Vocal Cord Dysfunction

Etiologies and Treatment

Michael J. Morris, COL, MC, USA,* Patrick F. Allan, MAJ, MC, USAF,†
and Patrick J. Perkins, MD*

Abstract: Vocal cord dysfunction, a syndrome of paradoxical inspiratory closure of the vocal cords, is becoming more frequently recognized and diagnosed recently since its initial modern description 30 years ago. Initially described as single case reports, the first case series in 1983 helped to clarify the typical patient and findings of vocal cord dysfunction. Recent investigations have elucidated specific etiologies such as gastroesophageal reflux, exercise, and irritants as causative factors in addition to the numerous associated psychologic factors. Speech therapy and psychotherapy have been used extensively with vocal cord dysfunction patients, but the optimal treatment has yet to be prospectively studied. This manuscript provides a comprehensive review of the reported causative factors and treatments for vocal cord dysfunction.

Key Words: vocal cord dysfunction, stridor, asthma, exercise, gastroesophageal reflux, irritants, psychological testing

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Vocal cord dysfunction (VCD) is a well-described condition characterized by paradoxical vocal cord adduction with inspiration. A variety of etiologies, including exercise, airway irritants, gastroesophageal reflux disease (GERD), and psychiatric or emotional causes, have been implicated.[1,2] The acute presentation of VCD can closely resemble an asthma exacerbation with cough, wheezing, chest pain, and dyspnea but is commonly characterized by the presence of inspiratory stridor. The diagnosis is frequently unrecognized, and many patients have been incorrectly treated for asthma as long as 15

years.[3] Because of its dramatic presentation, VCD has resulted in both endotracheal intubation and emergent tracheostomy. Although infrequently diagnosed prior to the early 1990s, increased awareness and recognition of VCD have led to descriptions of the various etiologies and treatment of this disorder. Evidence of the increasing importance of this disorder is highlighted in the National Heart, Lung and Blood Institute Guidelines for the Diagnosis and Management of Asthma that discuss VCD as an important differential diagnosis for asthma.[4] Previous reviews of some retrospective series and numerous case reports have provided some integrated understanding of VCD.[1,2,5–10] However, few systematic investigations have defined the prevalence of this disorder or the most advantageous course of treatment.

We completed a comprehensive review of the VCD literature to include all case reports and studies describing VCD in its various nomenclatures. A total of 116 case reports and small case series (including 4 abstracts),[3,11–125] 17 letters to the editor,[126–142] 24 retrospective studies and large case series (including 5 abstracts),[143–166] 20 reviews of VCD,[1,2,5–10,167–178] and 7 prospective studies and 8 prospective study abstracts[179–193] were reviewed. As part of this review, information on patient demographics, pulmonary function testing, etiology and treatment course was obtained. This information is listed in Table 1 and is presented throughout the text of this manuscript.

History

As some authors have discussed in their historical reviews, VCD has been described with a variety of names over the past 160 years.[132,139] VCD was first described by Dunglison in a medical textbook in 1842. He described disorders of the laryngeal muscles in hysterical females and termed it "hysteric croup." Recommended treatments included cold water thrown over the face or ammonia spirits.[194] Williams in 1845 described "hysterical aphonia" in females and Austin Flint in 1868 described this syndrome in 2 male adults and termed the condition "laryngismus stridulus."[195,196] Other textbooks by Guttman in 1880 commented that it occurred "most frequently in children, and next most frequently in the hysterical attacks of adults" and by Ingals in 1892 stated, "spasm of the larynx is much less frequent in adults than false croup in children, and is most commonly observed in nervous women."[197,198] Evidence of VCD by laryngoscopic evaluation was demonstrated in 1869 by Mackenzie who visualized the vocal cords in hysterical patients and noted paradoxical closure of the vocal

TABLE 1. Review of Current VCD Literature

	No. of Patients	% of Patients
Demographics	1161	—
Adults (18 yr and older)	603	70.8
Pediatrics (<18 yr)	176	29.2
Male	232	36.4
Female	637	63.6
Pulmonary testing		
Flow volume loop truncation	327	28.2
Normal spirometry	311	66.2
Abnormal spirometry	159	33.8
Reactive bronchoprovocation	90	72.6
Nonreactive bronchoprovocation	34	27.4
Etiology		
Asthma	380	32.7
Exercise	211	18.2
Gastroesophageal reflux	208	17.9
Irritant	76	6.5
Psychiatric/emotional	183	15.8
Upper respiratory infection	102	8.8
Treatment	499	
Sedation	31	6.2
Reassurance	23	4.6
Intubation	62	12.4
Tracheostomy	31	6.2
Heliox, CPAP	20	4.0
Speech therapy	181	36.3
Psychotherapy	278	55.7
Biofeedback	37	7.4
Hypnosis	35	7.0
Botulinum toxin	21	4.2

CPAP indicates continuous positive airway pressure.

cords with inspiration.[199] Osler further expanded on this syndrome in 1902 with his description of "spasm of the muscles may occur with violent inspiratory efforts and great distress, and may even lead to cyanosis. Extraordinary cries may be produced, either inspiratory or expiratory."[200] Little additional information was published in the medical literature until the 1970s when Patterson et al described a 33-year-old woman with a history of 15 hospital admissions for what they termed "Munchausen's stridor."[89] Several more case reports and small series were published over the next decade. In 1983, Christopher et al reported their comprehensive evaluation of 5 patients and described the syndrome as "vocal cord dysfunction."[147] Since 1983, more than 1100 additional patients have been reported in the medical literature.

