Vocal Cord Dysfunction in Patients With Exertional Dyspnea*

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Study objectives: To evaluate patients for vocal cord dysfunction (VCD) in a military population presenting with symptoms of exertional dyspnea.

Design: Cross-sectional, controlled study.

Setting: Pulmonary disease clinic at an army tertiary care center.

Patients: Forty military patients with complaints of exertional dyspnea and 12 military asymptomatic control subjects.

Intervention: Patients underwent direct visualization of vocal cords with flexible laryngoscopy before and after exercise to evaluate for presence of inspiratory vocal cord adduction.

Measurements and results: Complete evaluation for all patients consisted of spirometry with flow-volume loops, lung volumes, diffusing capacity, and maximum voluntary ventilation at rest; chest radiograph; methacholine bronchoprovocation testing; and a maximal cardiopulmonary exercise test with expiratory gas analysis. Fifteen percent of patients studied prospectively were found to have VCD, whereas all control subjects were negative for VCD. There was minimal difference in pulmonary function testing between VCD-positive and VCD-negative patients, whereas control subjects had higher spirometric values. Twenty percent of VCD-positive patients had abnormal flow-volume loops compared with 14% of patients without VCD, but after methacholine, 60% of VCD-positive patients developed abnormal flow-volume loops. In the VCD-positive group, 60% had a positive methacholine response, but there was less decrease in FEV₁/FVC ratio compared with either VCD-negative patients or control subjects.

Conclusions: Paradoxical inspiratory vocal cord closure is a frequent occurrence in patients with symptoms of exertional dyspnea and should be strongly considered in their evaluation.

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Key words: exercise testing; exertional dyspnea; flow-volume loops; laryngoscopy; methacholine; pulmonary function testing; vocal cord dysfunction

Abbreviations: CPEX = cardiopulmonary exercise testing; DLCO = diffusing capacity of the lung for carbon monoxide; FVL = flow-volume loop; MVV = maximum voluntary ventilation; PFT = pulmonary function testing; RR = respiratory rate; VAT = ventilatory anaerobic threshold; VCD = vocal cord dysfunction; VCO₂ = carbon dioxide production; VE = minute ventilation; VO₂ = oxygen uptake; VT = tidal volume; VT/IC = tidal volume to inspiratory capacity ratio

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Vocal cord dysfunction (VCD) is a well-described condition characterized by the paradoxical inspiratory closure of the vocal cords. Adduction of the vocal cords during inspiration limits airflow, and individuals complain of wheezing, stridor, shortness of breath, or dyspnea on exertion. The acute presentation is sometimes dramatic, leading to intubation or tracheostomy for upper airway obstruction. More commonly, these patients present with wheezing and frequently receive a misdiagnosis of poorly controlled asthma. Treatment with β-agonists and corticosteroids generally tends to be unsuccessful. Many of these patients are asymptomatic at rest and require exercise challenge to elicit symptoms and vocal cord abnormalities.
tensioning or truncation of the inspiratory limb of the flow-volume loop (FVL) has been a characteristic finding with this entity. However, this classic FVL abnormality has been shown to be absent in the majority of patients without acute symptoms. The “gold standard” for the diagnosis of VCD remains laryngoscopy with direct visualization of the abnormal movement of the vocal cords. The classic findings on laryngoscopy are inspiratory vocal cord closure with posterior “chinking” (a small opening at the posterior aspect of the cords), although closure of the vocal cords can be seen on expiration.

The majority of the literature concerning VCD consists of small retrospective series and case reports. The largest series is a chart review from the National Jewish Center for Respiratory Disease. Ninety-five cases of VCD proven by laryngoscopy were reviewed between the years 1984 and 1991. Fifty-three of the 95 patients (56%) were also found to have either airflow limitation or reactive bronchoprovocation testing consistent with asthma. Another retrospective review from an army tertiary medical center described 20 patients found to have VCD. Only seven of these patients (35%) also had underlying obstructive lung disease. McFadden and Zawadski described seven elite athletes with VCD who had received a diagnosis of exercise-induced asthma. Multiple bronchoprovocation tests including cold-air isocapnic hyperventilation and methacholine did not provoke symptoms, but exercise challenge did elicit VCD in six of seven patients. Many other case reports describe VCD in relationship to symptoms of exertional dyspnea, in which it has been initially misdiagnosed as exercise-induced asthma.

