



Work-related laryngeal syndromes

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Purpose of review

This review summarizes recent literature regarding the association of nonorganic laryngeal dysfunction with occupational exposures. Laryngeal dysfunction may masquerade as asthma and is an important consideration in patients with work-associated respiratory symptoms.

Recent findings

Although there is lack of consensus regarding clinical features, vocal cord dysfunction (VCD) is the most well appreciated form of nonorganic laryngeal dysfunction. There are significant gaps in the literature regarding the occupational epidemiology of laryngeal dysfunction, however, occupational exposures such as upper airway irritants may be associated with the onset of symptoms. Recurrent work-associated laryngeal dysfunction has been described in occupational groups including the military and professional athletes. Recent theories have considered that VCD may be a state of laryngeal hyperresponsiveness associated with both intrinsic and extrinsic factors.

Summary

Laryngeal dysfunction is an important consideration in patients with work-associated respiratory symptoms. Clinicians should have a high index of suspicion, in particular, if symptoms are associated with exposure to a respiratory irritant. Situations of high psychological stress may also be associated with recurrent symptoms. There is a requirement for evidence-based guidelines for the diagnosis and management of laryngeal dysfunction, which should also address work-related factors.

Keywords

laryngeal dysfunction, vocal cord dysfunction, work-related asthma

INTRODUCTION

Recently, there has been growing recognition of the importance of laryngeal dysfunction, specifically vocal cord dysfunction (VCD), as an important cause of episodic respiratory symptoms, and as a condition which may masquerade as asthma [1[•]]. Workplace exposures have been reported as precipitants for the development of nonorganic laryngeal dysfunction and also as triggers for recurrent symptoms [2–4]. Work-related asthma is the most common occupational lung disease in developed countries and as laryngeal dysfunction may be difficult to clinically differentiate from asthma, it is important to consider this diagnosis as a cause of work-related respiratory symptoms [5,6].

VCD is likely to be the most well appreciated manifestation of nonorganic dysfunctional laryngeal behaviour [7]. The larynx is a complex structure responsible for maintaining an airway to the lower respiratory tract, protection of the lower respiratory tract and phonation [2]. Protection of the lower respiratory tract following exposure to irritant inhaled stimuli involves the involuntary cough reflex and the laryngeal/glottic closure reflex.

Inappropriate laryngeal closure by involuntary adduction of the vocal cords (folds) during respiration results in variable upper airway obstruction and the most typical symptoms of VCD.

Unfortunately, there is lack of consensus regarding clinical features and terminology of laryngeal dysfunction [8^{••},9] as listed below:

- (1) emotional laryngeal wheezing;
- (2) episodic laryngeal dyskinesia;
- (3) episodic paroxysmal laryngospasm;
- (4) exercise-induced laryngospasm;
- (5) factitious asthma;
- (6) functional laryngeal dyskinesia;

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Curr Opin Allergy Clin Immunol 2012, 12:95–101

DOI:10.1097/ACI.0b013e328350fdaa

KEY POINTS

- Occupational exposures, such as respiratory irritants, may both precipitate and perpetuate recurrent symptoms of laryngeal dysfunction.
- Work-related asthma is the most common occupational lung disease in developed countries and as laryngeal dysfunction may be difficult to clinically differentiate from asthma, it is important to consider this diagnosis a cause of work-related respiratory symptoms.
- Substantial further research is required to better understand the pathophysiology and epidemiology of laryngeal dysfunction, and to validate diagnostic approaches.

- (7) functional laryngeal obstruction;
- (8) functional laryngeal stridor;
- (9) functional upper airway obstruction;
- (10) irritable larynx syndrome (ILS);
- (11) irritant-induced vocal cord dysfunction (IVCD);
- (12) munchausen stridor;
- (13) nonorganic upper airway obstruction;
- (14) paradoxical vocal cord motion (PVCMD);
- (15) periodic occurrence of laryngeal obstruction;
- (16) pseudoasthma;
- (17) psychogenic upper airway obstruction;
- (18) psychogenic stridor;
- (19) VCD;
- (20) work-associated irritable larynx syndrome (WILS).

