & Thibeault, 2015).

VCD Versus Asthma

VCD is most often confused with an acute asthma exacerbation. Symptomatic pediatric patients who have a central airway obstruction in early life usually have congenital pathology, whereas asthma is unlikely in early life (Benedictis et al., 2015). Patients with asthma will complain of wheezing on expiration, a cough that may cause nocturnal awakenings, and cough or wheezing with exercise or activity (Gimenez & Zafra, 2011).

Patients who have VCD and asthma may report some, but not complete, relief of symptoms with the use of SABAs (Gimenez & Zafra, 2011). Low et al. (2016) proposed that unstable asthma may induce dysfunctional breathing patterns, leading to activation of laryngeal reflexes, resulting in VCD. In patients with true VCD, asthma medications can further cause increased laryngeal irritation that may worsen symptoms. If VCD continues to be misdiagnosed and incorrectly controlled with asthma medications, lack of cessation of symptoms can prompt further inappropriate medication usage, propagating the cycle (Kramer, DeSilva, Forrest, & Matrk, 2016).

VCD Versus EIB

VCD and EIB have distinct pathophysiologic mechanisms; however, it continues to be difficult to differentiate between the two based on history alone (Rundell & Weiss, 2013). In children who have a history that is uncharacteristic of or inconsistent with EIB or who show no benefit from pretreatment with SABAs, VCD should be considered (Pandit et al., 2011). VCD and EIB are both provoked by exercise and typified by dyspnea (Landwehr et al., 1996); however, exercise may not always trigger symptoms in children

diagnosed with VCD (Tilles, Ayars, Picciano, & Altman, 2013). In a patient with EIB, the symptoms are usually more pronounced after cessation of exercise than during exercise, with maximal bronchoconstriction typically occurring 5 to 30 minutes after exercise has stopped (Rundell & Weiss, 2013). Patients who present with more severe EIB may become symptomatic before an exercise-induced catecholamine release, occurring just before the point of anaerobic threshold. During exercise, there is an increased tidal volume that provides mechanical protection for patients with decreased lung function, although a steady decrease in lung function is seen in exercise of 30 minutes or more (Rundell & Weiss, 2013).

VCD Versus Laryngomalacia and EIL

LM refers to inspiratory prolapse of supraglottic structures (Tilles et al., 2013). In EIL, there are typically anterior and medial movements of the arytenoids and aryepiglottic folds during symptoms of dyspnea and/or stridor during exercise (Tilles et al., 2013). LM is a well-recognized cause of airway obstruction and inspiratory stridor in infants, presenting shortly after birth and subsiding after 1 to 2 years (Bent et al., 1996).

EIL is also associated with inspiratory stridor, with symptoms that manifest during exercise (Abu-Hasan, Tannous, & Weinberger, 2005). Athletic individuals participating in competitive sports may generate enough inspiratory force to draw the aryepiglottic folds into the endolarynx, causing a subtotal glottic obstruction (Bent et al., 1996). EIL, as with VCD, does not respond to typical asthma therapy. Visualization of the larynx and vocal cords by laryngoscopy is needed to distinguish between VCD and EIL, because both EIB and VCD can elicit stridor and reduce the ratio of forced inspiratory flow (FIF) to forced expiratory flow (FEF) at 50% of vital capacity (FIF50:FEF50; Lee, Kim, Yu, & Hong, 2009).

Types of VCD

Morris and Christopher (2010) proposed a classification system to account for the variety of conditions that are associated with VCD. Types of VCD include exertional VCD, psychogenic VCD, irritant-associated VCD, GERD-related VCD, and rhinitis/sinusitis-triggered VCD.

Exertional VCD

Exertional VCD is commonly described as a choking sensation during exercise with normal baseline spirometry and no response to SABAs. In exertional VCD, symptoms most often improve rapidly once exercise is stopped (Rundell & Weiss, 2013). Exertional VCD can be diagnosed by laryngoscopy, post-exercise flow volume loop, and/or tidal volume loop flattening. There is a reported gender bias, with the occurrence of reported dyspnea-associated exertional VCD in female

athletes greater than male athletes (Idrees & FitzGerald, 2015).