At present time, there are no prospective studies that have evaluated the frequency of VCD. The purpose of this study is to evaluate a group of young military subjects with complaints of exertional dyspnea for evidence of VCD. The majority of these patients had been treated as exercise-induced asthma by the referring physicians. By undergoing a standard evaluation for exertional dyspnea including direct laryngoscopy before and after exercise, the percentage of patients with VCD in this group could be established. The results of other studies to evaluate for exertional dyspnea, including pulmonary function testing (PFT), methacholine testing, and exercise testing, could then be compared with normal control subjects and other patients without VCD.

Materials and Methods

Any active-duty patient between the ages of 18 and 50 years referred to the Pulmonary Disease Clinic at Brooke Army Medical Center, Fort Sam Houston, TX, for evaluation of new symptoms of exertional dyspnea was considered eligible for the protocol. Exertional dyspnea was defined as shortness of breath that occurred primarily with running, a regular part of physical training for soldiers. Any patient with an established diagnosis of pulmonary or cardiac disease was excluded from the study. All patients had to be capable of performing full PFT maneuvers and complete cardiopulmonary exercise testing (CPET) on a graded exercise treadmill. One hundred five patients were recruited for the standard protocol to evaluate exertional dyspnea. Seven patients from the initial dyspnea group (n = 72) were evaluated for VCD because of symptoms that suggested this disorder. Five patients had positive laryngoscopic examinations with evidence of inspiratory vocal cord adduction (VCD positive), and two patients were negative (VCD negative). The final 33 patients with exertional dyspnea were evaluated prospectively for evidence of VCD. An additional five patients were found to have VCD, and the remaining 28 patients were negative for VCD. The distribution of patients is shown in Figure 1.

Control subjects consisted of 12 asymptomatic active-duty volunteers with no history of cardiac and pulmonary disease who were able to complete the army physical fitness 2-mile run > 75% of the standard (60% is the minimum score required to pass). Both patients and control subjects were evaluated in an identical manner as described below. All study participants underwent a complete history and physical by a pulmonary physician before entrance into the protocol. A standard posteroanterior and lateral chest radiograph was also reviewed for any abnormalities.

PFT

All patients performed full PFT maneuvers on the 6200 Body Box (Sensormedics; Yorba Linda, CA) with flow-sensor spirometer. Predicted values were those of Morris et al. Spirometry was first performed to evaluate for FEV₁, FVC, FEV₁/FVC ratio, and appearance of the FVL. If there was evidence of airways obstruction, postbronchodilator testing was performed. Lung volumes were determined using body plethysmography to determine total lung capacity and residual volume. Diffusing capacity of the lung for carbon monoxide (DLCO) was determined using the single-breath carbon monoxide technique. Completion of PFT included measurement of the maximum voluntary ventilation (MVV).

Bronchoprovocation Testing

Bronchoprovocation testing was performed using a modified methacholine challenge study. All patients and control subjects

**Figure 1.** Flow diagram demonstrating the distribution of patients with exertional dyspnea and the method of evaluation (eval) for VCD.
were required to have refrained from caffeine and any inhaled bronchodilators at least 12 h before the test. The patient performed three FVC maneuvers at baseline. The patient was administered increasing doses of methacholine mixed in normal saline solution at the following concentrations: 0.025, 2.5, and 25 mg/mL. Each was given via five breaths through a Salter model 0700 dosimeter (Salter Labs; Arvin, CA) using an inspiratory time of 0.6 s. The patient waited 3 min and performed two FVC maneuvers. This was repeated for all concentrations of methacholine until the maximal concentration was reached or there was a 20% drop in the FEV1. If there was a > 10% decrease in FEV1, then the patient received two puffs of albuterol followed by repeat FVC maneuvers 5 min after the administration of the β-agonist to demonstrate responsiveness to a bronchodilator.11

**Cardiopulmonary Exercise Testing**

All patients performed a graded exercise test using an incremental protocol on the Series 2000 treadmill (Marquette Electronics; Milwaukee, WI). They were continuously monitored during the test with transcutaneous oxygen saturation monitoring with the Lifesat 1600 pulse oximeter (PhysioControl; Redmond, WA) and 12-lead ECG monitoring by the Marquette 2000 during the test. BP was taken before the test and immediately on completion of exercise. All subjects were exercised using a Bruce incremental treadmill protocol with an increase in grade and speed every 3 min. The subjects were asked to continue exercising until limited by symptoms or completion of the maximum Bruce stage. During the entire warm-up, exercise, and recovery phases of the test, inspiratory and expiratory gas analysis was performed through the 2900 Series Metabolic Cart (Sensormedics). This specifically measured breath-by-breath analysis of oxygen uptake (V\(\text{VO}_2\)), carbon dioxide production (V\(\text{CO}_2\)), tidal volume (V\(\text{T}\)), respiratory rate (RR), and minute ventilation (V\(\text{E}\)).