Some authors have a generalized view of dysfunctional laryngeal behaviour and include features such as globus pharyngeus and dysphonia, whereas others restrict the diagnosis to acute upper airway obstruction due to paradoxical vocal cord adduction [7,8¹¹,10]. Recently, there has been a recommendation to utilize the term periodic occurrence of laryngeal obstruction (POLO), encompassing VCD, paroxysmal vocal fold (cord) motion and laryngomalacia [8¹¹]. This review will focus on nonorganic laryngeal dysfunction in which VCD is a central feature.

CLINICAL FEATURES

Laryngeal dysfunction may masquerade with symptoms suggestive of asthma and may coexist with asthma [11–14]. Symptoms of intermittent dyspnea, wheeze and cough are common to both asthma and VCD. Patients may have been treated for asthma for several years with poor response to therapy [11]. Medical utilization and oral steroid therapy have

been demonstrated to be high in patients with VCD [15,16].

Features which suggest VCD include a sensation of inspiratory limitation, rather than expiratory as is usual in asthma, and tightness in throat rather than chest. During an acute episode, the wheeze may be loudest over the neck and upper thorax but transmitted to the chest wall. Hyperventilation during an episode may lead to symptoms such as tingling of the perioral area and digits, dizziness and light-headedness. Frequently, episodes have a rapid onset and resolution [8¹¹]. WILS is a condition which has been proposed to include other laryngeal symptoms such as acute dysphonia (muscular tension dysphonia), sensation of tension in the throat (globus pharyngeus), recurrent cough and sometime dysphagia, with a specific sensory workplace trigger present [4,10].

Clinically, the association of laryngeal dysfunction with occupation may be with both the onset of the condition and a trigger for ongoing symptoms [3,4,17,18]. Exposure of the upper respiratory tract to an irritant at the workplace, such as caused by an accidental spill of a water-soluble agent, may be associated with the onset of symptoms. The event may have been psychologically traumatic and required emergency medical treatment. Perkner *et al.* [3] described a variable latency between occupational irritant exposure and the onset of VCD symptoms, ranging from 1 h or less to 24 h.

Recurrent symptoms of laryngeal dysfunction may be primarily related to occupational factors including irritant exposures, physical exertion and psychogenic factors [4,8¹¹,13,18,19]. Andrianopoulos *et al.* [7] noted 10 of 27 patients with VCD having sensory triggers for symptoms including exposures to environmental stimuli, which may be present in a work environment such as perfumes, air pollutants and chemical agents. Members of the military involved in combat and elite athletes at sports competitions may experience periods of acute extreme stress, and may be a trigger for episodes of VCD [13,19].

Nonoccupational factors, which have been associated with laryngeal dysfunction, include post-nasal drip, gastrooesophageal reflux, coexistent asthma and the presence of coexistent psychopathology [8¹¹,10,14,15,20].

EPIDEMIOLOGY

There are no large prospective population or industry-based epidemiological studies investigating the prevalence of laryngeal dysfunction. The data for laryngeal dysfunction are primarily based on case studies and series. There is some evidence that the

prevalence of the condition is more common in women than men; however, the difference is not as significant as may be commonly assumed. The available literature suggests a female-to-male ratio of 2:1–3:1 [8[■],9].

The prevalence of VCD in association with asthma is currently unclear. One study [21] of 96 patients with asthma and 40 controls noted the presence of VCD in 19% of patients based on direct laryngoscopy compared with 5% of controls. No known studies have addressed the prevalence of VCD in association with work-related asthma. One retrospective study [4] of 340 patients assessed for work-related respiratory symptoms over a 5-year period identified 90 patients with work-related asthma (including four with irritant-induced occupational asthma) and 30 WILS. Five patients had concurrent diagnoses of WILS and work-related asthma.

OCCUPATIONAL IRRITANT EXPOSURE

Occupational exposures to airborne irritants may result in inflammation of the upper respiratory tract. Very high-level acute irritant exposure may cause laryngeal oedema with associated respiratory compromise [22]. Meggs [23] described 'reactive upper airway dysfunction syndrome' (RUDS), a syndrome of chronic rhinosinusitis (nasal and/or sinus inflammation) initiated by high-level exposure to inhaled irritants, with recurrence of symptoms after re-exposure to irritants. RUDS does not, however, include symptoms of laryngeal dysfunction, such as VCD.

