

Drug-Induced Paradoxical Vocal Fold Motion



Marlene Garcia-Neuer, MS^a, Donna Marie Lynch, MSN, FNP-BC^a, Kathleen Marquis, PharmD, PhD^b,
Jayme Dowdall, MD^{c,d}, Mariana Castells, MD, PhD^{a,d}, and David Edward Sloane, MD, EdM^{a,d} Boston, Mass

Vocal cord dysfunction, also known as paradoxical vocal fold motion (PVFM), is a disorder characterized by abnormal vocal cord adduction during inspiration. PVFM is commonly misdiagnosed as asthma because of the similarity of symptoms: cough, wheezing, chest pain, and dyspnea. We present the clinical vignette of a 36-year-old woman with juvenile rheumatoid arthritis and multiple adverse drug reactions who presented with recurrent episodes of unrecognized PVFM during skin testing for drug allergy, omalizumab treatment, and tocilizumab desensitization. Before the diagnosis of PVFM, these episodes were treated as anaphylaxis, including the administration of epinephrine. Once diagnosed and treated for PVFM, the patient did not present any further events and continued treatment for drug allergy. PVFM may be underreported in hypersensitivity reactions because of the similarity to Type 1-mediated respiratory symptoms and comorbid asthma. © 2017 American Academy of Allergy, Asthma & Immunology (J Allergy Clin Immunol Pract 2018;6:90-4)

Key words: Anaphylaxis; Adverse drug reaction; Drug hypersensitivity; Vocal cord dysfunction; Paradoxical vocal fold motion; Desensitization

CASE REPORT

A 36-year-old woman with asthma, gastroesophageal reflux disease, generalized anxiety disorder, rheumatoid arthritis, and chronic idiopathic urticaria (with daily intake of up to 200 mg of diphenhydramine per day) had adverse reactions to 15 different

medications, including 7 that reportedly induced anaphylaxis, shortness of breath, and/or throat symptoms (Table I). The patient had a history of 15 emergency department (ED) visits in 2016 alone, and she frequently received epinephrine because of concern for anaphylaxis (Table I and Figure 1). She was initially evaluated in the allergy clinic for possible hypersensitivity to tocilizumab after she developed dyspnea during an infusion of this agent for the treatment of rheumatoid arthritis. As she had positive skin testing to tocilizumab at the second intradermal level (20 mg/mL), tocilizumab desensitization was recommended. Despite desensitization, she continued to present with severe inspiratory stridor during infusion, and she received epinephrine for this on multiple occasions. She had similar symptoms during her initial omalizumab injection and during skin testing to mepivacaine, resulting in a code being called and in the administration of epinephrine during each episode (Figure 1). Her predominant symptoms during these events were severe stridor, dyspnea, gagging, hoarseness, and difficulty speaking, all without documented vital sign changes (Figure 1). Importantly, on physical examination during her acute reaction to local anesthetic skin testing (mepivacaine), she had stridor but no wheezing. She had a transient loss or impairment of consciousness twice, during which time the stridor completely resolved, only to recur when she regained full consciousness. Her local anesthetic skin test areas were completely negative for any wheal or flare reaction. She was transferred to the ED, and visualization of the upper airway was attempted, but she could not tolerate this procedure.

Through repeated observation of the patient during reaction episodes, it became clear that she was presenting with a rapidly reversible process without mucocutaneous signs or symptoms, repeatedly negative serum tryptase, and an absence of mast cell activation that was inconsistent with true anaphylaxis. The time course of the development, resolution, and redevelopment of symptoms suggested a primary neurological or neuromuscular process. Using stridor as the guiding central symptom, and seeing its rapid resolution with loss of consciousness, paradoxical vocal fold motion (PVFM) was suggested as an alternative diagnosis. Evaluation by otolaryngology confirmed this diagnosis by direct visualization of the upper airway (see Video 1, available in this article's Online Repository at www.jaci-inpractice.org). The patient received speech therapy focusing on respiratory control of PVFM. The patient did not receive continuous psychiatric care.