The following specific parameters were analyzed in each patient for evidence of limitation to exercise: maximum V\(\text{VO}_2\), ventilatory anaerobic threshold (VAT), V\(\text{E}\) to MVV ratio (V\(\text{E}/\text{MVV}\)), RR, ventilatory equivalent for carbon dioxide production (V\(\text{E}/\text{VCO}_2\)), and V\(\text{T}\) at peak exercise to inspiratory capacity ratio (V\(\text{T}/\text{IC}\)).

**Vocal Cord Evaluation**

Before the CPEX, each patient underwent direct laryngoscopy performed with the Kay Elemetrics Swallowing Workstation 7100 (Kay Elemetrics; Lincoln Park, NJ), using a flexible rhinoscope. Each patient received topical 2% viscous lidocaine in the right naris for anesthesia. The posterior pharynx was specifically not anesthetized to avoid involvement of the vocal cords. The laryngoscope was directed to the posterior pharynx several centimeters above the glottis to prevent stimulation of the area and induce adduction of the vocal cords. Videotape records of vocal cord movement were made during the evaluation. Observation was made of the vocal cords while the patient demonstrated normal motion of the cords with speech and performed rapid breathing for 10 s. Patients were specifically examined for evidence of vocal cord adduction during inspiration. Each patient then completed the CPEX, and immediately on discontinuation of exercise, the flexible rhinoscope was reinserted through the nares without repeat anesthesia. Again, videotape records were made of the study, and the presence or absence of vocal cord adduction during inspiration was noted. The confirmation of VCD was made by the speech pathologist who was blinded to the identity of each patient. Those patients in whom VCD was present were referred for further evaluation and treatment.

In addition to the above testing procedures, all patients also completed other studies to evaluate for other potential causes of exertional dyspnea. Laboratory tests included arterial blood gas analysis, CBC, serum electrolytes, and thyroid screening. Patients were evaluated for cardiac disease with a standard 12-lead ECG and echocardiography with Doppler flow imaging to evaluate pulmonary artery pressures.

**Results**

A comparison was performed of the PFT results, methacholine testing, and CPEX between three groups of patients. A total of 10 patients were considered VCD positive based on findings with direct laryngoscopy. There was agreement between the pulmonologist and speech pathologist in all cases. In the initial symptomatic evaluation group, five patients were identified: two patients had characteristic findings with both pre- and postexercise laryngoscopy, whereas the remaining three patients were only abnormal after exercise testing. The remaining five VCD-positive patients from the prospective group only had evidence of VCD after exercise. Of the patients evaluated prospectively, 5 of 33 patients (15.2%) had evidence of VCD. Thirty patients had normal vocal cord examinations before and after exercise and were considered VCD negative. The 12 control subjects also underwent an identical evaluation and all had negative examinations.

In the VCD-positive group (n = 10), there were 3 male and 7 female patients with an average age of 22.5 ± 2.4 years. In the VCD-negative group (n = 30) there were 20 male and 10 female patients with an average age of 25.6 ± 7.1 years. The control group (n = 12) consisted of 8 male and 4 female patients with an average age of 24.3 ± 4.4 years. The diagnoses in the VCD-negative group varied: five patients had asthma on the basis of baseline spirometry and a 20% decrease in FEV1 after methacholine, three other patients were treated for exercised-induced asthma who had > 15% decrease in FEV1 after methacholine, significant response to a bronchodilator, and all other studies negative. Three patients had gastroesophageal reflux disease, one had hyperventilation syndrome, and 11 patients had conditions that remained undiagnosed after completion of the preliminary exertional dyspnea evaluation.