Perkner *et al.* [3] described the condition of 'irritant-associated VCD' as listed below:

- (1) documented absence of preceding vocal cord dysfunction or laryngeal disease;
- (2) onset of symptoms after a single specific exposure or accident;
- (3) exposure to an irritating gas, smoke, fume, vapour, mist, or dust;
- (4) onset of symptoms within 24 h after exposure;
- (5) symptoms of wheezing, stridor, dyspnea, cough, or throat tightness;
- (6) abnormal direct laryngoscopy for vocal cord dysfunction either in the asymptomatic state, during symptoms, or with a provocative study;
- (7) exclusion of other types of significant vocal cord disease.

The authors investigated the development of persistent symptoms of laryngeal dysfunction following an acute irritant exposure affecting the upper airway and performed a case–control study [3]

comparing 11 cases of laryngoscopically confirmed irritant-associated VCD with 33 VCD controls. Cases of irritant-associated VCD developed following single-episode exposures to environmental ($n=1$) or occupational ($n=10$) irritants. Exposures included ammonia vapour, fumes from inappropriate mixing of flux and solder, an aerosolized cleaning solution, a noxious odour while cooking 'Cajun salmon' and ceiling tile dust and smoke [3].

Fourteen of 30 patients with WILS identified a specific precipitating event at the workplace at the time of the onset of their symptoms, and five of these patients presented to the emergency department following this event [4].

WORLD TRADE CENTRE RESPONDERS

Rescue and recovery workers at the World Trade Centre (WTC) site following the terrorist attacks of September 11 2001 were exposed to high levels of dust capable of causing irritation and inflammation of the upper respiratory tract. The dust present at the site, evaluated 4 days after the disaster, was very alkaline (pH 9.2–11.5) due to the quantity of pulverized cement [24,25[■]].

Analysis of spirometry performed on former WTC rescue and cleanup workers, and volunteers attending the WTC Health Effects Treatment Program, identified inspiratory flow-volume loop limitation consistent with variable extrathoracic air-flow obstruction in 18.6% (32 of 172) of patients [26]. VCD was diagnosed in 10 workers by flexible laryngoscopic examination. All patients had coexistent findings of laryngopharyngitis suggestive of acid reflux-related disease. It has been proposed that upper airway dysfunction in this group is likely to have been precipitated by upper airway inflammation due to acute irritant dust exposure and perpetuated by gastroesophageal reflux, rhinitis and coexistent psychopathology, stemming from the psychological stressors of the WTC disaster [25[■],26].

HEALTHCARE WORKERS

Healthcare workers, specifically nurses, are often cited as a group with a high incidence or predisposition for VCD [11,14,17,18]. Review of the literature, however, fails to find substantial evidence to support this commonly held belief [9]. Frequently, review articles are cited which do not provide specific epidemiological data. The perception appears to be based on a small number of early case studies and case series without objective substantiation of the diagnosis of VCD [12,27].

PROFESSIONAL ATHLETES

VCD has frequently been reported in elite and professional athletes [13,28]. VCD may be difficult to clinically differentiate from exercise-induced bronchospasm and often may coexist. Exercise-associated VCD may present with exertional dyspnoea with associated inspiratory stridor. It has been reported that symptoms of throat rather than chest tightness may be present, with symptoms beginning soon after exercise starts and resolution within 5 min of stopping exercise [28].

The prevalence of inspiratory stridor was investigated in 370 athletes from a wide range of predominately winter sports (169 competed in outdoor sports, such as biathlon, kayaking and cross country skiing, and 201 indoor sports, such as skating and ice hockey) at the US Olympic training centre in Lake Placid [28]. Participants provided a medical history and underwent spirometry and exercise challenge in cold, dry ambient conditions. Exercise-induced bronchospasm was present in 30% of patients and inspiratory stridor in 5.1% (18 women and one man). Ten of the patients (52%) with inspiratory stridor also had exercise-induced bronchospasm. Inspiratory stridor was also more common in outdoor athletes than indoor athletes (8.3 vs. 2.5%, $P < 0.05$), suggesting exercising in cold, dry ambient air may increase sensitivity of the extrathoracic airway resulting in VCD [28]. Others have speculated that VCD is a conversion reaction associated with personality traits, which may be common to some elite athletes such as success orientation and vulnerability to internal and external pressure not to fail [13].