The patient underwent 6 standard tocilizumab desensitizations without adverse reaction after speech therapy. She responded so well to treatment that her antihistamine pre-medication dose was lowered, and the dose of tocilizumab was increased to improve control of her rheumatoid arthritis. The

^aDivision of Rheumatology, Allergy and Immunology, Brigham and Women's Hospital, Boston, Mass

^bPharmacy Services, Brigham and Women's Hospital, Boston, Mass

^cDivision of Otolaryngology, Department of Surgery, Brigham and Women's Hospital, Boston, Mass

^dHarvard Medical School, Boston, Mass

No funding was received for this work.

Conflicts of interest: M. Castells is a member of the American Academy of Allergy, Asthma, and Immunology Board; has received consultancy fees from Sanofi, Merck, Contrafact, Arete Discoveries, and Bentham Science; has received lecture fees from American College of Allergy, Asthma, and Immunology, Colombian Allergy Association; and receives royalties from an Anaphylaxis book and UpToDate. The rest of the authors declare that they have no relevant conflicts of interest.

Received for publication May 3, 2017; revised July 20, 2017; accepted for publication August 8, 2017.

Available online October 14, 2017.

Corresponding author: David Edward Sloane, MD, EdM, Brigham and Women's Hospital, 60 Fenwood Rd., Boston, MA 02115. E-mail: dsloane@bwh.harvard.edu. 2213-2198

© 2017 American Academy of Allergy, Asthma & Immunology
<http://dx.doi.org/10.1016/j.jaip.2017.08.020>

Abbreviations used

ACE- Angiotensin converting enzyme

CT- Computed tomography

ED- Emergency department

EI PVFM- Exercise-induced paradoxical vocal fold motion

ICU- Intensive care unit

PVFM- Paradoxical vocal fold motion

patient discontinued daily antihistamines, and has not received epinephrine in the 5 months since the diagnosis and management of PVFM (Figure 1). In addition to decreasing the pre-medication doses, the resolution of the patient's previous reliance lorazepam is reflective of the success of speech therapy.

INTRODUCTION

PVFM is characterized by paradoxical vocal cord adduction during inspiration. Two distinct phenotypes of PVFM have been postulated: exercise-induced PVFM (EI PVFM) and sudden onset PVFM.¹ EI PVFM has a clear trigger (exercise), whereas the triggers for sudden onset PVFM are not well defined.¹ Although the precise etiology of PVFM is unknown, the incidence is higher among women than men (2 to 3:1) and PVFM is associated with comorbidities including gastroesophageal reflux disease, irritable bowel syndrome, and psychiatric diagnoses.^{2,3} PVFM may be under-reported in hypersensitivity reactions because of the similarity to Type 1-mediated respiratory symptoms and comorbid asthma.⁴ PVFM is commonly misdiagnosed as asthma because of the similarity of symptoms, namely cough, wheezing, chest pain, and dyspnea.^{2,4} Therefore, PVFM can be difficult to identify as the primary culprit during an acute drug hypersensitivity reaction, resulting in misdiagnosis, over usage of medical resources, and increased health care costs.^{2,4,5} PVFM presents mostly during inspiration, but expiratory PVFM has been described as well, with exaggerated adduction of vocal cords during expiration. Acute episodes of PVFM can result in endotracheal intubation for presumed severe upper airway obstruction, as patients present with stridor, hoarseness, tachypnea, chest tightness, and shortness of breath.^{3,6-9} Appropriate treatment of PVFM during an episode includes recognition of the condition, reassurance that the condition is self-limiting, and sedation with benzodiazepines, whereas long-term treatment includes management of underlying comorbidities, which may be triggers, as well as speech and behavioral therapy.^{1,2,10}

