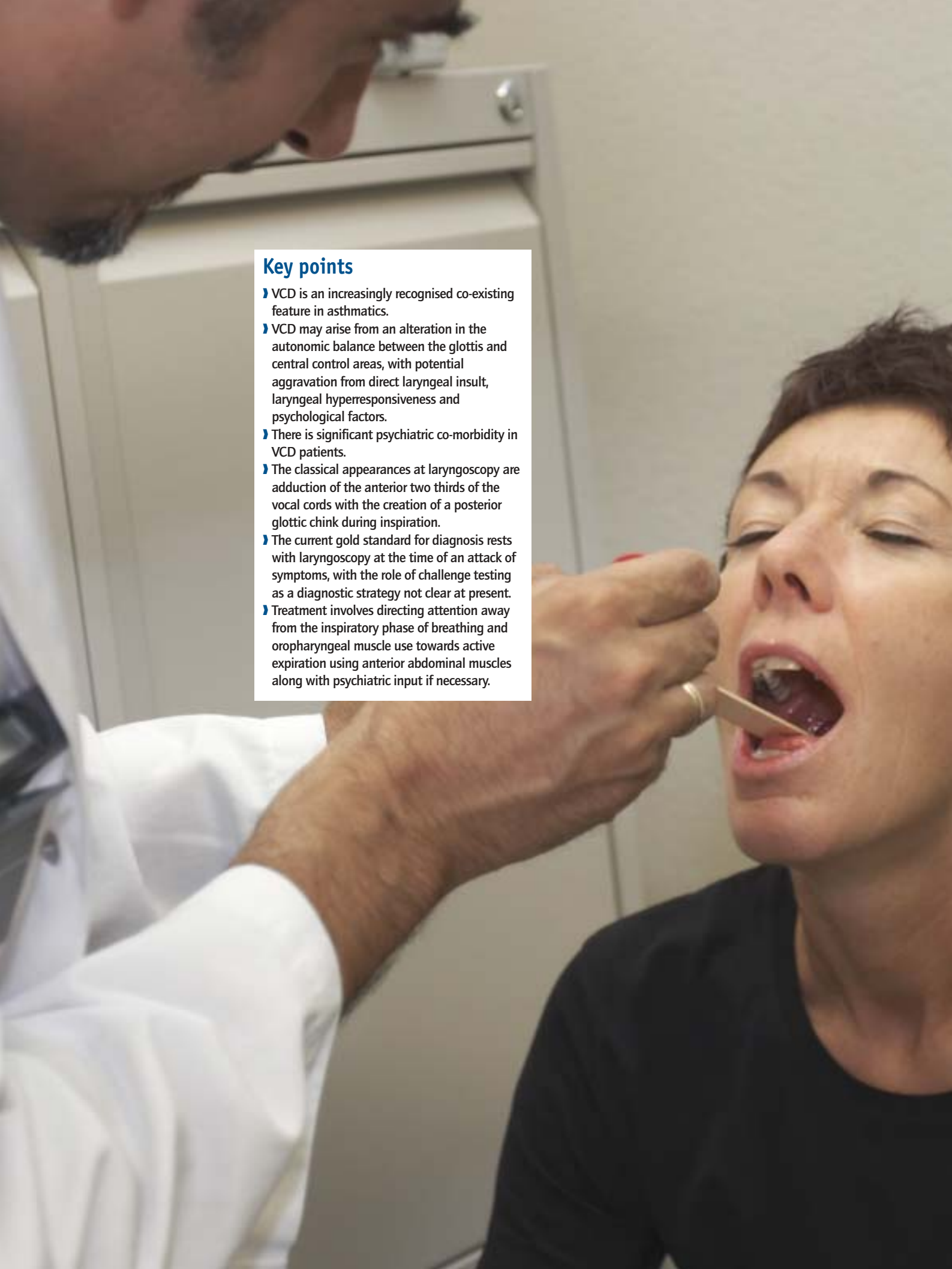

Key points

- VCD is an increasingly recognised co-existing feature in asthmatics.
- VCD may arise from an alteration in the autonomic balance between the glottis and central control areas, with potential aggravation from direct laryngeal insult, laryngeal hyperresponsiveness and psychological factors.
- There is significant psychiatric co-morbidity in VCD patients.
- The classical appearances at laryngoscopy are adduction of the anterior two thirds of the vocal cords with the creation of a posterior glottic chink during inspiration.
- The current gold standard for diagnosis rests with laryngoscopy at the time of an attack of symptoms, with the role of challenge testing as a diagnostic strategy not clear at present.
- Treatment involves directing attention away from the inspiratory phase of breathing and oropharyngeal muscle use towards active expiration using anterior abdominal muscles along with psychiatric input if necessary.

