

## REVIEW

# Paradoxical vocal cord motion disorder: past, present and future

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Paradoxical vocal cord motion disorder (PVCM), also called vocal cord dysfunction, is an important differential diagnosis for asthma. The disorder is often misdiagnosed as asthma leading to unnecessary drug use, very high medical utilisation and occasionally tracheal intubation or tracheostomy. Laryngoscopy is the gold standard for diagnosis of PVCM. Speech therapy and psychotherapy are considered the cornerstone of management of this disorder. The aim of this article is to increase the awareness of PVCM among doctors, highlighting the main characteristics that distinguish it from asthma and discuss the recent medical achievements and the possible future perspectives related to this disorder.

carefully unravel a patient's symptom and pinpoint the disorder in the larynx. The aim of this article is to increase the awareness of PVCM, highlight the main characteristics that distinguish it from asthma and discuss the recent medical achievements and the possible future perspectives related to it.

## HISTORICAL PERSPECTIVE AND NOMENCLATURE

A medical textbook published in 1842 by Dunglison<sup>6</sup> described a disorder of the laryngeal muscles brought on by "hysteria" and named it as "Hysterical croup". In 1869, Mackenzie<sup>7</sup> was the first to visualise the paradoxical movement of the vocal cords during inspiration in patients with stridor using a laryngoscope; he elicited the closure as the cause of the stridor. In 1902, Sir William Osler<sup>8</sup> gave an insightful description of a condition, which he thought to be an unusual disease. He wrote: "spasm of 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". In 1970s, Patterson *et al*<sup>9</sup> described a 33-year-old woman with a history of 15 hospital admissions for what they termed "Munchausen's stridor". During this period the cause for PVCM was considered purely psychogenic and was a topic confined to psychiatric literature; literature that published virtually nothing on the topic until 1980s when Christopher *et al*<sup>10</sup> reported in the *New England Journal of Medicine* their comprehensive evaluation of five patients treated for uncontrollable asthma and described the condition as "vocal cord dysfunction". Since that time manuscripts and abstracts describing >1400 patients with PVCM have been published in the medical literature.<sup>2 11 12</sup> Over the years, this disorder has had numerous labels such as "episodic laryngeal dyskinesia", "Munchausen's stridor", "pseudoasthma", "functional upper airway obstruction", "spasmodic croup", "emotional laryngeal wheezing", "irritable larynx syndrome", "vocal cord dysfunction", etc.<sup>2 13</sup>

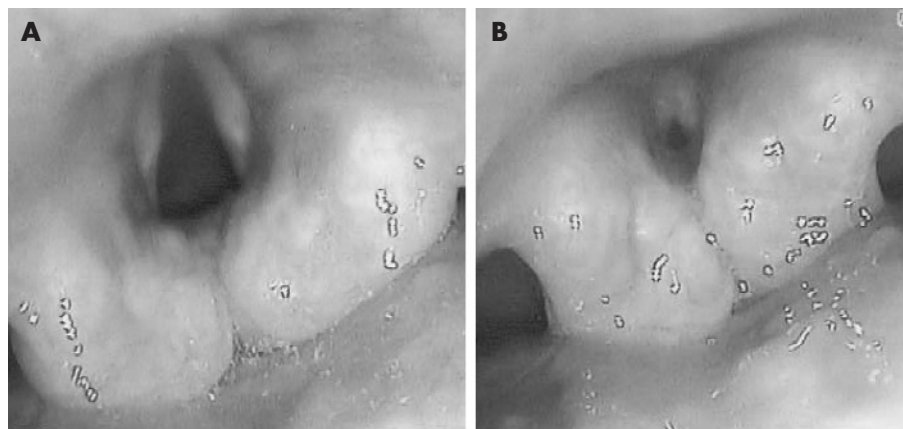
The term "vocal cord dysfunction" was originally selected to identify this very specific clinical syndrome that has defined endoscopic features and mimics asthma. However, there are several additional disorders that are manifested by

Asthma is a common respiratory problem, and doctors are often asked to treat patients with acute onset of wheezing and dyspnoea, suggestive of acute exacerbation. The first question when confronted by a patient with severe asthma-like symptoms despite conventional asthma treatment is: does the patient really have asthma? One of the most important differential diagnoses that has been recognised with increasing frequency over the past 20 years, is paradoxical vocal cord motion disorder (PVCM), also called vocal cord dysfunction. Evidence emphasising the increasing importance of this disorder is highlighted in the National Heart, Lung and Blood Institute guidelines for the diagnosis and management of asthma which discusses PVCM as an important differential diagnosis for asthma.<sup>1 2</sup> PVCM is characterised by paroxysmal periods of vocal cord adduction during inspiration and/or expiration, thereby restricting the airway opening leading to episodic dyspnoea, wheezing and/or stridor that is usually mistaken for asthma (fig 1). It may be precipitated by exercise, intense emotion or may occur spontaneously. The literature is rich with several case reports of PVCM being misdiagnosed as asthma for very long periods of time and many patients have been incorrectly treated for asthma for as long as 15 years.<sup>2-4</sup> The consequences of treating unrecognised PVCM as asthma can be dangerous. In many instances, failing to recognise PVCM has led to unnecessary drug use and hospitalisation. Furthermore, the acute presentation of this condition sometimes dramatically leads to tracheal intubation or tracheostomy.<sup>5</sup> Therefore, it is crucial for doctors particularly those beyond the pulmonary and ENT community to recognise this condition and develop a high index of suspicion to

