

# Laryngeal Sensory Deficits in Patients With Chronic Cough and Paradoxical Vocal Fold Movement Disorder

Thomas Murry, PhD; Ryan C. Branski, PhD; Kathy Yu, MD; Sabrina Cukier-Blaj, MS;  
Suzy Duflo, MD, PhD; Jonathan E. Aviv, MD

**Objectives/Hypothesis:** Although the diagnostic accuracy of paradoxical vocal fold movement disorder and chronic cough has improved, the underlying pathophysiology remains relatively unknown. We hypothesize that one potential etiological factor in these patients is an aberrant laryngeal sensory response and sought to determine if respiratory retraining in addition to antireflux therapy alters this aberrant response.

**Study Design:** Retrospective, outcomes.

**Methods:** Sixteen patients who had been on at least 3 months of twice-daily proton pump inhibitors with no subjective improvement in their primary complaint of cough, self-reported symptoms of gastroesophageal and laryngopharyngeal reflux, and concurrent paradoxical vocal fold movement (PVFM) were included in the current study. In addition to continuing twice daily pharmacological therapy, subjects underwent a course of respiratory retraining. Outcome measures including the Reflux Symptom Index (RSI), transnasal flexible laryngoscopy, and laryngopharyngeal sensory discrimination thresholds were obtained prior to and following a course of respiratory retraining.

**Results:** Mean bilateral laryngeal sensory response improved significantly after combined respiratory retraining and aggressive proton pump inhibitor therapy ( $P = .01$ ). In addition, mean RSI score decreased significantly following treatment ( $P = .02$ ).

Specifically, 13 of 16 patients experienced improved sensory response, corresponding with patient reports of improved PVFM symptoms following treatment.

**Conclusions:** Aberrant laryngeal sensation was identified in patients with PVFM and chronic cough. This response, however, normalized following a limited course of respiratory retraining, corresponding with improved patient symptoms.

**Key Words:** Paradoxical vocal cord movement, chronic cough, laryngopharyngeal reflux disease, laryngeal sensation.

**Level of Evidence.** 2c

*Laryngoscope*, 120:1576–1581, 2010

## INTRODUCTION

Paradoxical vocal fold movement (PVFM) is a dystonic, nonpulmonary laryngeal disorder resulting in disordered breathing and cough related to involuntary vocal fold adduction during inspiration and/or expiration. Triggers for PVFM are classified as 1) inhaled (smoke, fumes, or steam), 2) temperature (cold air or high humidity), 3) activity (talking, laughing, deep breathing, swallowing, or exercise), and 4) intrinsic (throat sensations, shortness of breath, or stress).<sup>1</sup> Identifying PVFM, often a diagnosis of exclusion, typically involves multidisciplinary evaluation and might include pulmonary function tests, sinus computed tomography scans, allergy testing, and pH monitoring and/or esophageal motility testing.<sup>1–4</sup> Cough and PVFM might be discrete entities or features within the spectrum of one common, underlying syndrome as described previously by Vertigan et al.<sup>1</sup> In addition, a more causal relationship has been proposed, such as coughing as an adaptive mechanism to maintain glottic patency during PVFM. Regardless, chronic cough has been described in approximately 80% of patients with PVFM,<sup>5</sup> and is often the primary symptom.<sup>6</sup>

Lee and Woo<sup>7</sup> suggested that intractable, chronic cough might also be a manifestation of laryngeal sensory neuropathy. In that study, 71% of patients with sudden-onset cough, laryngospasm, or throat clearing that presented after viral illness, surgery, or unknown trigger

From the Department of Otorhinolaryngology, Weill Cornell Medical College, New York (T.M., R.C.B.); Department of Head and Neck Surgery, Memorial Sloan-Kettering Cancer Center (R.C.B.), New York; Department of Otolaryngology/Head and Neck Surgery, Columbia Presbyterian Medical Center, New York (K.Y., S.C.-B.); Department of Otolaryngology, Mt. Sinai Medical Center, New York (J.E.A.), New York, U.S.A.; Department of Otolaryngology/Head and Neck Surgery, Timone Hospital University, France (S.D.).

Editor's Note: This Manuscript was accepted for publication February 19, 2010.

The authors have no funding, financial relationships, or conflicts of interest to disclose.