Vocal cord dysfunction

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Educational aims

- To be aware of the possible co-existence of VCD in patients with poorly controlled asthma.
- To recognise the various clinical presentations of VCD.
- To be aware of the unanswered questions regarding the pathophysiology of VCD.
- To be aware of treatment strategies for VCD.

Summary

Recently, there has been increasing recognition and understanding of vocal cord dysfunction (VCD), a condition characterised by abnormal adduction of the vocal cords, most often during inspiration, leading to airway obstruction and associated symptoms. VCD is recognised as a co-existing factor or an alternative diagnosis in patients with asthma, but the true prevalence and incidence are not entirely clear. VCD may arise from interrelationships between laryngeal hyperresponsiveness and autonomic imbalance. Diagnosis can be difficult and, although challenge testing may be helpful, there is no non-invasive alternative to direct visualisation of the larynx during an attack as a current gold standard. This review aims to summarise current understanding of the epidemiology, proposed underlying pathophysiological mechanisms, diagnosis and treatment of this disorder.

Also previously termed paroxysmal vocal cord motion, paroxysmal vocal cord dysfunction, paradoxical movement of vocal cords, episodic paroxysmal laryngospasm and irritable larynx syndrome [1], a condition that continues to present both diagnostic and therapeutic dilemmas to pneumonologists is best brought under the term vocal cord dysfunction (VCD). In this condition, there is abnormal adduction of the vocal cords during the respiratory cycle, leading to variable airflow obstruction and symptoms mimicking asthma.

Epidemiology

True population figures for incidence and prevalence of VCD are not known. However, in a group of 1,025 patients evaluated for exertional dyspnoea, 29 (2.4%) were found to have VCD [2]. In a smaller study of 105 army recruits evaluated for dyspnoea, 10 (9.5%) were found to have VCD [3].

The incidence in patients with asthma has been explored in more detail, but still remains unclear. One tertiary referral centre evaluated patients with refractory asthma and found 22 out of 132 (16.7%) had VCD in addition to asthma [4]. In one of the largest case series of 95 patients with VCD, 53 also had asthma [5]. This case series also suggested a high incidence of psychiatric problems in patients with this condition. In the 42 patients with pure VCD, nine had experienced psychiatric hospitalisations, 73% had a major psychiatric disorder and 37% had a personality disorder. Some 38% of these patients also had a history of

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sexual, physical or emotional abuse. Similar degrees of psychiatric morbidity were found in those with VCD and asthma. CHRISTOPHER *et al.* [6] proposed that VCD is a form of conversion disorder, a view supported by a small series by SELNER *et al.* [7]. This was not, however, confirmed by later work [8, 9]. The series by NEWMAN *et al.* [5] and a further series of 22 patients [10] both suggest that this is typically a condition of younger females. Other case reports implicate occupationally inhaled irritants [8], child abuse [11], brain-stem compression [12], cystic fibrosis [13], working in healthcare [5, 14] and gastro-oesophageal reflux [15].

Pathophysiology

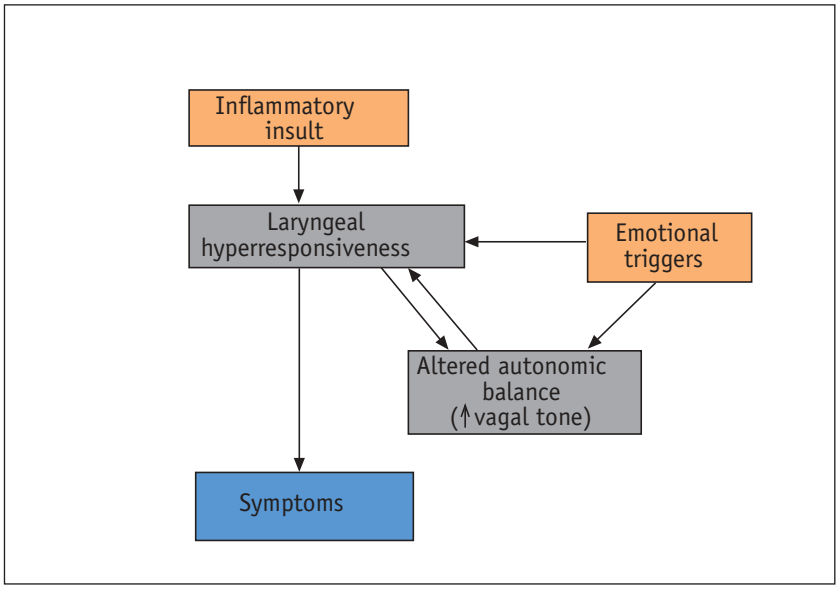
There is no clear consensus on how this condition arises. First of all, the innervation of the larynx must be considered. Sensory information is transmitted *via* the vagus nerve to the medulla. A variety of other factors, such as stress, emotion and ambient temperature, also input to this part of the central nervous system. These may influence the motor outflow, also *via* the vagus. Consequently, a baseline autonomic balance can be said to exist [16]. AYRES and GABBOTT [16] have previously proposed that this can become "imbalanced" by either laryngeal hyperresponsiveness (initiated by some form of inflammatory insult) or perhaps from a central stimulus, such as ill-defined psychological factors (see figure 1). MORRISON *et al.* [17] proposed a similar mechanism, whereby the threshold for stimulating glottic spasm is lowered by chronic irritation of the larynx by gastro-oesophageal reflux. Such an imbalance may favour adduction of the vocal cords.

