

# Review article

## Vocal cord dysfunction

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Vocal cord dysfunction (VCD), a condition that frequently mimics or confounds asthma, is characterized by a paradoxical adduction of the vocal cords on inspiration. The apposition of the vocal cords produces airflow obstruction sufficient to cause wheezing, chest tightness, shortness of breath, and cough. The clinical presentation is often dramatic, and its misdiagnosis as asthma has led to inappropriate treatment, most notably with high-dose corticosteroids.<sup>1</sup> Mistaken for anaphylaxis or angioedema, VCD has led to intubation and tracheostomy.<sup>1-3</sup> Because of improved treatment of asthma, VCD is now seen almost routinely among patients referred for consultation. Its prevalence is unknown, but it may be present in as many as 40% of patients seeking evaluation of asthma that fails to respond to aggressive therapy.<sup>1</sup>

### CLINICAL EXPERIENCE

Newman et al.<sup>4</sup> recently reviewed clinical experience at the National Jewish Center for Immunology and Respiratory Medicine from 1984 through 1991. Ninety-five patients met the criteria for the diagnosis of VCD. Among them, 42 had VCD alone, and 53 had VCD associated with asthma. In the course of their disease, 28% of the patients with VCD had been intubated.<sup>4</sup> Patients with uncomplicated VCD had been misdiagnosed as having asthma for an average of 4.8 years. Their prescribed medications were similar to those of 42 matched control patients with severe asthma. Thirty-four of the 42 patients with only VCD were receiving daily oral corticosteroids at an average

#### Abbreviation used

VCD: Vocal cord dysfunction

dose equalling 29.2 mg of prednisone. Moreover, in the year preceding their evaluation, the patients with uncomplicated VCD averaged 9.7 emergency department visits and 5.9 hospital admissions. The figures for patients with combined VCD and asthma were 5.5 and 6.7. Thus it is most important for physicians to be knowledgeable about VCD and to consider this diagnosis in patients who are treated aggressively for asthma but continue to experience bouts of dyspnea. These patients may have VCD, either alone or complicating asthma.

Common complaints of patients with VCD are throat tightness, voice change, and difficulty on inspiration. Attacks occur predominantly during the day; they most often begin and resolve abruptly. Patients with VCD can usually hold their breath despite dyspnea, wheezing, and an inability to speak. Their wheezing does not worsen with cough or panting, and their symptoms may be temporarily relieved when their attention is diverted.<sup>3</sup> History of cyanosis, hypoxia, or respiratory arrest is inconsistent with uncomplicated VCD.<sup>3</sup> The distress manifested by patients with VCD ranges from profound agitation to la belle indifférence. During acute episodes inspiratory wheezing may be heard. It is loudest over the larynx but may be transmitted across the chest.<sup>3</sup> The administration of  $\beta$ -agonists not only fails to relieve symptoms but may actually aggravate them.

Vocal cords are unique in that they fall under both autonomic and voluntary control, and a functional component of VCD has been proposed.<sup>3,5</sup> Christopher et al.<sup>6</sup> suggested that this condition may be a form of conversion disorder. It is not factitious because patients do not consciously control the process and cannot volitionally reproduce the characteristic vocal cord motion.<sup>2,6,7</sup> The majority of patients are single women between 20 and 40 years old who have had difficulty emancipating from their families.<sup>7</sup> Psychiatric conditions fre-

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quently encountered in adult patients include depression, obsessive-compulsive personality, borderline personality, passive-dependent personality, adjustment reaction, and somatization disorder.<sup>7</sup> Recently, VCD has been studied in large groups of adolescents, predominantly female athletes.<sup>8,9</sup> Among these patients, perfectionism, depression, and anxiety were common.

Diagnostic studies commonly performed on patients with asthma may suggest the presence of VCD. The chest x-ray films of patients with asthma typically show peribronchial thickening and, during periods of exacerbation, hyperinflation. The films in uncomplicated VCD are normal. Spirometry of patients with symptomatic asthma shows evidence of airflow obstruction, whereas the abnormalities most likely to be observed in patients with symptomatic VCD are blunting or truncation of the inspiratory portion of the flow-volume curve.<sup>7</sup> Because of the episodic nature of VCD, the flow rate patterns may vary, and during asymptomatic periods normal flow-volume curves are likely to be found. It is possible to replicate symptoms and spirometric findings of VCD by exercise or inhalation challenge; however, a negative challenge result does not preclude the diagnosis. Hypoxemia and widened alveolar-arterial oxygen gradients, which may occur in acute asthma, are not encountered in uncomplicated VCD.<sup>3,10</sup> Finally, in a large series in which most patients had received high doses of oral corticosteroids before laboratory evaluation, peripheral eosinophil counts were significantly lower in patients with VCD alone.<sup>4</sup>

