Paradoxical vocal cord motion (PVCM), or vocal cord dysfunction, is a descriptive term for inappropriate adduction of the vocal folds during respiration. The laryngeal mistiming leads to breathing difficulty and is often misdiagnosed as refractory asthma. The etiology of PVCM has been unclear but has long been hypothesized to be psychological. The present thesis is a prospective study of 170 patients older than 18 years being evaluated for PVCM, with 117 of the 170 (68.8%) identified as having PVCM by video laryngoscopy. Laryngeal edema (P = .021) and reflux (P = .026) were increased in patients with PVCM. A flat inspiratory arm of the flow volume loop during spirometry testing was a predictor of PVCM (P = .034). A subgroup of 47 newly diagnosed patients with PVCM underwent psychological analysis. The psychological profiles were elucidated using the Minnesota Multiphasic Personality Inventory and the Life Experiences Survey to evaluate stress. Compared to established normative data, PVCM demonstrated a conversion disorder pattern (P < .01) but not an anxiety disorder or a correlation with stress. A subgroup, 11 of the 47 (23.4%), had normal psychological outcomes, and two of the 47 (4.3%) were identified as malingering. Previous studies have suggested that PVCM is strictly a psychological disorder. It is proposed that PVCM is a descriptive term that is multifactorial and the etiology should direct treatment. A classification scheme divides PVCM into primary, or psychological, and secondary. The secondary form consists of medical disorders divided into irritable larynx syndrome and neurologic disorders.

**Key Words:** Paradoxical, paradoxical vocal cord motion, psychological, asthma, vocal cord dysfunction.

**Level of Evidence:** 2c

INTRODUCTION

Historically, the etiology of paradoxical vocal cord motion (PVCM) has been unclear. The term PVCM has assumed many names and has been attributed to various symptoms. It is most often referred to as PVCM or as vocal cord dysfunction. These are descriptive terms reflecting the abnormal physical findings on laryngoscopy. A normal respiratory cycle has wide abduction on inspiration and slight adduction on expiration. Patients with PVCM have inappropriate adduction of the true folds, and often of the supraglottic structures, during the respiratory cycle. As the angle made by the true vocal folds at the anterior commissure (the glottic angle) becomes more acute, the airway becomes more restricted, with creation of turbulent airflow. This can occur during inspiration, expiration, or both phases of the cycle. To be appropriately labeled as PVCM, at some point during the examination, normal complete abduction should be evident. The procedure of choice for the diagnosis of PVCM is fiberoptic laryngoscopy. This is used to identify the abnormal paradoxical motion and to rule out other causes of upper airway obstruction. A standard testing protocol for evaluating these patients has not been universally adopted. This makes comparing results between centers difficult. The characteristic findings of adduction during respiration can be identified at rest, even when the patient is asymptomatic.¹ ² It is important to note that fiberoptic laryngoscopy can also fail to document the characteristic findings, even in patients who are known to have PVCM.³ The false-negative results are due to the intermittent nature of the disorder. Therefore, the use of provocation with exercise or noxious stimuli is commonly utilized at our center to increase the likelihood of capturing an episode of PVCM during laryngoscopy. The PVCM will present with episodic shortness of breath, or even respiratory distress, that is usually provoked by exertion or chemical exposures. Patients can also develop symptoms at rest. These can vary in length from a few minutes to several hours, and often patients have wheezing, or audible respiration, with throat and upper chest tightness. Rarely, the obstruction can be severe enough to precipitate emergent intubation or tracheotomy. The attacks typically do not respond to the short-acting beta antagonists that are efficacious for reactive airway disease. When combined with other conditions, such as asthma or reflux, the response can be exaggerated and present with an associated cough or laryngeal globus sensation. Because of the
similarity in presentation, PVCM is most frequently misdiagnosed as refractory asthma.3–5

Vocal cord dysfunction has always been strongly attributed to be a psychological disorder and not a physical abnormality. Patterson et al. originally described the condition in 1974 as Munchnausen's stridor, implicating a psychological and volitional etiology.9,10 Leo and Konakanchi, in a meta-analysis, reviewed 171 cases of PVCM from various reports.7 A multitude of psychological conditions associated with PVCM were implicated and included conversion disorders (12%), anxiety disorders (11%), histrionic and other personality disorders (6%), depression (4%), psychosomatic disorders (2%), factitious disorders (2%), and somatization disorders (1%).7 These diagnoses were determined by psychiatric evaluation but not confirmed by testing. The majority of publications have concluded that PVCM represents a psychological conversion reaction.6,7–10 Conversion disorders are characterized by alterations in voluntary motor or sensory functioning rooted in psychological conflict without a physiological basis.11,12 According to the Diagnostic and Statistical Manual for Mental Disorders IV, the symptoms are not intentionally produced and cannot be fully explained by a general medical condition.11 In the case of PVCM, the abnormal laryngeal movement enables the patient to avoid confrontation with an unpleasant life situation and/or yields attention and sympathy (secondary gain).5,10

Several reports have attributed PVCM to etiologies other than psychological causes. Clinical studies have documented an association with gastroesophageal reflux disease (GERD)5,13–15 and even resolution of PVCM symptoms with a daily proton pump inhibitor.13 A neurologic etiology has been suggested that is mediated through the vagus nerve with a lowering of the afferent receptor threshold for stimuli producing an efferent motor response and vocal cord contraction.16 Others have found between 7% and 30% of those diagnosed with PVCM not having an identifiable psychological disorder.7,17 These reports would suggest that PVCM is multifactorial and is not strictly a psychological disorder.

