

THE VISIBLE VOICE

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Professional Voice Users, and People with Voice Disorders

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CHRONIC COUGH



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I recently examined the chief complaints of my patients. I was surprised to discover that almost 20% had “chronic cough,” and that those patients were referred to me by pulmonologists and gastroenterologists as well by otolaryngologists. The commonest reason I was consulted was to rule-in or rule-out laryngopharyngeal reflux (LPR), aka “atypical” or “silent reflux.”

Based upon laryngeal electromyography and reflux-testing, the most common 1^o diagnosis was “neurogenic cough” with or without concomitant LPR. And in treating those patients, many were more responsive to medications such as amitriptyline, tramadol, and gabapentin than to proton pump inhibitors and H2-antagonists. This VV issue is about reflux-related and neurogenic cough.

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THURSDAY, DECEMBER 18, 2008 • 6:00 – 9:00 P.M.
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At VINY in January, first monthly *Laryngology Case Study Group*
Focus: Vocal Fold Paresis: Thurs., Jan. 15, 2009, 6:30 – 8:00 p.m.

CHRONIC COUGH *THAT'S NOT LUNG DISEASE*

Even though I was trained as an otolaryngologist, in my history taking I have always included questions about breathing, cough, asthma, and “choking” because the details of those symptoms and the pattern hold the key to the accurate diagnosis of many airway disorders. Chronic cough¹⁻⁵ is a common complaint in otorhinolaryngologic (ORL, ENT) practice; however, it almost never occurs as a symptom in isolation. In other words, ENT patients with chronic cough almost always have associated symptoms such as globus, dysphagia, dysphonia, dyspnea, and/or stridor.

For most chronic cough patients the symptom pattern has diagnostic relevance. For example, awakening from sound sleep in the night violently coughing (usually with accompanying dyspnea and stridor) is virtually pathognomonic of laryngopharyngeal reflux (LPR).

Patients may refer to several heterogeneous symptoms as “choking,” including patients with dysphagia, aspiration, and stridor. Precise characterization of the choking episodes should be elicited by further questioning, since the treatment for each condition is different.

TABLE 1: COMMON DIAGNOSTIC “RED FLAGS” IN THE PATIENT’S HISTORY

Symptom/Pattern	Likely Diagnosis
Choking episodes	
Solid food bolus getting stuck	LPR, esophageal dysmotility, stricture
Coughing after drinking or eating	Aspiration, glottal incompetence
Shortness of breath, difficulty getting air in	LPR, paroxysmal laryngospasm
Being awakened coughing from sound sleep	LPR
Vocal fatigue and breathy dysphonia after URI	Vocal fold paresis, laryngeal neuropathy
Painful speaking (“odynophonia”) after URI	Neuropathic pain, laryngeal neuropathy
Nagging dry cough weeks-to-months after URI	LRP, neurogenic cough, or both

Another key area of the history has to do with the question of prior upper respiratory infection (URI), i.e. URI at the time of onset of cough or other symptom(s). Interestingly, a retrospective review of 80 consecutive patients with laryngeal and voice disorders revealed that 19% (15/80) specifically dated symptoms to an URI. (Koufman JA, Unreported data)

REACTIVE UPPER AND LOWER AIRWAY DISEASE

The population of patients with reactive airway disease is bimodal, with the larger group having asthma (*reactive lower airway disease*) and the smaller group having *reactive upper airway disease*, i.e., variable airway obstruction at the laryngeal level. The hallmark of the former is wheezing, and the latter is stridor. Despite some medical literature to the contrary, there is not a lot of overlap between the stridor and wheezing groups.⁵

Of the roughly 10-15% of my patients present with symptoms described as “choking,” most turn out to have LPR-related *laryngospasm* (LS), as opposed to dysphagia or aspiration (**Table 1**). In addition, many LS patients, as well as those with *paradoxical vocal fold movement* (PVFM), are mistakenly diagnosed as having asthma. In such cases, careful history taking will almost always reveal that such patients have inspiratory stridor and not wheezing.⁵

There are patients who have both LPR and asthma; however, the combination of “asthma” with intractable chronic cough increases the likelihood that LPR is the cause or is a factor. As mentioned above, many of my LS and PFVM reactive upper airway disease patients have been misdiagnosed as having adult-onset asthma; but coincidentally, the breathing problems often began at the time of other LPR symptoms. To the point, some “asthma” patients are cured by antireflux treatment. In any event, defining respiratory/airway symptoms accurately is the first job of the clinician; and some patterns are associated with specific diagnoses (**Table 2**).