Terms

VCD has been described by many terms that refer to a functional airway obstruction associated with inspiratory vocal cord adduction. VCD was initially used by Christopher et al and remains the most frequently used term to describe this syndrome.[147] Paradoxical vocal cord (or fold) motion more correctly describes this syndrome and has also been commonly used.[56] VCD was initially described by several terms to include Munchausen's stridor, pseudoasthma, nonorganic or functional upper airway obstruction, factitious asthma, spasmodic croup, and emotional laryngeal wheezing.[12,27,28,33,35,89,98] Other terms used to describe VCD include psychogenic upper airway obstruction,[17] episodic laryngeal dyskinesia,[165] episodic paroxysmal laryngospasm,[151] psychogenic or hysterical stridor,[67,74] functional laryngeal stridor,[110] exercise-induced laryngospasm,[63] functional laryngeal obstruction,[92] and irritable larynx syndrome.[156] Most of these terms have been used to describe paradoxical inspiratory vocal cord adduction in the absence of other etiologies. A recent series by Andrianopoulos et al equates paradoxical vocal cord motion, paroxysmal VCD, episodic paroxysmal laryngospasm, and irritable larynx syndrome as the same entity.[145] The use of the term "irritable larynx syndrome" by Morrison et al proposes a unifying hypothesis to VCD and laryngospasm in the presence of a sensory trigger. This definition, however, eliminates an identifiable psychiatric diagnosis as the etiology for VCD.[156]

Glottic Function

In addition to phonation, the glottis plays an active role in adjusting airflow in healthy and diseased lungs. The glottis is a highly innervated structure that can adjust its size under both voluntary and reflex control of activated striated muscles. Normal respiration causes little vocal cord movement, although there is slight abduction of the true vocal cords during both inspiration and expiration.[201] One function of the glottis is to prevent damage to the airways by limiting the inflow of noxious material through either increasing laryngeal resistance or by cough. Another potential function of the glottis is to alter respiratory flow and thereby change inspiratory and expiratory times along with lung volumes in certain disease states.[202,203]

Studies have demonstrated a relationship between glottic abnormalities and asthma. Histamine challenge testing has been shown to produce narrowing of the glottic width during both inspiration and expiration.[204] Since histamine should not affect the laryngeal musculature, the effect was thought to be reflex narrowing due to reduction of intrapulmonary airway caliber. This effect was further demonstrated to correlate with decrease in FEV_1 and be more prominent in expiration.[205] Other studies demonstrated increased inspiratory resistance during panting maneuvers in 7 of 16 asthmatic patients due to inspiratory narrowing of the glottis and extrathoracic airway.[206] Further evaluation by Collett et al showed a small change in inspiratory glottic dimension but substantial changes during expiration.[207] It is thought that expiratory glottic narrowing may benefit asthma and allow hyperinflation by reducing inspiratory muscle activity that persists during expiration. However, there is no theorized benefit to inspiratory narrowing, and some authors theorize that there may be an overrecruitment of glottic constrictor muscles.[37]

Demographics

Our review of all available literature found 1161 patients reported with VCD. There is some potential overlap with the large number of VCD patients reported from both the National Jewish Medical and Research Center and military medical cen-

ters. The majority of VCD literature consists of case reports and retrospective series. There is a reported 2:1 female predominance with 63.6% of female patients and 36.4% male patients. Seventy-one percent of reported patients were adults (n = 603). The remaining 29% of patients were below the age of 18 and predominantly in the adolescent years.[153,164] However, VCD has also been reported in neonates because of the presence of GERD.[148] The true incidence of VCD in the general population has not been defined, although it is relatively uncommon. A study of 1025 patients with dyspnea prescreened and studied 63 patients. Twenty-nine patients, or 2.8% of all dyspnea patients, were found to have VCD.[190] A retrospective study of 236 patients admitted for asthma exacerbations to an inner city hospital only diagnosed 4 patients with VCD, approximately 2% of admissions.[188] Our study of 120 active duty military patients with exertional dyspnea did find an overall higher prevalence of 12%.[181]

Differential Diagnosis

VCD may present with a variety of symptoms but most commonly includes cough, wheezing, dyspnea, and inspiratory stridor. The differential diagnosis for these symptoms may be localized to the upper airway (oropharynx, larynx) or lower airway (trachea or bronchi).[6] The various other causes of airway obstruction are listed in Table 2. Examination of the oropharynx and upper airway is required to eliminate these etiologies as the cause of dyspnea or stridor in a given patient.[128]

There are several neurologic causes of paradoxical vocal cord movement that have been outlined by Maschka et al.[154] These neurologic causes include brainstem compression (eg, Arnold-Chiari, cerebral aqueductal stenosis); upper motor neuron injury (eg, severe cerebrovascular accident, static encephalopathy); nuclear or lower motor neuron injury (eg, myasthenia gravis, amyotrophic lateral sclerosis); and movement disorders (eg, adductor laryngeal breathing dystonia, myoclonic disorders, parkinsonism-plus syndromes, drug-induced brainstem dysfunction).[154] Cunningham et al also described 3 children with a history of neonatal stridor in whom no etiology was determined but was hypothesized to be an abnormal neuromuscular pathway that matured with age.[32] VCD has been reported in association with a posterior fossa arachnoid cyst and also brainstem abnormalities manifested by cranial nerve palsies.[56,90] Patients with neurologic deficits should have central nervous system abnormalities ruled out before a diagnosis of VCD can be established.

Pathogenesis

The pathogenesis of VCD is not well understood. Both organic (eg, GERD, upper airway irritation) and nonorganic (eg, psychologic disorders or emotional stressors) causes can precipitate episodes of VCD and may have different pathophysiology. VCD is characterized by laryngeal hyperresponsiveness.[168] Bucca et al evaluated 441 patients with cough, wheeze, or dyspnea without documented asthma or bronchial obstruction. Extrathoracic airway hyperresponsiveness was determined by a 25% fall in maximal midinspiratory flow and was found in 67% of patients. Disease associations included postnasal drip and pharyngitis in 55%, laryngitis in

TABLE 2. Differential Diagnosis of VCD

Location	Cause
Oropharynx	Angioedema
	Goiter
	Ludwig's angina
	Pharyngeal abscess
	Neoplasm
	Trauma
Larynx	Anaphylactic laryngeal edema
	Bilateral vocal cord paralysis
	Cricoarytenoid arthritis
	Croup
	Epiglottitis
	Laryngospasm
	Laryngomalacia
	Myasthenia gravis
	Papillomatosis
	Spastic dysphonia
Trachea	Foreign body aspiration
	Neoplasm
	Tracheobronchitis
	Tracheal stenosis
	Tracheomalacia
	Trauma
	Subglottic stenosis
	Vascular anomalies
Endobronchial	Asthma
	Carcinoid
	COPD
	Exercise-induced asthma
	Extrinsic airway compression
	Foreign body aspiration
	Neoplasm

COPD indicates chronic obstructive pulmonary disease.