MATERIALS AND METHODS

Study Design

Institutional review board approval was obtained for the study. The cohort in this prospective study consisted of all patients older than 18 years who presented to an academic voice center for breathing difficulty. All of the patients evaluated for possible PVCM completed a medical questionnaire and were tested using a standard protocol. Medical and social histories were reviewed, with a particular focus on asthma, GERD, psychologic/psychiatric diagnoses, and reports of past sexual/physical abuse. The medical histories and associated physical findings on laryngoscopy were noted to help identify contributing factors and develop a differential diagnosis. The source of the referral was also noted. A subset of the cohort with recently confirmed PVCM by laryngoscopy had psychological testing performed. All of the subjects completed the entire Minnesota Multiphasic Personality Inventory-2 (MMPI-2) and Life Experiences Survey (LES). Consent forms were completed, and each patient was tested individually under appropriate testing conditions. The study psychologist read an introductory statement explaining the testing and then sequentially administered the MMPI-2 and LES assessment. The psychological testing period lasted 2 to 3 hours. Upon completion, patients were given a debriefing sheet that explained the main purpose of the study and provided contact information.

Inclusion Criteria

All of the subjects were evaluated by one of three experienced speech language pathologists with the diagnoses confirmed by a fellowship trained laryngologist. Flexible video laryngoscopy in patients with an appropriate history of breathing difficulty was used to make the diagnoses of PVCM. Continuous pulse oximetry and heart rate monitoring was used during the testing. The primary criteria for diagnosing PVCM on the video study included:

1. Observing respiration in the absence of gagging or coughing
2. Inappropriate adduction of the vocal cords during inspiration or during both inspiration and expiration with at least two episodes per 10 respiratory cycles
3. Evidence of normal abduction at some point during the exam
4. Absence of other causes of airway obstruction
5. In the absence of inappropriate adduction during quiet respiration, provoking probing with exercise or chemical challenge was performed. Exercise challenge was done through the use of a stationary recumbent bike, sprinting, or climbing stairs until the patient became symptomatic. The fiberoptic scope was then immediately reinserted to examine the laryngeal motion. The chemical challenge was performed with the fiberoptic scope already in position. The patients were challenged with a liquid cleaning solution, dry laundry detergent, potpourri, and strong cologne. If the patient gave a history of sensitivities to certain stimuli, then that scent was used.

The complete standardized protocol used is listed in Table I. If the patient was diagnosed with PVCM, then an information sheet was provided to invite the person to participate in the psychological study. Subjects were excluded from the psychological study if the patient had already completed laryngeal control therapy for PVCM or for unacceptable scores on the MMPI-2 validity scales.

Psychological Assessment Instruments

MMPI-2. The MMPI-2 was used to screen for various types of psychopathology. The MMPI-2 is the most widely used personality instrument in psychology.16,19 There are a total of 567 items on the MMPI-2, all of which are written in a true-or-false format.16,19 The items are written at an eighth-grade reading level.19 These items are scored with regard to several validity indices, 10 clinical scales, and several additional supplementary, content, and research scales.19 All scale scores are expressed as T scores, which are calculated on the basis of normative data calculated from 2,600 normal individuals (1,138 men and 1,462 women) from five regions of the United States.19 For all MMPI-2 assessments, the clinical and supplementary scales were computer scored, and raw scores and T scores were calculated. For each scale on the MMPI, the mean T score is 55, with a standard deviation (SD) of 10.18,19 Scores are considered elevated at 65 or higher.18,19 Some of the significant scales included the following:

- Scale 1 (Hs: Hypochondriasis) was developed on a group of patients who had many somatic complaints with little or no organic basis and great concern about their physical health.19
- Scale 2 (D: Depression) was initially developed on psychiatric patients with various forms of depression.19 The items in
All PVCM evaluation patients will have check in by a nurse and time out completed. Baseline vitals will be obtained prior to the exam. If baseline O$_2$ Sat is $<92\%$, the staff physician will be notified. Nurse will exit the room until the time of the procedure. The SLP will collect a case history including triggers for dyspnea. Patients will be informed of the procedure and sign an informed consent form. The room will be set up for the procedure including a flexible endoscope, gloves, barrier for counter, and surgical lubrication. Universal precautions and hand hygiene completed by the SLP and nurse. The patient will be offered topical anesthetic, which will be administered by the state, nurse, or physician. The flexible scope will then be passed via the nasal cavity to observe respiration at rest. The patient will be directed to breathe in and out through the nose, in through the nose and out through the mouth, and in and out through the mouth. The patient will hold his or her breath for 5 seconds then be directed to “let it go.” The patient will be directed to count 1 to 10 on one breath. The patient will be directed to count for as long as he/she can on one breath. If constriction is observed during normal respiration, the SLP will direct the patient in various therapeutic breathing techniques to discover what achieves full abduction. If constriction is observed during normal respiration, the SLP will direct the patient in various therapeutic breathing techniques to discover what achieves full abduction. The exam will end at this point if they have shown constriction during normal respiration, and they will be enrolled in laryngeal control therapy. If constriction is not noted and strong odors are a reported trigger, the nurse will hold a container of various scents in front of the patient. If a reaction is noted to strong odors, the patient will be directed in breathing therapy techniques and the exam will end.