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**Figure 1** (A) Normal appearance of vocal cords during inspiration. (B) Vocal cords during inspiration in a patient with paradoxical vocal cord motion disorder showing paradoxical adduction and formation of posterior chink.

abnormal vocal cord motion during breathing and confusion will continue if a spectrum of disorders is collectively lumped together as “vocal cord dysfunction”. In an editorial, Christopher<sup>14</sup> emphasised the need to move away from using the term “vocal cord dysfunction” as a catchall descriptor for vocal cord adduction during breathing. We believe that the term “paradoxical vocal cord motion disorder” gives a better and more specific description of the condition, and therefore it will be used throughout this paper.

### VOCAL CORD ANATOMY AND FUNCTION

The larynx is a valve separating the trachea from the upper aero-digestive tract. The Glottis consists of the true and false vocal cords and the opening between them, the rim glottides. The principal muscle for vocal cord abduction is the posterior cricoarytenoid (PCA) muscle. Adduction is performed mainly by the lateral cricoarytenoid (LCA) muscle. During normal inspiration, the glottic opening is controlled through the medullary respiratory centre (via the vagus nerve), which leads to contraction of the PCA muscle and therefore to vocal cord abduction.<sup>15–17</sup> During normal expiration there is a decrease in the tonic activity of the PCA muscle and contraction of the LCA muscle, resulting in a 10–40% narrowing of the rim glottides.<sup>18</sup> This narrowing starts just before expiration and continues for almost 95% of the expiratory phase.<sup>19, 20</sup> Of note; the vocal cords also appear to be sensitive to changes in airway calibre.<sup>17</sup>

### EPIDEMIOLOGY OF PVCMD

The overall incidence of PVCMD in general population is not well defined. Different studies have reported variable incidence of PVCMD. A study of 1025 patients with dyspnoea found an overall 2.8% prevalence of PVCMD.<sup>21</sup> In a cohort study of military patients evaluated for exertional dyspnoea by Morris *et al*,<sup>22</sup> the prevalence of PVCMD was surprisingly higher at 12%, more than what was anticipated. Knowledge about the exact incidence of PVCMD as exercise-induced asthma is lacking, but it is suspected to occur in approximately 3% of intercollegiate athletes having exercise-induced asthma.<sup>23, 24</sup> There is no sport-specific predilection for PVCMD. It has been observed in skiing, swimming, track and cross-country, figure skating, boxing, wrestling, soccer, basketball, softball, football, baseball, racquetball and taekwondo.<sup>23</sup> In a recent retrospective study of 142 subjects with exercise-induced asthma by Abu-Hasan *et al*,<sup>25</sup> 9% were found to have PVCMD. A prospective study of 52 Vancouver school children referred for poorly controlled exercise-induced asthma, found that only 8 (15.4%) children fulfilled the diagnostic criteria for exercise-induced asthma. Of the remainder, 14 (26.9%) patients had PVCMD.<sup>26</sup> The famous 1994 study by the National Jewish Centre for Immunology and Respiratory

medicine<sup>27</sup> evaluated patients diagnosed with refractory asthma and reported findings of PVCMD alone, in up to 10% of patients, while an additional 30% of patients had both PVCMD and asthma. O’Connell *et al*<sup>28</sup> did a retrospective chart review of 164 patients and reported that 20% of all females who underwent rhinolaryngoscopy for any indication were found to have PVCMD.

PVCMD affects mainly children and young adults with average age at diagnosis of 14.5 years in juvenile patients and 33 years in adults.<sup>28, 29</sup> Surprisingly, the disease has also been reported in neonates because of the presence of gastro-oesophageal reflux disease (GORD).<sup>30</sup> There is a reported 2:1 female predominance, with 63.6% of reported cases being females and 36.4% being males.<sup>2</sup>

We believe that PVCMD is largely underreported and the true incidence and prevalence in the general population might be even higher than anticipated. The reason for this belief is that primary care doctors, who are not aware of PVCMD, are caring for many cases of what is thought to be asthma; in addition, many doctors may not consider laryngoscopy to prove or exclude the presence of PVCMD in poorly controlled “asthma” patients.