TABLE 2: CHARACTERIZATION OF STRIDOR (NOISY BREATHING)

Inspiratory Stridor -- Usually Laryngeal Obstruction

Bilateral (medial vocal fold position) laryngeal paralysis
Swelling/edema due to fungal, bacterial, or viral infection, angioedema or reflux
Obstructing benign growths (e.g., papillomas, cysts, granulomas, cancer, etc.)
Glottic stenosis, e.g. posterior and subglottic stenosis, webbing
Paradoxical vocal fold movement (PVFM)

Inspiratory & Expiratory (Biphasic) Stridor -- Usually Tracheal Obstruction

Extrinsic compression from neck masses or mediastinal masses, e.g., thyroid cancer
PVFM with asthma

Expiratory Stridor (Wheezing) -- Usually Intrathoracic Obstruction

Asthma and chronic obstructive lung disease (COPD)
Benign and malignant tumors at or near the thoracic inlet

Incidentally, the diagnosis *vocal cord dysfunction* (VCD) should probably be abandoned as a nebulous and clinically worthless term.⁵ It is rather like using a vague and non-specific term like “airway obstruction.” With all of the conditions that fall under PVFM (“VCD”), specific anatomic and etiologic diagnoses are needed if the subsequent treatment is to be effective. [The differential diagnosis of paradoxical vocal fold movement](#) is summarized in **Table 3**.

TABLE 3: DIFFERENTIAL DIAGNOSIS OF PVFM

Laryngopharyngeal reflux (LPR)
Respiratory-type laryngeal dystonia
Asthma-associated laryngeal dysfunction
Brainstem abnormalities, e.g., Chiari malformations
Drug-induced dystonic reactions, e.g., phenothiazines
Psychogenic stridor (Rare)

CAUSES OF NON-PULMONARY CHRONIC COUGH

Cough is a vagally-mediated reflex, the general purpose of which is to protect the airway by the forceful expulsion of material from the larynx, trachea and lungs. The material may be exogenous (foreign) liquid/food that has been aspirated, or it may be endogenous (autologous), product of normal or variously altered biophysiology e.g., mucus, phlegm, inflammatory exudate. Cough associated with primary lung disease is usually productive.

Sometimes cough is unproductive and appears to have no apparent defensive function. It may be the troublesome, inappropriate and disruptive symptom of an occult (somewhat difficult-to-diagnose) medical condition. Non-pulmonary cough has many causes (**Table 4**).

TABLE 4: AUTHOR'S TOP 10 CAUSES OF (NON-PULMONARY[†]) CHRONIC COUGH

1. Laryngopharyngeal reflux (LPR) aka *extraesophageal reflux*^{1,5-8}
2. Neurogenic cough (e.g., post-viral vagal neuropathy)^{2,3}
3. Asthma / Allergy / Sinusitis / Post-nasal drip^{5,9}
4. Aspiration due to glottal insufficiency (paralysis)
5. Angiotensin converting enzyme (ACE) inhibitors⁹
6. Zenker's or distal esophageal diverticulum
7. Esophageal achalasia
8. Tracheoesophageal fistula
9. Occult bronchial foreign body
10. Tracheobronchial (e.g., carcinoid) tumor

Honorable mention: Cerumen impaction, Ortner's syndrome,¹⁰ *Syngamus laryngeus*,¹¹ esophagopharyngeal reflux¹²

[†] This article is not about COPD, bronchitis, bronchiectasis, smoking, viral, bacterial or fungal infections or pneumonia, benign or malignant pulmonary tumors, tuberculosis, sarcoidosis, or any other granulomatous or primary pulmonary (lung) diseases. This table represents the author's differential diagnosis of "corticosteroid-resistant non-eosinophilic cough."