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**TABLE I.**

<table>
<thead>
<tr>
<th>PVCM</th>
<th>O$_2$ Sat</th>
<th>SLP</th>
<th>2L/NC</th>
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</thead>
<tbody>
<tr>
<td>paradoxical vocal cord motion</td>
<td>oxygen saturation</td>
<td>speech language pathologist</td>
<td>2 liters per nasal cannula</td>
</tr>
</tbody>
</table>

### Medical and Social History

Medical charts were reviewed for each patient. All patients filled out a medical and voice questionnaire prior to the initial PVCM evaluation. Patients were specifically asked if there was a history of any verbal, emotional, physical, or sexual abuse and if there was prior psychological history. Patients also listed medical diagnoses, surgical history, and current and past medications. Patients were determined to have a positive psychological history if they had an affirmative response on their medical questionnaire or if they reported being on medication with psychopharmacological effects, including antidepressants and/or antianxiety medications.

### RESULTS

All of the patients evaluated for breathing complaints with suspected PVCM were studied prospectively to evaluate the frequency of PVCM, incidence of associated medical conditions, and differential diagnosis. A
total of 170 consecutive patients with potential vocal cord dysfunctions were studied using the standard protocol previously outlined. In the study, 117 of the 170 (68.8%) adult patients were identified as having PVCM. The primary sources of referrals were from pulmonologists (89 patients), primary care physicians (55 patients), otolaryngologists (21 patients), and allergists (5 patients). The pulmonologists had the highest percentage of suspected PVCM confirmed on examination with 68 of 89 patients (76.4%), followed by the otolaryngologists with 16 of 21 (76.2%), primary care with 31 of 55 (56.4%), and allergists with two of five (40%). The high rate of confirmed PVCM stems from the detailed testing protocol used and an astute referral base of pulmonologists who are well acquainted with the signs and symptoms of PVCM. Overall, 132 of the 170 patients were female (77.6%) with 93 of the 117 (79.5%) patients with PVCM being female. Only 13 of the 170 (7.6%) patients, two with PVCM and 11 without, had completely normal findings on laryngoscopy. The most common finding in all patients evaluated was laryngoscopic evidence of laryngeal edema, which was found in 123 of the 170 (72%) patients. In the patients with PVCM, 90 of the 117 (76.9%) had laryngeal edema compared to 33 of the 53 (62.3%) who were negative for PVCM. A Fisher exact test 2 × 2 calculation gives a \( P = .021 \), signifying a significant difference in the proportion of patients presenting with breathing difficulty with laryngeal edema and PVCM. The majority of the patients believed to have edema consistent with reflux disease. Reflux complaints were elicited in 69% (81 of 117) of patients with PVCM. This represented 90% of those found to have laryngeal edema. This was compared to 55% (29 of 53) in those without PVCM (\( P = .026 \)). Of the patients with PVCM, 51 of the 117 (43.6%) had confirmed asthma by testing compared to 17 of the 53 (32.1%) that were found to be negative for PVCM (\( P = .105 \)). Patients with proven asthma referred for PVCM evaluation had a confirmed diagnosis of PVCM in 75% (51 of 68) of cases, and this was statistically significant (\( P < .0001 \)) when compared to nonasthmatics identified as having PVCM. Of the 68 asthmatics referred, 30 had a flat inspiratory arm on the flow volume loop. Twenty-five of the 30 (83.3%) were identified as having PVCM. Only five of the 17 asthmatics who were negative on testing had a flat inspiratory arm, versus 25 of the 51 who were positive for PVCM (\( P = .034 \)). In the cohort with identified PVCM, 39 of the 117 (33.3%) had a positive psychiatric history, which was not different from the 18 of 53 patients (34%) who were suspected of having PVCM but were negative by testing (\( P = .54 \)). Laryngeal sicca was identified in 15 of the positive cases and nine of the negative cases (\( P = .309 \)). Other physical findings in those referred for shortness of breath and suspected PVCM included vocal cord granulomas (\( n = 15 \)), bilateral vocal cord paralysis (\( n = 3 \)), subglottic stenosis (\( n = 3 \)), tracheomalacia (\( n = 3 \)), laryngeal candidiasis (\( n = 2 \)), unilateral vocal cord paralysis (\( n = 1 \)), and laryngomalacia (\( n = 1 \)).