There remains the possibility that more than one of these factors may exist in any one patient and that, depending on the persistence or degree of such factors, a vicious cycle encouraging persistence may be created. The ultimate result is of abnormal adduction of the vocal cords with creation of a characteristic posterior glottic "chink" visualised at laryngoscopy [5, 6, 14]. This occurs during the respiratory cycle leading to upper airway obstruction and symptoms. This most commonly occurs during inspiration, but can also occur during expiration in addition or in isolation [18–20]. Due to the variable nature of the factors described above, the symptoms are also variable.

If laryngeal hyperresponsiveness is the basis of VCD, what provides the initial insult? This may be due to upper airway hyperresponsiveness occurring in association with the lower airway hyperresponsiveness of asthma. BUCCA *et al.* [21] have demonstrated that histamine provocation testing can produce extrathoracic (upper) airway narrowing as measured by a 25% decrease in mid-inspiratory flow, in 25–40 patients with episodic breathlessness with wheeze and/or cough. This extrathoracic airway hyperresponsiveness (EA-HR) was observed with or without lower airway bronchial hyperresponsiveness (BHR), as measured by a 20% fall in forced expiratory volume in one second (FEV_1) in response to histamine. Although laryngoscopy was only performed in seven out of the 25 patients in the study, it is interesting that, in addition to mucosal oedema and pharyngoconstriction, adduction of the vocal cords during forced inspiration was seen in all seven of these patients. Five out of the 15 patients who had no evidence of EA-HR also had laryngoscopy, but with normal findings. This study did not state how many patients had asthma and so it is unclear if EA-HR was a phenomenon distinct from or part of the spectrum of asthma. The same group later showed that isolated EA-HR was responsible for asthma-like symptoms in 117 out of a larger sample of 441 patients, but laryngeal examination was not undertaken in this study [22]. EA-HR occurred in association with BHR in a further subgroup of 179 patients in this study. This raises the possibility that a reflex can be triggered by stimulation of pharyngo-laryngeal receptors independent of the lower airways, and this is supported by the finding of EA-HR in 72% of patients with sinusitis [23].

Other workers have suggested that stimuli,

Figure 1
VCD pathogenesis.