Chronic cough is so ubiquitous and multifactorial that its diagnosis and treatment are fragmented. In addition to pulmonologists, the medical doctors who see patients with chronic cough include family practitioners, pediatricians, infectious disease specialists, allergy/immunologists, gastroenterologists, and otolaryngologists. Each field has a different approach and bias, and no one specialty has sovereignty over the chronic cough patient.

There are some bizarre and interesting causes of chronic cough; my personal favorite is *Syngamus laryngeus*.¹¹ A small, round-worm indigenous to the Caribbean, *Syngamus laryngeus* is acquired by ingesting a contaminated fruit or vegetable. Apparently, a pair of worms, a male and female, take up residence in the subglottic larynx; and there they remain tenaciously adherent to the mucosa, except when mating. Thus, the pair may be coughed up *in copula*; otherwise they can be endoscopically removed with resolution of the host's chronic cough.¹¹

Other uncommon otolaryngologic causes of chronic cough. A large, cerumen impaction can account for chronic cough. The mechanism is vagal nerve stimulation via the afferent branch to the ear known as *Arnold's Nerve*.

Proximal- (*Zenker's*) and distal-esophageal diverticula and *esophageal achalasia* can cause chronic cough by the aspiration of the undigested contents of the sac, or in the case of achalasia by the esophageal contents, i.e., by *esophagopharyngeal reflux*.¹² The key element of the history for those conditions is that the patients almost always have cough when they lie down.

Ortner's syndrome is intermittent left vocal fold paresis/paralysis as a result of cardiac ptosis.¹⁰ When the heart is edematous and in failure; it pulls down on the ipsilateral recurrent laryngeal nerve. On one occasion, I have seen such a condition associated with chronic-intermittent cough.

Laryngopharyngeal-Reflux-Related Cough

There are many reports associating chronic cough with reflux (LPR and GERD); however, a causal relationship is not always easily established. Because there are multiple mechanisms for reflux-related cough (**Table 5**); no single testing method may be diagnostic. It is not clear, for example, that the refluxate must reach the larynx to cause cough. In performing pH monitoring or esophageal impedance testing, we have found some patients have cough associated with esophageal reflux without actual LPR. The mechanism of cough is still presumably vagally-mediated, stimulated by esophageal afferents triggered by esophageal distension.

Notwithstanding the above exceptional situation, in my practice most patients with chronic cough have LPR. The initial diagnosis is based upon the history and clinical parameters, [reflux symptom index](#) and [reflux finding score](#); the definitive diagnosis is made by *ambulatory 24-hour (simultaneous pharyngeal and esophageal) pH monitoring* (or esophageal impedance testing). In either case, an event marker is employed so that we establish if symptoms correlate with actually-measured reflux events.

Indeed, the *symptom index* discussed here is different from the *reflux symptom index* above. The *symptom index for pH monitoring* is an important element of pH-testing for which the patient is instructed to press an *event marker* on the pH recording device when a symptom occurs, e.g., when coughing. When the data are analyzed, the relationship between reflux events and cough events are analyzed. The *symptom index* is expressed as a percentage with the number of reflux-related events being the numerator and the total number of symptom events being the denominator. The threshold (pH criteria) for pharyngeal reflux have changed. In the past, we only considered a pH <4 drop to be significant; at present we consider reflux pH <5 events.

In the near future, we will examine all reflux events, because with chronic cough, even neutral-pH reflux, can be problematic. In practice, as many as 10-15% of chronic my cough LPR patients don't improve on "maximal" medical treatment and eventually require antireflux surgery. Over the years I have had cases that did not stop coughing until after fundoplication.