A subset of 62 patients more than 18 years old diagnosed with definite PVCM was invited to participate in the psychological study. Forty-seven patients completed the psychological testing. Forty-five of the 47 were included in the psychological study after the validity scores for the MMPI-2 assessment were interpreted. Thirty-seven (81%) patients were women and eight (19%) were men. The average age was 53 years, with 40% between the ages of 18 and 49 years and 60% being 50 years or older. This group averaged 2.4 visits to the emergency department for PVCM symptoms within the previous 12 months. A history of physical or sexual abuse was revealed in 38% of these patients.

A very detailed analysis of the psychological testing was reported previously. A summary of the significant results for the psychological testing will be emphasized. Mean T scores and SDs for the four MMPI-2 scales were separated by sex. There were significant differences between study patients and the general population on the hypochondriasis scale, depression scale, and the hysteria scale for both males and females. The mean for the study group for the hypochondriasis scale was significantly higher than the mean for the general population (\( M = 55, SD = 10 \); for the males, \( M = 68.5, SD = 17.7 \) \( z = 3.20, P < .001 \); for the females, \( M = 68.1, SD = 10.6 \) \( z = 3.20, P < .001 \)). The effect size for this comparison was large \( (d = 1.13) \) for males and very large \( (d = 1.51) \) for females. The mean for the study group on the depression scale was also significantly higher than the mean for the general population (\( M = 55, SD = 10 \); for the males, \( M = 60.5, SD = 13.8 \) \( z = 3.20, P < .001 \); for the females, \( M = 58.6, SD = 13.0 \) \( z = 2.33, P < .01 \)). The effect size for this comparison was not as great as the hypochondriasis scale, being moderate \( (d = 0.55) \) for males and small for females \( (d = 0.36) \). This would indicate a small difference between females in this study and females in the general population. For the hysteria scale, the mean for the study group was significantly higher than the mean reported for the general population (\( M = 55, SD = 10 \); for the males, \( M = 67.9, SD = 7.3 \) \( z = 3.20, P < .001 \); for the females, \( M = 65.9, SD = 11.3 \) \( z = 3.20, P < .001 \)). The effect size for this comparison was large for both sexes \( (d = 1.29 \) for males, \( d = 1.09 \) for females), indicating a large difference between scores on the hysteria scale for the study group and the general population. The mean for the anxiety scale in the study group (\( M = 56.5, SD = 20.0 \) for males; \( M = 52.2, SD = 10.8 \) for females) was not significantly higher than the general population (\( M = 55, SD = 10 \)).

According to MMPI-2 criteria, a “conversion-V” profile involves elevated scores on the hypochondriasis (1) and hysteria (3) scales and a lower score on the depression (2) scale. This is also known as the “1-3” or “3-1” profile with a slightly lower 2 (the classic conversion-V). For both the male and the female groups in this study, the overall results on average were consistent with the conversion-V pattern. An important subset of patients was also identified through the psychological testing. Although the mean scores for the entire study group were consistent with a conversion disorder, 11 of the 45 patients, or 24.4%, had normal scores on all the scales of the MMPI, suggesting an absence of any psychopathology.

Laryngoscope 122: April 2012 Forrest et al.: Paradoxical Vocal Cord Motion
Comparison means for the LES in the PVCM subgroup showed significantly lower scores than the general population for both males and females. For males, the mean positive stress score (M = 2.25, SD = 2.1) was significantly lower than the mean for the normative population (M = 6.9, SD = 6.0, z = 2.59, P < .01). For females, the mean positive stress score (M = 4.9, SD = 4.7) was significantly lower than the mean for the general population (M = 6.7, SD = 5.5, z = 1.96, P < .05). The effect size was large for the males (d = 0.77) and was small for the females (d = 0.33). The mean negative stress score for the male study group (M = 2.1, SD = 2.6) was also significantly lower than that reported for the general population (M = 4.7, SD = 4.4, z = 2.59, P < .01). Conversely, the mean negative stress score for the females (M = 8.5, SD = 10.7) was significantly higher than the reported mean for the normative female group (M = 5.6, SD = 6.4, z = 2.59, P < .01). The effect size for this comparison was moderate (d = 0.40), indicating a moderate difference between females in the study cohort and females in the normative sample. Regarding total stress scores, the mean (M = 4.4, SD = 3.7) for the males in the study group was significantly lower than the mean for males in the normative group (M = 11.53, SD = 8.0, z = 2.59, P < .01). However, the total stress score for the female study group (M = 13.0, SD = 13.1) was not significantly different than the mean for the general population (M = 12.4, SD = 8.8).