Psychogenic VCD

Psychogenic VCD can be caused by emotional stress, depression, and/or sexual abuse (Morris & Christopher, 2010). Psychogenic VCD can have a role in both symptoms at rest and exercise-induced symptoms (Morris & Christopher, 2010). A case-control study of pediatric patients diagnosed with VCD compared with patients diagnosed with asthma by Gavin, Wamboldt, Brugman, Roesler, and Wamboldt (1998) found higher levels of anxiety and anxiety-related diagnoses in patients with VCD. VCD is also seen in patients who are extremely motivated, the high achiever-type A personalities. Newman, Mason, and Schmaling (1995) showed that 73% of patients with the sole diagnosis of VCD (not in conjunction with asthma or EIB) had a diagnosis of a major psychiatric disorder, with 37% having a diagnosis of personality disorder. A history of abuse (sexual, emotional, or physical) was reported in 38% of these patients (Newman et al., 1995). Powell et al. (2000) noted that 55% of adolescent girls with VCD had severe social stressors (the highest reported stressor was due to organized sports). Any periods of extreme stress can trigger VCD at any age, such as test taking, public speaking, life events, and many more. Adolescents experience tremendous amounts of stressors as they develop into adulthood and may be particularly vulnerable to psychogenic VCD.

Irritant-Associated VCD

Morris and Christopher (2010) found that irritant-associated VCD has been tied to intrinsic causes (laryngopharyngeal reflux, sinusitis, rhinitis, and/or GERD) and extrinsic causes (sensory and/or chemical irritants). With irritant-associated VCD, the irritant, either extrinsic or intrinsic, will trigger glottic closure as a protective response.

GERD-Related VCD

GERD has been closely linked to the development of VCD in infants. Denoyelle, Garabedian, Roger, and Tashjian (1996) examined nine infants with highly intense stridor that occurred during calm breathing, crying, or sleep. In all cases, supraglottic fiber-optic endoscopy showed vocal cord failure to abduct during inspiration, suggesting vocal cord paralysis. True paralysis of the abductor muscles was ruled out in all infants because normal glottis motions were observed during calm breathing or induction of anesthesia.

Rhinitis- and Sinusitis-Triggered VCD

Upper respiratory infection and allergic rhinitis are less specifically defined triggers that can contribute to VCD (Morris & Christopher, 2010). It is reported that nasal drainage resulting from common viral illnesses and

allergic rhinitis can cause upper airway irritation, resulting in VCD (Morris & Christopher, 2010).

DIAGNOSTICS

Proposed guidelines for the diagnosis of VCD include appropriate clinical history, flexible laryngoscope visualization of the vocal cords, and confirmatory pulmonary function test findings of an abnormal flow volume loop, in particular the inspiratory limb (Li, Singh, Windom, Gorman, & Bernstein, 2016; Morris & Christopher, 2010).

Laryngoscopy

Once VCD is suspected, the criterion standard for confirmation requires visualization of paradoxical vocal cord motion. Although visualization of VCD may be possible during laryngoscopy in the absence of symptoms, in some patients, in the absence of a trigger, normal vocal cord motion in between attacks can occur (Traister, Fajt, Landsittel, & Petrov, 2014). In these cases, patients' symptoms should be triggered so the abnormal cord motion can be visualized (Tilles, 2003). Provocation techniques include exercising on a bicycle ergometry or treadmill or noxious stimuli such as methacholine or odor irritant (Frank-Ito, Schulz, Vess, & Witsell, 2015). Visualization of the vocal cord motion can be done by performing fiber-optic videolaryngoscopy during bicycle ergometry (Tervonen et al., 2009). More commonly, laryngoscopy is performed before and after exercise (Rundell & Weiss, 2013). Visualization of paradoxical vocal cord motion by laryngoscopy does not eliminate the diagnosis of VCD, and taking a thorough patient history is of extreme importance.

Spirometry

Spirometry is most commonly used to diagnose patients with asthma and is not necessary in confirming the diagnosis of VCD. In patients with VCD, spirometry may depict a flattening of the inspiratory flow volume loop, which can be beneficial in guiding the provider to elicit a diagnosis of VCD. Spirometry can also be used to rule out asthma or laryngomalacia. In a patient with VCD, results would differ than in a patient with asthma or laryngomalacia. When assessing spirometry, it is important to assess the inspiratory flow, because airflow obstruction as a result of VCD results in difficulty on inspiration. Most classically, patients with VCD will show an increase in FEF50/FIF50 (Tilles, 2003). In a healthy patient without VCD, an FEF50/FIF50 ratio is usually less than 1; however, patients with VCD show a truncation of the inspiratory loop because of the inspiratory air flow obstruction, reducing the FIF50 and consequently causing the FEF50/FIF50 ratio greater than 1 (Dunn, Katial, & Hoyte, 2015). In a patient with an acute VCD exacerbation, spirometry can show a reduced forced vital capacity (FVC) and expiratory volume in 1 second (FEV₁) resulting in a normal FEV₁/FVC ratio

(Morris & Christopher, 2010). The accuracy of spirometry is based on patient performance and compliance and is recommended for patients older than 5 years.

