

# THE VISIBLE VOICE

A Newsletter for Physicians, Speech-Language Pathologists,  
Professional Voice Users, and People with Voice Disorders

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## WHY AND HOW PROFESSIONAL SINGERS AND ACTORS LOSE IT

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Even though voice problems in singers and actors seem to occur suddenly, in fact, they may start slowly over a prior period of months or years; and often in retrospect, there may have been protracted warning symptoms that were minimized or ignored. The “obvious” causes of voice problems may be bogus. Acute voice strain is infrequently the underlying cause of acute vocal collapse; although having a perennially vocally-demanding occupation is a key risk factor.

*Note to the reader: I recognize that phonotrauma from chronic hard-use is a kingpin variable; however, the point of this article is that occult (hidden, undiagnosed) underlying neuromuscular and inflammatory diseases are often central to the process and progression of vocal destabilization, independent of “vocal technique” and vocal abuse/misuse/overuse issues.*

Many singers and actors blame themselves for “over-doing it” when something goes wrong. In taking the medical history of a Broadway performer, I am struck by how often I hear a string of self-deprecating *non sequiturs* that frequently end with, “and I know that I shouldn’t have done that.” “Thats” may refer to booze, marijuana, other drugs, dietary indiscretions, such as a chocolate binge or a pepperoni pizza, yelling or singing loudly, or singing/acting during a cold.

It is true that screaming can cause acute vocal fold hemorrhage or that a fudge-bomb brownie can exacerbate reflux laryngitis (laryngopharyngeal reflux, LPR); however, in truth most performers take good care of themselves most of the time; although vocal professionals are often inappropriately enchanted by the magic of holistic and herbal medicine. Frequently, I have patients ask me about the benefits of vitamins, herbs, enzymes, acupuncture, etc. to treat their voices, especially for LPR, but to my knowledge, none have been shown to be therapeutic.

### **Help! I Lost My Voice**

For performers, any significant voice loss is a medical emergency. But what brings a singer or actor to the office in a panic? The most common *bona fide* out-of-the-blue precipitant of acute problems is the upper respiratory infection (URI). The medical term *laryngitis* -- often mistaken

as a synonym for hoarseness – generally refers to a viral infection of the larynx (voice box). Viral laryngitis can cause mild, moderate or severe hoarseness; it typically lasts 3-7 days; and it can be nasty enough to cause cancellation of performances by a sick singer. More often, however, the show can go on with some modifications in the program. Doctors sometimes use an injection of anti-inflammatory steroid to reduce vocal fold swelling, which may improve the voice for few days. Laryngitis accounts for few patients seen at the Voice Institute of New York. What are the underlying causes of voice disorders?

### **Voice Disorders Are Usually Multifactorial**

Recently, I reviewed (IRB-approved protocol) the medical records of 80 unselected, consecutive of my patients with voice disorders. There were 31 males and 49 females with a mean age 56 years; and 81% (65/80) were professional voice users.

Eighty-eight percent (70/80) of the population had hyperkinetic biomechanics (muscle tension dysphonic) on transnasal flexible laryngoscopy (TFL). By clinical and/or pH-monitoring criteria 71% (57/80) had laryngopharyngeal reflux (LPR). Of those, it was the primary diagnosis in 40% (23/57) and an incidental, secondary diagnosis in 60% (34/57).

Excluding mild, asymptomatic presbylaryngis (“old age voice”), 55% (44/80) had neuromuscular disease. Of those, 7 patients had spasmodic dysphonia, 6 had vocal fold paralysis, and 26 had vocal fold paresis. It was the primary diagnosis in 52% (23/44) and it was an incidental, secondary diagnosis in 48% (21/44). Fifty percent (40/80) of the patients had histopathologic lesions including nodules, polyps, cysts, pseudocysts, papillomas, granulomas, dysplasia and carcinoma. Some lesions were incidental, e.g., some small granulomas.

The data of **Table 1** indicate that the average patient had 2.68 different diagnoses. As mentioned, in some cases, paresis, reflux, or a vocal fold lesion were primary diagnoses, and in others cases, they were secondary or incidental findings (diagnoses).