All patients completed full PFT, which included spirometry, lung volumes, DLco, and MVV. The results for all patients and control subjects are shown in Table 1. None of the VCD-positive patients had evidence of a baseline obstructive process on spirometry consistent with asthma. Five VCD-negative patients with asthma were obstructed at baseline,
and one control subject was noted to have mild obstruction by PFT. The values for all PFT measurements were similar for VCD-positive patients and VCD-negative patients. However, FEV₁ and FVC for all patients were decreased when compared with normal control subjects. In the VCD-positive group, 100% of the patients had an MVV < 70% of predicted, and there is a notable difference between VCD-positive (52.7 ± 12.2%) and VCD-negative (67.6 ± 17.8%) patients. In the VCD-negative group, 66% of patients also had an MVV < 70% of predicted, whereas no control subjects were abnormal.

All patients and control subjects completed methacholine bronchoprovocation testing as previously described. For the VCD-negative group, eight patients (27%) were methacholine positive (defined as a 20% decrease in FEV₁ from baseline), whereas only two patients (14%) in the control group reacted to the methacholine. In the VCD-positive group, 6 of 10 patients had a positive methacholine test. Figure 2 shows the mean decrease in both FEV₁ and FEV₁/FVC for all methacholine bronchoprovocation tests for each group. There is less decline in the control (11%) and VCD-negative groups (17%) than the VCD-positive group (23%) because of fewer patients with increased airway reactivity. However, the mean decrease in FEV₁/FVC is very similar. Figure 3 shows the same information for only those patients with a positive methacholine bronchoprovocation test. There is a definite trend in the decrease of FEV₁/FVC with a much smaller decrease for the VCD-positive patients (8%) when compared with the VCD-negative (13%) or control group (20%).

FVL on both baseline spirometry and with methacholine testing were reviewed and were considered abnormal if there was evidence of flattening or truncation of the inspiratory limb. Both findings are consistent with a variable extrathoracic obstruction to inspiratory flow. None of the control subjects had evidence of abnormal FVL on either baseline spirometry or after methacholine testing. Table 2 shows the percentage of total patients with evidence of abnormal FVL during baseline spirometry or change with methacholine testing. Twenty percent of VCD-positive patients had an abnormal baseline FVL, whereas 13% of VCD-negative patients had an abnormal baseline FVL. In VCD-positive patients, there was a significant increase in alteration of the FVL after methacholine.

CPEX was completed on all patients, with the mean values for measurements shown in Table 3. All

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>VCD Positive</th>
<th>VCD Negative</th>
</tr>
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<tbody>
<tr>
<td>FEV₁, % predicted</td>
<td>99.8 ± 11.1</td>
<td>85.7 ± 13.8</td>
<td>85.6 ± 11.2</td>
</tr>
<tr>
<td>FVC, % predicted</td>
<td>101.9 ± 8.7</td>
<td>89.3 ± 16.5</td>
<td>88.6 ± 12.5</td>
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<tr>
<td>FEV₁/FVC, % predicted</td>
<td>97.7 ± 6.3</td>
<td>95.7 ± 6.1</td>
<td>96.7 ± 5.1</td>
</tr>
<tr>
<td>TLC, % predicted</td>
<td>101.0 ± 12.6</td>
<td>89.0 ± 14.9</td>
<td>90.9 ± 14.3</td>
</tr>
<tr>
<td>DLco, % predicted</td>
<td>93.5 ± 13.2</td>
<td>84.3 ± 15.1</td>
<td>84.7 ± 16.9</td>
</tr>
<tr>
<td>MVV, % predicted</td>
<td>91.9 ± 13.7</td>
<td>52.7 ± 12.2</td>
<td>67.6 ± 17.8</td>
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*Comparison of mean values ± 1 SD for each PFT measurement. All values are given as percent of normal predicted; TLC = total lung capacity.
patients except one in the VCD-positive group reached VAT. Only one other patient was diagnosed with deconditioning on the basis of CPEX. Ninety percent of VCD-positive, 47% of VCD-negative, and 25% of control subjects had one value (Ve/MV > 80%, RR > 50, Ve/VCO₂ > 40, or Vt/IC > 80%) that suggested respiratory limitation to exercise. Asymptomatic control patients demonstrated an ability to exercise longer (higher maximum \( \dot{V}O₂ \)) and reach anaerobic threshold later (higher VAT) than patients with exertional dyspnea. The difference seen with Ve/VCO₂ reflected those patients with obstructive airways disease in the VCD-negative group. With respect to other respiratory measurements on CPEX, little difference was seen. All groups have a normal Ve/MV ratio (normal < 80%) and Ve/VCO₂ (normal < 40). VCD-positive patients did have a mean elevated RR of 54.2 ± 12.3 breaths/min (normal < 50) and elevated Vt/IC ratio of 86.7 ± 20.9% (normal < 80%) but there was minimal difference between VCD-negative patients and control subjects.