40%, and sinusitis in 30% of patients.[208] The presence of laryngeal hyperresponsiveness may be explained by a variety of the aforementioned inflammatory condition but do not likely serve as the sole reason for paradoxical vocal cord motion. For instance, Ayres and Gabbot suggested that an altered autonomic balance maintained by activity of more central brain regions (medulla, midbrain, and area 25 of the prefrontal cortex) may play a role in VCD.[168] A combination of laryngeal hyperresponsiveness from an initial inflammatory component and alerted autonomic balance may lead to an exaggeration and perseveration of the laryngeal response in lung protection.[168] Thus, either an inflammatory condition or central input from emotional stresses may lead to VCD episodes. Another hypothesis in irritant-induced VCD is that direct stimulation of sensory nerve endings in the upper or lower respiratory tract may initiate local reflexes that lead to paradoxical laryngeal closure.[169] Morrison et al used the term "neural plasticity" in describing the pathogenesis of "irritable larynx syndrome." It is suggested that repeated noxious stimulation leads to permanent alteration of the cell genome; thus, brainstem laryngeal controlling neuronal networks are

held in a perpetual hyperexcitable state.[156] VCD also overlaps with hyperventilation syndrome where tachypnea may lead to glottic narrowing due to underlying laryngeal hyperresponsiveness.[160] Underscoring this association, Bucca et al correlated hyperventilation with hypocalcemia and the development of VCD in 8 female patients during nonisocapnic hyperventilation.[187]

Organic VCD

Asthma

The association between VCD and asthma is unclear and lacks sufficient prospective evaluation. The literature is replete with numerous reports of VCD being misdiagnosed as asthma for very long periods of time.[3,76] This review found 32.7% (n = 380) of VCD patients were diagnosed with asthma prior to the correct diagnosis of VCD. In many case reports, the patient only had VCD and did not require further asthma therapy after diagnosis and treatment. It is not well defined if some patients also had underlying reactive airways disease or, more importantly, that underlying asthma is a trigger for VCD. The first large case series of 95 patients reported by Newman et al found that 56% of patients had coexistent asthma based on bronchoprovocation testing or peak flow variability.[158] O'Connell et al reported coexistent asthma in 35% of their 20 patients.[159] Our study of exertional dyspnea noted that 6 of 10 patients with VCD also had reactive methacholine challenge testing (MCT).[181] In a study evaluating VCD in an emergency room setting, Jain et al found 10 of 50 patients (20%) with laryngoscopic evidence of VCD. Sixty percent of these patients had coexistent asthma based on portable spirometry measurements.[189]

The reliability of spirometry and bronchoprovocation testing in VCD is questionable. A reduction in inspiratory flow due to vocal cord adduction may lead to misinterpretation of spirometry as evidence of airways obstruction. We previously examined 10 VCD patients before and after MCT. Six patients had a reactive MCT; 3 of these patients had evidence of bronchial hyperresponsiveness without evidence of vocal cord adduction. Two patients with normal vocal cord movement at baseline had a reactive MCT and evidence of vocal cord adduction post-MCT.[182] No other studies have examined VCD patients during spirometry or bronchoprovocation testing to confirm the presence or absence of VCD. Inspiratory vocal cord adduction may lead to incorrect interpretation of spirometry or bronchoprovocation testing. One recent study suggested that the absence of sinus disease might help differentiate VCD from asthma. Peters et al compared 13 patients with VCD to 77 acute asthmatic patients and found that the lack of extensive sinus disease in the VCD patients differentiated these patients from asthmatics.[163]

Exercise

Exercise is a commonly recognized precipitant of VCD and is reported as the underlying etiology in 18.2% of patients (n = 211). It was first recognized as a cause of VCD in 1984 in a 32-year-old woman with wheezing during exercise that was treated for 10 years as exercise-induced asthma. MCT was negative and postexercise flow volume loops (FVLs) showed characteristic flattening of the inspiratory limb.[60] Several more case reports over the next decade described a similar clinical presentation.[16,40,58,63,102,125] In 1996, McFadden and Zawadski reported a series of 7 elite athletes with a "choking" sensation during exercise, normal baseline pulmonary function testing (PFT), and negative bronchoprovocation testing. The diagnosis of VCD was made on the basis of laryngoscopy and postexercise FVL.[155] A similar study by Landwehr et al the same year diagnosed 7 adolescent athletes with VCD on the basis of postexercise FVL inspiratory limb flattening.[152] These first case series demonstrated an important characterization of VCD related to exercise. These patients are usually either elite athletes who exercised competitively or active duty military required who exercise regularly.

Rundell and Spiering evaluated 370 athletes for evidence of VCD by the presence of inspiratory stridor and noted a prevalence of 5% in this group.[183] Sullivan et al reported the successful treatment of 20 adolescent athletes with exercise-induced VCD by a program including counseling and speech therapy.[184] We conducted the first prospective study of patients with exertional dyspnea and found 12% of active duty military subjects had VCD precipitated by exercise.[181] Similarly, 2 Army tertiary care facilities reported that 52% of their 176 VCD patients had exercise-induced VCD.[161] The use of exercise tidal FVL in conjunction with cardiopulmonary exercise testing in 26 patients found inspiratory flow limitation in 74% of these patients.[192] Diamond et al also reported 11% of patients in a suburban pulmonary practice had VCD precipitated by exercise. However, there was no further characterization of the athletic lifestyle of these patients.[149]

The etiology of VCD during exercise remains unexplained. Even though there is evidence of reduction of the glottic width in some forms of bronchospasm, it serves to reduce the work of breathing.[204,207] This reduction in glottic width is not reported to cause paradoxical movement of the vocal cords or upper airway obstruction. The glottic aperture widens during hyperpnea and remains widened even in the presence of exercise-induced bronchospasm.[209,210] There have been no investigations to determine the mechanism of VCD in exercise, although the above studies do not specifically associate VCD with exercise-induced bronchospasm.

