Laryngopharyngeal Reflux and Singers: Diabolus in Gula?

Martin L. Spencer

LARYNGOPHARYNGEAL REFLUX (LPR) occurs when gastric juices, chiefly hydrochloric acid and the enzyme pepsin, seep out of the stomach, traverse the esophagus, and irritate the voice box (larynx) and throat (pharynx). LPR is a common, if not the most common source of laryngeal pathology, and may be a contributor to disorders ranging from slight, but distressing voice change to laryngeal cancer.¹

Persons experiencing reflux report differing symptomatic presence and intensity. Singers affected by LPR typically describe difficulties such as increased tension, vocal fatigue, harder tonal onset, reduced quality, persistent throat clearing, loss of high range, and register dissociation.² Additional reflux-related symptoms may include speaking voice changes (particularly in the morning), sore throat, hacking cough, nocturnal choking, metallic taste, swallowing difficulty, and throat “tickle” or fullness.³ To aid in diagnostic verification, a physician or voice pathologist will correlate these symptoms to characteristic tissue changes (see Figure 1). Note that allergies, infection, and postnasal drainage from rhinosinusitis may produce similar symptoms; clinical dialogue and judicious usage of treatment modalities should parse out the relative contributions of each potential source of irritation.

Singers are particularly predisposed to reflux through sustained diaphragmatic compression of the stomach during “supported” singing, frequent late night eating, and physiologic manifestation of performance stress.⁴ Reflux-based laryngeal inflammation may be relatively widespread in both symptomatic and asymptomatic singing instructors.⁵

Laryngopharyngeal Reflux Versus Gastroesophageal Reflux

Gastroesophageal reflux disease (GERD) is presently a more conspicuous media presence via “the Purple Pill” than its relatively unknown cousin, laryngopharyngeal reflux. Although GERD and LPR may be considered as separate diagnostic entities, intervention modalities for both kinds of reflux are similar. Also, one condition is not necessarily a precursor to the other. It is important to note that many patients suffering from LPR do not present with esophagus-related heartburn, or stomach upset/ulceration.⁶ Indeed, many patients may exclaim, “I don’t have reflux . . . I don’t have heartburn!” when first diagnosed with LPR. Reflux may selectively affect relatively delicate throat tissue to the exclusion of tougher esophageal tissue.
Causes of Laryngopharyngeal Reflux

The upper esophageal sphincter is a thickened muscular ring, posterior to the larynx, which primarily facilitates food entry into the gullet (Figure 2). The lower esophageal sphincter primarily seals digestive processes within the stomach. Secretions must infiltrate through both rings to cause LPR:

- Chemically, through smoking and certain foods that may relax the grip of the lower esophageal sphincter; spicy or acidic foods may compound existing tissue irritation (see Figure 2 for further specification).
- Mechanically, through factors such as obesity-related abdominal pressure, tightly fitted clothing, carbonated drinks, and recumbent or inverted posture (e.g., sleeping or gardening).

As the muscular esophagus is caudally propulsive, it is possible that muscular dysmotility, transient sphincteric relaxations, or weakened architecture of the cardia (gastroesophageal junction) may facilitate reflux movement. In a contrasting manner, increased life stressors or nervousness may overly activate the digestive system and precipitate reflux.

Gravity-fed reflux flow during recumbent sleep has been significantly implicated in reflux excursion. Therefore, eating within three hours of bed is cautioned as digesting food has not fully passed into the small intestine within this interval. Structurally, suboptimal stomach alignment along a relatively horizontal axis and "weak" architecture of the cardia (gastroesophageal junction) also may facilitate reflux. Hiatal herniation, in which the LES and adjacent stomach protrude through the diaphragm, has been implicated in reflux via destabilization of the peri-sphincter cradle.

Research into pathologic linkage of the digestive and respiratory systems has yielded solid evidence of relationships between reflux and asthma through "silent" microaspiration and connecting vagal innervation of the esophagus and bronchi. More remotely, links have been found between reflux, sinusitis, and inner ear inflammation (otitis media). These far reaching reflux effects are logical when one considers that the stomach is an intensely harsh environment in which from three to four and a half liters of hydrochloric acid are secreted on a daily basis, in addition to numerous digestive enzymes. (Acidity refers to the sufficient presence of protons, also called hydrogen ions, in a compound to lower...
pH <7.0.) Somewhat surprisingly, an acidic environment is not necessary for the effective digestion of food; note that modern reflux medications are very effective at suppressing acid production. The purpose of stomach acidity is to comb out potentially harmful microbes in our food, and activate pepsinogen into the primary digestive enzyme, pepsin.