The results of other laboratory studies to evaluate for other causes of exertional dyspnea in the patients evaluated for VCD were all within normal limits. Echocardiography results in all groups revealed no significant abnormalities and normal pulmonary artery pressures. All ECGs and chest radiographs were without significant abnormalities. Arterial blood gas analyses also showed no evidence of hypoxemia or hypercarbia. All other laboratory values were found to be within normal limits for our institution. Calcium concentrations were found to be 9.6 ± 0.3 mg/dL for the VCD-positive group compared with 9.5 ± 0.3 mg/dL for VCD-negative group.

Table 2—FVLs With Inspiratory Limb Truncation

<table>
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<tr>
<th>Group</th>
<th>Baseline Spirometry</th>
<th>After Methacholine</th>
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</thead>
<tbody>
<tr>
<td>VCD Positive</td>
<td>20.0%</td>
<td>60.0%</td>
</tr>
<tr>
<td>VCD Negative</td>
<td>13.3%</td>
<td>10.0%</td>
</tr>
<tr>
<td>Control</td>
<td>0%</td>
<td>0%</td>
</tr>
</tbody>
</table>

Research over the last 2 decades has resulted in a greater understanding of laryngeal dyskinesias to include paradoxical VCD. Only in the last few years has the concept of VCD as a cause of dyspnea been generally accepted by the medical community. Our present study examined VCD in a cohort of young military patients presenting with a primary complaint of exertional dyspnea and an inability to pass a required army physical fitness test. Many of the patients were referred to our clinic with a presumptive diagnosis of exercise-induced asthma before spirometry or bronchoprovocation testing. VCD should be considered in any individual who presents with exertional dyspnea in whom other pulmonary, cardiac, and anatomic laryngeal abnormalities have been excluded.

Many investigators consider VCD to represent a conversion reaction with various other psychiatric conditions that is triggered by emotional and possibly physical stress. VCD is an involuntary disorder inasmuch as patients cannot produce the abnormal laryngeal movements voluntarily. Extremely stressful events such as wartime have also been documented to elicit VCD symptoms. Others suggest that VCD may not be solely a conversion reaction and can be associated with a wide variety of psychological disorders. Many of the patients in our study population were young Army recruits undergoing advanced individual training, which is generally considered to be a time of great emotional, psychological, and physical stress. Individual recruits are encouraged by their superiors to achieve high scores in both the classroom and during physical training. As such, the cohort we studied may have a built-in selection bias.

Although there have been prior case reports describing paradoxical VCD as a cause of dyspnea in young athletes, this is the first study that prospectively evaluated VCD in a cohort of patients presenting with a primary complaint of exertional dyspnea. Our data were surprising in that the prevalence of VCD is higher than we anticipated, present in 15% of the patients we prospectively studied. The majority of patients were all found to have VCD by direct laryngoscopy after exercise and were asymptomatic at rest. Our study patients are young and required to engage in regular strenuous exercise programs. This is similar to the seven elite athletes reported by McFadden and Zawadski who were referred for evaluation of dyspnea and a “choking sensation” during competitive physical activity. We may have also identified VCD in association with other pulmonary processes. None of the VCD-positive patients

Table 3—Cardiopulmonary Exercise Testing*

<table>
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<tr>
<th>Variable</th>
<th>Control</th>
<th>VCD Positive</th>
<th>VCD Negative</th>
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<tbody>
<tr>
<td>Max ( \dot{V}O₂ ), % pred</td>
<td>106.2 ± 9.8</td>
<td>84.7 ± 12.6</td>
<td>87.9 ± 13.9</td>
</tr>
<tr>
<td>VAT, % Max ( \dot{V}O₂ )</td>
<td>80.9 ± 9.0</td>
<td>72.0 ± 13.0</td>
<td>68.1 ± 10.7</td>
</tr>
<tr>
<td>Ve/MVV</td>
<td>67.0 ± 10.5</td>
<td>67.9 ± 11.8</td>
<td>72.1 ± 14.1</td>
</tr>
<tr>
<td>RR, breaths/min</td>
<td>46.7 ± 7.2</td>
<td>54.2 ± 12.3</td>
<td>49.1 ± 13.9</td>
</tr>
<tr>
<td>Ve/VCO₂</td>
<td>31.1 ± 3.3</td>
<td>36.3 ± 5.5</td>
<td>33.7 ± 4.6</td>
</tr>
<tr>
<td>Vt/IC</td>
<td>78.9 ± 12.1</td>
<td>86.7 ± 20.9</td>
<td>79.2 ± 25.6</td>
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</table>