MILITARY PERSONNEL

In a study of 40 active duty military patients undergoing evaluation for exertional dyspnoea, Morris *et al.* [29] performed flexible laryngoscopy with direct visualization of the vocal cords before and after exercise to evaluate the presence of inspiratory vocal cord adduction. VCD was present in 15% of prospectively studied patients. Sixty percent of patients with VCD had a positive methacholine challenge. The cause of VCD in military personnel has not been determined; however, extreme psychological stress during wartime and training may contribute, as may the high level of regular strenuous exercise required [19,29].

PATHOGENESIS

The pathophysiology of VCD and laryngeal dysfunction is undetermined; however, it is

hypothesized to involve interactions of intrinsic and environmental (potentially workplace) factors. Psychopathology has commonly been proposed as the etiological basis of VCD; however, more recent theories have placed emphasis on organic causes, such as altered neurological control of laryngeal reflexes and the role of olfaction [3,4,7,10,30,31].

Morrison *et al.* [10] hypothesized that in ILS the laryngeal controlling neurons are held in a 'spasm-ready' state. Nociceptive stimuli (such as airborne irritant exposure or gastroesophageal reflux) may lead to 'plastic' changes in central neurons, lowering their threshold for initiating the glottic closure reflex [10]. Symptoms of cough, dysphonia, globus and laryngospasm may then occur at relatively low levels of stimuli. Similarly, Ayres and Gabbott [30] suggested a state of 'laryngeal hyper-responsiveness' underlying VCD and that an initial inflammatory insult to the upper airway may result in altered autonomic balance. The altered balance may be transient or persistent. Further stimuli, such as irritant exposures or stress, then result in triggering local parasympathetic reflexes causing airway narrowing at the glottis. Ayres proposes that this is an exaggeration of the larynx's role in protective of the lower respiratory tract. At the workplace exposure to 'low-level' stimuli, such as perfumes, cleaning products, odors and irritants, may then result in triggering the laryngeal reflex inappropriately [4].

Further research is required to determine whether intrinsic factors, such as psychological characteristics or gastroesophageal reflux, may increase the risk of the development of laryngeal dysfunction. Similarly, better characterization of precipitating events is required including the nature of precipitating exposures. The role of psychological factors such as acute stress at the time of the precipitating exposure, as may be experienced in an industrial accident, also needs further investigation.

DIAGNOSIS

There are no known validated recommendations regarding the diagnosis of work-associated laryngeal dysfunction. The American College of Chest Physicians consensus statement for the diagnosis and management of work-related asthma recommends consideration of nonasthma causes of work-related respiratory symptoms including upper airway dysfunction [6]. The development of respiratory symptoms following an irritant exposure at the workplace may be due to a range of conditions which require consideration, including irritant-induced asthma, RUDS, VCD and WILS.

Objective evaluation of pulmonary function is necessary for the diagnosis of work-related asthma [6]. The performance of spirometry may identify features of VCD including truncation of the inspiratory limb of the flow-volume curve. This finding is more likely to be present in the setting of active symptoms; however, Newman *et al.* [15] reported that 25% of their patients with asymptomatic PVCM showed the characteristic inspiratory cut-off. Methacholine and histamine challenges have been reported to induce VCD; however, truncation of the inspiratory flow-volume loop during methacholine challenge test has not been shown to be diagnostic for the presence of inspiratory vocal cord adduction [32]. Normal lung function tests in the setting of a clinical suspicion of asthma should raise the possibility of VCD as the cause of symptoms [8^{***}].

Morris and Christopher [8^{***}] have proposed diagnostic criteria for VCD including: clinical symptoms of noisy breathing and dyspnoea, laryngoscopic evidence of vocal cord adduction and confirmatory pulmonary function test findings of an abnormal flow-volume loop (most commonly the inspiratory limb) or lack of airway hyperreactivity (Table 1). They stress the importance of laryngoscopy to identify abnormal vocal fold movement, but also to exclude anatomical upper airway disorders, such as paralysis of vocal cords, tumours and laryngomalacia. Laryngoscopy may also identify rhinitis or gastroesophageal reflux, which may contribute to ongoing symptoms. Limitations of laryngoscopy are the lack of access to appropriately skilled physicians, subjective nature of some diagnostic features and the procedure may be

poorly tolerated in patients with acute symptoms [33].