Send correspondence to Thomas Murry, PhD, Department of Otorhinolaryngology, Weill Cornell Medical College, 1305 York Avenue, 5th Floor, New York, NY 10021. E-mail: thm7001@med.cornell.edu

DOI: 10.1002/lary.20985

had concomitant superior or recurrent laryngeal nerve motor neuropathy, documented by electromyography and/or videostroboscopy. All patients were treated with gabapentin (100–900 mg/d) with favorable outcomes. In addition, the relatively high incidence of chronic cough in patients with PVFM and autosomal dominant hereditary sensory neuropathy further suggests some link between cough and sensory neuropathy.<sup>8</sup>

In spite of these data, hyposensitivity or hypersensitivity of the laryngopharyngeal (LP) mucosa in this population remains controversial. Some conclude that the combination of chronic cough and PVFM is due to upper airway hypersensitivity, presumably secondary to vagal injury sustained as a result of viral infection.<sup>3</sup> Morrison et al.<sup>9</sup> described chronic cough as a component of irritable larynx syndrome, suggesting that the sensitivity threshold for the cough behavior is thought to be associated with neural remapping. Wani et al.<sup>10</sup> described a cohort of patients with known laryngeal nerve injury who presented with cough and stridor, in which blockade with botulinum toxin to the thyroarytenoid muscle temporarily mitigated or relieved cough, but did not abolish stridor or prevent abnormal irritative sensations. The authors suggested that abnormal laryngeal sensory function likely resulted from aberrant sensory nerve repair, regeneration, or central changes resulting in hyperesthesia.

The relationship between chronic cough and PVFM has also been associated with hyposensitivity of the larynx. Recurrent irritation of the larynx and pharynx by inflammatory stimuli, most notably gastric refluxate, has been hypothesized as one likely cause.<sup>1–3,11</sup> Phua et al.<sup>4</sup> used the calibrated air pulse technique originally described by Aviv et al.<sup>12</sup> to identify the lowest air pressure required to elicit the laryngeal adductor reflex (LAR) in patients with chronic cough and gastroesophageal reflux disease (GERD) and 10 healthy subjects without GERD. The mean baseline LAR threshold was significantly higher (9.5 mm Hg) in the group with cough and GERD when compared to controls (3.68 mm Hg), suggesting impaired laryngopharyngeal sensation.

Chronic cough and PVFM often lead to substantive disruptions in quality of life, and these conditions often remain untreated despite numerous specialty consultations and extensive testing. Recently, Murry et al.<sup>6</sup> proposed a combined treatment paradigm of proton pump inhibitor (PPI) plus respiratory retraining to control the chronic cough when reflux medication alone offered no change in symptom severity. This paradigm included PPI dosing twice daily, 30 to 60 minutes before breakfast and dinner. Respiratory retraining consisted of a series of breathing exercises focusing on increasing abdominal control of the breath while breathing in rhythm without breath holding.

In the current investigation, we sought to quantify the laryngeal sensory response in a cohort of patients with PVFM and chronic cough. Furthermore, we sought to determine if this aberrant laryngeal sensory response was altered following an aggressive therapeutic regimen of both pharmacological antireflux therapy and a limited course of respiratory retraining. We hypothesized that

the laryngeal sensory response will, in fact, improve following intervention, consistent with patient reports of improved symptoms.

## MATERIALS AND METHODS

### Subjects

This retrospective study was approved by the institutional review board. Inclusion criteria were adults on twice-daily PPI therapy for at least 3 months with no subjective improvement in their primary symptom, chronic cough. Patients with a history of active sinonasal disease, laryngeal surgery, or documented pulmonary disease were excluded. Sixteen patients from 29 to 69 years of age were included. No subject had any history of breathing exercises or speech therapy prior to their participation in the current study. Some patients had been on daily antacid therapy prior to initiating twice daily treatment. The mean duration of the primary symptom of cough was 8.4 months (standard deviation = 5.3 months).

### Clinical Outcome Measures

Clinical suspicion of reflux disease was determined via the Reflux Symptom Index (RSI).<sup>13</sup> The RSI is a reliable patient assessment of symptoms associated with laryngopharyngeal reflux (LPR). According to Belafsky et al.,<sup>13</sup> a score below 11 suggests little or no evidence of LPR. For this study, a score of <11 was considered normal, a score between 11 and 22 was indicative of moderate LPR symptoms, and from 23 to 45 was indicative of severe LPR symptoms. All patients completed the RSI prior to treatment; only 12 of the 16 completed it following treatment. For the purpose of determining the efficacy of intervention as it relates to cough, particular attention was dedicated to statement 7 (“troublesome or annoying cough”).