Radiographic and Laboratory Studies

Radiographic imaging, such as a chest x-ray, is not a part of routine diagnostics for VCD. Ordering a chest x-ray for a patient suspected of VCD should not be a part of the patient's diagnostic workup. A chest x-ray should be ordered only to rule out other diagnoses or if another diagnosis is suspected. Radiographic imaging of the chest is typically normal in patients with VCD; however, in patients with asthma, hyperinflation or peribronchial thickening can be seen (Morris et al., 2006). There are no helpful laboratory studies.

MANAGEMENT

The management of VCD involves focus on breathing techniques and treatments, as well as identifying, eliminating, or treating the patient's VCD triggers. The goal of acute management is to improve breathing. Immediately, the patient should be instructed to breathe with pursed lips or instructed to breathe through only the nose. When a patient is having a VCD exacerbation, it is important to get the patient to relax; agitation and restlessness can cause the spasm to worsen.

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Speech Therapy

Treatment of pediatric patients diagnosed with VCD involves child and parent education and timely referral to a speech therapy specialist (Rameau, Foltz, Wagner, & Zur, 2012). During speech therapy, the child performs various breathing exercises and participates in relaxation techniques (Rameau et al., 2012). The therapy is focused on expiration rather than inspiration, and the use of diaphragmatic breathing rather than laryngeal breathing (Morris et al., 2006). Two common types of breathing exercises performed in speech therapy are paused breathing and belly breathing. In paused breathing, a patient must breathe in through the nose and stick the tongue out of the mouth, past the teeth and lower lip. The forward stretch of the tongue forces the airway to open at the vocal cords. With the tongue out, the patient is to exhale only through the mouth in paused/spaced breaths. This breathing will help decrease a vocal cord spasm. In belly breathing as the patient exhales, the patient is to place the tip of the tongue along the upper gingiva on the roof of the mouth. The patient is to make an "S" sound during

exhalation. This creates pressure in the back of the airway to keep the airway open. The patient is to exhale, allowing the hand and stomach to move inward to a resting position and make the “S” sound as the air is being pushed between the tongue and the teeth.

Patients must practice and perform various breathing exercises routinely and when they are not experiencing VCD (such as the ones discussed). When a VCD exacerbation presents, patients who have practiced these exercises can use these techniques to help mitigate the vocal cord spasm.

Mitigating Exacerbating Factors

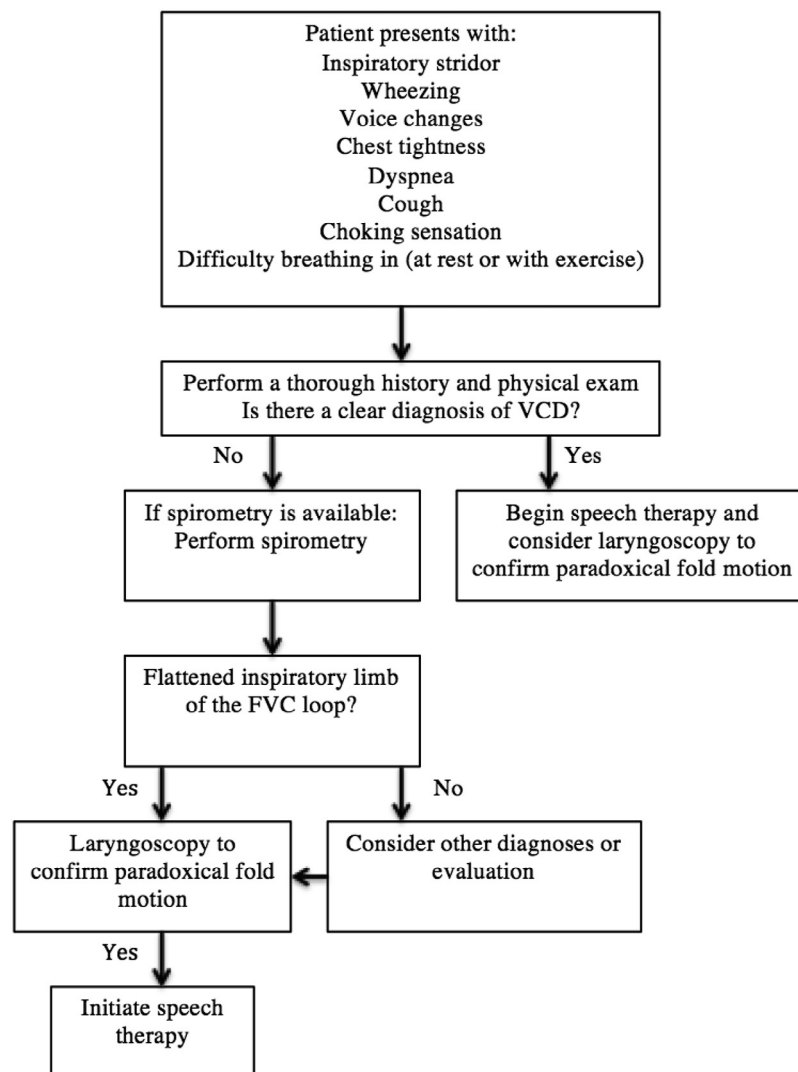
The patient with VCD and an associated condition (asthma, EIB, GERD, or extrinsic factors) needs management of the associated condition in conjunction with VCD speech therapy. Management of VCD depends on the etiology. For example, if VCD has a psycho-