I do not always get diagnostic reflux testing on the front end. If the patient has symptoms and findings of LPR, and assuming that he/she has not already been on high-level ("maximal") medical treatment, I will put him/her on twice-daily proton pump inhibitors (before the morning and evening meals) and an H2-antagonist at bedtime and reasonable dietary and lifestyle modifications. Then, I'd reserve reflux testing (later on) for apparent medical treatment failures.

When needed, the reflux testing must be done right. In my opinion, single-probe pH-monitoring (esophageal or pharyngeal) is inadequate. I recommend pharyngeal/UES/esophageal manometry followed by ambulatory 24-hour (simultaneous pharyngeal and esophageal) pH monitoring. Incidentally, having used impedance testing for two years, the data appear no more useful than those of pH monitoring using expanded pH threshold criteria; see the August 2008 Visible Voice, [Laryngopharyngeal Reflux: A Religious War](#). If anything, I prefer dual-sensor (pharyngeal and esophageal) pH monitoring to impedance testing, because with pH monitoring, pharyngeal reflux events are measured. Impedance doesn't work in the pharynx.

Illustrative Case Example 1: LPR-Related Chronic Cough. A 63-year-old physician presented in April 2008 with a 23-year history of chronic cough. She would cough every night when she lay down; and she'd frequently awake from sound sleep coughing. She'd also intermittently cough during the day. She saw many physicians, and never received an effective treatment. One otolaryngologist treated her for LPR with proton pump inhibitors (PPI), but she didn't improve. In February 2008, she had impedance-pH-monitoring that showed (1 acid and 1 "non-acid") proximal reflux events; the study was reported as normal, as was a barium swallow/esophagram.



Figure 1: Chronic Cough Case 1 -- Initial Laryngeal Examination (March 2008)

Right [vocal fold paresis](#) and LPR. The RFS was 12 (normal <5); including pseudosulcus, partial ventricular obliteration, edema, and erythema

May 22, 2008, I performed high-definition pharyngeal/UES/esophageal manometry that revealed moderately-severe esophageal dysmotility; and the lower esophageal sphincter (LES) showed an intermittent achalasia pattern; it didn't open with more than half of the swallows. Ambulatory 24-hour dual-sensor (simultaneous pharyngeal and esophageal) pH monitoring showed three episodes of delayed pharyngeal acid exposure, long-after esophageal acid was detected by the esophageal sensor, suggesting *esophagopharyngeal reflux*.¹² The patient was initially treated with Nexium 40 mg b.i.d. and ranitidine 150 mg q.h.s., but she didn't stop coughing, but when baclofen 10 mg t.i.d. was added, she did.

Comment: Most LPR-related chronic cough cases are not this complicated; and most respond to antireflux treatment alone. In this case, esophageal motor and LES dysfunction were presumably improved by baclofen, and apparently that was the difference that made the difference.

Neurogenic Cough

I frequently see patients who are referred with recalcitrant "LPR" who have pristine laryngeal examinations. Such patients' symptoms are typically (burning) sore throat and/or chronic cough. Certain foods and fumes typically trigger symptoms. The laryngeal examinations usually show evidence of [vocal fold paresis](#) but no LPR. Some of these patients feel very certain that the problem is LPR, but reflux testing turns out to be normal. I refer to this syndrome as *pseudoreflux* or *pseudo-LPR*. This group of patients has *neurogenic cough* and/or *neurogenic sore throat* due to neuropathic vagal dysfunction.

Illustrative Case Example 2: Neurogenic Cough. In 1999, a thin, otherwise healthy 19-year-old woman presented to my clinic with chronic cough, sore throat, globus, and throat discomfort after speaking. Two years prior, all of her symptoms had begun following a typical URI. Three weeks after the URI, she was hoarse, coughing incessantly, and was diagnosed by an ORL as having severe LPR. Typically, she would cough all day long, but never at night during sleep. She reported that she “had a hair-trigger for cough,” that anything like laughing, yelling, or chuckling would cause coughing, as well as exposure to perfumes and diesel fuel fumes. After almost a year being unresponsive to maximal antireflux treatment, she underwent antireflux surgery (laparoscopic fundoplication). Unfortunately, her symptoms did not improve after the surgery.