All subjects underwent transnasal flexible laryngoscopy with sensory testing. Once comfortable, subjects were asked to breathe through their nose and then through their mouth. They were also asked to produce an “ee” sniff maneuver to reflect bilateral symmetry of vocal fold abduction/adduction. Finally, they were asked to repeat the sentence, “We see three green trees,” several times. This was followed by sensory testing according to the air pulse technique procedure described by Aviv et al.<sup>12</sup> The calibrated air pulse test was used to determine LP sensory discrimination thresholds by endoscopically delivering air pulse stimuli to the aryepiglottic mucosa innervated by the superior laryngeal nerve. Briefly, a calibrated 50-ms air pulse was delivered to the anterior wall of the pyriform sinus. Surface sensitivity was determined by varying air pressure with constant pulse duration. The LAR was determined by direct visualization of vocal fold medial movement. If at 9.0 mm Hg no LAR was elicited, a suprathreshold stimulus consisting of a continuous air stream was given. LP sensory discrimination thresholds were defined as either normal (<4.0 mm Hg air pulse pressure [APP]), moderately aberrant (4.0–6.0 mm Hg APP), or severely aberrant (>6.0 mm Hg APP).<sup>12</sup>

Following endoscopic examination, two observers, independently but not blinded to the subject, determined the presence of PVFM. The criteria set forth for identifying PVFM was >50% adduction of the vocal folds during quiet respiration and/or adduction of the vocal folds after repetition of the sentence. Agreement was unanimous among both raters for all subjects (inter- and intra-rater reliability was not assessed). All subjects in this study showed paradoxical motion during oral and/or nasal quiet breathing, and 13 of 16 subjects showed paradoxical motion following repetition of the sentence. Thus, diagnosis of PVFM was made on the basis of the video-recorded

**Quiet rhythmic breathing.** Patients are instructed to exhale with shoulders relaxed and abdominal movement in and out, consistent with continuous exhalation and inhalation.

**Breathing with vocal resistance.** Patients are instructed to exhale while sustaining one of several sounds (sh, f or z), for increasing lengths of time.

**Pulsed exhalation.** Patients are instructed to produce a pulse of air using "ha" or "sha" followed by sniffing through the nose with the mouth closed.

**Abdominal focus at rest.** Patients are instructed to lie flat and put a small book on their abdomen and focus on elevating the book on inhalation and lowering the book on exhalation. Once this task is mastered, patients are instructed to breathe through a straw providing resistance by squeezing or occluding the straw. Patients are instructed to focus on the abdominal movement in the context of increased resistance. These exercises may be performed either upright or prone.

Fig. 1. Sequence of exercises employed in the respiratory retraining program, based on the early work of Christopher et al.<sup>14</sup> Subjects advanced from low breathing effort to breathing against increased resistance.

laryngeal examination and independent agreement by two authors. Flow loop spirometry (Koko Spirometer, Longmont, CO) was then performed, and only patients with normal measures of vital capacity, forced expiratory flow (FEF), and forced expiratory flow 25% to 75% (FEF 25–75) were included in the current study.

Following treatment, improvement of cough was defined by the rating of statement 7 (troublesome or annoying cough) on the RSI and subjective patient report. Resolution of PVFM was defined as no pathologic adduction of the vocal folds during quiet inhalation and/or exhalation or following the repetition of the sentence, again judged by two investigators who were unaware of their previous ratings.

### Respiratory Retraining

Each patient was instructed in a series of breathing exercises focusing on the rhythm and location of breathing following

diagnosis, as originally described by Christopher et al.<sup>14</sup> (Fig. 1). Patients were instructed to perform these exercises twice a day for 10 to 15 minutes as described previously by the first author.<sup>6</sup> In addition to the home exercise program, all patients were scheduled for quasiregular treatment sessions. All subjects completed at least two treatment sessions. All patients remained on twice-daily PPI treatment throughout the duration of the retraining program. In addition, follow-up evaluation was scheduled for approximately 3 months following their initial visit.