logical etiology, treatment of the underlying psychological disorder is essential. It may be necessary to keep a record of what the patient is doing before and when exacerbations occur to reduce potential exacerbating triggers. The patient may need to be referred to a specialist if the exacerbating factor of VCD is poorly controlled. For example, the patient may need to be referred to an allergist if the VCD is triggered by an extrinsic allergen, and the patient with psychogenic VCD should be referred to a mental health specialist.

Botulinum Toxin

Case reports have described the use of botulinum toxin for patients with severe VCD (past tracheal intubation and/or tracheostomy) who did not respond to conventional therapy (Morris et al., 2006). The off-label use of botulinum toxin in the pediatric population (> 2 years of age) may be considered when there is

FIGURE 1. Algorithm for the nurse practitioner showing the pathway for steps in diagnosing vocal cord dysfunction in patient in the primary care setting. FVC, forced vital capacity; VCD, vocal cord dysfunction.



failure of VCD resolution after speech therapy or if the patient is unable to begin psychiatric treatment (Shogan, Rogers, Hartnick, & Kerschner, 2014). Botulinum toxin is used for VCD by injecting the botulinum toxin in the thyroarytenoid muscles, which would require a referral to an ear, nose and throat specialist (Shogan et al., 2014).

ROLE OF THE NURSE PRACTITIONER

It is important to gain an understanding of VCD to prevent exhaustive testing and inappropriate medication prescribing. The diagnosis of VCD can be easily made if the nurse practitioner asks the appropriate questions when taking the patient's history and uses appropriate testing and therapies.