**Healthy Larynx**

Entrance to esophagus
Open vocal folds for breathing
Closed vocal folds for phonation

**Reflux Signs**

Infraglottic edema
TVF erythema/edema
Posterior cricoid edema
(Reflux dispersion indicated by arrows)
Infrrarytenoid pachydermia
Characteristic buckling of pachydermia
Granuloma
Pre-granuloma

Healthy laryngeal mucosa (moist skin), adjacent to esophageal entry, should drape finely over the underlying muscular and cartilaginous frame. Physical findings indicative of LPR include tissue edema (swelling), erythema (redness), pachydermia (tissue thickening), and granuloma (benign inflammatory lesion). Granuloma, or early stage pre-granuloma, is generally found on the medial arytenoid fascia (inner and back-placed vertical laryngeal walls). Other signs that may be associated with LPR include ulceration, infraglottic edema (swelling around the vocal folds), pseudosulcus (swollen vocal folds which may appear to be horizontally divided), tracheal stenosis (airway narrowing), vocal fold webbing, and ventricular atrophy (loss of tissue above the vocal folds).

**Figure 2. Healthy larynx and reflux signs.**

**Treatment of Laryngopharyngeal Reflux**

There are two contrasting philosophic approaches to reflux-related pharmacotherapy: "stepping up" from a small dosage of a less powerful medication to increasingly find an adequate level of control, or "stepping down" from a more aggressive dosage and/or type of medication.14

A proton pump inhibitor (PPI) irreversibly jams intracellular acid production sites (proton pumps) within gastric parietal cells. This powerful class of medication includes Nexium, Prilosec (both prescriptive and OTC), Prevacid, AcipHex, and Protonix. As PPIs bind only to active pumps they should be taken 30 to 60 minutes before a meal to optimize absorption rate. (Note that accompanying product literature approves of Protonix and AcipHex intake without regard to timing of meals, whereas it is specified that Nexium and Prevacid should be taken one hour before eating, and that Prilosec OTC be taken before eating without further specification.) PPIs offer the most effective mechanism for acid suppression, yet may also produce the most adverse reactions in users; ≥1% experience headache, diarrhea or constipation, abdominal pain, flatulence (gas), nausea/vomiting, or dry mouth.15

Relatively less aggressive and, correspondingly less effective H2-receptor agonists ("H2-blockers") such as Zantac, Tagamet, and Pepcid exert a different mechanism of acid control via suppression of histamine, a chemical messenger that activates proton pumps. A disadvantage of sustained H2-blocker usage, especially in comparison with PPIs, is that their effect rapidly wanes (an effect called tachyphylaxis) via "rebound" acid hypersecretion.16 Both PPIs and histamine...
blockers are effective for between one half to three quarters of a 24 hour period, therefore double dosing each day may be necessary.\textsuperscript{17}

Common antacids have a buffering effect, and create a foam barrier in the upper stomach and lower esophagus which aids in reflux containment for up to ninety minutes. (Remember mixing an acid and base in chemistry?) They are the least aggressive class for reflux intervention, offering rapid symptomatic relief, particularly after a meal, but with little effect on overall disease progression.\textsuperscript{18} There may be additional merit to a drug such as Pepcid Complete which combines both a histamine blocker and antacid. Likewise, in refractory reflux it is possible to combine both a PPI 30 to 60 minutes before breakfast and before dinner with an after dinner H\textsubscript{2} blocker. This aggressive strategy may ensure acid production blockage via overlapping PPI duty cycles, with expanded H\textsubscript{2}-blocker control of nocturnal acid "breakthrough."\textsuperscript{19} (Breakthrough is defined as one hour of continuous intragastric pH <4.0.) Cases that continue to be refractory to this schedule may be judged suitable for transnasal esophagoscopy and/or GI (gastrointestinal) referral.

All the aforementioned medication classes are now available over the counter (Prilosec OTC is a half dosage of standard Prilosec); however, it would be most prudent for a singer to seek initial medical evaluation as a physician will be the best judge of course duration, timing of medication intake across the day, and level of control. Note that there is little difference in the mechanism of activity within each class, and therefore patients should utilize a prescription that is in the preferred tier specified by their particular insurer.

Chewing gum, particularly containing bicarbonate, has been implicated in reducing reflux acidity (note that normal saliva is slightly alkaline).\textsuperscript{20} Walking after meals may even be beneficial to the reflux-prone population.\textsuperscript{21} Basic recommendations from the American Academy of Otolaryngology specify head of bed elevation and antacid use prior to sleep.

**Long Term Reflux Control**

A recent comprehensive survey of LPR intervention recommended a minimum three month period of combined proton pump inhibitor and behavioral change before further reassessment of intervention strategy.\textsuperscript{22} ENT clinicians would not generally view several weeks of usage as an adequate interval in which to judge intended effect.