### RESULTS

The treatment duration ranged from 4 to 23 weeks with a mean number of treatment visits of 5.7 (range, 2–13 visits). As shown in Table I, prior to treatment all 16 patients had a primary complaint of cough. However, following therapy, only one patient had persistent cough. Similar

TABLE I.  
Patients' Symptoms Pre- and Post-Proton Pump Inhibitor and Respiratory Retraining.

Patients	Symptoms Pretreatment				Symptoms Post-Treatment			
	Cough	Throat Clearing	Hoarseness	Dyspnea	Cough	Throat Clearing	Hoarseness	Dyspnea
1	x	x						
2	x	x	x					
3	x			x				
4	x	x	x					
5	x			x				
6	x	x	x		x	x	x	
7	x	x	x	x				
8	x	x		x				x
9	x	x						
10	x	x						
11	x	x		x				
12	x	x	x					
13	x	x		x		x		
14	x	x	x					
15	x	x	x			x		
16	x	x	x	x		x		x
Total	16	14	8	7	1*	4*	1*	2

Patients had been on at least 3 months of twice daily proton pump inhibitor therapy prior to initiating respiratory retraining and remained on this regimen throughout the duration of the treatment.

\* $P \leq .01$  after treatment.

TABLE II.  
Pre- and Post-Proton Pump Inhibitor and Respiratory Retraining Treatment for Each Subject.

Treatment	Subject	Follow-Up, mo	RSI	Degree of RSI	Air Pulse, Right	Degree of Right LAR	Air Pulse, Left	Degree of Left LAR	PVFM
Pre	1		N/A		Cont	Severe	Cont	Severe	
Post		5	N/A		5.6*	Moderate	5.6*	Moderate	Improved
Pre	2		13	Moderate	Cont	Severe	7.5	Severe	
Post		7	7*	Normal	6.5	Severe	6.6	Severe	Resolved
Pre	3		9	Normal	5.2	Moderate	8.4	Severe	
Post		7	N/A		3.8*	Normal	3.8*	Normal	Improved
Pre	4		23	Severe	3.8	Normal	3.8	Normal	
Post		8	N/A		4.5†	Moderate	4.5†	Moderate	Resolved
Pre	5		32	Severe	6	Severe	6	Severe	
Post		6	10*	Normal	5.1*	Moderate	5.1*	Moderate	Resolved
Pre	6		9	Normal	5.8	Moderate	5.8	Moderate	
Post		17	9	Normal	Cont†	Severe	3.2*	Normal	Resolved
Pre	7		36	Severe	3.8	Normal	3.8	Normal	
Post		12	21*	Moderate	3.6	Normal	3.6	Normal	Same
Pre	8		18	Moderate	Cont	Severe	3	Normal	
Post		19	N/A		Cont	Severe	Cont†	Severe	Same
Pre	9		23	Severe	7.2	Severe	7.2	Severe	
Post		7	1*	Normal	5*	Moderate	5*	Moderate	Improved
Pre	10		16	Moderate	4.2	Moderate	4.2	Moderate	
Post		7	19	Moderate	3.2*	Normal	3.2*	Normal	Resolved
Pre	11		10	Normal	3.5	Normal	3.5	Normal	
Post		7	#12	Moderate	3.8	Normal	3.8	Normal	Improved
Pre	12		37	Severe	7	Severe	7	Severe	
Post		6	30	Severe	3.8*	Normal	3.8*	Normal	Same
Pre	13		24	Severe	7.2	Severe	7.2	Severe	
Post		9	*4	Normal	5.6*	Moderate	5.6*	Moderate	Resolved
Pre	14		18	Moderate	Cont	Severe	7.5	Severe	
Post		12	21	Moderate	3.7*	Normal	3.7*	Normal	Resolved
Pre	15		N/A		6.2	Severe	6.2	Severe	
Post		7	N/A		4.2*	Moderate	4.2*	Moderate	Resolved
Pre	16		30	Severe	Cont	Severe	Cont	Severe	
Post		9	14*	Moderate	6.5	Severe	6.5	Severe	Resolved

The features presented are months of follow-up treatment, number of respiratory retraining sessions, reflux symptom index (RSI), degree of RSI, air pulse stimulus threshold to elicit right laryngeal afferent reflex (mm Hg), air pulse stimulus threshold to elicit left laryngeal afferent reflex (mm Hg), degree of both sides of laryngeal afferent reflex, and presence of paradoxical vocal fold movement.