Clinical Role

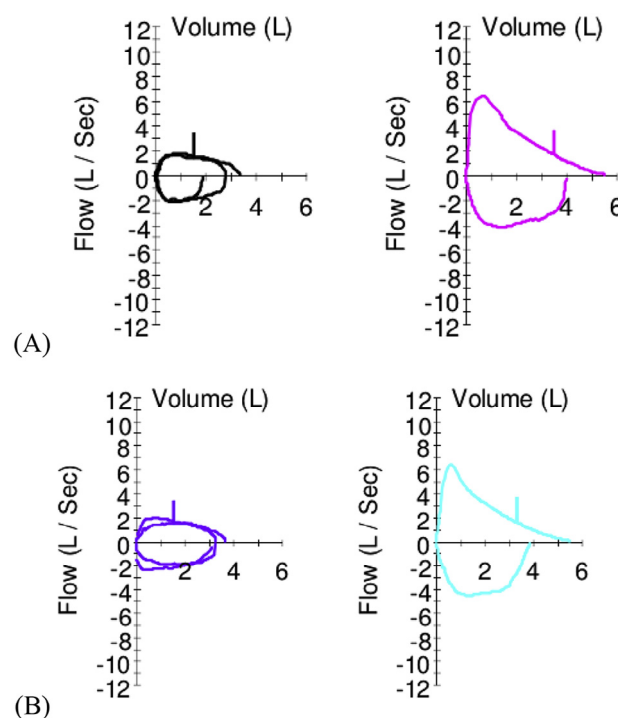
The value of a good clinical history and physical examination cannot be overstated. Nurse practitioners care for patients with a wide range of respiratory symptoms and diagnoses, including the following: pharyngeal abscess, anaphylactic laryngeal edema, croup, epiglottitis, foreign body aspiration, tracheomalacia, asthma, and EIB. The clinical role as nurse practitioners should be to include VCD in the common differential for common respiratory symptoms. It is important to ask where the patient's symptoms occur (throat/upper chest), if the airway feels restricted past a certain point, if the attacks are sudden, if there is a change in the quality of the patient's voice, if there are specific triggers causing the attacks, if the patient feels pressure in the throat with movement of the neck, or if any medications help during the exacerbation. By keeping VCD high on the nurse practitioner's differential, simple inclusion or elimination of the diagnosis can be made, and mismanagement of VCD can be avoided. In the primary care setting, if after a thorough history and examination the provider cannot elicit a diagnosis of VCD, if the patient is stable and spirometry is available, spirometry should be performed during complaints of symptoms to rule in/out upper respiratory diagnoses. Again, spirometry is not necessary for diagnosis of VCD but rather can help guide diagnosis. The criterion standard for diagnosis would be for the patient to undergo laryngoscopy to confirm the paradoxical vocal fold motion, although as stated, a negative laryngoscopy study result does not exclude VCD. Therefore, the most important role of the nurse practitioner is taking a thorough history and physical examination. A useful algorithm for nurse practitioners is depicted in Figure 1. The clinical role of the nurse practitioner is to avoid patients undergoing unnecessary treatment with inappropriate medications, alleviate the disease burden, reduce health care costs, and promote improvement in quality of life.

Education

Providers need to know the appropriate history questions to ask to elicit the diagnosis of VCD. Educating providers on how to perform and interpret spirometry testing can be useful to rule in/out a diagnosis, because it is a noninvasive and easily accessible test to perform. Because many patients do not exhibit signs of VCD during testing, clinical history and physical examination are keys to proper management. Also, once the diagnosis is confirmed, educating the patient and parents on appropriate breathing techniques is necessary. In a society where patients gravitate

In a society where patients gravitate to medications for symptomatic relief, using speech/breathing therapy when in an acute breathing exacerbation may be hard for patients and their families to understand.

FIGURE 2. Tidal breaths and flow volume loops, before and after exercise. Tidal breaths and flow volume loops, respectively, in a patient with a diagnosis of VCD before treatment. (A) Pre-exercise spirometry was taken before an 8-minute exercise challenge test. (B) Post-exercise spirometry was taken 15 minutes into recovery after completion of the exercise protocol. All pictures show flattening of the inspiratory loops. Sec, second; VCD, vocal cord dysfunction.



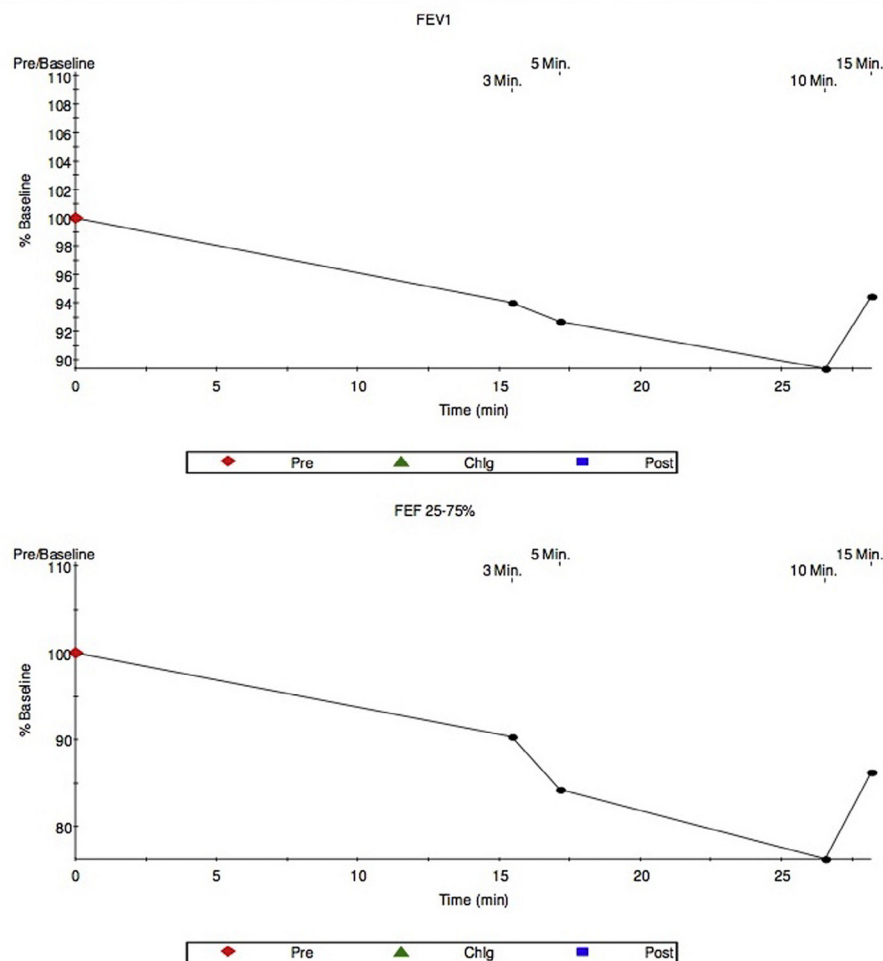
to medications for symptomatic relief, using speech/breathing therapy when in an acute breathing exacerbation may be hard for patients and their families to understand. The role of the nurse practitioner should be to educate families as to why breathing and speech therapy are so important for patients with VCD.