ETIOLOGY AND CLINICAL PRESENTATION

Etiology

Originally, PVFM was thought to be psychological in nature, with stress and anxiety as the main triggers. Currently, it is considered a functional disorder that may relate to the role of glottic closure in protecting the trachea and lungs.⁵⁻⁷ The exact mechanisms of PVFM remain unknown; however, the proposed mechanisms include altered autonomic input from central brain regions resulting in laryngeal hyperresponsiveness, other changes in brainstem laryngeal control, motor neuropathy, and inflammation and irritation of the vocal cords from processes such as gastroesophageal reflux.^{3,6,8-11} Most patients have associated medical conditions that can also be acute triggers. These include asthma, hyperventilation, exposure to tobacco smoke, allergic laryngitis, viral infection, untreated sleep apnea, gastroesophageal reflux disease, and rhinitis.^{3,8,12-15}

TABLE I. Drug allergies

Drug	Reaction
Bupivacaine*	Shortness of breath, stridor, difficulty speaking, and swallowing, SVS
Ceftriaxone*	Cough, respiratory distress, SVS
Gadolinium-containing contrast media	Angioedema
Morphine	Shortness of breath, rash
Penicillins	Rash
Sumatriptan	Shortness of breath
Tocilizumab*	Shortness of breath, SVS
Divalproex sodium	Hives, throat itchiness
Isoniazid	Rash
Lidocaine	Rash
Metoclopramide	Akathisia
Latex	Rash
Prochlorperazine	Akathisia
Omalizumab	Nasal congestion, shortness of breath, difficulty speaking
Mepivacaine*	Throat itching, stridor, and difficulty speaking, coughing, gagging, SVS

SVS, Stable vital signs.

*Listed as anaphylaxis in the electronic medical record.

Clinical presentation

Episodes of PVFM typically present abruptly and last from seconds to minutes, but have been reported to last longer. Symptoms resemble other causes of upper airway obstruction such as laryngeal angioedema, and include cough, choking, and dyspnea, but without other clinical signs of a true allergic reaction (such as accompanying urticaria).^{6,8,16,17} Patients often report feelings of throat tightness, and have greater difficulty with inhalation than exhalation, but have no response to rapid acting bronchodilators. The hallmark physical findings include audible stridor, respiratory distress with accessory muscle use, inability or difficulty speaking, hoarseness, and voice change.^{12,18} Vital signs are typically stable, without hypoxemia, and dyspnea appears and resolves more rapidly in PVFM than in asthma. Loss of consciousness has been reported with PVFM, but whether there was resolution of stridor during loss of consciousness has not been previously reported.^{19,20} Careful auscultation of the neck and lungs allows the clinician to differentiate PVFM's stridor from the true wheezing of asthma. Commonly reported triggers include perfumes, odors, changes in humidity and/or temperature, exposure to chemicals, and strong emotions.^{17,18} Patients typically have multiple ED visits and/or intensive care unit (ICU) admissions, and some have a concomitant diagnosis of asthma or idiopathic allergic reactions.^{10,21-23} The presence of a tracheostomy scar or a history of intubation does not exclude a diagnosis of PVFM.⁶

DIAGNOSIS

Transnasal laryngoscopy is key to visualizing vocal cord movement and rapidly ruling out other upper airway pathology. However, laryngoscopy often occurs when patients are asymptomatic.⁶ During an active episode of PVFM, there is narrowing on inspiration as well as marked narrowing on exhalation with