In analyzing medical history, some differences were found. In this psychological subset, the incidences of asthma, GERD, psychologic/psychiatric history, and history of abuse were 65%, 50%, 40%, and 38%, respectively. A two-way analysis of variance (ANOVA) with two factors was used to perform multiple comparisons between different subgroups of patients on four different scales of the MMPI-2. Patients were categorized into one of four non–mutually exclusive groups: 1) medical: patients with a history of asthma, GERD, or a comorbid condition of both, n = 35 (78%); 2) nonmedical: patients without a history of asthma or GERD, n = 10 (22%); 3) psychological: patients with a psychological history, n = 18 (40%); and 4) nonpsychological: patients without a psychological history, n = 27 (60%). The results for the hypochondriasis scale showed those in the medical group (M = 70.6, SD = 11.9) had a significantly higher mean on the hypochondriasis scale than those in the nonmedical group (M = 59.9, SD = 5.9, F = 8.328, P < .05). There was no main effect for the psychological subgroup, meaning there were no significant differences found between those with a psychological history and those without one.

The results of the ANOVA for the depression scale and anxiety scale indicates a significant difference between the means for those that had a psychological history and those that did not. Specifically, the mean for the psychological subgroup on the depression scale (M = 65.4, SD = 13.5) was significantly higher than the mean for the nonpsychological subgroup (M = 54.7, SD = 10.9, F = 7.58, P < .05). The effect size for this comparison was medium (F² = 0.14). The mean for the psychological subgroup on the anxiety scale (M = 59.7, SD = 12.7) was significantly higher than the mean for the nonpsychological subgroup (M = 47.9, SD = 10.1, F = 7.31, P < .05). The effect size for this comparison was also medium (F² = 0.15), indicating a moderate difference between those with a psychological history and those without.

There was no main effect for the medical subgroup, meaning there were no significant differences found between patients with a history of asthma/GERD and those without.

The ANOVA for the hysteria scale reveals no interaction effects between the medical and psychological subgroups, as well as no main effects for both the medical and psychological groups. The absence of any main effects signifies no significant differences between patients with a medical history and those without, and there are no significant differences between patients with a psychological history and those without.

**DISCUSSION**

Vocal cord dysfunction has been considered an uncommon etiology in patients with symptoms of shortness of breath. The identification of patients with PVCM is on the rise with better understanding and education of those who care for patients with breathing difficulty. On the order of 300 patients per year are evaluated at our institution for shortness of breath that is not responding to conventional therapy. Nearly 70% of the patients evaluated in this study were found to have PVCM as the sole cause, or as a contributing factor, to their symptom complex. Historically, the majority of patients with PVCM have been treated for refractory asthma. Ninety-five percent of the patients in this cohort carried a diagnosis of asthma at some point. Thirty-five percent of those labeled as asthmatic subsequently had normal pulmonary function testing and bronchial reactivity testing. The patients are subjected to polypharmacy and multiple courses of steroids. Emergency department visits are frequent, and PVCM can be disabling and adversely affect the quality of life for many individuals.

The etiology of PVCM appears to be multifactorial (Table II). This thesis includes the first prospective study of our knowledge to examine PVCM as a psychological disorder and to use psychological assessment instruments. There is a strong psychological basis for PVCM, and testing indicates this is a classic conversion disorder (P < .01). The psychological aspect appears to represent the majority of these patients. However, there is clearly a subset of PVCM patients that do not fit that profile. Results of psychological testing were completely normal in nearly 25% of patients with PVCM. In addition, the ANOVA revealed a difference between the medical and psychological groups stratifying patients with PVCM on the hypochondriasis and hysteria scales. Comorbid factors such as asthma, reflux, and laryngeal sicca can exacerbate the symptoms. Detailed analysis of the data helps us to separate the etiology into primary PVCM (75%) and secondary PVCM (25%). Primary PVCM is psychologically based. Secondary PVCM can be divided into laryngeal hyperreactivity disorders and a neurologic basis.
Primary, or psychologically based, PVCM is either a conversion disorder or malingering. Early descriptions of this disorder labeled it as Munchausen’s stridor, alluding to the malingering association. The majority of patients have a conversion disorder, with only a small percentage representing malingering. Two of the 49 (4.3%) patients with PVCM in this study would fit a malingering profile on the MMPI-2 testing. Based on our clinical experience, this is a fair representation of the patients seen with PVCM. The use of laryngeal control therapy through a speech language pathologist is of minimal, if any, value in this subset and they should be sent for psychological counseling.