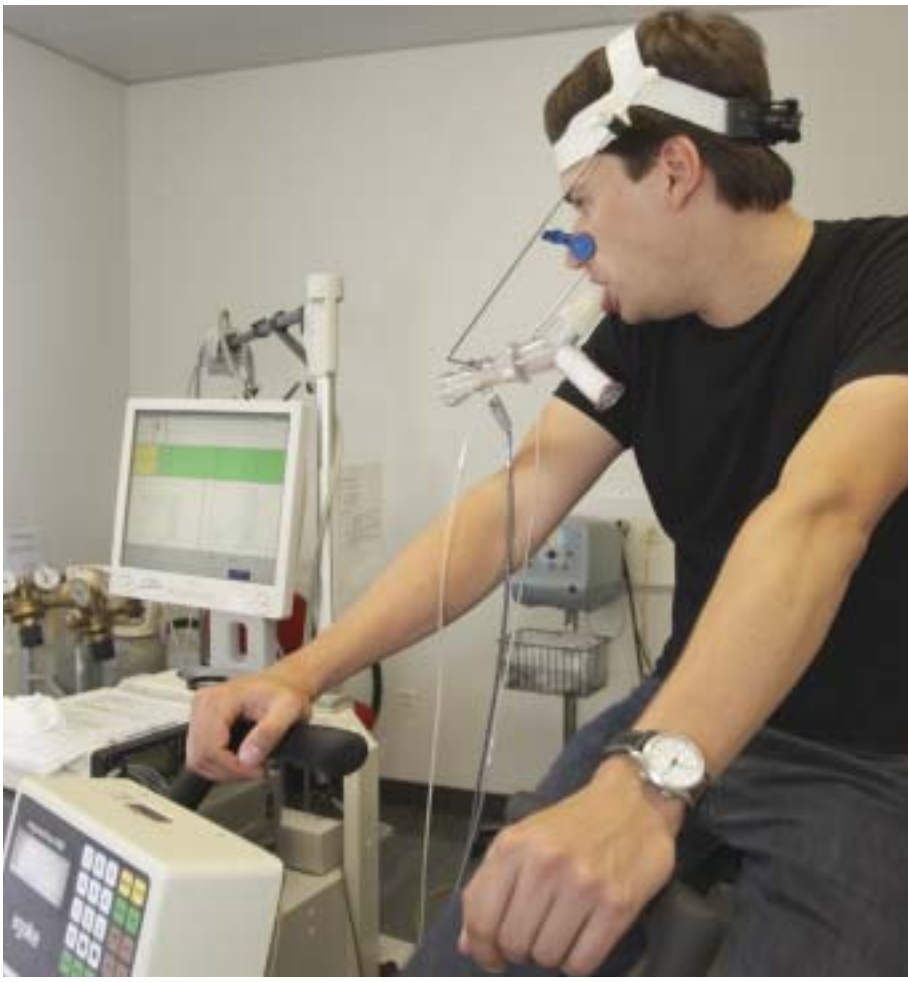


such as acid reflux or inhaled irritants, could initiate or contribute to laryngeal hyperresponsiveness. PERKNER *et al.* [8] described nine patients from a cohort of 127 VCD patients with symptoms relating to irritant exposure to ammonia and fumes from cleaning fluids. In this same group, 28 had symptomatic gastro-oesophageal reflux disease (GORD), but this was not defined objectively. In the cohort of a study by POWELL *et al.* [10], 19 out of 22 patients had laryngoscopic changes suggestive of reflux disease. The relationship of GORD and, perhaps even more importantly, laryngopharyngeal reflux disease to VCD has not been prospectively evaluated.

Clinical features

CHRISTOPHER *et al.* [6] used the term VCD to describe five patients with dramatic episodes of wheezing, previously thought to have asthma. Further investigation demonstrated no objective evidence of asthma, but each had marked flattening of the inspiratory limb of a flow–volume loop and characteristic laryngoscopic abnormalities. Other case reports [24–30] and the large series mentioned above have described the clinical features of VCD in detail. The patient may complain of wheezing, "noisy breathing", stridor, dyspnoea, cough or throat tightness. VCD has been shown to account for "choking" during athletic activities in patients previously felt to have exercise-induced asthma [31]. As a result of these symptoms, asthma is commonly misdiagnosed, as several case reports describe, and the patients may have been on long-term high-dose steroids resulting in a Cushingoid appearance. Other clues to the diagnosis are inspiratory stridor heard over the trachea, the absence of typical asthma features (in particular BHR) and lack of response to conventional asthma therapy. If a patient is ventilated for presumed severe asthma and found to have normal inflation pressures, this would also suggest a diagnosis of VCD.

Examination may be unhelpful. Careful listening to a patient's breathing may reveal inspiratory stridor rather than expiratory wheeze, but the timing of abnormal vocal cord adduction can be inspiratory or expiratory. Similarly, pronounced inspiratory noise heard by auscultating over the trachea may help in some cases. Presumably, as a result of airflow obstruction, hypoxia can rarely occur, and, in some cases, may lead to intubation and mechanical ventilation [27].



Diagnosis

Perhaps the most significant problem in the diagnosis of VCD lies in the episodic nature of symptoms. Visualisation of the cords, with characteristic adduction of the anterior two thirds and creation of a posterior glottic chink during inspiration and/or expiration, must be regarded as the gold standard for diagnosis [5, 8]. In between attacks, the cords may be normal. Other diagnostic tests detailed below can be helpful in suggesting VCD as a diagnosis.

Spirometry

Measurements of FEV_1 and forced vital capacity are likely to be normal unless lower airway obstruction is present. FEV_1 is not a sensitive measure of extrathoracic airway narrowing [32].