## DIAGNOSIS

Definitive criteria are needed to differentiate between asthma and VCD and to identify those patients who have either or both. The diagnosis of VCD is best established by flexible fiberoptic rhinolaryngoscopy of a patient experiencing either spontaneous or induced symptoms.<sup>11-13</sup> The examination can be videotaped or photographed for the medical record (Fig. 1). Complications are rare, and discomfort is minimal.<sup>11-13</sup> In VCD the vocal cords adduct anteriorly from the vocal process, and the posterior glottic chink remains open (Fig. 1, A). The adduction occurs during inspiration or in both the inspiratory and expiratory phases. Adduction that occurs only during the last half of exhalation is not pathologic. In normal subjects during quiet breathing, the vocal cords are abducted throughout the respiratory cycle, although there may be a slight adduction at the end-expiratory phase.<sup>14</sup> During forced inspiratory or expiratory maneu-

vers, such as pulmonary function testing, the cords may hyperabduct briefly at the initiation of either phase.

The adduction of vocal cords with an open glottic chink in a patient experiencing dyspnea unequivocally establishes the diagnosis of VCD. An important caveat is that the apposition of the vocal cords must not result from the insertion of the laryngoscope into the upper airway. In such cases, adduction of the vocal cords usually occurs without the posterior glottic chink (Fig. 1, B). A common mistake is to interpret as VCD the closure of the vocal cords caused by the gag reflex provoked by the scope. This error can be avoided by using the correct technique. For children or adults who may be fearful of the procedure, diazepam may be given, 30 minutes to 1 hour before the examination. The more patent naris is chosen. Three milliliters of 1% ephedrine is sprayed into the nose in two bursts by using an atomizer. After 2 minutes, 3 ml of 4% aqueous lidocaine is delivered in the same manner. When the patient notes anesthesia of the hypopharynx, the scope is inserted slowly to the level of the midepiglottis to allow a clear view of the vocal cords. If the patient experiences any pain or discomfort, the scope should be withdrawn, and one more burst of lidocaine should be administered. The sequence may be repeated, and laryngoscopy should only proceed in the absence of coughing, gagging, or discomfort when the scope tip stimulates the hypopharynx. The scope should be held still for 2 minutes while the patient breathes quietly. The patient is then asked to repeat low-pitched "e," high-pitched "e," and "ha-ha-ha." The patient is then instructed to open his or her mouth widely, take a deep breath as rapidly as possible, and exhale forcefully without making a sound (Table I).

If adduction of the vocal cords, characteristic of VCD, is seen, the patient is asked to perform two maneuvers with the scope still in place. The first consists of breathing in and out through tightly pursed lips. In the second, the patient breathes through his or her nose and performs a "relaxed throat" maneuver in which the jaw is lowered, the tongue is placed behind the lower teeth, and the lips are closed. If either of these techniques alleviates the adduction, the patient is likely to benefit from speech therapy.

The adduction of the vocal cords may not be seen in the absence of symptoms, and a normal examination does not exclude the diagnosis. For this reason it is useful to provoke symptoms.