A conversion disorder is a specific somatoform disorder. Somatoform disorders are psychological difficulties in which there are symptoms of a physical disorder without a physical cause. A conversion disorder is defined as a deficit in voluntary motor or sensory functioning, other than pain, with symptoms that are not intentionally produced. Thus, the findings of abnormal constriction of the vocal folds during respiration would be consistent with the classification of a conversion disorder. As such, PVCM patients should demonstrate specific psychopathology on psychometrically sound, psychological assessments. Compared to the general population, PVCM patients’ scores were highly elevated on the hypochondriasis scale (1), mildly elevated on the depression scale (2), highly elevated on the hysteria scale (3), and not elevated on the anxiety scale (A). These results for the first three scales look like a “V” when graphed, and so this is known as the conversion V profile. Thus, PVCM appears to be, on average, consistent with a conversion disorder ($P < .01$). It is known that on the MMPI-2, the conversion V profile is more common among women and older subjects than among men and younger individuals. This is consistent with the results, as 81% of the patients were female and 60% were more than 50 years old. Patients with a conversion disorder prefer medical explanations for their symptoms and often underrate the psychological aspect, do not show appropriate concern about their symptoms, and may even present an overly optimistic view of their situation. The authors have found this to be true with PVCM patients, as one of the most difficult aspects is to help them understand that it is a change in behavior and thinking, rather than a quick medication or surgery, that is most likely to alleviate the condition. The most effective treatments to date for PVCM are psychologically based techniques with speech therapy (laryngeal control therapy) focusing on relaxation techniques, comprehension of mental control over breathing, and stress reduction. This involves deep or pulsed nasal breathing (“sniffs”) combined with pursed lip expiration (“blowing out candles”) or even blowing out through a straw. These techniques should be performed during the laryngoscopy to identify the most effective techniques for keeping the airway patent. The use of biofeedback is helpful in demonstrating the findings and effects of treatment to the patient. Biofeedback is accomplished by having the patient face the monitor with a flexible scope in place while performing tasks to maintain abduction of the vocal folds. When laryngeal control therapy fails, then it is advisable for the patient to be referred for psychological counseling. Psychotherapy, including cognitive-behavioral therapy, personal construct therapy, and patient education, has been shown to be effective for the treatment of PVCM.

Medical comorbidities are very common in patients with PVCM. In the cohort, 76% of the subjects with PVCM had laryngeal edema, and 68% had reflux symptoms. The ANOVA identified a statistically significant effect of medical conditions on the hypochondriasis scale.
A statistically significant difference was also identified in the association of laryngeal edema ($P = .021$) and reflux ($P = .026$) with PVCM when compared to those with breathing difficulty without PVCM. The largest percentile of patients with PVCM had a conversion disorder with an associated comorbid medical condition such as laryngeal edema/chronic laryngitis, reflux, laryngeal sieca, or asthma. Of the 70% of patients with PVCM that would fit the conversion profile, we estimate based on the data that close to 70% have associated medical conditions that contribute to the PVCM. This would therefore represent 50% of all patients with PVCM with the remaining 20% representing a conversion disorder as the only etiology. Medical management of the comorbid conditions becomes an important adjunct to the treatment protocol. This can be done as the initial treatment or in conjunction with laryngeal control therapy.

Asthma deserves special mention owing to the strong association with PVCM. Seventy-five percent of asthmatics evaluated for breathing difficulty were positive on PVCM testing. Having asthma was a strong predictor for having PVCM compared to nonasthmatics with PVCM ($P < .0001$). Asthmatics that persist with breathing difficulty despite treatment should raise the suspicion for PVCM. There was a significant association of PVCM in asthmatics with a flat inspiratory arm on a flow volume loop ($P = .034$), and this was a strong predictor of a positive finding on laryngoscopy. On spirometry, the flow volume loop consists of an expiratory phase and an inspiratory phase. A flat inspiratory arm is indicative of a fixed obstruction that is external to the thorax (Fig. 1). The classic symptoms of PVCM, such as episodic stridor, occur overall infrequently in patients with both PVCM and asthma and do not distinguish asthmatics with PVCM from those without PVCM. In fact, patients with PVCM and asthma present with dyspnea, cough, and wheezing, and these are all nonspecific symptoms used in evaluating asthma control. As a result, PVCM may be underdiagnosed in patients with asthma as the symptoms may be misinterpreted as poorly controlled asthma resulting in escalating asthma therapy. Physicians need to have a high index of suspicion for PVCM in poorly controlled asthmatics, independent of the level of asthma severity. In addition, patients with PVCM have a more rapid onset of shortness of breath than those with exercise-induced asthma. Rapid onset of symptoms should raise the suspicion for PVCM. This is more commonly associated with high-exertion sprinting activities and not as much with distance running with a lower intensity level. Exercise-induced PVCM can mimic exercise-induced asthma, and specific testing, such as a methacholine challenge, needs to be performed to help distinguish them from each other.

Asthmatics could have a predisposition to developing PVCM. Asthmatics have been demonstrated to have an altered autonomic response on exposure to inhaled sulphur dioxide, with a reduction in the parasympathetic tone, and normal controls have an increase in tone. The use of bronchodilator and steroid inhalers can produce laryngeal irritation and edema, and this may predispose asthmatics to a hypersensitivity response. In those patients with equivocal asthma based on pulmonary function testing, a methacholine challenge should be considered. If the methacholine challenge is negative, then the unnecessary use of inhalers should be eliminated, as inhalers can escalate the symptoms of PVCM. In asthmatics with PVCM, an attempt should be made to decrease inhaler frequency or strength in coordination with the patient’s pulmonologist.