Specific inhalational challenges are considered to be the gold standard for the diagnosis of sensitizer-induced occupational asthma. The role of specific challenges in the diagnosis of work-associated laryngeal dysfunction has not been determined. Huggins *et al.* [18] reported the case of a 46-year-old woman with episodic dyspnoea that appeared primarily at work with exposure to a eucalyptus plant. An initial challenge was performed with exposure to eucalyptus and observation of the vocal cords with flexible fiberoptic nasolaryngoscopy. Obvious adduction of the cords was observed. A second masked challenge was performed 1 month later with control irritant substances which did not induce cord adduction, but also eucalyptus masked with ammonia which did have a positive response.

The investigation of exercise-associated VCD has been performed during and after treadmill and bicycle ergometry exercise tests [29,34,35]. Exercise tests with continuous fiberoptic laryngoscopy were noted to be easy to perform, well tolerated and useful in the diagnosis of exercise associated VCD [34,35].

MANAGEMENT

The management of laryngeal dysfunction requires careful explanation of the condition and feedback regarding the cause of dyspnoea [9]. This should be done without the implication that the disorder is purely psychological. The management of acute VCD episodes may include relaxation

Table 1. Proposed diagnostic criteria for vocal cord dysfunction

Clinical symptoms	Prolonged symptoms
	Recurrent, intermittent episodes
	Shortness of breath
	Upper airway stridor or wheezing
	Reproducible causative or inciting factor
Laryngoscopy	Adduction of vocal cords during inspiration, or both inspiration and expiration
	>50% closure of cords
	Intermittent findings
	May be normal when asymptomatic
	May normalize with vocalization
Pulmonary function tests (criteria for suspected VCD)	'Posterior chinking'—variable finding
	Normal spirometry (no response to bronchodilator)
	Negative bronchoprovocation testing
	Abnormal (truncated or flattened) inspiratory flow-volume loop

VCD, vocal cord dysfunction. Reproduced with permission from [8^{***}].

techniques such as pursing lips and active reassurance despite the sensation of severe dyspnoea [7,9,36]. Anxiolytic medications may be considered [37]. Continuous positive airway pressure and Heliox (a gas mixture of 80% helium and 20% oxygen) inhalation has been shown to be effective in a proportion of patients during acute episodes [38,39].

Chronic therapy is usually necessary and requires a multidisciplinary approach [7,9,40[■]]. Patients with work-associated symptoms should be questioned regarding specific triggers and measures should be in place to minimize exposure to triggers. Patients should be educated that stress can be a factor contributing to VCD episodes. The success of this approach, however, has not been investigated. Speech therapy and relaxation techniques may be useful to manage ongoing symptoms [40[■],41]. Specific recommendations include focusing attention away from inhalation, increasing the patient's awareness of 'early warning signs' of an attack so they can institute breathing exercises and empowering patients to take voluntary control of their breathing [9]. In a study [42] of 20 adolescent female athletes, 95% were able to control their symptoms after speech therapy. Biofeedback has been found to be beneficial in some athletes with VCD [43]. Psychological evaluation and therapy may be required to address anxiety, affective, personality or posttraumatic stress disorders [7,10,40[■]].

CONCLUSION

Unfortunately, there are clear deficiencies in the literature regarding the epidemiology, pathogenesis and diagnosis of laryngeal dysfunction. This deficiency is more significant in work-associated laryngeal dysfunction. Further research is clearly required. Clinicians should, however, consider the diagnosis of laryngeal dysfunction when assessing patients with work-associated respiratory symptoms. Evidence-based guidelines need to be developed to assist clinicians in the diagnosis and management of laryngeal dysfunction.

Acknowledgements

Thanks to Professor Michael Abramson for reviewing this article.

Conflicts of interest

R.H. has no conflicts of interest to disclose.

R.H. has received grant funding from the National Health and Medical Research Council (NHMRC) of Australia.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

■ of special interest

■ of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 211).