Irritant

The pathophysiology of irritant-induced VCD is hypothesized to be an accentuation of the glottic closure reflex and that various extrinsic and intrinsic stimuli trigger closure as a protective response.[169] Other causes of chronic irritation of the larynx such as GERD, sinusitis, or postnasal drip may predispose individuals to VCD attacks. Numerous irritants have been implicated as a causative factor in the etiology of VCD. A retrospective review of VCD cases diagnosed at National Jewish Medical and Research Center by Perkner et al defined 11 patients with irritant-induced VCD.[162] These patients all had VCD symptoms within 24 hours of single exposure to smoke, dust, fumes, gas, or vapors. Agents that were found to induce symptoms included ammonia, flux

fumes, cleaning chemicals, smoke, odors, and ceiling tile dust. Andrianopoulos et al reported that VCD was triggered in 10 of 27 patients by a variety of stimuli to include foods, perfumed products, air pollutants, and chemical agents.[145] The triggering agents as defined by Morrison et al in "irritable larynx syndrome" included perfumes, foods, and airborne irritants.[156] Several reports include the use of olfactory challenge to induce VCD such as chlorine exposure in a swimmer and perfume odors in a 10-year-old boy.[19,119] Some irritants that trigger VCD symptoms may not have a direct airway effect. Selner et al noted the importance of irritant or sensory triggers and used provocation challenges in 3 patients to elicit and treat VCD symptoms. Each patient had a reactive provocation challenge (methacholine, cooked corn, and petrochemical fumes) that became negative after psychologic treatment.[104] A more thorough discussion of potential mechanisms for irritant causes of VCD is provided in several reviews.[156,167,169]

GERD

GERD is well described for its close association to asthma due to the regurgitation of gastric acid worsening underlying inflammation. It frequently occurs without symptoms of heartburn. Relatively little information is available on the relationship between GERD and VCD. Although not implicated as a causative factor in every patient, 208 patients (or 17.9% of all VCD patients) were reported to have underlying GERD in this review. In the pediatric population, especially in infants, the etiology of VCD is most closely linked to GERD. Several authors have described case reports of infants with VCD in the absence of central normal nervous system abnormalities. Denoyelle et al examined 9 infants, aged 1 to 13 months, with a history of "laryngeal dyskinesia" and were able to document vocal cord adduction.[148] Eight of the 9 infants had a history of GERD and 5 patients underwent 24-hour pH monitoring with a positive study in 4 patients. All the infants were treated for GERD and were noted to have symptomatic improvement.[148] Heatley and Swift also reported a 4-month-old infant with intermittent stridor that responded to treatment of GERD.[49]

Several retrospective studies have described GERD as the etiology of VCD. Powell et al evaluated 22 adolescent VCD patients and noted posterior laryngeal changes of arytenoid edema, interarytenoid edema, or interarytenoid pachyderma typically seen in GERD; no definitive testing such as 24-hour pH monitoring was performed.[164] Morrison et al reviewed their 39 patients diagnosed with "irritable larynx syndrome." Twenty-five of the patients were noted to have laryngospasm (but not defined as inspiratory vocal cord adduction) and 92% of patients had the diagnosis of GERD. Proton pump inhibitors were routinely used, but it was not mentioned if the patients' symptoms improved.[156] In a similar study of laryngospasm (defined as sudden upper airway obstruction associated with stridor and choking spells), a complete evaluation including 24-hour pH monitoring, barium swallow, and laryngoscopy was performed. Twelve patients were prospectively identified and found to have typical laryngeal findings associated with GERD; 10 patients also

had positive pH monitoring. Despite the laryngospasm symptoms, only one third of patients had complaints of heartburn or regurgitation.[211] Several other studies have noted GERD as a common underlying disorder in their cohort of patients.[143,145,161,182] The large number of VCD patients with GERD should prompt evaluation for this disorder as the potential etiology of their symptoms. Consideration should also be given for treatment with proton pump inhibitors.

Upper Respiratory Infection

A less well-defined entity as a trigger for VCD is upper respiratory infection (URI). It has been suggested that allergic rhinitis with postnasal drip may cause upper airway irritation in a similar manner. The nasal drainage from either allergic rhinitis or a viral illness may irritate the upper airway and cause VCD. This review of reported cases found 102 VCD patients or 8.8% who were reported to have an antecedent history of a viral URI prior to an episode of VCD. Brugman reported a total of 213 VCD patients or 19% with postnasal drip as a trigger for VCD; none of these patients was in the pediatric age group.[146] In the large series of 176 patients seen at military tertiary care facilities, concomitant rhinosinusitis was noted in 31% of the patients.[161] Andrianopoulos et al also identified URI as comorbidity in 17 of 27 patients evaluated for VCD.[145] Several early reports identified URI as the only antecedent history prior to onset of VCD symptoms.[3,22,56,99]

Postoperative Period

Six cases of VCD diagnosed in the postoperative period have been reported in the literature.[14,46,47,72,97,114] Five patients were female, and only 1 patient was reported to have a previous history of asthma or VCD. They underwent a variety of surgical procedures; only in 2 cases were procedures related to the upper airway (thyroidectomy and nasal turbinectomy). These patients developed inspiratory stridor within 30 to 60 minutes after extubation from general anesthesia, and 3 patients were reintubated due to lack of response to treatment of possible laryngospasm. Because of the use of supplemental oxygen postoperatively, hypoxia was only noted in 1 case. In every case, the diagnosis of VCD was made by laryngoscopic evidence of inspiratory vocal cord adduction with no evidence of laryngeal edema. The episode was usually self-limited and resolved without further reported episodes after discharge. One patient did have a tracheostomy placed and maintained for 6 weeks, and another patient required 3 months of speech therapy. The etiology of postoperative VCD may be related to mechanical stimulation of the airway. Psychiatric evaluation may reveal underlying stress or emotional disturbance as described in some of these cases.