Of particular interest, 11 (23.4%) patients had normal results on the MMPI-2, suggesting that there is a subset of PVCM patients in whom the disorder is not associated with any psychopathology. This subset can be labeled as secondary PVCM. This group is further divided into hypersensitivity reactions that are sensory based and neurologic conditions that are more of an abnormal motor response. The majority of these patients have conditions that lead to laryngeal hypersensitivity. This results in involuntary constriction as an overprotective mechanism. The secondary PVCM patient population tends to have more chronic symptoms as opposed to the episodic attacks seen with primary PVCM.

Laryngeal hyperreactivity can be divided into irritation of the laryngeal mucosa or alteration of the sensation. Laryngeal constriction is a normal physiologic response to irritation of the airway, swallowing, aspiration, and emesis. In this condition, the airway becomes hypersensitive and assumes a constricted posture. This can be considered to be on a continuum between throat clearing/cough and laryngospasm. The irritable larynx syndrome has been described as having episodic laryngospasm, dysphonia, and cough. This emphasizes that laryngeal irritation produces a symptom complex similar to PVCM. GERD was felt to be a contributing factor in more than 90% of the patients studied, and one-third had viral associated triggers. Laryngeal hyperresponsiveness has been demonstrated to produce laryngeal constriction. The inhalation of histamine and methacholine in patients with and without asthma produces
laryngeal narrowing.32 Hyperresponsiveness can be induced by chemical exposures, fumes, and strong scents, and irritant induced PVCM has been reported.53,34 Laryngeal edema and PVCM is an important cofactor in patients with industrial or occupational asthma due to inhalation exposures. GERD, allergic laryngitis, laryngeal sicca, recent intubation, laryngeal candidiasis, untreated obstructive sleep apnea, and tobacco can also contribute to hypersensitivity that can complicate the treatment of PVCM. The initial treatment of this subgroup of patients with PVCM consists of implementing behavioral or medical management directed at decreasing the laryngeal irritation. Inquiring about exposure is important information in the management of PVCM. Avoidance therapy is imposed as well as medical management for reflux and/or dryness. Reflux is usually treated with a proton pump inhibitor and the dryness with a cholinergic agonist such as pilocarpine or cevimeline. Sucralfate elixir in our opinion can be helpful as a coating agent for the posterior larynx as well. Laryngeal candidiasis can be a product of inhaled or oral steroids, especially when combined with broad-spectrum antibiotics. This produces laryngeal irritation with edema and should be treated with fluconazole.35 Medical management can be done in conjunction with laryngeal control therapy. In cases where an irritable larynx appears to be the primary factor, improving the health of the mucosa precedes the laryngeal control therapy and can obviate the need for therapy.

Just as with a chronic cough and asthma, chronic rhinosinusitis can contribute to laryngeal irritation. This is consistent with the unified airway model.36 Nasal breathing is an important aspect to laryngeal control therapy to achieve maximal abduction. Chronic mouth breathing can produce desiccation of the laryngeal mucosa, especially with rapid breathing during exertion. This was identified in three different patients with exercise-induced PVCM who were not included in this cohort. In all three cases, a septoplasty corrected the PVCM after the patients had failed laryngeal control therapy.

Laryngeal sensory neuropathy (LSN) has gained acceptance as a cause of a chronic cough.37–39 This may also be a factor in patients with PVCM. In patients presenting with unusual triggers such as talking, laughing, or touching the neck, LSN needs to be considered. This is especially true in those patients that present with a prodromal upper respiratory tract infection. Neuromodulating agents, such as gabapentin and pregabalin, have been used with success in LSN,37–39 and some success has been achieved with these agents in our PVCM population.

Certain neurologic disorders have also demonstrated abnormal vocal cord adduction during respiration. Focal respiratory dystonia has been described and is capable of producing laryngeal mistiming.40 This appears to be a relatively rare cause of PVCM. Onabotulinum toxin A has been used in less than 5% of our patients with PVCM, but it has a very specific role in the treatment protocol. It is used for cases that are refractory to conventional treatment and in those patients with other symptoms that would suggest a dystonia such as spasmodic dysphonia. The patients with dystonia respond well to the use of onabotulinum toxin A. We have historically identified four patients, not involved in this cohort, with multiple sclerosis with intermittent PVCM that coincided with a flare-up of the multiple sclerosis. Two of these patients required a tracheotomy at least on a temporary basis. Autonomic dysfunction of multiple system atrophy, formally known as Shy-Drager syndrome, also has an associated laryngeal abductor paralysis.41

Fiberoptic laryngoscopy has a critical role in the diagnosis and treatment of PVCM. This can be used to confirm the diagnoses and also to rule out other causes of breathing difficulty. Findings in this study included bilateral vocal cord paralysis, unilateral vocal cord paralysis, vocal cord granulomas, subglottic stenosis, laryngomalacia, and tracheomalacia. The differential diagnosis needs to also include glottic stenosis, benign or malignant neoplasm of the larynx, adenotonsillar hypertrophy, and lingual tonsil hypertrophy. A continuous pulse oximeter can help distinguish patients with PVCM from those with a pulmonary or cardiac etiology. This is also helpful in determining if it is still safe to continue with the exertion-provoking portion of the protocol. Patients with PVCM rarely achieve an oxygenation level below 90%. In young healthy individuals, a pulse oximeter reading below 90% should raise the suspicion for exercise-induced arterial hypoxemia.32