**Table 1: Multifactorial Causes of Voice Disorders (N = 80)**

Hyperkinetic biomechanics (e.g., abnormal laryngeal tension)	88%
Inflammatory disease (e.g., reflux and respiratory infections)	75%
Neuromuscular disease (e.g., paralysis, paresis, SD, tremor)	55%
Neoplastic growths (e.g., polyps, nodules, papillomas, cysts)	50%
<b>Total</b>	<b>268%</b>

Interestingly, 28% (22/80) of the patients had onset of glottal closure symptoms that were associated with a self-identified precipitating event: 15 had a history of upper respiratory infection (URI); 7 dated the onset of symptoms to the time of endotracheal intubation (a breathing tube for surgery) or surgery of the head, neck, or thyroid.

In addition, over half of the vocal professionals dated the onset of symptoms to a performance event. However, while voice strain was reported to be “the precipitant” in many patients, those same patients were virtually always also found to other diagnoses (as shown in **Table 1**).

## Vocal Decompensation and the Last Straw Principle

As it turns out, most singers and actors who present with seemingly sudden-onset voice problems actually have had preceding chronic symptoms that may have been minimized. Some of these often-ignored warning signs are summarized in **Table 2**.

### **Table 2: Warning Signs That the Voice / Larynx May Need Attention**

Effortful or painful speaking  
 Vocal fatigue at the end of the day  
 Loss of the high range of the voice  
 Hoarseness of the speaking voice  
 Voice breaks in the singing mid-range  
 Chronic throat-clearing and/or too much throat mucus  
 Chronic or morning cough, awaking from sleep coughing  
 Difficulty swallowing and/or a sensation of a lump in the throat

Many vocal professionals wait until they experience acute decompensation before seeking medical attention. As you might expect, most voice problems are best prevented. While I do not wish to over-intellectualize here, **Table 3** is my general theory (model) of life and vocal collapse.

### **Table 3: The Laws of Decompensation**

1st Axiom: Before: The composition of a system is in dynamic balance  
 1st Corollary: Conflicting elements are held together by function and purpose

2nd Axiom: Decompensation is preceded by often ignored warning signs  
 2nd Corollary: During early destabilization, imbalance is assessable (measurable)

3rd Axiom: Last straw principle: When threshold is exceeded, collapse occurs  
 3rd Corollary: Recovery requires stabilization and repair of all essential elements

The *Laws of Decompensation* provide a loose framework for approaching the practice of laryngology, because for practical purposes, excluding congenital abnormalities and rare diseases, voice disorders have just five (interrelated) etiologies:

- (1) **H**yperkinetic laryngeal behaviors (aka muscle tension dysphonia)
- (2) **I**nflammation
- (3) **N**euromuscular disease
- (4) **N**eoplasia (including benign, malignant, and traumatic growths)
- (5) **T**rauma, i.e., phonotrauma that causes striking-zone pathology, and “growths” (that include nodules, polyps, cysts, and granulomas)

I refer to this as the HINNT model, and I try to evaluate each of those factors in every patient, every visit. As a result, I actively look for that which is still hidden, and I am willing to discuss potential trouble before it manifests, even though that makes some people uncomfortable.

The overarching principle is that vocal decompensation occurs when compensation fails. In other words, decompensation is a threshold event. Not only that, the interactions of (adaptive and

maladaptive) behavioral vocal use patterns with vocal fold inflammation and glottal closure problems, are the common pathway to decompensation.

### **HINNT: Voice Disorders are Complex and Cumulative**

Under normal circumstances, the vocal folds come together along their lengths, like two hands clapping on a hinge. Additionally, phonation (voicing) depends upon a steady stream of (subglottic) air pressure provided by the bellows of the lung/diaphragm apparatus.

Voice quality is largely dependant upon the symmetry of vibrating layered structures of vocal folds, and the glottis (the two vocal folds) acts in many ways like a double-reed instrument. The oscillation of the soft-tissue reeds should be symmetrical in both frequency and amplitude. That is the normal physiologic state.