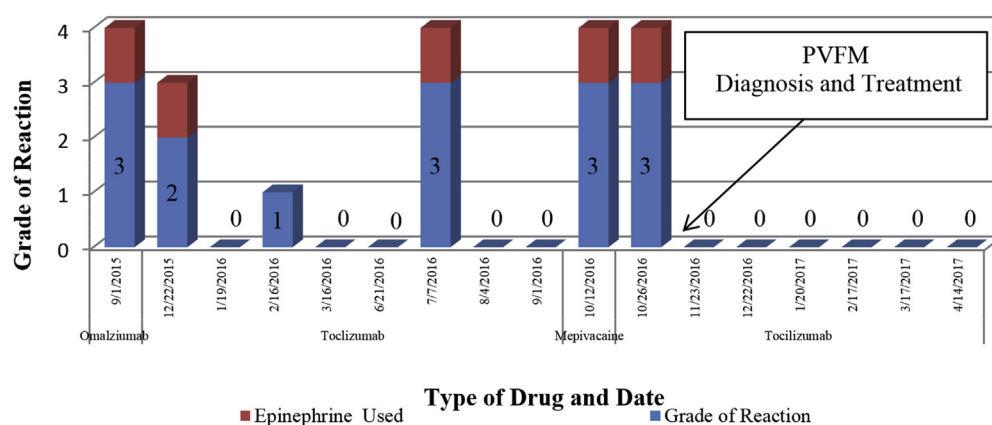


FIGURE 1. Reactions during skin testing, omalizumab injection, and toclizumab desensitizations with description in Table II. Hypersensitivity reactions are graded as mild/grade I (cutaneous symptoms or with only 1 organ system), moderate/grade II (2 or more systems involved without vital sign changes), and severe/grade III when more than 2 systems are affected with vital sign changes. *PVFM*, Paradoxical vocal fold motion.

TABLE II. Reactions during skin testing, omalizumab injection, and toclizumab desensitization with description of protocol, pre- and intramedications, reaction, and epinephrine use

	Protocol	Premedication	Intraprotocol medication	Reaction	Epinephrine
Omalizumab injection September 1, 2015	Injection	None	N/A	Gagging, nasal congestion, shortness of breath, difficulty speaking	Yes
Desensitization to toclizumab #64 December 22, 2015	3 bag 12 step	Acetaminophen 650 mg PO, montelukast 10 mg PO, diphenhydramine 50 mg IV, famotidine 20 mg IV, ondansetron 8 mg PO PRN	Between steps 8 and 9 Famotidine 20 mg IV, diphenhydramine 25 mg IV, albuterol 2 puffs, lorazepam 1 mg IV	Dry heaving, chest tightness rash, voice change, congestion, difficulty speaking	Yes
Desensitization to toclizumab #70 July 7, 2015	3 bag 12 step	Acetaminophen 650 mg PO, montelukast 10 mg PO, diphenhydramine 50 mg IV, famotidine 20 mg IV, ondansetron 8 mg PO PRN	Between steps 8 and 9 Famotidine 20 mg IV, diphenhydramine 25 mg IV, albuterol 2 puffs, lorazepam 1 mg IV	Wretching, shortness of breath, SVS	Yes
Mepivacaine Skin test October 12, 2016	Skin test	None	N/A	Throat itching, stridor, and difficulty speaking, coughing, gagging, SVS	Yes
Desensitization to toclizumab #73 October 12, 2016	3 bag 12 step	Acetaminophen 650 mg PO, montelukast 10 mg PO, diphenhydramine 50 mg IV, famotidine 20 mg IV, ondansetron 8 mg PO PRN	Between steps 8 and 9 Famotidine 20 mg IV, diphenhydramine 25 mg IV, albuterol 2 puffs, lorazepam 1 mg IV	Shortness of breath, voice hoarseness, stridor, SVS	Yes
PVFM diagnosis and treatment					
Desensitization to toclizumab #74-80 November 23, 2016, to April 14, 2017	3 bag 12 step	Acetaminophen 650 mg PO, montelukast 10 mg PO, diphenhydramine 50 mg IV,* famotidine 20 mg IV, ondansetron 8 mg PO PRN	Between steps 8 and 9 Famotidine 20 mg IV, diphenhydramine 25 mg IV, albuterol 2 puffs, lorazepam 1 mg IV	No reaction	No

IV, Intravenous; PO, per os (by mouth); PRN, pro re nata (as needed); *PVFM*, paradoxical vocal fold motion; SVS, stable vital signs.

*Desensitizations 76-80 had a reduction in antihistamine pre- and intramedication.

adduction or prolapse of the supraglottic laryngeal structures.^{12,18} Normally (ie, in the absence of *PVFM*), the vocal folds abduct during inspiration and adduct slightly during exhalation.