### PATHOGENESIS OF PVCMD

The exact pathogenesis of PVCMD remains uncertain, however both organic (eg, GORD, irritant-induced) and non-organic (eg, psychological or emotional stress) causes have been identified as precipitating factors. Four pathogenetic mechanisms have been suggested in the literature to underlie PVCMD—namely, laryngeal hyper-responsiveness, altered autonomic balance, direct stimulation of the sensory nerve endings in the upper or lower respiratory tract and hyperventilation.<sup>2</sup>

Laryngeal hyper-responsiveness (analogous to bronchial or nasal hyper-responsiveness in asthma or allergic rhinitis) may underlie vocal cord dysfunction as shown by Bucca *et al*.<sup>31, 32</sup> They found laryngeal hyper-responsiveness in 67% of patients who presented with recurrent episodes of wheeze (without documented asthma) by showing deformation of the changes in the flow-volume loop after a histamine challenge. The inhalation of histamine or methacholine has also been shown to cause laryngeal narrowing in both normal subjects and subjects with asthma.<sup>31</sup>

Inflammatory and irritant stimuli can cause a transient or a long-lasting laryngeal narrowing; that is the laryngeal hyper-responsiveness, which contributes to stridor, transmitted wheezing and breathlessness. In addition, antigen-handling cells can be found in the laryngeal mucosa,<sup>33</sup> which suggests that allergens may be important in the development of laryngeal hyper-responsiveness.<sup>31</sup> A recent editorial suggested

an altered state of autonomic balance, maintained by the activity of more central brain regions as the underlying pathogenesis for PVCCM.<sup>31</sup> These central areas include sites in the medulla, midbrain and the prefrontal cortex that are polysynaptically linked with the larynx. Both true and false vocal cords derive motor innervations from the vagus, while sensory fibres from M3 muscarinic receptors in the laryngeal mucosa pass via the vagus to the medulla. PVCCM often overlaps with the hyperventilation syndrome, where tachypnoea may lead to glottic narrowing due to underlying laryngeal hyper-responsiveness. Underscoring this association, Bucca *et al*<sup>34</sup> correlated hyperventilation with hypocalcaemia and the development of PVCCM in eight female patients during non-isocapnic hyperventilation. Ayers and Gabbott<sup>31</sup> described the most plausible hypothesis regarding pathogenesis of PVCCM. They suggested that PVCCM may be caused by laryngeal hyper-responsiveness initiated by an initial inflammatory insult and resulting in altered autonomic balance, which may be short lived or become persistent. If persistent, subsequent stimuli (such as psychological stresses or cold air) will induce local presynaptic reflexes causing airway narrowing, either at the glottic level or in patients with asthma in the lower airways.

## AETIOLOGY OF PVCCM

### The role of psychogenic factors

The role of psychogenic factors in the aetiology of PVCCM has been elucidated in many case reports and clinical studies. Various names reflecting the importance of psychogenic factors were initially given to PVCCM like “hysteric croups”, “hysterical aphonia”, “Munchausen’s stridor”, etc. Psychiatric consultation for further therapy and consideration of underlying somatoform disorders has also been strongly recommended.<sup>35</sup> Selner *et al*<sup>36</sup> described three patients as having spontaneous onset of symptoms attributed to psychological factors and a psychological evaluation was recommended. Gavin *et al*<sup>37</sup> described 12 patients with PVCCM occurring only at times of anxiety with no relationship to activity or exercise. Newman *et al*<sup>38</sup> reported a previous psychiatric diagnosis in 73% of patient with PVCCM. This observation has also been noted in other studies suggesting a higher incidence of PVCCM in female patients with underlying psychological conditions. Powell *et al*<sup>29</sup> noted that 55% of 20 adolescent females with PVCCM had severe social stresses such as competitive sports. McFadden and Zawadski<sup>39</sup> noted similar findings in describing eight patients with exercise-induced PVCCM, all of whom were elite athletes involved in competitive sports. Prior history of sexual abuse has also been reported to play a part in the development of PVCCM. Freedman *et al*<sup>40</sup> in a retrospective study of 47 cases with PVCCM found 14 patients with a definite history of sexual abuse and 5 patients with suspected childhood sexual abuse. Hypnosis has also been used as treatment for patients with PVCCM with good results.<sup>41</sup> Psychogenic factors are thus considered major precipitating factor for PVCCM.

### Association with asthma

The association between PVCCM and asthma is still unclear and further prospective evaluation is required. PVCCM may present alone or in association with asthma. Newman *et al*<sup>38</sup> found that 56% of the 95 patients fulfilling the criteria of laryngoscopically proved PVCCM, had coexistent asthma. They also noted that patients with PVCCM only (without asthma) were misdiagnosed as asthma and had used prednisone on a regular basis for >4 years before admission. O’Connell *et al*<sup>28</sup> found that 35% of patients with PVCCM have coexistent asthma. Jain *et al*<sup>42</sup> prospectively studied 50 patients who sought emergency room treatment for sudden onset of wheezing and shortness of breath. After being diagnosed with acute asthma, the patients had a spirometry test and laryngoscopy. Five (10%) patients

had classic PVCCM and 6 (12%) patients had concomitant asthma and PVCCM.