Measurement of flow–volume loops is more helpful if VCD is present. Truncation of the inspiratory limb is characteristic (although not specific for VCD), resulting in a mid-expiratory flow/mid-inspiratory flow (MEF_{50}/MIF_{50}) ratio

exceeding 1.5 [33]. The flow–volume loop may only be abnormal in about a fifth of asymptomatic patients [5]. An atypical expiratory limb with abrupt drop and rise has been described previously [28, 34], presumably due to expiratory VCD.

Estimation of MIF_{50} is a more scientific method of measuring extrathoracic airflow obstruction and, in a small sample of patients, has been shown to correlate well with mid-inspiratory glottis area measured laryngoscopically [21].

Specific challenge testing

Given the episodic nature of symptoms, if a particular precipitant can be identified, it would seem logical to attempt provocation testing to aid diagnosis. SELNER *et al*. [7] reported reproduction of symptoms with cooked corn in one VCD patient, but also with placebo during food challenge in another patient initially felt to have symptoms related to egg products. In this latter patient, methacholine also produced stridor. In the description of irritant-associated VCD by PERKNER *et al*. [8], these were all diagnosed by laryngoscopy within 24 hours of exposure, but no formal challenge tests were subsequently performed.

Bronchial provocation tests

Methacholine and histamine are bronchoconstrictors that act directly on bronchial smooth muscle. The ability of histamine challenge to detect extrathoracic airway hyperresponsiveness has been discussed. It seems simplistic, however, to presume that upper airway obstruction demonstrated in this way will always be due to VCD. In a cohort in the study by NEWMAN *et al*. [5], methacholine challenge testing (MCT) induced VCD in nine out of 12 subjects with normal laryngoscopy. MORRIS *et al*. [3] demonstrated changes in inspiratory limbs of flow–volume loops in four out of 10 VCD patients with MCT, but did not correlate these findings with laryngoscopy. There has only been one prospective evaluation of MCT in the diagnosis of VCD. In this study [35], 10 known VCD patients, 12 patients with exercise-induced asthma (EIA) and 12 controls underwent laryngoscopy before and after MCT challenge testing. The findings in the 10 known VCD patients were as follows: two had VCD changes before and after MCT; two had VCD changes induced by MCT; six had no VCD changes, but three demonstrated truncation of the inspiratory limb of the flow–volume loop, suggesting extrathoracic airway hyperresponsiveness. In addition, seven out of the 10 patients had BHR with MCT. None of the control group or EIA patients developed VCD post-MCT, although one EIA patient developed inspiratory flow–volume loop flattening with MCT. This study highlights the importance of correlating any flow–volume loop abnormalities with laryngoscopic appearances.

Exercise testing

Case reports have described VCD in association with exercise [36, 37]. MCFADDEN *et al*. [31] described seven elite athletes who developed VCD during sporting competitions. Attempts were made to recreate symptoms by exercise testing. This was only successful in three out of seven patients (two on a treadmill and one by bicycle ergometry), with three further patients being examined after their individual sporting activity and the remaining patient positive by hyperventilation testing. The diagnosis was made by laryngoscopy in only three of these patients, with the flow–volume loop used in the others. Interestingly, MCT did not provoke symptoms in any of these patients. In the same study previously quoted by MORRIS *et al*. [3], 40 patients and 12 controls were evaluated for exertional dyspnoea. Progressive cardiopulmonary exercise testing with pre- and post-test laryngoscopy was performed in all patients. Two patients had evidence of VCD pre- and post-exercise. Exercise provoked VCD in a further eight patients, with the remaining 30 patients and 12 controls having no evidence of VCD. It is not clear how many of these patients had asthma, although six of the VCD patients had BHR on MCT.

More recently, the use of continuous transnasal laryngoscopy during exercise has been described in the diagnosis of exercise-induced laryngeal dysfunction in a group of patients predominantly with laryngomalacia, rather than VCD [38]. This technique may be helpful to clarify the role of challenge testing in the diagnosis of VCD in future studies.

Forced oscillation technique

The forced oscillation technique (FOT) uses small oscillating forces in the form of sound waves to measure the impedance (the opposition to flow of these forces) of the respiratory system. The input is usually applied *via* a mouthpiece through which the subject performs tidal breathing. Impedance and, hence, resistance are calculated from the mouth pressure and flow after the effects of breathing have been removed by signal processing. Its merits include the rapid acquisition of

data and that it does not require maximum effort manoeuvres [39]. It is, therefore, easier for some patients to perform than routine pulmonary function tests which involve forced manoeuvres. This may be particularly relevant for patients with suspected upper airway symptoms who anecdotally often have difficulty with these.