Miscellaneous Conditions

Several other etiologies are reported as underlying conditions associated with VCD. Two cases of VCD in adolescent patients with cystic fibrosis have been reported. Both patients had significant long-term underlying lung disease to include spirometric evidence of reactive airways disease. No specific trigger or psychiatric cause could be iden-

tified.[101,107,127] VCD as a mimic of anaphylaxis has also been reported. A patient with a remote history of bee sting allergy developed airway symptoms during skin testing.[43] After blinded administration of a placebo caused airway symptoms, another patient with reported allergy to levothyroxine was found to have VCD.[85] There is a reported possible association with sleep. The original patient reported by Patterson with "Munchausen's stridor" awoke frequently with self-limited episodes of stridor.[89] Kryger et al[131] described 4 male patients with random awakenings in conjunction with stridor. Polysomnography and evaluation of the upper airway were normal but suggestive of VCD.[131] A more recent report from 1997 described nocturnal awakening in 4 patients as part of their overall symptom complex.[95]

Psychogenic Causes of VCD

The first case reports of VCD emphasized the underlying psychologic conditions in patients with this disorder. The initial terminology of "hysteric croup," "Munchausen's stridor," and "emotional laryngeal wheezing" suggested an underlying psychologic disorder as the etiology for inspiratory vocal cord adduction.[89,98,194] Early VCD reviews described the condition primarily occurring in young females in the medical profession with significant emotional stress or psychiatric disease. A variety of functional disorders to include depression, sexual abuse, factitious disorder, and conversion disorder have all been recognized to be associated with VCD. Newman noted that the majority of his 95 VCD patients had underlying psychiatric disorders. Nine patients had prior psychiatric hospitalizations and 73% had a DSM-III diagnosis.[158] A comparison of 12 adolescent VCD patients with controls found 8 patients had significant psychiatric diagnoses to include major depression, separation anxiety, overanxious disorder, and dysthymia.[179] Lacy and McManis[174] performed a review of 48 VCD cases from the literature from 1965 to 1994. They described 45 cases in which a psychiatric disorder was reported. The majority (52%) of patients were diagnosed with a conversion disorder, 13% had a major depression, 10% a factitious disorder or "Munchausen's," 4% with obsessive-compulsive disorder, and the remaining 4% with adjustment disorder. Underlying all these diagnoses was the presence of significant emotional stress.[174]

Conversion Disorder

The first series by Christopher et al in 1983 emphasized that their patients were unaware of their upper airway obstruction and were not purposely causing inspiratory vocal cord adduction. The patients were diagnosed with psychiatric disorders such as obsessive-compulsive disorder but overall were thought to have a conversion disorder.[147] The review of the 48 cases of VCD found in the early literature prior to 1994 noted that 52% of patients had a conversion disorder.[174] An essential feature of conversion disorder is an alteration of physical functioning that has no underlying physical disorder but is an expression of psychologic conflict or need. The authors commented that the increased female-to-male ratio, history of multiple hospitalizations, and numerous psychologic symptoms found in conversion disorder correlate with the findings in VCD.[174]

Emotional Stress

Emotional stress is frequently mentioned as a common precipitant of VCD in patients with psychiatric disorders. Numerous case reports also comment on emotional stress as the causative factor in patients who are otherwise normal.[3,23,33,35,59,67,70,80] Several studies highlight the importance of emotional stressors in VCD. Gavin et al conducted a case-control study of pediatric VCD patients compared with pediatric asthma; they found that family functioning did not vary between groups, but VCD patients tended to have significantly higher levels of anxiety and more anxiety-related diagnoses.[179] Powell et al noted that 55% of 20 adolescent females with VCD had significant social stresses such as competitive sports.[164] McFadden and Zawadski noted similar findings in describing 8 patients with exercise-induced VCD, all of whom were elite athletes involved in competitive sports.[155] It has also been reported that stressful situations such as a combat environment may trigger VCD.[31]

Depression

Depression has been a common underlying psychiatric diagnosis in many VCD patients, and major depression may be a component of a conversion disorder.[17,21,41,54,109,165] In the literature review by Lacy and McManis, major depression or dysthymia was present in 13% of VCD patients.[174] Other large studies evaluating psychiatric disorders noted similar findings with 33% to 40% of VCD patients with major depression.[165,179] In addition to psychologic counseling, the use of antidepressants may be indicated to control VCD symptoms.

Sexual Abuse

Only 3 articles discussed a prior history of sexual abuse as the underlying psychiatric cause of VCD. Freedman et al conducted a retrospective chart review of 47 cases of VCD at National Jewish Center and noted 14 patients with a reported history of sexual abuse and 5 patients with suspected childhood sexual abuse.[39] Two other case reports also noted a history of sexual abuse in association with other psychiatric disorders such as depression.[21,116] Psychotherapy was reported to be helpful, but some patients had difficulty in accepting the diagnosis of VCD.[21,39] None of the recent literature in the past 10 years has described any further cases of sexual abuse associated with VCD. The series of 95 patients by Newman et al did report a history of physical, sexual, or emotional abuse in 38% of their patient cohort.[158]

Clinical Presentation

The clinical presentation of VCD is well described in the numerous case reports and reviews of VCD. Several large reviews have tabulated the predominant symptoms in VCD that are listed in Table 3.[146,158,161] Predominant symptoms are dyspnea, wheezing, stridor, and cough. An isolated chronic cough in the absence of stridor has also been reported.[76] These symptoms are commonly mistaken for asthma, and patients may be treated for numerous years without the correct diagnosis being considered.[3] Several key points about the clinical presentation should be highlighted. First, many patients will have recurrent visits to the emergency department or hospi-

TABLE 3. Symptoms of VCD

	Brugman[146]	Newman et al[158]	Perello et al[161]
No. of patients	1140	95	176
Dyspnea (%)	95	88	76
Wheeze (%)	51	84	61
Stridor (%)	51	10	NR
Cough (%)	42	70	NR
Voice changes (%)	25	25	NR
Chest tightness (%)	15	9	76
Throat tightness (%)	15	NR	56
Choking (%)	8	8	NR

NR indicates not reported.

talizations for their VCD symptoms. Although the presentation can be quite dramatic, it is not a life-threatening illness and is usually self-limited. The failure to recognize VCD has led to intubation and tracheotomy in numerous patients. Second, these patients will be taking numerous asthma medications without significant effect on asthma control. The first case series by Christopher et al reported 5 patients with asthma refractory to treatment.[147] Third, the VCD episodes are usually abrupt in onset and resolution without the typical worsening symptoms over several hours to several days in asthmatics. Exercise-induced bronchospasm occurs after 6 to 12 minutes of high intensity exercise and lasts for 60 minutes after cessation of exercise if left untreated. Fourth, careful evaluation of patients will demonstrate high-pitched inspiratory sound that is commonly mistaken for wheezing. Physical examination will disclose the sound to be inspiratory and localizing over the trachea consistent with stridor and not peripheral airway expiratory wheezing found in asthma. This clinical presentation may be less evident in patients with concomitant asthma and VCD. Patients will have a normal examination when they are asymptomatic. Another component of the VCD symptom complex is hyperventilation. A review of 54 patients by Parker and Berg noted that 76% of VCD patients had symptoms of lightheadedness, visual changes, extremity numbness, and tingling during episodes. Forty-eight percent of patients also had a low end-tidal carbon dioxide levels less than 30 mm Hg.[160]