### The role of GORD

The role of GORD as an aetiological factor for PVCCM is a good media for future research. There is much speculation for a strong link;<sup>29-43</sup> however, the final story is not in at this time. In a study of 22 patients with PVCCM aged  $\leq 18$  years, Powell *et al*<sup>29</sup> found a high proportion of these patients to show posterior glottic changes typically seen in GORD. Of the 22 patients, 21 (95%; all non-smokers) showed notable arytenoids oedema and interarytenoid oedema or pachyderma on endoscopic examination of the posterior glottis; 36% showed frank interarytenoid pachyderma, which is typically associated with chronic reflux laryngitis.<sup>44</sup> These abnormalities would not be expected in healthy adolescent larynx, and support the association between PVCCM and GORD.

Koufman<sup>45</sup> reported that acid reflux triggered PVCCM in a series of 12 patients whose gastro-oesophageal reflux was confirmed by 24 h dual-probe pH monitoring. Acid-suppression drug and dietary and life-style modifications led to resolution of the PVCCM symptoms within 4 weeks. A study conducted using an animal model by Loughlin *et al*<sup>46</sup> showed that an infusion of hydrochloric acid into the oesophagus to lower the pH to  $\leq 2.5$  could induce laryngospasm by sensitisation of subglottic chemoreceptors through a vagally mediated mechanism. Suttithawil *et al*<sup>47</sup> recently reported an interesting case of a 36-year-old woman with PVCCM whose acute symptoms could be provoked by infusion of 0.1 N hydrochloric acid into her oesophagus and resolved completely when pH was raised by isotonic sodium chloride infusion. PVCCM may represent a low-grade form of laryngospasm in response to irritation from reflux events, which have been documented to cause vocal cord adduction and apnoeas in infants.<sup>44-48-49</sup> The laryngeal irritation associated with GORD may contribute to the bronchial constriction of asthma as well.<sup>50</sup> It is thought that bronchial constriction is a vagally mediated reflex in response to laryngeal irritation.<sup>10</sup> Therefore, the role of GORD in patients with PVCCM and coexistent asthma needs to be carefully elucidated in future studies.

### Irritant-induced PVCCM

Numerous respiratory tract irritants, such as smoke, gases, vapours, dust, airborne pollutants and odours have been linked to the onset of PVCCM. Other irritants include GORD, sinusitis

#### Box 1: Some important differential diagnosis of paradoxical vocal cord motion disorder

Differential diagnosis of paradoxical vocal cord motion disorder

- Asthma
- Anaphylaxis
- Aspiration of foreign body
- Angioedema
- Chronic obstructive pulmonary disease
- Croup
- Epiglottitis
- Extrinsic airway compression
- Laryngomalacia
- Laryngospasm
- Laryngeal tumour
- Spastic dysphonia/laryngeal dystonia



and postnasal drip. Perkner *et al*<sup>43</sup> conducted a case-control study and found 11 cases of PVCMM temporally associated with environmental or occupational irritant exposure. Galdi *et al*<sup>51</sup> reported a case of PVCMM that developed secondary to glutaraldehyde exposure. Bhargava *et al*<sup>52</sup> reported additional case of chlorine-induced PVCMM in a 17-year-old male associated with multiple episodes of swimming. PVCMM was also triggered by different irritants including perfumes, air pollutants and chemical agents in 10 of 27 patients reported by Andrianopoulos *et al*.<sup>53</sup> The exact mechanism of irritant-induced PVCMM is still unclear, however it is postulated that a reflex adduction of the vocal cords, which might be protective, is responsible for the development of PVCMM in these patients. It is recommended that patients who develop respiratory symptoms after irritant exposure should be evaluated for PVCMM.

### Other possible aetiologies for PVCMM

Other possible aetiologies of PVCMM that have been reported in literature include extubation after general anaesthesia for some surgical procedures and following viral upper respiratory tract infection.<sup>3 54-62</sup>

On rare occasions, central neurological diseases like Arnold-Chiari malformation, cerebral aqueduct stenosis, Parkinson-plus syndromes or amyotrophic lateral sclerosis may be associated with PVCMM.<sup>63</sup>

### CLINICAL FEATURES AND DIAGNOSIS OF PVCMM

A common pattern of presentation that is often encountered in the literature is that of a patient reporting shortness of breath and asthma-like symptoms during exercise or intense emotion, which does not respond to asthma drugs. Box 1 shows some disorders that may manifest like PVCMM.