*Comparison of mean values ± 1 SD for each cardiopulmonary exercise testing measurement.
had asthma on the basis of normal baseline spirometry, but six had positive responses during methacholine challenge studies.

Making the diagnosis of VCD other than with direct visualization of the vocal cords can be difficult. The sensitivity of FVL is very low; previous studies have reported only a 23% incidence of abnormal inspiratory limbs in asymptomatic patients with documented VCD. The low utility of FVL screening is demonstrated in our current study inasmuch as only 20% of patients with documented VCD exhibited inspiratory limb truncation during routine spirometry. We noted that 60% of VCD-positive patients exhibited changes in the FVL after methacholine. This may represent evidence that VCD can be elicited by bronchoprovocation testing.

We reported that 60% of patients with evidence of VCD by flexible rhinolaryngoscopy had a positive methacholine challenge test. This is similar to the 56% incidence of coexistent asthma and VCD reported at National Jewish Hospital in Denver. This contrasts with the 29% incidence of positive methacholine challenge tests in the VCD-negative group and 16% in the control group. These patients may in fact have a combination of reactive airways disease and VCD as previously reported. However, the patients with VCD and a positive methacholine challenge (20% decrease in the FEV1) were more likely to have a decrease in the FEV1 and the FVC after methacholine challenge when compared with the control and the VCD-negative groups with positive methacholine challenge tests. This suggests that patients with VCD may be more prone to developing a smaller decrease in FEV1/FVC because of a reduced inspiratory volume and not increased airways obstruction. Although patients with reactive airways disease and positive bronchoprovocation tests can show decreases in both FEV1 and FVC, this significant difference between our groups may indicate that VCD may play some role in altering the test. This trend, in combination with the significant number of patients with FVL changes during methacholine challenge testing, indicates several possibilities. It also may be possible that methacholine testing could induce vocal cord closure, thereby limiting airflow through the trachea. This suggests that individual FEV1 and FVC values obtained during methacholine challenge should be examined closely for similar degree of change. Many patients previously diagnosed with asthma by methacholine challenge may in fact have VCD. Previous publications have not closely evaluated the interaction between bronchoprovocation testing and VCD.

The “gold standard” for the diagnosis of VCD is direct observation of the paradoxical inspiratory vocal cord closure while the patient is in the midst of an acute attack. We have attempted to elicit an individual patient’s symptoms by performing CPEX. This is a similar type of exercise, although the environment is more controlled. Increased humidity or extremes of temperature could potentially increase the number of VCD-positive patients. It is also possible that exercising on a treadmill with a mouthpiece to facilitate respiratory monitoring triggers mild vocal cord spasm and increases the number of patients diagnosed with VCD. There may be other factors involved such as situational stressors that were not elucidated in the laboratory environment. An additional limitation is that a single exercise study may not elicit symptoms in those patients with intermittent symptoms. Furthermore, the CPEX data clearly do not help to distinguish VCD patients from other causes of exertional dyspnea.

In conclusion, paradoxical VCD should be considered in a young individual presenting with exertional dyspnea, particularly those with asthma or exercise-induced asthma that is difficult to treat or unresponsive to therapy. Additionally, many patients may not report associated wheezing or stridor and may complain of only dyspnea on exertion or a choking sensation with exercise. Spirometry is usually normal, and baseline FVL infrequently demonstrates truncation of the inspiratory limb. A careful review of the results of methacholine bronchoprovocation testing for FVL changes and changes in FEV1/FVC should be performed in patients in whom VCD is suspected. Flexible rhinolaryngoscopy with direct visualization of the vocal cords is necessary to make the diagnosis. In our group of young active-duty patients with exertional dyspnea, we found that exercise helps to elicit the symptoms. Their level of regular exercise makes them symptomatic at higher levels of exercise than the normal population. However, the relatively common occurrence of VCD should prompt clinicians to evaluate young patients with exertional dyspnea for this entity.

References