Diagnostic Studies

Radiographic and Laboratory Studies

There are no definitive radiologic or laboratory studies that can confirm the diagnosis of VCD. Chest radiography findings are typically normal and will not demonstrate hyperinflation or peribronchial thickening seen in acute asthma exacerbations.[94,106] It may be helpful in eliminating other potential etiologies of stridor such as foreign body aspiration, neoplasm, or tracheal abnormalities. Soft tissue films of the neck may also rule out an upper airway obstruction in certain conditions such as croup. Arterial blood gases had been advocated as potential instrument to differentiate VCD from other conditions, especially asthma, in early case series.[147] This has not been substantiated in any studies, and hypox-

emia has been reported in association with 10 cases of VCD.[12,14,24,46,82,83,117] Appleblatt and Baker documented mild hypoxemia during an acute VCD episode that resolved when the patient was asymptomatic. Three of 4 VCD patients with concomitant asthma reported by Nolan et al were also noted to have hypoxemia during attacks.[83] However, the finding of hypoxemia does not eliminate the possibility of VCD in any given patient.

Pulmonary Function Testing

There is limited utility in PFT in diagnosing VCD apart from inspiratory FVL, which will be discussed later. First, VCD is intermittent and may be asymptomatic at rest so both the PFT and FVL will be normal. Symptomatic VCD may result in reduction in both the forced expiratory volume at one second (FEV_1) and forced vital capacity (FVC) with no change in the FEV_1/FVC ratio. It has been suggested that the limitation in inspiratory flow will cause an increase in the forced expiratory to inspiratory flow at 50% of vital capacity (FEF_{50}/FIF_{50}).[131,158,173] This ratio will be normal if both expiratory and inspiratory closure is present. Second, as previously discussed, VCD can overlap with asthma in a significant percentage of patients. In the patient undergoing evaluation for asthma, the presence of normal PFT with a normal FEV_1/FVC ratio should raise the possibility of VCD as a potential diagnosis. However, reduction in the FEV_1/FVC ratio consistent with airways obstruction does not eliminate concomitant VCD. Vhalakis et al used plethysmography on a VCD patient to demonstrate inspiratory plateau in flow on the open shutter loop with an elevated airway resistance.[121]

FVLs

A reported characteristic finding in VCD is the truncation of the inspiratory limb of the FVL consistent with a variable extrathoracic obstruction.[212] In symptomatic patients, this can be extremely helpful in suggesting the diagnosis of VCD. VCD is intermittently symptomatic, and the FVL may be normal in the absence of symptoms. VCD may also be expiratory and has been demonstrated to have expiratory FVL changes in several VCD patients.[1,15,58,126] In this review, FVL truncation was reported in only 28.1% of all VCD patients. The utility of FVL in diagnosing VCD has not been evaluated in a prospective manner. When we performed our prospective study of VCD in exertional dyspnea, 20% of patients had an abnormal FVL at rest.[181] Newman et al[158] reported only a 23% incidence of abnormal FVL in asymptomatic patients with documented VCD, whereas Perkner et al reported 25% of patients with irritant-induced VCD had inspiratory FVL truncation at baseline.[162] Much higher numbers were reported in several abstracts of 56% in a community-based setting to 79% in a military setting.[149,161] However, it was not stated whether these patients were symptomatic during testing.

Laryngoscopy

Visualization of the vocal cords with flexible fiberoptic laryngoscopy during an acute VCD episode is the current

gold standard to confirm the diagnosis. Patients should be instructed to perform a variety of maneuvers, including phonation, normal breathing, panting, and repetitive deep breaths. The classic finding is inspiratory vocal cord adduction of the anterior two thirds with a posterior diamond-shaped "chink."[9] Inspiratory closure of the vocal cords is sufficient for diagnosis, and not all patients are reported to have the finding of posterior chinking. VCD is seldom reported to be only expiratory as initially described in a 41-year-old psychologically disturbed woman.[98] Newman et al noted that 11% of their VCD patients had expiratory closure, 31% with both inspiratory and expiratory closure.[158] Several other case reports have documented both inspiratory and expiratory closure in patients with VCD.[1,58] However, early expiratory closure may also be a compensatory physiologic mechanism in those patients with asthma.[9,173]

Care should be taken during examination to avoid anesthetizing the vocal cords or stimulating the posterior pharynx. Many patients with VCD have normal findings when they are asymptomatic and may only be abnormal during an acute episode. In those patients with exercise-induced VCD, provocation with exercise is required to evoke symptoms.[155,181] As previously mentioned, either methacholine or histamine may also provoke symptoms separate from airway hyperreactivity.[162,182] Olfactory challenge has also been used in those VCD patients with irritant-induced VCD to provoke symptoms.[19,119]

Methacholine

MCT is problematic in differentiating VCD from asthma. Several studies have documented the common association of VCD and asthma.[158,159,178] There is ample evidence that methacholine itself may have an effect on the vocal cords. Selner et al in 1987 described a patient who underwent 3 MCT.[104] During the first MCT, stridor resulted at 5 mg/mL without change in peak flow and during the second MCT; the patient developed acute VCD documented by laryngoscopy during the normal saline control. A third MCT after psychologic intervention resulted in no symptoms or VCD. A case report suggests the presence of VCD induced by methacholine based on FVL, but direct laryngoscopy was not performed during the patient's acute symptoms.[87] Another case report noted acute flattening of the inspiratory FVL at the maximum dose of methacholine that returned to normal several minutes later.[60] Our initial study of VCD and exertional dyspnea noted that 60% of VCD patients had evidence of FVL changes post-MCT. VCD patients likewise tended to have a smaller decrease in the FEV_1/FVC ratio when compared with patients with a reactive MCT.[181] Our prospective study of VCD during MCT demonstrated that MCT may cause an acute episode of vocal cord adduction, and positive MCT results may not reflect underlying reactive airways disease. Truncation of the inspiratory FVL during MCT was not diagnostic for the presence of inspiratory vocal cord adduction.[182] However, those patients with reactive MCT, changes in FVL, and a physical examination suggesting upper airway obstruction should likely undergo further evaluation for VCD.