Symptoms associated with PVCMM are listed in (table 1).<sup>38</sup>

As mentioned earlier, patients with PVCMM are usually misdiagnosed as having asthma leading to unnecessary morbidity, including intubation,<sup>38 64-66</sup> tracheostomy,<sup>10 67-69</sup> high-dose corticosteroid use with attendant adverse effects,<sup>27 70</sup> multiple hospitalisations and long-term psychological and behavioural dysfunction.<sup>71 72</sup> A recent retrospective case-control study<sup>73</sup> showed that ambulatory patients with undiagnosed PVCMM have considerably more visits to doctors and multiple prescriptions than patients with moderate persistent asthma, a group known to have high medical utilisation. When inconsistencies with asthma are encountered, the index of suspicion for the diagnosis of PVCMM must be raised to avoid unnecessary and exhaustive testing. Some important presentations should raise the clinical suspicion of PVCMM (box 2).

Some features that help to distinguish PVCMM from asthma are summarised in (table 2).

**Table 1** Symptoms associated with paradoxical vocal cord motion disorder

Symptom	PVCMM (%)	Asthma (%)
Wheeze	80	100
Cough	77	87
Dyspnoea	83	97
Chocking sensation	6	28
Chest pain	12	45
Stridor	18	6
Better with bronchodilators	10	90
Reflux	33	47
Voice changes	29	39
Difficulty in speaking	6	26
Sputum	3	80

PVCMM, paradoxical vocal cord motion disorder.

In the patient's history, the best discriminators appear to be the lack of symptomatic response to bronchodilators and the lack of sputum production. As for stridor, it was reported in patients with PVCMM in 20% of cases, however, it was also reported in control groups with asthma, although not very often. This may be a reflection that wheezing and stridor have the same frequencies and differ only by their timing in the respiratory cycle.<sup>38 74</sup> In contrast to asthma, symptoms of PVCMM can sometimes be abated by distraction.<sup>75</sup> The diagnosis of PVCMM becomes more difficult if it co-occurs with asthma or exercise-induced bronchospasm.<sup>28 38</sup> These patients show signs of asthma but experience intermittent airway obstruction symptoms that are not adequately responsive to the usual regimen of bronchodilators and steroids.<sup>76 77</sup> The physical examination in patients with PVCMM is usually normal when they are not experiencing an acute attack. During symptoms, the high-pitched wheezing produced by the nearly apposed vocal cords is heard all over the chest and is loudest over the larynx or larger airways. However, the wheezing may not be loudest over the larynx or larger airways, may not be high pitched, and often mimics wheezing in asthma.<sup>24</sup> The degree of respiratory distress varies from minimal to severe.<sup>64</sup> A flexed neck posture may be assumed during acute attacks.<sup>78</sup> Despite signs and symptoms of upper airway obstruction or bronchospasm, arterial hypoxemia is usually lacking. Patients have normal oxygen saturation and normal alveolar-arterial gradient.<sup>24 64 72 79</sup> However, the presence of hypoxemia does not completely eliminate the possibility of PVCMM, as it has been reported in association with few cases of PVCMM.<sup>57-59 69</sup>

### Laryngoscopy

Direct visualisation of the vocal cords while the patient is having symptoms is the gold standard for diagnosing PVCMM. The classic findings described initially by Christopher *et al*<sup>10</sup> are the hallmarks for diagnosis. Complete adduction of the vocal cords during inspiration with formation of a small posterior diamond-shaped glottal chink is diagnostic (fig 1). These findings may also be present during expiration. Newman *et al*<sup>38</sup> reported laryngoscopic findings diagnostic of PVCMM in 100% of their patients with symptoms and 60% of their asymptomatic patients. Similarly, Powell *et al*<sup>29</sup> identified true vocal cord adduction during quiet respiration in 55% of their asymptomatic juvenile patients. If the patient is asymptomatic at the time of laryngoscopic examination, having the patient pant, breathe deeply and phonate may sometimes elicit symptoms.<sup>10</sup> During laryngoscopy it is important to assure that the laryngoscope does not come in contact with vocal cords as this may trigger vocal cord adduction.<sup>75</sup> Although Lidocaine seems not to affect the behaviour of the vocal cords during the procedure,<sup>80</sup> it is recommended to avoid local Lidocaine infusion. Benzodiazepines and other sedatives should be avoided during laryngoscopy as they may cause relaxation of the vocal cords. Continuous laryngoscopic examination during exercise is a newly reported technique that may be useful in diagnosing subjects with asymptomatic PVCMM. Heimdal *et al*<sup>81</sup> evaluated the use of continuous laryngoscopy during treadmill exercise in diagnosis of asymptomatic patients with PVCMM. The results seem promising as the test was easy to perform, well tolerated and may serve as an important future tool in diagnosis for patients with respiratory symptoms during exercise.