- Benninger C, Parsons JP, Mastronarde JG. Vocal cord dysfunction and asthma. *Curr Opin Pulm Med* 2011; 17:45–49.
- This is a very good review of the current understanding of the association of asthma with VCD, and its diagnosis and management.
- Balkissoon R. Occupational upper airway disease. *Clin Chest Med* 2002; 23:717–725.
- Perkner JJ, Fennelly K, Balkissoon R, *et al.* Irritant-associated vocal cord dysfunction. *J Occup Environ Med* 1998; 40:136–143.
- Hoy RF, Ribeiro M, Anderson J, Tarlo SM. Work-associated irritable larynx syndrome. *Occup Med* 2010; 60:546–551.
- Petsonk EL. Work-related asthma and implications for the general public. *Environ Health Perspect* 2002; 110 (Suppl 4):569–572.
- Tarlo SM, Balmes J, Balkissoon R, *et al.* Diagnosis and management of work-related asthma. American College of Chest Physicians Consensus Statement. *Chest* 2008; 134:1S–41S.
- Andrianopoulos MV, Gallivan GJ, Gallivan KH. PVCN, PVCD, EPL, and irritable larynx syndrome: what are we talking about and how do we treat it? *J Voice* 2000; 14:607–618.
- Morris MJ, Christopher KL. Diagnostic criteria for the classification of vocal cord dysfunction. *Chest* 2010; 138:1213–1223.
- Following an extensive literature review, the authors have developed diagnostic criteria for VCD based on clinical features, laryngoscopy and pulmonary function tests. Although further validation of the proposed diagnostic criteria is required, this is a very important contribution to a condition distinguished for being poorly understood and diagnosed.
- Brugman SM. What's this thing called vocal cord dysfunction? 2005; 20. www.chestnet.org/accp/pccsu/pccsu-archives [Accessed 1st October 2011].
- Morrison M, Rammage L, Emami A. The irritable larynx syndrome. *J Voice* 1999; 13:447–455.
- Bahrainwala AH, Simon MR. Wheezing and vocal cord dysfunction mimicking asthma. *Curr Opin Pulm Med* 2001; 7:8–13.
- Thomas PS, Geddes DM, Barnes PJ. Pseudo-steroid resistant asthma. *Thorax* 1999; 54:352–356.
- McFadden ER, Zawadzki DK. Vocal cord dysfunction masquerading as exercise induced asthma. *Am J Respir Crit Care Med* 1996; 153:942–947.
- Christopher KL, Wood RP II, Eckert RC, *et al.* Vocal-cord dysfunction presenting as asthma. *N Engl J Med* 1983; 308:1566–1570.
- Newman KB, Mason UG III, Schmalzing KB. Clinical features of vocal cord dysfunction. *Am J Respir Crit Care Med* 1995; 152:1382–1386.
- Mikita J, Parker J. High levels of medical utilization by ambulatory patients with vocal cord dysfunction as compared to age- and gender-matched asthmatics. *Chest* 2006; 129:905–908.
- Tonini S, Dellabianca A, Costa CM, Lanfranco A, *et al.* Irritant vocal cord dysfunction and occupational bronchial asthma: differential diagnosis in a healthcare worker. *Int J Occup Med Environ Health* 2009; 22:401–406.
- Huggins JT, Kaplan A, Martin-Harris B, Sahn SA. Eucalyptus as a specific irritant causing vocal cord dysfunction. *Ann Allergy Asthma Immunol* 2004; 93:299–303.
- Craig T, Sitz K, Squire E, *et al.* Vocal cord dysfunction during wartime. *Mil Med* 1992; 157:614–616.
- Poelmans J, Tack J, Feenstra L. Paroxysmal laryngospasm: a typical but underrecognized supraesophageal manifestation of gastroesophageal reflux? *Dig Dis Sci* 2004; 49:1868–1874.
- Yelken K, Yilmaz A, Guven M, *et al.* Paradoxical vocal cord motion dysfunction in asthma patients. *Respirology* 2009; 14:729–733.
- Sala E, Hoytenen E, Tupasela O, *et al.* Occupational laryngitis with immediate allergic or immediate type specific chemical hypersensitivity. *Clin Otolaryngol* 1996; 21:42–48.
- Meggs WJ. RADS and RUDS: the toxic induction of asthma and rhinitis. *J Toxicol Clin Toxicol* 1994; 32:487–501.
- Lioy PJ, Weisel C, Millette JR, *et al.* Characterization of the dust/smoke aerosol that settled east of the World Trade Center (WTC) in Lower Manhattan after the collapse of the WTC 11 September. *Environ Health Perspect* 2002; 110:703–714.
- de la Hoz RE, Shohet MR, Cohen JM. Occupational rhinosinusitis and upper airway disease: the World Trade Center experience. *Curr Allergy Asthma Rep* 2010; 10:77–83.
- On the basis of studies performed on recovery workers after the WTC disaster, this study provides a comprehensive review of irritant-induced upper airway disorders in this occupational group. Diagnostic and management algorithms for individuals with chronic rhinosinusitis and laryngopharyngitis symptoms are presented.
- de la Hoz RE, Shohet MR, Bienenfeld LA, *et al.* Vocal cord dysfunction in former World Trade Center (WTC) rescue and recovery workers. *Am J Ind Med* 2008; 51:161–165.