Figure 2: Chronic Cough Case 2 -- Initial Laryngeal Examination
Mildly hypomobile (paretic) right vocal fold, but no inflammation (LPR)

On laryngeal examination, she had a hypomobile right vocal fold, but no findings of LPR. There was sluggish mobility (adduction) of the right vocal fold and increased amplitude on stroboscopy suggesting decreased tone. Laryngeal electromyography (LEMG) showed “old” bilateral recurrent laryngeal neuropathies, and pH testing was negative. She was successfully treated for with gabapentin 100 mg q.i.d. and amitriptyline 10 mg q.h.s.

Comment: When certain particular symptoms – chronic cough, sore throat, especially a burning throat sensation, or odynophonia (painful speaking) – are out of proportion to the laryngeal findings, the problem is probably neurogenic, e.g., PVVN. These neuropathic conditions are sometimes referred to as “hyperirritable vagus syndrome” or “sick nerve syndrome.”

My criteria for diagnosis of “neurogenic cough” are: (1) Almost constant during-the-day, non-productive (idiopathic) cough with or without a history of antecedent URI; (2) Unilateral or bilateral vocal fold paresis on laryngeal exam; (3) Laryngeal electromyographic confirmation of paresis; and (4) Response to treatment with resolution of symptoms.

Treatment of neurogenic chronic cough is somewhat different than other neuropathic conditions. For isolated cough (no-LPR), I usually start treatment with tramadol 25 mg q.i.d. p.r.n. or amitriptyline 10 mg q.h.s. If LPR is also present or there are other symptoms, then I tend to start with gabapentin 100 mg q.i.d. and then escalate the dose as needed (usually to 300-500 mg q.i.d.) depending on the side-effects and results. Those medications may be used alone or in combination, and the most common combination is gabapentin with a small dose of amitriptyline at bedtime. In my practice, pregabalin and baclofen are second-line choices for special situations.

Post-Viral Vagal Neuropathy

After 30 years as a laryngologist with research interests in LPR and laryngeal electromyography, I have concluded that LPR and laryngeal neuropathy (alone or in combination) are the commonest causes of chronic cough, and many patients have an apparent viral etiology.

Post-viral vagal neuropathy (PVVN) provides an important clinical and theoretical connection between laryngopharyngeal reflux and vocal fold paresis. PVVN offers a causal link for different manifestations of vagal dysfunction. It makes good common sense although the diagnosis PVVN is difficult to prove. Unfortunately, there is no test that proves a viral infection caused disruption of various vagal branches or that aberrant regeneration after such neuropathy can cause pain.

I have been using the term *post-viral vagal neuropathy* since the 1980s, but we didn't publish it until 2001.² We wanted to call our paper by that name, but the reviewers thought the term PVVN was conjectural. As an alternative, we came up with [*Vagal Neuropathy after Upper Respiratory Infection: A Viral Etiology?*](#)

Five years later, Jeyakumar *et al.*³ from the Cleveland clinic published a paper *Effectiveness of amitriptyline versus cough suppressants in the treatment of chronic cough resulting from postviral vagal neuropathy*. Not only did they not give us credit for the term and concept of PVVN, they actually plagiarized from our paper. Example:

“Cranial nerves are known to be affected by inflammatory neuropathic processes. Bell's palsy, trigeminal neuralgia, and glossopharyngeal neuralgia are examples of such cranial neuropathies. These represent isolated nerve injuries that result in motor or sensory dysfunction (e.g., paralysis, pain) depending on the nerves affected. Other cranial neuropathies have also been described. Of particular interest to the otolaryngologist is the existence of vagal neuropathy.”

Here's the most relevant case from our original paper (*Am J Otolaryngol* 22:253, 2001) that emphasizes the relationship between the neuropathic event and the onset of both LPR and cough.