CONCLUSION

Vocal cord dysfunction can be an important factor in patients with refractory breathing difficulty. The true incidence is not known, but it appears to be underdiagnosed. Laryngeal-based pathology needs to be considered by physicians who treat patients with refractory shortness of breath. The term PVCM is descriptive for inappropriate adduction of the larynx during respiration. One of the confounding factors has been that the literature for PVCM has reported predominately level 3 evidence. The psychological findings in this study represent the only prospective psychological evidence to support PVCM as a conversion disorder, but that represents only a proportion of those diagnosed with PVCM. As such, treatment of patients with PVCM needs to be individualized and focused on treating the proper etiology, or the contributing medical conditions, and not just the standard therapy that assumes this to be a conversion disorder. Treating all patients with PVCM as a conversion disorder will only result in unacceptable high failure rates. Although the majority of the patients have a psychological etiology, this entity needs to be considered as multifactorial. Vocal cord dysfunction can be divided into primary PVCM (75%) and secondary PVCM (25%).

Fiberoptic laryngoscopy is the procedure of choice in diagnosing PVCM. A standardized protocol with provocative testing should be followed with the supplemental use of a pulse oximeter to follow the oxygenation levels and heart rate. This will ensure that the exertion provocation is safe and can assist in the
diagnosis, as hypoxemia is not a characteristic of PVCM. Laryngoscopy will identify the characteristic finding of adduction during respiration and evaluate for other causes of upper airway obstruction. To optimize response to treatment, laryngeal control therapy techniques, such as deep nasal inspiration and pursed lip blowing, should be performed to observe which of the techniques maximize glottal opening.

Primary PVCM has a psychological basis. Malingering is included in the psychological grouping and represents 5% of patients with PVCM. The remaining 70% are patients with a conversion disorder. This is not an anxiety disorder or stress related based on our data. This group can be subdivided into isolated conversion (20%), or conversion with an associated comorbid medical condition. The largest percentile of patients with PVCM would fall into the category of conversion disorder with an associated medical condition (70% of the conversion group or 50% of all patients). Treatment needs to be individualized to the history and physical findings, and both the comorbid medical conditions and the psychiatric aspects need to be treated to optimize outcomes. If malingering is suspected, then treatment needs to be directed toward psychotherapy. In those without associated medical comorbidities, respiratory retraining through laryngeal control therapy directed by a speech language pathologist should be the initial form of treatment, followed by psychotherapy or a trial of onabotulinum toxin A in refractory cases. Laryngeal edema and GERD have a significant association with PVCM. Laryngeal edema has multiple causes including inhaler use, inhaled irritants, reflux, or allergies. Medical management has an important role in the treatment of this subset of primary PVCM. This can be done prior to, or in conjunction with, laryngeal control therapy.

Asthma is strongly associated with PVCM, and identifying asthmatics with PVCM is important in their treatment protocol to avoid escalating medical management. Refractory asthma with a flat inspiratory arm of the flow volume loop is a strong indicator of PVCM. Asthmatics may have a predisposition to developing PVCM. Collaboration with a pulmonologist is essential in the treatment of asthmatics or suspected asthmatics. In the patients studied, 95% carried a diagnosis of asthma at some point in their management, with 35% of those previously labeled as asthmatic subsequently found to be normal on pulmonary function testing and bronchial reactivity testing. Formal pulmonary function testing should be obtained if a definitive diagnosis of asthma has not been confirmed. This is followed by bronchial reactivity testing with a methacholine challenge if equivocal results are found. If asthma can be excluded, then the use of inhalers should be eliminated to decrease laryngeal irritation.

The remaining 25% of patients have secondary PVCM. This represents those patients who tested normal on the MMPI-2. This subgroup can be further divided into laryngeal hypersensitivity and neurologic disorders. The vast majority of the patients are in the hypersensitivity subgroup. The hypersensitivity stems from mucosal changes with edema due to irritants, dryness, or reflux. This could also be a virus-induced sensory neuropathy lowering the afferent response of a stimulus to produce the characteristic efferent reaction of laryngeal contraction. This is an exaggeration of a normal physiologic response. Medical management needs to be directed at the various medical disorders that are found in this subgroup. Laryngeal control therapy has a role in this group but is not the primary mode of treatment.

Laryngeal-based disorders, such as PVCM, need to be considered in patients with refractory breathing difficulty despite adequate medical management. The use of a standardized protocol will improve the diagnostic capabilities and facilitate identifying those patients with PVCM. The history and laryngeal findings will allow the treatment to be individualized to improve the response to treatment. Patients with PVCM cannot be considered homogeneous. Treatment needs to be directed by categorizing the patients into primary or secondary PVCM, with therapy and medical management directed at the appropriate cause.

BIBLIOGRAPHY