27. Downing ET, Braman SS, Fox MJ, Corroa WM. Factitious asthma. Physiological approach to diagnosis. *JAMA* 1982; 248:2878–2881.
 28. Rundell KW, Spiering BA. Inspiratory stridor in elite athletes. *Chest* 2003; 123:468–474.
 29. Morris MJ, Deal LE, Bean DR, *et al.* Vocal cord dysfunction in patients with exertional dyspnea. *Chest* 1999; 116:1676–1682.
 30. Ayres JG, Gabbott PLA. Vocal cord dysfunction and laryngeal hyperresponsiveness: a function of altered autonomic balance? *Thorax* 2002; 57:284–285.
 31. Shusterman D. Review of the upper airway, including olfaction, as mediator of symptoms. *Environ Health Perspect* 2002; 110:649–753.
 32. Perkins PJ, Morris MJ. Vocal cord dysfunction induced by methacholine challenge testing. *Chest* 2002; 122:1988–1993.
 33. Jain S, Bandi V, Officer T, *et al.* Role of vocal cord function and dysfunction in patients presenting with symptoms of acute asthma exacerbation. *J Asthma* 2006; 43:207–212.
 34. Tervonen H, Niskanen MM, Sovijärvi AR, *et al.* Fiberoptic videolaryngoscopy during bicycle ergometry: a diagnostic tool for exercise-induced vocal cord dysfunction. *Laryngoscope* 2009; 119:1776–1780.
 35. Heimdal JH, Roksund OD, Halvorsen T, *et al.* Continuous laryngoscopy exercise test: a method for visualizing laryngeal dysfunction during exercise. *Laryngoscope* 2006; 116:52–57.
 36. Pitchenik AE. Functional laryngeal obstruction relieved by panting. *Chest* 1991; 100:1465–1467.
 37. Blager FB. Paradoxical vocal cord movement: diagnosis and management. *Curr Opin Otolaryngol Head Neck Surg* 2000; 8:180–183.
 38. Morris MJ, Allan PF, Perkins PJ. Vocal cord dysfunction: etiologies and treatment. *Clin Pulm Med* 2006; 13:73–86.
 39. Reisner C, Borish L. Heliox therapy for acute vocal cord dysfunction. *Chest* 1995; 107:757–760.
 40. Kenn K, Balkissoon R. Vocal cord dysfunction: what do we know? *Eur Respir J* 2011; 37:194–200.
- An excellent review of VCD current concepts including pathophysiology and management.
41. Ibrahim WH, Gheriani HA, Almohamed AA, Raza T. Paradoxical vocal cord motion disorder: past, present and future. *Postgrad Med J* 2007; 83:164–172.
 42. Sullivan MD, Heywood BM, Beukelman DR. A treatment for vocal cord dysfunction in female athletes: an outcome study. *Laryngoscope* 2001; 111:1751–1755.
 43. Banez GA, Culbert T. An integrative approach to vocal cord dysfunction in young athletes. *Biofeedback* 2005; 33:54–57.