“A 50-year-old woman presented in April 1999 with a 5-month history of hoarseness, vocal fatigue, effortful phonation,odynophonia, cough, dysphagia, globus, and heartburn. All of these symptoms began after a viral URI with severe cough. Persistent chronic cough and dysphagia were her most troubling symptoms. Before her illness, the patient denied ever having any of the above symptoms, and her past medical history was otherwise noncontributory.”

“Examination showed the typical inflammatory findings of LPR and sluggish mobility and bowing of the left vocal fold. Chest radiographs were normal. The patient underwent LEMG, which confirmed a left thyroarytenoid muscle paresis (ie, left recurrent laryngeal neuropathy). Double-probe pH testing was grossly abnormal, with the finding of severe esophageal and pharyngeal acid exposure. Esophageal manometry was also abnormal.”

“In this case, the onset of severe LPR after URI suggests the possibility that viral inflammation may involve vagal branches supplying the esophageal body and its sphincters.”

Leaving aside the plagiarism and inappropriate-citation-of-references issues by the authors, the 2006 Laryngoscope paper³ is an interesting paper that supports the premises of this article.

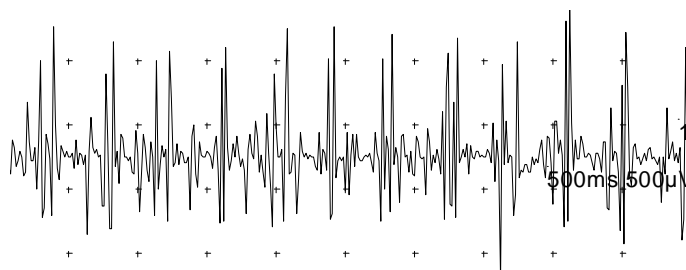
It is now clear that chronic cough patients can have post-viral (PVVN) with or without LPR, and that in almost all of those cases, the usual findings are clinical and electromyographic vocal fold paresis. Sometimes treatment of LPR will effectively stop the cough; sometimes it won't.

Illustrative Case Example 3: Post-Viral Vagal Neuropathy. A 39-year-old banker presented with a thirteen-month history of chronic cough, having had an URI at the onset. His cough was triggered by voice use. In addition to cough, he complained of voice changes, vocal fatigue, post-nasal drip, and chronic throat-clearing. His glottal closure index was 16 (normal <10), and his reflux symptom index was 20 (normal <15). Laryngeal examination showed right vocal fold paresis (hypomobile, foreshortened, and increased amplitude on stroboscopy suggesting decreased tone), and the LPR. The reflux finding score was 11 (normal <5); see below.



Fig. 1: Laryngeal Examination (Right Vocal Fold Paresis)

Laryngeal electromyography – bilateral testing of the cricothyroid muscles (supplied by the superior laryngeal nerves) and the thyroarytenoid muscles (supplied by the recurrent laryngeal nerves) – showed an “old” diffuse laryngeal polyneuropathy with the right recurrent division being most severely (1-2+ recruitment) affected (see below). There was no evidence of any ongoing denervation or reinnervation in any of the muscles tested.



**Fig. 2: Representative Laryngeal Electromyography (Right Thyroarytenoid Muscle)
Reduced (2+) recruitment with high-amplitude polyphasic motor units**

Patient was treated with ranitidine and relatively small doses of gabapentin (100 mg q.i.d.) with improvement of his cough; four weeks later, when the gabapentin dose was escalated to 500 mg q.i.d., the patient’s cough completely disappeared.

Comment: In this case, I targeted the neuropathic element and not so much the LPR, because the primary precipitant of the patient’s cough was voice use; that’s a neurogenic symptom pattern.

Post-viral vagal neuropathy is a group of syndromes. Voice, swallowing, and other vegetative vagal functions (i.e., cough), may be involved. Theoretically, PVVN may play a role in other conditions such as gastroparesis, irritable bowel syndrome, or possibly even cardiac problems. I

believe that viral neuropathic syndromes are probably much more important and common than the understanding that the current state of the art recognizes.