Other Modalities

Newer imaging modalities or noninvasive methods are being used to assist in confirming the presence of VCD. In many cases, the severity of symptoms or lack of cooperation by the patient may make flexible laryngoscopy impossible to perform. Ooi commented on the use of ultrasound to diagnose vocal cord paresis and suggested its use for VCD. B-mode real time was not accurate, but color Doppler imaging was as accurate as laryngoscopy.[136] Fluoroscopy has also been reported to demonstrate inspiratory vocal cord adduction.[106] A group of 50 adult VCD patients were examined with laryngoscopy and stroboscopy. Laryngoscopy first documented evidence of paradoxical vocal cord adduction in all patients. When asymptomatic, more than half of the patients demonstrated laryngeal abnormalities on stroboscopy, suggesting an underlying predisposition to VCD attacks.[185] Acoustic pharyngometry is currently being studied as another method to noninvasively study upper airway compliance. An abstract of 14 VCD patients noted 7 patients with abnormal inspiratory flow and 7 patients with normal inspiratory flow. The glottic and hypopharynx compliance as measured by acoustic pharyngometry noted lower compliance in the abnormal flow group. It was not stated if patients were examined during these measurements to document presence of inspiratory vocal cord adduction.[79] Rigau et al used inhaled carbon dioxide and a forced oscillation technique to distinguish VCD from controls in a small cohort of patients.[193] Finally, a retrospective analysis of multidimensional voice program analysis of VCD patients demonstrated abnormalities specifically in the soft phonation index.[166] While none of these modalities has been compared directly with laryngoscopy, they are other tools to allow a more rapid and noninvasive method to diagnose VCD.

Acute Management

The acute management of VCD first requires establishing the correct diagnosis in patients without a prior VCD diagnosis. Once recognized, treatment should then be directed at relieving the airway obstruction. Typically, there is minimal response with continued treatment of asthma using inhaled bronchodilators, corticosteroids, and other asthma medications. These medications should not be discontinued if underlying airways hyperreactivity is suspected. If there is clear laryngoscopic evidence of VCD, the first step is to reassure the patient that the condition is benign and self-limited. Case reports in 23 patients discuss the effectiveness of relieving acute airway symptoms simply with reassurance.[33,35,42,46,56,57,63,67,72,92,96,103,112,114,150] The second measure that has been used effectively is sedation with benzodiazepines such as midazolam. Andrianopoulos et al reported the use of sedatives in 5 of the 27 VCD patients in their retrospective series.[145] In addition, another 24 patients from various case reports have been reported to cease or decrease symptoms when given intravenous doses of sedatives.[13,21,24,25,28,34,35,38,46,56,64,66–68,73,78,92,96,97,99,112,120,122,124] We have noted administration of benzodiazepines to be very effective in terminating VCD episodes in our patients who present with acute symptoms. Other adjuncts to treat patients emergently to relieve airway obstruction are discussed below.

Heliox

Heliox has been recommended as an adjunct in the acute treatment and diagnosis of VCD.[95,129] Christopher et al commented in their original description of VCD "a mixture of helium and oxygen uniformly relieved dyspnea."[147] Either a mixture of 80% helium and 20% oxygen or 70% oxygen and 30% helium has been reported to have a dramatic and immediate response in VCD.[142] As with other upper airway diseases, reduction of air density decreases turbulent flow that reduces work of breathing associated with the airway obstruction. The increased ease of breathing results in decreased anxiety and aborts the worsening inspiratory vocal cord adduction. Weir conducted a retrospective review of VCD patients seen at their institution; he noted a favorable response in 4 of 5 adolescents treated for VCD with heliox both in the acute setting in the emergency department and with home use.[123] However, reports from the emergency medicine literature do not comment on the use of heliox.

Intubation

The severity of the symptoms seen in some acute VCD episodes has led to emergent intubation or tracheostomy. Our review of the available VCD reports found that a total of 62 patients or 6.2% were emergently intubated and another 31 patients underwent tracheostomy (both emergently and postintubation). Many case reports comment that, after intubation, stridor abates and the patients are "easy to ventilate." These patients are commonly extubated within 24 hours. Only 4 case reports commented on peak airway pressures on mechanical ventilation; 2 noted normal peak airway pressures (no values given), one was reported at 22 cm H_2O and another at 25 cm H_2O.[30,37,75,82] In our experience, resolution of wheezing and/or stridor postintubation with normal airway pressures should prompt laryngoscopic evaluation for the presence of VCD.

Chronic Management

Speech Therapy

Speech therapy is regarded as the primary therapy for VCD.[151] Our review of patients demonstrated that 36% of patients were referred for speech therapy as one method of treatment. The techniques for VCD have been adapted from treatment of functional voice disorders such as laryngeal dystonia. They are aimed at focusing on expiration and diaphragmatic breathing and removing the focus on inspiration and laryngeal breathing. Rogers and Stell were the first to report successful treatment with speech therapy.[3] Christopher et al noted that all 5 of their patients responded well to speech therapy. All patients were asymptomatic after treatment and no longer required used of asthma medications.[147] Martin et al provided a complete description of speech therapy and the specific techniques used at National Jewish Research and Medical Center.[8] Their technique uses 7 specific steps to teach the patient a behavioral approach to voice exercises utilizing diaphragmatic breathing, use of "wide-open" breath, and focusing on exhalation.[8] Sullivan et al performed a prospective treatment study of 20 VCD patients involved in competitive athletics.[184] Utilizing both speech therapy and counseling with family member involvement, patients were

followed up at 6 months for improvements. Ninety-five percent of patients were able to control symptoms and 65% had no further use of asthma medications.[184] Other retrospective studies have likewise demonstrated benefit. Andrianopoulos et al referred 21 of 27 patients for speech therapy as part of a 3-phase multifactorial treatment model.[145] Morrison et al also used speech therapy as a primary treatment in 14 of 25 VCD patients described with "irritable larynx syndrome."[156]