A unique marker for *PVFM* is the posterior glottic chink, a small opening at the posterior glottis during inspiration.^{6,8} *PVFM* may not be seen on routine examination because of its intermittent

character or due to the difficulty of performing a laryngoscopy during an acute episode secondary to patient distress, as occurred in our patient.²⁴ Provocation testing, which may include breathing maneuvers, cough, nonspecific irritants, and methacholine, may be of value to visualize and differentiate PVFM from asthma.^{25,26} If laryngoscopy has normal findings, other imaging modalities may be helpful to exclude other causes of stridor not visualized during laryngoscopy. The modalities focus on the trachea and distal airway: radiography of chest and neck, computed tomography (CT), and spirometry with flow volume measurements.^{5,16,21} PVFM typically occurs in the absence of abnormal findings on radiography of the chest and neck and CT, but imaging can be helpful in ruling out other causes of respiratory distress and stridor, such as deep neck space infections, tracheal stenosis, and foreign bodies. Spirometry and flow volume loop analysis can identify PVFM, with the most common finding being a truncated inspiratory flow volume curve.^{5,16,21}

DIFFERENTIAL DIAGNOSIS

Asthma attacks, infections, angioedema (either due to mast cell activation in the context of severe allergic reactions or due to bradykinin in the context of hereditary or acquired angioedema), bilateral vocal cord paralysis, laryngeal, subglottic and tracheal stenosis, and foreign body obstruction may resemble PVFM, but can be differentiated by history, physical examination, laryngoscopy, and (if necessary) additional imaging. Asthma is the most common misdiagnosis and comorbidity, but can be identified by resolution of symptoms with quick acting bronchodilator therapy, by the presence of expiratory wheezing, and by spirometric evidence of airflow obstruction.^{5,21,27} Symptoms of mast cell activation in severe allergic reactions, as suspected in the case above, generally are not limited to the larynx, and often present with angioedema as well as vital sign changes such as hypotension, tachycardia, tachypnea, hypoxemia, and urticaria. These symptoms are often accompanied by helpful confirmatory biomarkers such as elevated tryptase. Angioedema induced by angiotensin converting enzyme (ACE) inhibitors can be difficult to differentiate acutely because of low levels of laryngeal angioedema. However, a clue comes from their distinct kinetics: PVFM symptoms typically resolve within minutes with treatment, whereas ACE inhibitor reactions can take hours to resolve. Both infectious causes and laryngeal tracheal stenosis present with gradual onset of symptoms. Infectious causes would also be expected to have concomitant symptoms such as fever and odynophagia.^{6,8} Laryngeal and tracheal stenosis may present with stridor, either biphasic or only inspiratory. Subglottic stenosis either congenital or acquired can be visualized by laryngoscopy and x-ray. Bilateral vocal cord paralysis presents very similarly to PVFM, with the most notable difference being the lack of throat tightness that is reported with PVFM; also, bilateral vocal cord paralysis is commonly observed as a complication of surgery or neurological damage.^{6,8}

In our case, in which there was no obvious extrinsic inhalation exposure or direct intrinsic insult to the vocal cords that can easily explain the development of PVFM, we strongly suspect that anxiety on the part of the patient was the dominant precipitating factor. Biochemical evidence, such as the negative serum tryptase, argues against a true systemic hypersensitivity reaction. Although the patient never underwent blinded skin testing, the stereotypical response to a wide variety of structurally

unrelated drugs to which she was tested (including antibiotics, local anesthetics, and biologic agents such as tocilizumab and omalizumab) is most economically explained by attributing the reactions to the patient's psychological state, which was the one consistent factor among all her episodes. We suspect that a number of adverse reactions to medications early in her "allergy" history may actually have been anxiety-induced PVFM that we misdiagnosed as mast cell-mediated hypersensitivity.