Even if the patient has no clear history of having had a viral illness, it is sometimes logical to postulate PVVN as a diagnosis, for example when a healthy young adult rather suddenly develops vocal symptoms of vocal fold paresis and LPR. In such cases, laryngeal electromyography (LEMG)¹³ will usually show a diffuse polyneuropathic pattern (without spontaneous activity that implies on-going neural degeneration),^{2,13} an LEMG pattern that is characteristic of PVVN; see also [VV on LEMG](#) and [Wake Forest LEMG Experience paper](#).¹³

Chronic cough is a common symptom in medical practice, and its association with reflux (LPR) is still in large measure unproven. Nevertheless, it is likely that PVVN is an important cause of LPR. It is also likely that viral neuropathy with aberrant neural regeneration an important cause of neurogenic cough. The prevalence and mechanisms of LPR and chronic cough, as well as all of the other vagal neurogenic syndromes remain to be explored.

TABLE 5: POSSIBLE MECHANISMS FOR NON-PULMONARY CHRONIC COUGH

Laryngopharyngeal reflux	Posterior laryngeal inflammation (reflux laryngitis) Stimulation of supraglottic laryngeal receptors Endolaryngeal aspiration of gastric contents
Gastroesophageal reflux disease	Stimulation of vagal receptors by the refluxate Stimulation of esophageal stretch receptors (e.g., Eructation, bolus of the refluxate) Tracheobronchial aspiration of gastric contents
Upper respiratory infection	Acute inflammation of the laryngopharynx
Laryngeal/vagal neuropathy	Acute neuropathy due to viral inflammation
Post-viral vagal neuropathy	Aberrant neural regeneration following viral neuropathy; “hyperirritable vagus syndrome”

References

1. Koufman JA. The otolaryngologic manifestations of gastroesophageal reflux disease (GERD): Laryngoscope 101 (Suppl. 53):1-78, 1991
2. Amin MR, Koufman JA. Vagal neuropathy after upper respiratory infection: a viral etiology? Am J Otolaryngol 22:251-256, 2001.
3. Jeyakumar A *et al.* Effectiveness of amitriptyline versus cough suppressants in the treatment of chronic cough resulting from postviral vagal neuropathy. Laryngoscope. 116:2108-12, 2006.
4. Pavord ID, Chung KF, Management of chronic cough. Lancet 371:1375-84, 2008.
5. Koufman JA, Block C. Differential diagnosis of paradoxical vocal fold movement. Am J Speech Lang Pathol. 17:327-34, 2008.

6. Loughlin CJ, Koufman JA. Paroxysmal laryngospasm secondary to gastroesophageal reflux. *Laryngoscope* 106:1502-1505, 1996.
7. Loughlin CJ *et al.* Acid-induced laryngospasm in a canine model. *Laryngoscope* 106:1506, 1996.
8. Little JP *et al.* Extraesophageal pediatric reflux: 24-hour double-probe pH monitoring of 222 children. *Ann Otol Rhinol Laryngol Suppl* 169: 1-16, 1997.
9. Spector SL. Chronic cough: The allergist's perspective. *Lung*. 186 Suppl 1:S41-7, 2007.
10. Sengupta A, *et al.* Ortner's syndrome. *J Laryngol Otol*. 112:377-9, 1998.
11. Weinstein L, Molavi A. Syngamus laryngeus infection (syngamosis) with chronic cough. *Ann Intern Med*. 74:577-80, 1971.
12. Belafsky PC, Rees CJ, Rodriguez K, *et al.* Esophagopharyngeal reflux. *Otolaryngol Head Neck Surg* 138:57-61, 2008.
13. Koufman JA *et al.* Diagnostic laryngeal electromyography: the Wake Forest experience 1955-1999. *Otolaryngol Head Neck Surg* 124:603-606, 2001.

CASE OF THE MONTH



What is the most likely diagnosis?

- A. Cervical osteophytes
- B. Vertebral tuberculosis
- C. Metastatic small cell tumor
- D. M. Miles' granulomas
- E. Odontogenic cysts

Answer next month

[Answer for last case, October 2008 – D. Distal esophagus, C. Barrett's esophagus]

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