\* $P \leq 0.01$  after treatment; † $P \leq 0.05$  after treatment.

LAR = laryngeal adductor reflex; PVFM = paradoxical vocal fold movement; N/A = not available; Cont = continuous.

improvements in subjective patient complaints were observed for throat clearing and hoarseness. These subjective reports of improvement were corroborated by objective

data, including a significant decrease in the mean laryngeal sensory response bilaterally ( $P = .02$  and  $P = .02$ , left and right, respectively). Interestingly, no differences were

TABLE III.  
Shifts in Reflux Symptom Index (Item 7), Laryngeal Sensory Threshold, and Paradoxical Vocal Fold Movement After Treatment.

	RSI7		Air Pulse, Right		Air Pulse, Left		PVFM	
	Shift in score	% Improved	N	% Improved	N	% Improved	N	% Improved
Shift in score	-3.74	100*						
Same			2	12.5	2	12.5	1	6.25
Improved			2	12.5	1	6.25	3	18.75
Resolved			12	75.0	13	87.5	12	75.0

RSI7 = Reflux Symptoms Index, statement 7; air pulse = laryngeal sensory response; PVFM = paradoxical vocal fold movement.

\* $P < .01$ .

observed between the left and right laryngeal sensory response ( $P = .88$ ). Complete laryngeal sensory response data is located in Table II.

As summarized in Table III, of the 12 patients that completed the RSI before and after treatment, the mean difference in the score of statement 7 was 3.76, a statistically significant difference ( $P = .05$ ). All patients, including the four who did not complete the RSI, reported improved cough. When analyzing the total RSI score, no statistically significant differences between pre- and post-treatment were observed. Clinically, 12 of the 16 patients presented with resolution of paradoxical vocal fold motion following treatment. Three patients had improved motion and one patient did not appear to have any alteration in paradoxical movement.

## DISCUSSION

The pathophysiology of LPR-associated cough has been postulated to occur via two main mechanisms: 1) acid exposure in the distal esophagus stimulates the esophageal-tracheobronchial cough reflex via the vagus nerve, and 2) microaspiration of esophageal contents into the LP and tracheobronchial tree.<sup>3</sup> Although the first mechanism has been supported by prior studies,<sup>15,16</sup> there is little evidence to support the latter theory. The results of the present study suggest that cough associated with PVFM might be attributed to decreased mechanosensitivity resulting from the receptors buried in the edematous mucosa. This is a hallmark of LPR and can be expected to improve with treatment. In this group with PVFM, although the PPI might reduce edema, the behavioral response (cough) required further treatment with behavioral methods, namely respiratory retraining.<sup>2,3,6</sup> Decreased mechanosensitivity and chemosensitivity of the LP from chronic acid irritation results in increased collection of particulate or irritant matter in the LP, and the chronic cough reflex might be simply an adaptive mechanism that has evolved through habit to clear the larynx. Paradoxical adduction of the vocal folds during inspiration in this context might be acting as a protective response to prevent further inhalation of particulate matter in patients whose cough is not controlled by pharmacological treatment alone.

We proposed that chronic cough in patients with PVFM was associated with aberrant laryngeal sensation, mainly caused by LPR. However, we further hypothesized that this response might improve with a combination of PPI treatment and respiratory retraining. Phua et al.<sup>4</sup> also used the calibrated air pulse technique to evaluate LP sensation in patients who presented with chronic cough and GERD, and compared those patients to healthy subjects and found decreased sensation. Similar to the present finding, they concluded that patients with chronic cough and GERD had impaired LP mechanosensitivity. Our data suggest that respiratory retraining not only alters the respiratory mechanics, but also has the capacity to shift the inherent laryngeal sensory response. Although interesting, we

acknowledge that the current study has not elucidated potential mechanisms for these findings.

Admittedly, this study is limited in terms of the number of patients and the lack of complete follow-up data for four of the 16 patients. Regardless, we propose that chronic cough in patients with PVFM is associated with aberrant sensation of the larynx mainly caused by LPR, and might be amendable to a combination of PPI treatment and respiratory retraining. These data also concur with a previous investigation suggesting that patients with chronic cough and LPR had impaired LP sensitivity.<sup>3</sup>

In the current study, patients with chronic cough showed improvement in sensation and a reduction in cough following a behavioral training program and maintenance of a reflux medication regimen. Although previous studies have examined cough in patients with PVFM,<sup>4,11</sup> the results of this study lead to a further understanding of the importance of targeting sensation to reduce cough. Previous studies have shown that in some patients, treatment with PPIs alone does not resolve cough.<sup>1,6,15</sup> Because all subjects in the current study were previously on twice daily PPI therapy without improvement of cough and shortness of breath, the addition of respiratory retraining proved valuable to cough reduction.