MANAGEMENT

Short term

Once the diagnosis has been made, the treatment of acute PVFM should be focused on relieving airway obstruction with a combination of patient reassurance and breathing techniques.^{8,28,29} Patient education and reassurance are effective in relieving acute airway symptoms, but breathing techniques have been shown to provide the most rapid relief.^{7,8,28} Education includes showing the patient normal vocal cord motion as well as the patient's own laryngoscopy video.²⁸ Effective breathing techniques include pursed-lip exhalation, breathing through a large-diameter straw, and panting. The purpose of the breathing techniques is to disrupt the irregular respiratory pattern or spasm and relax the vocal folds.^{8,18,22,27} Benzodiazepines have been shown to be effective in relaxing patients and terminating acute symptoms of PVFM but have a risk of oversedating the patient and suppressing respiratory drive.^{13,27,30,31} Another effective treatment is the use of heliox, a mixture of helium and oxygen that is less dense than air and reduces turbulence on inhalation, and thus requires less effort to breathe. This has no effect on the vocal cords themselves, but decreases the patient's work to breathe.^{21,27,28,30} Ketamine and nebulized lidocaine have also been reported to help treat acute symptoms.^{6,8}

Long term

Multidisciplinary follow-up is critical for long-term management of PVFM and includes a combination of speech pathology and otolaryngology. Psychiatric care is important when there is underlying psychopathology, but is not universally required. Goals associated with long-term care include managing triggers and exacerbations brought on by illness or stress, cessation of unnecessary medication, and avoidance of frequent ED admissions.^{9,12} Speech pathology with respiratory training has been shown to be the most effective, with symptom control in up to 90% of patients.^{6,20}

CONCLUSION

Identifying patients with PVFM during acute symptoms is critical to provide appropriate treatment and prevent unnecessary use of resources. A high index of suspicion should be maintained in patients with adverse drug reactions requiring the use of epinephrine, ED visits, ICU admissions, or intubation with symptoms limited to airway complaints and a normal serum tryptase. The key to treating patients with PVFM is identifying the typical symptoms of dyspnea, stridor, difficulty speaking, and hoarseness without angioedema or vital sign changes. We recommend early evaluation by laryngoscopy or referral to otolaryngology, as this can prevent unnecessary use of medications and hospital resources.