### Chest radiography

Chest radiographs are not helpful in diagnosing PVCMM or in helping discriminate it from asthma, since they are usually normal in both entities. Hyperinflation and peribronchial thickening raise suspicion of asthma but does not rule out concurrent PVCMM.<sup>23 82</sup>

### Box 2: Clinical features that should raise the possibility of paradoxical vocal cord motion disorder

- Asthma-like symptoms that do not respond to bronchodilators or corticosteroids
- Asthma-like symptoms that are precipitated by stress, emotional factors or anxiety
- Exercise-induced asthma
- Athletes with choking sensation during exercise
- Irritant-induced asthma-like symptoms

#### Airway fluoroscopy

Airway imaging using fluoroscopy may show paradoxical vocal cord motion. It is non-invasive and may provide a rapid means of diagnosing PVCMM; however, it has not been standardised against laryngoscopy, and it cannot rule out organic lesions of the vocal cords.<sup>22 83 84</sup>

#### Pulmonary function tests

Pulmonary function tests with flow volume loops are often used to support the diagnosis of PVCMM. When patients are asymptomatic, the flow volume loop is usually normal. In patients with symptoms, it may show a cut-off or flattening of the inspiratory limb suggestive of extrathoracic obstruction. In patients with expiratory type of PVCMM, reduction in the expiratory limb may also be seen.<sup>85-87</sup> Pulmonary function testing in patients with only PVCMM shows normal total lung capacity without evidence of lung hyperinflation during acute attacks. Although the flow volume loop tends to be normal when subjects are asymptomatic, Newman *et al*<sup>38</sup> reported that 25% of their patients with asymptomatic PVCMM showed the characteristic inspiratory cut-off. Morris *et al*<sup>22</sup> also found abnormal flow volume loops in 20% of patients with PVCMM at rest.

Methacholine challenge test can be misleading and should not be used to distinguish PVCMM from asthma. In addition to the association between PVCMM and asthma reported in literature,<sup>28 38 88</sup> methacholine has been shown to induce acute

attacks in patients with PVCMM. Perkins *et al*<sup>89</sup> in a prospective study showed that methacholine challenge test may cause an acute episode of PVCMM and positive results may not reflect underlying reactive airway disease. Truncation of the inspiratory flow volume loop during methacholine challenge test was not diagnostic for the presence of inspiratory vocal cord adduction. Two patients with normal vocal cord movement at baseline had a reactive methacholine challenge test and evidence of vocal cord adduction after methacholine test.

#### Ultrasound of vocal cords

Use of colour Doppler ultrasound imaging of the vocal cords during attack may become an important tool in diagnosing PVCMM in the future. Ooi<sup>90</sup> suggested that colour Doppler imaging was as accurate as laryngoscopy; however, this requires confirmation by further studies.

#### MANAGEMENT OF PVCMM

##### Therapies with established benefits

##### Reassurance and on-site manoeuvres

A calm reassuring manner has been described in many case reports as an effective method of terminating acute attack of PVCMM.<sup>60 78 91-97</sup> Asking the patient to focus on making a soft "s" sound while exhaling has a dual effect of diverting attention from inhalation while giving auditory feedback on air movement.<sup>23</sup> Panting and coughing have been found to be helpful in acute attacks. Panting is the most practical on-site manoeuvre to abort a PVCMM attack. It activates the PCA muscle (the principal abductor of the vocal cords) and widens the glottic aperture to near maximal diameter.<sup>23 80</sup>

##### Benzodiazepines

Benzodiazepines used to sedate patients and relieve their anxiety, have been shown to be effective in terminating acute symptoms of PVCMM.<sup>2 5 98-100</sup> It is prudent to confirm normal oxygen saturation and exclude hypercapnoea before administering these drugs.

##### Heliox

Heliox is a gaseous mixture of oxygen and helium in various ratios. Because heliox is less dense than air, inhalation reduces turbulence in the airway and eliminates respiratory

**Table 2** Features distinguishing paradoxical vocal cord motion disorder from asthma

	PVCMM	Asthma
Incidence	Less common	More common
Age and sex	Young, female	Any
Triggers	Usually exercise or emotional stress	Many triggers
Psychological factors	Important role	Less important role
History of allergy	Usually absent	May be present
Family history	Usually absent	May be present
Sensation of tightness	Throat	Chest
Inspiratory stridor	More common, heard loudly over larynx	Rare
Sputum production	Rare	Common
Nocturnal awakening with symptoms	Rare	Common
Response to bronchodilators	No response	Good response
Hypoxemia	Rare	Common
Eosinophilia	Rare	Common in allergic asthma
Chest radiograph	Usually normal	May show hyperinflation and peribronchial thickening
Residual volume and Total lung capacity	Normal	May be increased
Flow volume loop	Flattening of inspiratory loop	Obstructive pattern
Bronchial provocation test	May be positive	Usually positive
Laryngoscopy	Inspiratory adduction of the anterior 2/3 of vocal folds with posterior chink	Usually normal

PVCMM, paradoxical vocal cord motion disorder.

noise.<sup>23 101 102</sup> It provides short-term relief from dyspnoea in patients with PVCMM and other forms of upper airway obstructive disease.

### Nebulised Lignocaine

Nebulised lignocaine has been used in one centre with good symptomatic relief.<sup>103</sup> A trial of nebulised lignocaine may be used for acute attack.