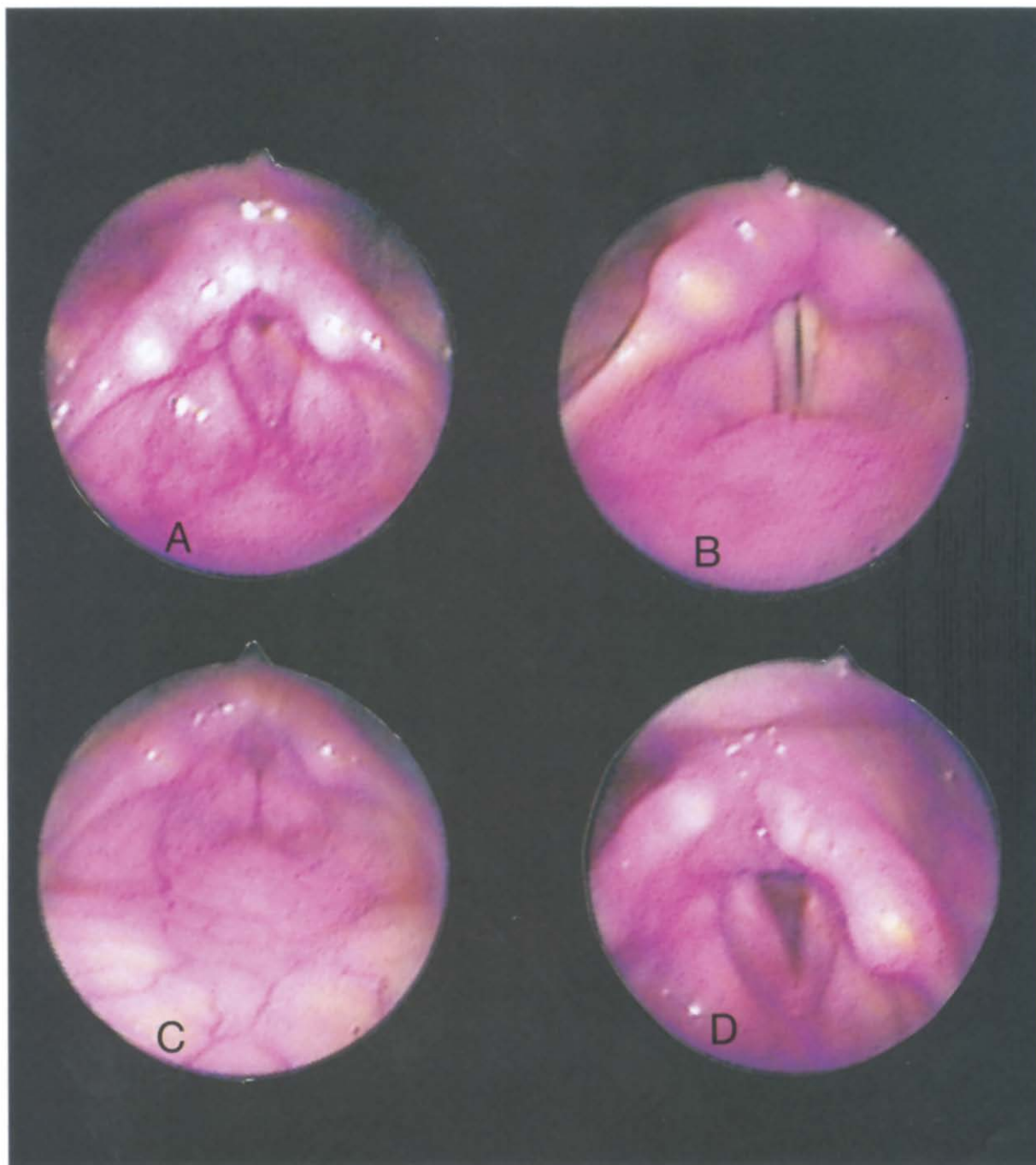


**FIG. 1.** **A**, Vocal cords of patient with VCD; note diamond-shaped posterior chink. **B**, Normal vocal cords in adducted position. **C**, Vocal cords during laryngospasm. Fully adducted true vocal cords are obscured by the adducted false vocal cords. **D**, Vocal cords after left recurrent laryngeal nerve block; note left vocal cord in paramedian position.

Laryngoscopy may then be performed in conjunction with pulmonary function tests. The most common challenge procedures use methacholine, histamine, or exercise. It has been shown recently that the determination of maximal midinspiratory flow

in conjunction with histamine challenge can be used to reflect extrathoracic airway responsiveness.<sup>15</sup> This technique was used in a study of patients with normal pulmonary function test results who were first seen with a 2-month history of

**TABLE 1.** Procedure for fiberoptic rhinolaryngoscopy

1. Select more patient nares.
2. Administer 3 ml of 1% ephedrine in 2 bursts; wait 2 minutes.
3. Administer 3 ml of 4% aqueous lidocaine in 2 bursts; wait 2 minutes; repeat if adequate anesthesia has not resulted.
4. Pass scope to the level of mid-epiglottis; hold in place for 2 minutes.
5. Observe cords during: quiet breathing; repetition of low-pitched "e," high-pitched "e," and "ha-ha-ha"; "relaxed throat" breathing; and "pursed-lip" breathing.

wheezing, attacks of dyspnea, and/or nonproductive cough. Extrathoracic airway hyperresponsiveness was documented in 26.5% of patients, bronchial hyperresponsiveness in 11.1%, both in 40.6%, and neither in 21.8%.<sup>16</sup>

VCD is commonly encountered during exertion and must be differentiated from exercise-induced bronchospasm.<sup>8,9</sup> Because many patients who experience VCD during exercise are young and fit, standard exercise-induced bronchospasm protocols frequently fail to provoke symptoms. For this reason, at the conclusion of a negative exercise-induced bronchospasm challenge, the patients should continue to exercise until they experience symptoms or become exhausted. It is useful to attempt to duplicate the inciting activities (e.g., running, biking) or exposures (perfume, smoke, cleansers, cold air, or occupational agents).