Research

It is not known what the frequency, duration, or severity of paradoxical vocal cord motion has to be for symptoms of VCD to occur, and this remains a diagnostic challenge (Low et al., 2016). Current research reports the need for new alternative testing for

FIGURE 3. Spirometry during EIB exercise challenge test, showing the five stages of spirometry during an 8-minute exercise challenge test to diagnose EIB. Spirometry is performed at baseline, 5 minutes after exercise, 10 minutes after exercise, and 15 minutes after exercise. Spirometry measures were forced vital capacity (FVC), forced expiratory volume in 1 second (FEV1), forced expiratory flow at 25% to 75% vital capacity (FEF_{25-75%}), and maximum forced expiratory flow (FEF_{MAX}). Chlg, challenge; EIB, exercise-induced bronchospasm; Post, after exercise; Pre, before exercise.

Stage	Pre/Baseline	3 Min.	5 Min.	10 Min.	15 Min.
---- SPIROMETRY ----					
FVC (L)	5.56	5.58	5.55	5.33	5.48
% Change	+0	+0	+0	-4	-1
FEV1 (L)	3.52	3.31	3.26	3.14	3.32
% Change	+0	-5	-7	-10	-5
FEF 25-75% (L/sec)	2.16	1.95	1.82	1.65	1.87
% Change	+0	-9	-15	-23	-13
FEF Max (L/sec)	6.43	6.80	6.24	5.34	6.39
% Change	+0	+5	-2	-17	+0



diagnosing the pediatric patient because of poor test compliance at young ages.

CONCLUSION

VCD remains highly underdiagnosed and is commonly misdiagnosed as asthma or EIB. By asking pertinent questions, performing a thorough history, and using appropriate diagnostics, nurse practitioners can elicit the diagnosis of VCD and reduce the number of patients with improper management. Patients who do not respond to traditional asthma treatment strategies should be further evaluated for VCD. This is especially true for patients who present with inspiratory stridor, dyspnea, wheezing, throat or chest tightness, change in voice quality, and/or cough. VCD remains a diagnosis of exclusion, but if providers ask appropriate questions and perform appropriate therapies, it does not have to be mismanaged. Misdiagnosis or unrecognized VCD has led to mismanagement and overtreatment, increased health care costs, and increased mortality. The clinical improvement of these patients with proper management of VCD is remarkable, and therefore VCD should be considered in all patients complaining of acute changes in breathing, those with difficult-to-treat asthma, or those with significant respiratory histories.

Denouement

This patient presented with recent shortness of breath associated with exercise, which was unresponsive to SABAs. The patient resulted in having a definitive diagnosis of mild asthma and VCD with a history of anxiety and depression. Because of the history of anxiety and depression, this patient's VCD may be psychogenic in nature. Exercise stress testing with spirometry, tidal volume loops, and oxygen kinetics were performed before exercise, during exercise, and up to 15 minutes after exercise. Baseline and post-exercise spirometry showed indication of asthma; however, his tidal volume loops were significantly flattened. [Figure 2](#) is a depiction of tidal breaths and flow volume loops before and after exercise. [Figure 3](#) shows the results of his EIB exercise challenge testing, which was not significant. Laryngoscopy before and after exercise confirmed the diagnosis.

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