### Speech therapy

Speech therapy is regarded as the cornerstone of treatment for PVCMM. The primary role of speech therapy is to teach the patient with PVCMM how to control the laryngeal area and maintain an adequately open airway during respiration. The role of speech therapy extends to assessment, diagnostic input, patient education, supportive counselling, PVCMM symptom management and possible voice therapy.<sup>10</sup> Sullivan *et al*<sup>104</sup> found that 95% of patients with PVCMM treated with speech therapy were able to control their symptoms. Three to four sessions with a trained therapist are usually required for a patient to feel confident in the techniques, which must be practiced during asymptomatic periods to ensure success during attacks.<sup>23</sup>

### Psychotherapy and hypnosis

Psychotherapy remains an important mode of treatment in patients with PVCMM despite it not having been studied systematically. There are many forms of psychotherapy used in PVCMM, which include relaxation therapy to alleviate distress associated with symptoms, identification of stressors that contribute to PVCMM and development of new coping strategies for dealing with those stressors. Family therapy may also be indicated.<sup>10</sup> Hypnosis induces relaxation and an altered state of awareness and uses imagery, both specific and non-specific; to achieve a state of well-being that helps to resolve the PVCMM. Teaching patient to perform a self-hypnosis technique can be helpful.<sup>23 41 105 106</sup>

### Biofeedback

Biofeedback may be used in conjunction with psychotherapy for treating patients with PVCMM. Nahimas *et al*<sup>107</sup> have used biofeedback in 5 out of 15 female patients with "laryngeal dyskinesia", there was a reduction in the midmaximal flow rate in 2 patients and normalisation in the remaining 3 patients. Warnes and Allen<sup>108</sup> evaluated the effectiveness of electromyography biofeedback in a 16-year-old girl and showed an overall baseline muscle tension reduction by >60% with corresponding reduction in episodes of respiratory distress and chest pain.

### Therapies with possible benefits

#### Inhaled anticholinergic drugs

A recent retrospective study<sup>109</sup> to determine the long-term outcome of PVCMM in 49 patients found that anticholinergic aerosol in form of Ipratropium bromide was successful in preventing the exercise-induced symptoms when used before the activity. The rationale of using an anticholinergic agent to prevent exercise-induced PVCMM is also found in two case reports<sup>110 111</sup> that describe prolonged stimulation of the vagal nerve by vagal nerve stimulators, used in patients with intractable seizures, causing PVCMM as a complication. An editorial<sup>31</sup> has also suggested an altered autonomic balance as a cause of PVCMM, since true and false vocal cords derive motor innervation from the vagus nerve. Anticholinergic drugs may be a safe and effective measure for treating exercise-induced PVCMM and warrant a double-blind, placebo-controlled clinical trial.<sup>12</sup>

### Learning points

- Paradoxical vocal cord motion disorder (PVCMM) is an important differential diagnosis of asthma, that is widely unrecognised by many doctors
- If misdiagnosed as asthma, PVCMM can lead to high medical utilisation, unnecessary high-dose steroid use and other dangerous consequences
- Suspect PVCMM in patients with asthma-like symptoms that do not respond to conventional asthma therapy or induced by exercise or stress
- The gold-standard test for diagnosis of PVCMM is direct visualisation of the vocal cords by laryngoscopy
- The term "vocal cord dysfunction" has become a very loosely applied descriptor of the disorder and should be changed

### Inspiratory valve

Archer *et al*<sup>112</sup> developed a new approach for the treatment of inspiratory PVCMM by using a device in the form of a mask with a one-way adjustable pressure valve. The patient is asked to fix the mask to the face. The pressure through the valve is adjusted until an increase in resistance to inspiration occurs and the stridor cannot be heard. It is possible that the mask works for a variety of reasons: (1) by decreasing the inspiratory rate of airflow, the patient is not passing as much air through the vocal cords and therefore is unable to wheeze, (2) by decreasing the rate of airflow hitting the vocal cords, the cords are not stimulated to adduct abnormally and (3) since the patients are unable to hear themselves wheeze they do not feel as distressed, and therefore the spiral of distress causing more wheeze is reversed and the patients feel calmer. The use of this valve seems simple; however, its effectiveness in treating patients with PVCMM requires further investigation.

### Continuous positive airway pressure

Only few case reports have described the use of continuous positive airway pressure (CPAP) to relieve acute symptoms of PVCMM. Goldman and Muers<sup>113</sup> suggested that CPAP is effective when there is vocal cord adduction during expiration. They assumed that CPAP relieves dyspnoea by slowing the expiratory flow, thereby increasing lung volume, which in turn results in a more open glottis. CPAP is also administered for the more typical PVCMM where there is glottic closure on inspiration. In

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this case, CPAP effectiveness is attributed to reducing the effort needed for inspiration by establishing a favourable pressure gradient for inhalation.<sup>10-66</sup> Lloyd and Jones<sup>114</sup> reported a case of PVCMM with stridor that was successfully treated with intermittent CPAP and heliox.