REFERENCES

1. Doshi DR, Weinberger MM. Long-term outcome of vocal cord dysfunction. *Ann Allergy Asthma Immunol* 2006;96:794-9.
2. Allan PF, Abouchahine S, Harvis L, Morris MJ. Progressive vocal cord dysfunction subsequent to a chlorine gas exposure. *J Voice* 2006;20:291-6.
3. Traister RS, Fajt ML, Landsittel D, Petrov AA. A novel scoring system to distinguish vocal cord dysfunction from asthma. *J Allergy Clin Immunol Pract* 2014;2:65-9.
4. Owens G, Petrov A. Successful desensitization of three patients with hypersensitivity reactions to omalizumab. *Curr Drug Saf* 2011;6:339-42.
5. Kenn K, Balkissoon R. Vocal cord dysfunction: what do we know? *Eur Respir J* 2011;37:194-200.
6. Denipah N, Dominguez CM, Kraai EP, Kraai TL, Leos P, Braude D. Acute management of paradoxical vocal fold motion (vocal cord dysfunction). *Ann Emerg Med* 2017;69:18-23.
7. Kramer S, deSilva B, Forrest LA, Matrka L. Does treatment of paradoxical vocal fold movement disorder decrease asthma medication use? *Laryngoscope* 2017;127:1531-7.
8. Ng T-T. The forgotten cause of stridor in the emergency department. *Open Access Emerg Med* 2017;9:19-22.
9. Chiang WC, Goh A, Ho L, Tang JPL, Chay OM. Paradoxical vocal cord dysfunction: when a wheeze is not asthma. *Singapore Med J* 2008;49:e110-2.
10. Perkner JJ, Fennelly KP, Balkissoon R, Bartelson BB, Ruttenber AJ, Wood RP II, et al. Irritant-associated vocal cord dysfunction. *J Occup Environ Med* 1998;40:136-43.
11. Vertigan AE, Gibson PG, Theodoros DG, Winkworth AL. The role of sensory dysfunction in the development of voice disorders, chronic cough and paradoxical vocal fold movement. *Int J Speech Lang Pathol* 2008;10:231-44.
12. Matrka L. Paradoxical vocal fold movement disorder. *Otolaryngol Clin North Am* 2014;47:135-46.
13. Ibrahim WH, Gheriani HA, Almohamed AA, Raza T. Paradoxical vocal cord motion disorder: past, present and future. *Postgrad Med J* 2007;83:164-72.
14. Husein OF, Husein TN, Gardner R, Chiang T, Larson DG, Obert K, et al. Formal psychological testing in patients with paradoxical vocal fold dysfunction. *Laryngoscope* 2008;118:740-7.
15. Franca MC. Differential diagnosis in paradoxical vocal fold movement (PVFM): an interdisciplinary task. *Int J Pediatr Otorhinolaryngol* 2014;78:2169-73.
16. Newman KB, Mason UG III, Schmaling KB. Clinical features of vocal cord dysfunction. *Am J Respir Crit Care Med* 1995;152(Pt 1):1382-6.
17. Altman KW, Mirza N, Ruiz C, Sataloff RT. Paradoxical vocal fold motion: presentation and treatment options. *J Voice* 2000;14:99-103.
18. Hicks M, Brugman SM, Katial R. Vocal cord dysfunction/paradoxical vocal fold motion. *Prim Care* 2008;35:81-103. vii.
19. Kayani S, Shannon DC. Vocal cord dysfunction associated with exercise in adolescent girls. *Chest* 1998;113:540-2.
20. Sullivan MD, Heywood BM, Beukelman DR. A treatment for vocal cord dysfunction in female athletes: an outcome study. *Laryngoscope* 2001;111:1751-5.
21. Lawrence SG. Laryngeal dyskinesia: an under-recognized condition. *Emerg Med Australas* 2007;19:96-104.
22. Christopher KL, Wood RP II, Eckert RC, Blager FB, Raney RA, Souhrada JF. Vocal-cord dysfunction presenting as asthma. *N Engl J Med* 1983;308:1566-70.
23. Bahrainwala AH, Simon MR, Harrison DD, Toder D, Secord EA. Atypical expiratory flow volume curve in an asthmatic patient with vocal cord dysfunction. *Ann Allergy Asthma Immunol* 2001;86:439-43.
24. Frank-Ito DO, Schulz K, Vess G, Witsell DL. Changes in aerodynamics during vocal cord dysfunction. *Comput Biol Med* 2015;57:116-22.
25. Benninger C, Parsons JP, Mastronarde JG. Vocal cord dysfunction and asthma. *Curr Opin Pulm Med* 2011;17:45-9.
26. Forrest LA, Husein T, Husein O. Paradoxical vocal cord motion: classification and treatment. *Laryngoscope* 2012;122:844-53.
27. Goldman J, Muers M. Vocal cord dysfunction and wheezing. *Thorax* 1991;46:401-4.
28. Christopher KL, Morris MJ. Vocal cord dysfunction, paradoxical vocal fold motion, or laryngomalacia? Our understanding requires an interdisciplinary approach. *Otolaryngol Clin North Am* 2010;43:43-66. viii.
29. Ophir D, Katz Y, Tavori I, Aladjem M. Functional upper airway obstruction in adolescents. *Arch Otolaryngol Head Neck Surg* 1990;116:1208-9.
30. Heiser JM, Kahn ML, Schmidt TA. Functional airway obstruction presenting as stridor: a case report and literature review. *J Emerg Med* 1990;8:285-9.
31. Tousignant G, Kleiman SJ. Functional stridor diagnosed by the anaesthetist. *Can J Anaesth* 1992;39:286-9.

Reproduced with permission of copyright owner. Further reproduction prohibited without permission.