In very rare cases of severe VCD, pulmonary function cannot be assessed because of the glottic narrowing. To confirm or exclude a diagnosis of asthma in these patients, we have produced temporary unilateral vocal cord paralysis to reduce the upper airway obstruction. The left recurrent laryngeal nerve is injected with 5 ml of a 1:1 mixture of 2% plain lidocaine and 0.75% bupivacaine. The result is several hours of vocal cord paralysis, during which pulmonary function testing and bronchial challenges can be carried out.

Although VCD is most commonly misdiagnosed as asthma or anaphylaxis, its differential diagnosis includes other conditions characterized by sudden onset of airway obstruction. Most can be differentiated from VCD by clinical history. The presence of a foreign body lodged in the trachea or the esophagus is suggested by history and can be confirmed by x-ray studies.<sup>17</sup> Traumatic injury

caused by intubation or blows to the neck or chest can be established by questioning the patient.<sup>17</sup> Attacks of hereditary angioedema may be suspected on the basis of recurrence, family history, and results of diagnostic laboratory studies.<sup>18</sup> Infections such as croup, epiglottitis, and abscesses involving surrounding structures produce fever and generalized malaise.<sup>19-21</sup> Upper respiratory tract infection or exercise may cause sudden onset of dyspnea in the presence of an underlying anatomic defect, such as subglottic stenosis or laryngomalacia.<sup>22</sup> VCD can be distinguished from all of these conditions by flexible fiberoptic rhinolaryngoscopy.

Patients with a history of neck surgery may have damage to the vagus or recurrent laryngeal nerve, resulting in a cord paresis that predisposes them to developing VCD or laryngospasm. Laryngospasm is defined as a maladaptive exaggeration of the glottic closure reflex that results from a noxious supraglottic or glottic stimulation and persists beyond the duration of the stimulus<sup>23</sup> (Fig. 1, C). It is different from laryngeal obstruction associated with anaphylaxis that is characterized by angioedema. VCD and laryngospasm may be differentiated by the configuration and movement of the vocal cords. In laryngospasm the cords are adducted throughout their entire length, whereas in VCD an open posterior glottic chink is preserved. In severe VCD the vocal cords may be adducted throughout the breathing cycle, but on phonation they move normally, and both opening and closure are rapid and symmetric. Phonation of "e" and "ha-ha-ha" by the patient while the vocal cords are being observed confirms the diagnosis of paresis or paralysis, conditions that are usually unilateral, allowing for comparison between the two cords (Fig. 1, D). In paresis the affected cord lags in movement, and paralyzed cords do not move at all.

## TREATMENT

In our experience, the most successful treatment of VCD is derived from breathing exercises used for hyperfunctional voice disorders to decrease laryngeal muscle tone.<sup>7,24</sup> These techniques can be taught by a speech therapist in one or two half-hour sessions and are readily accepted by most patients who are eager to exert control over their symptoms. In some extreme cases hypnosis, biofeedback, and psychotherapy have been used successfully.<sup>7,25</sup> An approach reserved for acute attacks is the administration of a mixture of helium and oxygen.<sup>6,7</sup> More aggressive therapies under study for patients with intractable, recurrent symp-

**TABLE II.** Criteria for the diagnosis of VCD

1. Absence of gagging or coughing during rhinolaryngoscopy
2. Adduction of the vocal cords during inspiration or during both inspiration and expiration
3. Presence of an open glottic chink during adduction

toms include injection of botulinum toxin directly into one vocal cord or sectioning of the laryngeal nerve<sup>26</sup>; however, in our experience, the injection of botulinum toxin has not been effective.

Asthma therapy for patients with pure VCD is ineffective and may be deleterious. Patients who have asthma and VCD require treatment for both. Overlooking VCD in this group of patients has led to an unwarranted escalation of the use of bronchodilators and corticosteroids. It is equally important not to dismiss too readily the diagnosis of asthma because, as medications are withdrawn, previously controlled bronchospasm may reemerge.

The failure to recognize VCD will continue to lead to iatrogenic complications until we consider this diagnosis in every case of presumed asthma that is refractory to therapy. Because the treatment for VCD is benign, its overdiagnosis becomes of concern only when it is compounded by overlooking coexisting asthma. This latest paradox involving VCD results in undertreatment of otherwise manageable asthma. It is magnified by overdiagnosis of VCD by unsatisfactory endoscopy and the failure to adhere to consistent diagnostic criteria (Table II). Successful treatment of asthma and VCD is within the scope of practice today, but the physician must be alert to distinguish among his patients those who have either condition or both.

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