### Botulinum toxin

Botulinum toxin A acts at nerve terminals to prevent release of acetylcholine, resulting in chemical denervation. Botulinum toxin A is particularly useful in the treatment of dystonias, such as blepharospasm, spasmodic torticollis or spasmodic dysphonia.<sup>115</sup> More recently its use has been reported in isolated cases of PVCMM.<sup>116-119</sup> Maillard *et al*<sup>5</sup> described the successful use of botulinum toxin in treating a woman with severe PVCMM who required intubation and tracheostomy on several occasions.

The effect of botulinum toxin may last up to 14 weeks.<sup>117</sup> Its use may be advocated for severe cases of PVCMM that do not respond to conventional therapy.

### Prognosis of PVCMM

Disparate results have been reported in the literature about the prognosis of PVCMM. The long-term outcome of PVCMM is still unknown. Data from a recent retrospective study indicated that PVCMM is generally a self-limiting disorder with most patients having no long-term sequelae, once the diagnosis has been established.<sup>109</sup> In contrast, a 10 year follow-up of three patients with PVCMM failed to show improvement in their condition despite trials using various methods of treatment.<sup>72</sup> The presence of these contradictory results makes a prospective study of the long-term outcome of PVCMM a high priority.

### Prospects for the future

It has been >20 years since Christopher *et al*<sup>10</sup> published their landmark description of five patients with what they named as "vocal cord dysfunction". Since then, the term vocal cord dysfunction has been used widely despite a recent recommendation by Christopher<sup>14</sup> to change this name; he suggested that the term "vocal cord dysfunction" has become a very loosely applied descriptor for inspiratory or isolated expiratory vocal cord adduction. In addition, there are several additional diseases or disorders that are manifested by abnormal vocal cord motion during breathing. Christopher<sup>14</sup> recommended to move away from using the term "vocal cord dysfunction". Firstly, we feel that the term "paradoxical vocal cord motion disorder" may be a better description for the condition. Secondly, we need to improve the knowledge and awareness among doctors. PVCMM is usually mistaken for asthma with the consequence of unnecessary steroid use and a considerable negative effect on healthcare utilisation. We suggest that health departments, academic institutions and medical professional organisations should provide continuing education about PVCMM as important differential of asthma to their constituent healthcare providers. Thirdly, a double-blind, placebo-controlled trial using the anticholinergic "ipratropium" aerosol to prevent acute attacks of PVCMM is needed to confirm the findings of a recent retrospective study.<sup>109</sup> This will not only advocate the use of a cheaper drug but may also give a clue to the underlying pathogenesis responsible for PVCMM (the altered autonomic balance).<sup>12</sup> Prospective study is needed to compare colour Doppler ultrasound of the vocal cords with laryngoscopy for diagnosis of PVCMM. It is a non-invasive technique and has been shown in one case report to be as effective as laryngoscopy.<sup>90</sup> Another area of fertile research is the link between PVCMM and GORD. GORD has been proposed as a possible aetiology for PVCMM. As we mentioned above, the link seems strong, however further research is needed. We strongly recommend that GORD as an aetiological factor for PVCMM to

be scheduled as a research topic in future trials on PVCMM. Finally, the long-term outcome of PVCMM has not been discussed in prospective studies. What is now available in terms of a long-term prognosis, is a collection of contradictory case reports and small retrospective trials. It is important, therefore, to discuss this issue in a future prospective trial.

### MULTIPLE-CHOICE QUESTIONS (TRUE (T)/FALSE (F); ANSWERS AFTER THE REFERENCES)

- About paradoxical vocal cord motion disorder:
  - It is an important differential diagnosis of asthma
  - If unrecognised can lead to high medical utilisation
  - Stridor is the most common presentation
  - It is a purely psychogenic disorder
- In which of the following patients, should PVCMM be suspected:
  - Young patient with exercise-induced asthma
  - Young patient whose asthma does not respond to steroids
  - Patient with mainly nocturnal asthma
  - Patient with asthma symptoms precipitated by emotional stress
- The following are recognised associations with PVCMM:
  - Asthma
  - Gastroesophageal reflux disease
  - Psychological disorders
  - Exposure to irritants
- The following presentations are more in favour of PVCMM than asthma
  - Sputum production
  - Nocturnal symptoms
  - Presence of family history
  - Poor response to steroids
- Which one of the following is considered the gold standard test for diagnosis of PVCMM:
  - Pulmonary function test
  - Chest radiograph
  - Laryngoscopy

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## ANSWERS

1. (A)T (B)T (C)F (D)F; 2. (A)T (B)T (C)F (D)T; 3. (A)T (B)T (C)T (D)T; 4. (A)F (B)F (C)F (D)T; 5. (A)F (B)F (C)T

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