RIGAU *et al*. [40] mimicked VCD in a model using variable resistance to simulate normal respiratory anatomy and found that the changes in oscillatory resistance were in agreement with the degree of area reduction in the model.

There are no published data on this technique in the clinical setting of VCD. As with changes in the inspiratory limb of the flow–volume loop, changes in resistance will not be specific for obstruction at the cords, but rather more of a reflection of upper airways obstruction in general.

Other methods of diagnosis

One case report [41] demonstrated VCD by means of airway radiographs and fluoroscopy in a patient where laryngoscopy was not performed.

Another [42] described abnormalities in multidimensional voice programme analysis, whereby VCD patients had differences in soft phonation indexes compared with normal subjects. One case of VCD has been reported under hypnotic suggestion [43].

Treatment

There are no randomised controlled trials of any form of treatment for VCD. Evidence is limited to case reports and series describing the course of the condition.

The patients from the series by CHRISTOPHER *et al*. [6] were treated by a speech pathologist. They were taught to focus attention away from the larynx and inspiratory phase of breathing. Instead, they were taught to concentrate on active expiration using anterior abdominal muscles and to relax oropharyngeal muscles. Short-term psychotherapy was also administered to these patients and they all experienced a reduction in frequency and severity of attacks. The importance of thorough psychological assessment has also been emphasised [7], and the prevalence of psychiatric morbidity has been discussed above [5, 16]. This may not be appropriate in all cases and may be counterproductive in patients who have been dismissed as "mad" by doctors previously. Together with speech therapeutic strategies similar to those used for treatment of other voice disorders (such as laryngitis, hoarseness), NEWMAN *et al*. [5] has emphasised the importance of cessation of unnecessary medications, as patients misdiagnosed as having asthma may often have been prescribed significant doses of inhaled or oral corticosteroids resulting in side-effects. POWELL *et al*. [10] also treated his group with speech therapy and psychological counselling, as well as raising interesting questions about the role of anti-reflux therapy in cases where this can be implicated. More recently, SULLIVAN *et al*. [44] reported success of a speech therapy programme in 20 adolescent female athletes with VCD.

In the acute setting, a mixture of helium and oxygen (heliox) has been described as beneficial. WEIR [30] detailed dramatic results in four VCD patients. The mechanism of action of heliox is likely to relate to the low density of such a gas mixture, allowing easy movement of air through the adducted cords [45]. LISBOA *et al*. [46] found varying degrees of increased inspiratory resistance in asthmatics compared with normal, which was corrected by breathing heliox. Such a benefit from heliox has also been described in other patients with fixed upper airway obstruction [47].

Other therapies for which there is anecdotal evidence of benefit include intralaryngeal injection of botulinum toxin [48] and a portable facemask with adjustable resistance to inspiration but not expiration [26].

Conclusions

Recognition and description of VCD have improved over the last two decades. Pneumonologists are more aware of the possibility of VCD underlying or mimicking poorly controlled asthma and will consider the diagnosis in others with atypical asthma-like symptoms. Visualisation of the cords during an attack of symptoms is the current gold standard for diagnosis. It may be that VCD represents one end of a spectrum of "upper airways dysfunction" in patients who have extrathoracic hyperresponsiveness with or without associated asthma. Important questions remain regarding epidemiology within the general and asthmatic populations, as well as the pathological mechanisms underlying VCD.

Educational questions

1. In the epidemiology, pathophysiology and presentation of VCD, which of the following statements are true?
   a) The prevalence of VCD in the general population is 1 per 1,000.
   b) All patients with VCD have an underlying psychiatric disorder.
   c) Patients with VCD frequently present with episodic dyspnoea and stridor.
   d) Laryngopharyngeal acid reflux may provide the initial stimulus for VCD.
   e) VCD occurs exclusively in asthmatics.
2. In the diagnosis and treatment of VCD, which of the following statements are true?
   a) The flow–volume loop is frequently normal in between attacks.
   b) Challenge testing with methacholine will unmask VCD in all cases.
   c) The current gold standard for diagnosis rests with visualisation of the cords during an attack.
   d) There are large controlled clinical trials to support the use of speech therapy for the treatment of VCD.
   e) The use of heliox in the acute setting of VCD has been found to be helpful.

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Suggested answers

1. c and d
2. a, c and e