Although we are inherently limited by sample size to make broad conclusions, there is a trend that shows a relationship between improved LAR and reduction in cough and PVFM. The majority of patients in our case series who underwent combined pharmacological and behavioral treatment had improved PVFM, and improved LP sensation. The behavioral treatments were conducted in the clinic and at home; patient compliance with the exercise regimen was not assessed, providing a potential explanation as to why all subjects did not experience a complete change in their primary symptom of cough. Regardless, these preliminary data suggest a relationship between PVFM/cough, laryngeal sensory function, and behavioral therapy for these challenging patients.

## CONCLUSION

Patients with the primary complaint of cough and LPR following the diagnosis of PVFM exhibited improvement in laryngeal sensation with an associated resolution of PVFM and chronic cough after treatment with PPIs and respiratory retraining despite persistence of cough treated with PPIs alone. This suggests that improvement in sensory function is a key element in the reduction of chronic cough in patients with LPR and PVFM.

## BIBLIOGRAPHY

1. Vertigan AE, Theodoros DG, Gibson PG, Winkworth AL. The relationship between chronic cough and paradoxical vocal fold movement: a review of the literature. *J Voice* 2006;20:466-480.
2. Rogers JH, Stell PM. Paradoxical movement of the vocal cords as a cause of stridor. *J Laryngol Otol* 1978;92:157-158.

3. Altman K, Simpson C, Amin M, et al. Cough and paradoxical vocal fold motion. *Otolaryngol Head Neck Surg* 2002;127:501–511.
4. Phua SY, McGarvey LPA, Ngu MC, Ing AJ. Patients with gastro-esophageal reflux disease and cough have impaired laryngopharyngeal mechanosensitivity. *Thorax* 2005;60:488–491.
5. Newman KB, Mason UG III, Schmaling KB. Clinical features of vocal cord dysfunction. *Am J Respir Crit Care Med* 1995;152:1382–1386.
6. Murry T, Tabae A, Aviv JE. Respiratory retraining of refractory cough and laryngopharyngeal reflux in patients with paradoxical vocal fold movement disorder. *Laryngoscope* 2004;114:1341–1345.
7. Lee B, Woo P. Chronic cough as a sign of laryngeal sensory neuropathy: diagnosis and treatment. *Ann Otol Rhinol Laryngol* 2005;114:253–257.
8. Spring PJ, Kok C, Nicholson GA, et al. Autosomal dominant hereditary sensory neuropathy with chronic cough and gastro-esophageal reflux: clinical features in two families linked to chromosome 3p22-p24. *Brain* 2005;128:2797–2810.
9. Morrison M, Rammage L, Emami AJ. The irritable larynx syndrome. *J Voice* 1999;13:447–455.
10. Wani MK, Woodson GE. Paroxysmal laryngospasm after laryngeal nerve injury. *Laryngoscope* 1999;109:694–697.
11. Harding SM, Richter JE. The role of gastroesophageal reflux in chronic cough and asthma. *Chest* 1997;111:1389–1402.
12. Aviv JE, Martin JH, Keen MS, et al. Air pulse quantification of supraglottic and pharyngeal sensation: a new technique. *Ann Otol Rhinol Laryngol* 1999;102:777–780.
13. Belafsky PC, Postma GN, Koufman JA. The validity and reliability of the reflux finding score (RFS). *Laryngoscope* 2001;111:1313–1317.
14. Christopher KL, Wood RP II, Eckert RC, Blager FB, Raney RA, Souhrada JF. Vocal-cord dysfunction presenting as asthma. *N Engl J Med* 1983;308:1566–1570.
15. Mintz S, Lee JK. Gabapentin in the treatment of intractable chronic cough: case reports. *Am J Med* 2006;119:13–15.
16. Mishriki YY. Laryngeal neuropathy as a cause of chronic intractable cough. *Am J Med* 2007;120:5–7.