Psychotherapy

Psychotherapy or psychologic counseling has been used extensively in the treatment of VCD and remains a primary treatment modality along with speech therapy. This review of the literature found 278 patients (55.7% of treated patients) underwent various forms of psychiatric therapy. The effectiveness of psychotherapy in eliminating or reducing VCD episodes has not been prospectively studied. Many case reports and large case series describe extensive use of psychiatric evaluations and counseling; Newman et al noted that 73% of patients had a significant psychiatric diagnosis requiring intervention.[158] The National Jewish Medical and Research Center uses formal psychiatric evaluation and counseling as an integral part of the treatment plan for VCD.[179] Other studies have noted a referral rate ranging from 30% to 90% for formal psychotherapy.[145,150] The outcome study by Sullivan et al evaluating speech therapy for VCD used family counseling, but not formal psychotherapy, as part of an effective treatment strategy.[184]

Biofeedback

Biofeedback may be used as a component of psychotherapy in the treatment of VCD. The use of biofeedback was mentioned as treatment in 37 patients in our review of the literature.[36,48,59,108,110,126,143,150,155,157] There is one prospective study investigating the utility of electroencephalographic neurofeedback training as a treatment of VCD. Nahmias et al evaluated 15 female patients diagnosed with "laryngeal dyskinesis" and provided neurofeedback in 5 patients.[157] The authors were able to demonstrate a reduction in midmaximal flow rate (FEF_{50}/FIF_{50}) in 2 patients and normalization in the remaining 3 patients.[157] McFadden and Zawadski noted that 4 of the 9 patients with exercise-induced VCD had rapid resolution of symptoms after basic biofeedback training.[155] Ferris et al also used biofeedback in their series of 9 patients in combination with counseling and antidepressants with good success.[150] The authors noted that 1 patient had 3 episodes of stridor over a period of 5 years and used biofeedback techniques to abate the VCD episodes.

Hypnosis

Hypnotherapy has been occasionally used as an adjunct to psychotherapy in the treatment of VCD.[11,23,111] Smith initially reported the use of hypnotherapy with a relaxation technique in a 16-year-old in whom symptoms resolved with continued patient self-hypnosis in 6 months.[111] Based on its previous use in asthma, Caraon and O'Toole also reported the successful use of hypnosis in a 14-year-old boy with VCD and asthma.[23] A more general use of hypnosis in a pediatric

pulmonary center for various disorders described improvement in 80% of patients. Use of hypnosis in 29 VCD patients who were taught self-hypnosis by their pediatric pulmonologists demonstrated symptom improvement in 31% of patients and VCD resolution in another 38%.[11] The authors also describe its specific use in diagnosing VCD by using hypnotherapy to elicit symptoms and document the presence of inspiratory vocal cord adduction.

Inspiratory and Expiratory Valves

One case report describes another potential method to treat episodes of VCD. Archer et al[13] and MacDonald et al[133] developed an inspiratory device using a one-way adjustable pressure limiting valves from single use anesthesia circuits. By adjusting the inspiratory valve, a patient can increase resistance to inspiratory flow and eliminate stridor. The report noted initial use every 2 hours initially and then down to as needed use 2 weeks later as VCD symptoms abated. Goldman and Muers suggested that continuous positive airway pressure can be used to treat patients with expiratory VCD but did not provide any clinical experience on its effectiveness.[173] Lloyd and Jones commented on the use of intermittent continuous positive airway pressure and heliox in the acute setting for a patient unable to sleep due to severe inspiratory stridor.[65]

Botulinum Toxin

The experience with botulinum toxin in VCD is limited with its use only reported in 9 VCD patients.[40,65,68,121,143] Botulinum toxin prevents the release of acetylcholine at nerve terminals and causes a chemical denervation, thereby preventing vocal cord adduction. It has been used in the treatment of various dystonias such as blepharospasm, spasmodic torticollis, and spasmodic dysphonia. It is likewise reported to have good success in 9 patients with adductor laryngeal breathing dystonia, a disorder characterized by inspiratory stridor, cough, and normal voice with associated dystonias. After injection in the thyroarytenoid muscle, patients had maximal effect at 2 weeks and sustained improvement for 14 weeks.[44] Garibaldi et al reported the first use of botulinum toxin for VCD in an abstract in 1993. A competitive athlete with exercise-induced VCD remained asymptomatic for 2 months following injection of botulinum toxin.[40] Several other case reports noted the successful use of botulinum toxin for patients with repeated tracheal intubations and tracheostomy who did not respond to more conventional therapies.[65,68] One case series demonstrated good results with botulinum toxin for VCD. Altman et al injected botulinum toxin in the thyroarytenoid muscle in 5 patients (2 with other dystonias in addition to VCD). While preventing further urgent airway interventions in 3 patients, the 2 patients with a tracheostomy were unable to be decannulated.[143]

Prognosis

There is minimal information on the long-term prognosis of VCD. None of the large series comments on the long-term prognosis for VCD patients. The available literature describes a variety of clinical courses. Some patients have self-limited symptoms and respond rapidly to speech therapy and psychotherapy. Many of the 20 patients in one study had either complete resolution or reduction in symptoms at 6 months following intensive speech therapy and counseling.[184] Conversely, many patients do not respond to standard therapies and continue to have recurrent symptoms. Hayes et al described 3 VCD patients who were followed up over a 10-year period. All 3 patients failed to respond to speech therapy, psychotherapy, and hypnotherapy with recurrent VCD episodes and chronic use of asthma medications.[48] No studies have determined whether the underlying cause (organic versus nonorganic) makes a difference in the resolution of symptoms or long-term prognosis.

CONCLUSION

Vocal cord dysfunction is an important cause of acute and chronic respiratory symptoms and should be considered in the differential diagnosis of dyspnea. Recognition of this disorder is especially important in those asthmatics who fail to respond to standard treatment. Classically considered to be a psychologic disorder, new evidence is emerging that a variety of mechanisms may induce the glottis to be hypersensitive. The relationship between the physical and psychologic causes of vocal cord dysfunction is not well understood. The majority of vocal cord dysfunction knowledge is based on case reports and large retrospective cohorts. More prospective evaluation is required to better identify the mechanism of vocal cord dysfunction, the contributing physical and psychologic factors, and the most effective treatment of these patients.

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