In patients who develop vocal fold pathology, there is a common vicious cycle, a cascade of events: Increased laryngeal muscle tension leads to phonotrauma leads to increased vibratory vocal fold swelling and stiffness, which leads to further increases in laryngeal/neck muscle tension and subglottal driving pressures. This pressure-tension-trauma cycle escalates until further compensatory increases in subglottal pressure and muscle tension are no longer possible.

The term *phonotrauma* almost defines itself, “trauma of the vocal folds caused by phonation, vibration of the vocal folds to produce voice.” The striking-zones of the vocal folds by design impact each other on average thousands of times during one minute of speaking or singing. Is speaking and singing then intrinsically phonotraumatic? If the striking-zones of the vocal folds are colliding millions of times a day, why doesn't everyone have a voice problem?

The elements of the HINNT model are interactive. For example, patients with mild vocal fold paresis can manage for many months or years until they subsequently develop something else, too, like LPR. **Table 4** lists common precipitants of vocal decompensation, the “last straw” list.

#### **Table 4: Common Precipitants of Vocal Decompensation**

- Laryngopharyngeal reflux
- Upper respiratory infection
- Increasing vocal demands
- Change of occupation
- Endotracheal intubation
- Head and neck surgery
- Allergy and/or asthma

One of the greatest risk factors for vocal decompensation for singers (and actors, too) is success. How so? A singer/actor who is just barely surviving vocally doing a few performances per week may have to quadruple (or more) his or her voice demands, and thereby exceed the threshold for decompensation. This happens especially often to singers who go on tour with heavy performance schedules. Furthermore, it is really hard to eat a healthy diet when one is on tour. With travel and late-night eating, LPR frequently worsens, and this may be the “final straw” that precipitates vocal decompensation.

## Laryngopharyngeal Reflux (LPR)

The term *reflux* literally means *backflow* from the Latin *re-* (back) and the verb *fluere* (to flow). LPR is the backflow of stomach (gastric) contents into the throat (the laryngopharynx). [LPR is often silent](#), occurring without digestive symptoms such as heartburn or regurgitation; it does commonly cause hoarseness, difficulty swallowing, a sensation of a lump in the throat, too much throat mucus, and chronic cough. The [reflux symptom index](#), a validated outcomes instrument, is shown in **Appendix A**. If the sum of your symptoms (the RSI) is <10, you may have LPR and you should consider seeing a doctor.

Most people with voice disorders have *laryngopharyngeal reflux* (LPR), and it is the commonest cause of hoarseness and other throat symptoms. Approximately [half of voice patients have LPR](#). Usually LPR can be diagnosed by the laryngeal examination, but sometimes special testing is needed. LPR affects as tens of millions of Americans over age 50 years, and it commonly begins when people are in their thirties. Nevertheless, we do see LPR in young people, even in children and teens. (LPR is an important topic in laryngology and it has been the author's main research interest for 25 years; several of [my publications related to LPR](#) may be found on my website.)

## Vocal Fold Paresis

[Vocal fold paresis](#) refers to “partial paralysis” of the vocal folds. Such paresis may be associated with vocal fold weakness, thinning, atrophy, or bowing. In fact, I sometimes use the terms interchangeably; although strictly speaking there are differences. “Old age,” presbylaryngis, is associated with age-related vocal fold atrophy alone; whereas paresis implies a neuropathic (“sick nerve”) condition as the cause of muscle loss (atrophy). A [post-viral vagal neuropathy](#) that may be likened to *Bell's palsy of the larynx*, appears to be a common cause of bilateral (both-sided) vocal fold paresis. Another common cause of vocal fold paresis is an idiopathic (unknown) cause; see **Table 5**.

**Table 5: Causes of Vocal Fold Paresis (Partial Paralysis)† (N=415)**

Neck/chest surgery	128	31%
Viral infection*	66	16%
Neurologic disorder	38	9%
Neck/chest neoplasm	35	8%
Endotracheal intubation	34	8%
Neck/chest trauma	17	4%
Idiopathic*	97	23%
Total	415	100%

† Koufman *et al.* [Otolaryngol Head Neck Surg 124:603, 2001](#). This series may not be representative of the true prevalence of the different causes of paresis, because of referral patterns of the author. In other words, the percentage of patients with iatrogenic paresis may be over-represented. \* In most series, the groups would be combined making the idiopathic group (39%) the largest.

The [laryngeal findings of vocal fold paresis](#) on videostroboscopy are asymmetrical vocal fold bowing, hypomobility (decreased mobility) of one or both arytenoids (the moving parts), and asymmetry of videostroboscopy. Unilateral increased vibratory amplitude, a floppy vocal fold, may be the most common finding of paresis, but the diagnosis of paresis requires laryngeal EMG (electromyography) for confirmation.

Vocal fold paresis is common; about half of my patients seeking medical attention for a voice disorder have it. The consequences of paresis are related its severity, which nerves and muscles are affected, and the degree of impairment of vocal fold closure. Superior laryngeal nerve paresis may be associated with loss of the high range of the voice; and recurrent laryngeal nerve paresis tends to be associated with loss of power/volume and vocal fatigue. Indeed, paresis is associated with other glottal closure symptoms such as odynophonia (painful speaking) as well. The glottal closure index is shown in **Appendix B**. If the sum of your symptoms is <10, you may have vocal fold paresis and you should consider seeing a doctor.

To reiterate: From a biomechanical point of view, people with paresis have to compensate for incomplete glottal closure by squeezing the vocal folds together using extra-laryngeal muscles. The increased work of compensatory behaviors leads to increased vocal fold tension, trauma, and stiffness; and consequently, subglottal driving pressures must be increased. Thus, a vicious cycle ensues in which increased vocal fold tension and stiffness requires increased driving pressures. At some point, increased sheering-forces in the striking-zone overwhelm tissue pliability and injury occurs. Indeed, most striking-zone pathology is the result of compensatory laryngeal behaviors and not simply the result of vocal misuse.

### Conclusions

Professional singers and actors make high biomechanical demands on the larynx, and the development of phonotraumatic pathology appears to be multifactorial. The model of vocal decompensation presented in this article assumes that people generally compensate effectively for paresis and LPR for as long as possible. The combination of effortful closure and tissue inflammation and its sequelae leads to phonotrauma. The kind of striking-zone pathology that results from the paresis/LPR duo includes vocal nodules, polyps, cysts, and sulcus (scarring).

Like the rest of the body, wear and tear on the vocal folds, is cumulative. In my experience, it is a rare vocalist or actor who has a completely normal laryngeal examination after age 40. The keys to career longevity appear to be good genetics, good scheduling, and all-around good preventive maintenance. The old adage about diet, exercise, and moderation applies here.

### APPENDIX A: THE REFLUX SYMPTOM INDEX (RSI)

Within the last MONTH, how did the following problems affect you?	0 = No Problem    5 = Severe Problem						RSI
	0	1	2	3	4	5	
Hoarseness or a problem with your voice	0	1	2	3	4	5	
Clearing your throat	0	1	2	3	4	5	
Excess throat mucous or postnasal drip	0	1	2	3	4	5	
Difficulty swallowing food, liquids, or pills	0	1	2	3	4	5	
Coughing after you ate or after lying down	0	1	2	3	4	5	
Breathing difficulties or choking episodes	0	1	2	3	4	5	
Troublesome or annoying cough	0	1	2	3	4	5	
Sensations of something sticking or a lump in your throat?	0	1	2	3	4	5	
Heartburn, chest pain, indigestion, or stomach acid coming up?	0	1	2	3	4	5	

**APPENDIX B: THE GLOTTAL CLOSURE INDEX (GCI)**

Within the last MONTH, how did the following problems affect you?	0 = No Problem      5 = Severe Problem						GCI
	0	1	2	3	4	5	
Speaking took extra effort	0	1	2	3	4	5	
Throat discomfort or pain after using your voice	0	1	2	3	4	5	
Vocal fatigue (voice weakened as you talked)	0	1	2	3	4	5	
Voice cracks or sounds different	0	1	2	3	4	5	

**CASE OF THE MONTH – WHAT IS IT?**

- A. Granulomas
- B. Papillomas
- C. Sessile polyps
- D. Sjorgren's syndrome
- E. Laryngopharyngeal reflux

Answer next month

[Answer for last case, April 2008 – E. Pharyngeal polyps]

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