We do not have basic laboratory research beyond one year on the long term effects of gastric acid suppression (e.g., the author has not encountered discussion of how such suppression permits adequate pepsinogen activation for digestion). However, it is common for many reflux patients to use related medications for many years. All pharmaceutical products are potentially harmful; the Greek word \textit{pharmakon} indicates the potential of a medicine to act both as a remedy and a toxin. Adverse reactions to medication usage, and contraindications with other medications and medical conditions are far too numerous to list here. It may be preferential to control reflux activity, particularly when emerging, with behavioral targets such as dietary change, avoidance of late night eating, weight loss, smoking cessation, or stress reduction.\textsuperscript{23} Note that intended drug effects may be undermined if these precipitating conditions are not addressed. Novel approaches to long term reflux management include treatments aimed at decreasing transient LES relaxations (using Baclofen), and endoscopic antireflux procedures.\textsuperscript{24}

If the aforementioned methods of reflux control have not been effective, then alternative routes of exploration may center on esophageal dysmotility, structural defect, severe hiatal herniation, absence of H. pylori, or bile reflux.\textsuperscript{25}

**Antireflux Surgery**

Reflux-related surgery is an important current topic in long term control; the common premise across varying techniques is to reinforce LES structure. There is much conflicting research concerning the effectiveness of GERD- and LPR-related surgeries. The issues chiefly address continued postsurgical need for pharmacotherapy—with many advocates claiming no such need.\textsuperscript{26} There are also those who are ambivalent about surgical benefits,\textsuperscript{27} and the more recent emergence of detractors claiming little to no benefit as regards LPR-specific conditions.\textsuperscript{28}

Should GERD-related surgery be performed, it is noted that benefits may be relatively short term, and that physician-related surgical experience and careful patient selection may reduce complications and postsurgical reliance on PPIs.
The surgeries fall into two basic procedural types, the Nissen fundoplication and more recent emergence of noninvasive techniques:

1. In a Nissen fundoplication the upper compartment of the stomach, the fundus, is sutured or "wrapped" around the distal esophagus. (A plication is a folding of tissue.) Two methods of access may be utilized:
   - Traditional "open" approach (first used in the early 50s);
   - Laparoscopic/"keyhole" surgery (greatest popularity in the 90s and reportedly waning in the 00s).29

2. Endoscopic techniques in which an intraluminal approach to the gastroesophageal junction is made through the natural course of the esophagus. These outpatient procedures have been developing over the past decade, and are correspondingly in a preliminary phase of study (all the noted methods below have been FDA approved except for the Wilson-Cook Endoscopic Suturing Device).30 It is anticipated that evolution of current technologies will lead to more efficacious and common methodology.31 Two differing approaches are utilized:
   a. LES augmentation. Further subdivided into:
      1) The Stretta procedure which utilizes radiofrequency thermal bombardment to toughen ("denature") LES muscular tissue. The procedure has been noted to significantly reduce transient LES relaxations, albeit with a less significant increase in basal LES pressure.32 The Stretta procedure has been the most studied intraluminal technique: a three year follow-up has indicated only "modest" effectiveness.33
      2) Tightening of the sphincteric ring, by circular implantation of a biopolymer, the Enteryx procedure.34
   b. Sutured folding of the cardia ("valvuloplasty") via procedures such as EndoCinch, Full-thickness Plicator, and the Endoscopic Suturing Device. Overall discontinuation rate for PPI usage after these procedures, when performed for GERD, is currently about 70%.35

CONCLUSION
Reflux of gastric juices into the base of the throat is a very common source of laryngeal complaint. Symptomatic presence and intensity vary from individual to individual, and heartburn is not a qualifying factor. Relatively subtle injury may significantly impact a singer, and this article is intended to promote a greater awareness within the singing community of up-to-date reflux intervention protocols. The author stresses that antirefluxogenic behavioral and dietary controls should minimize the need for medications, and maximize the intended effect of medications. Readers of this article are strongly advised to seek medical evaluation via endoscopy and interview should atypical throat or voice problems persist for longer than several weeks.

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NOTES


17. Ford; Ulsalp and Touhill; Sandage and Zelazney; Harrill, et al.


22. Ford; Ulsalp and Touhill; Sandage and Zelazney; Harrill, et al.

23. Rosen and Murry.


33. Lutfi, et al.


35. Ozawa, et al.

**Martin Spencer** is a voice pathologist working with two surgical groups, Ohio ENT Surgeons and Central Ohio ENT, in Columbus, Ohio. He is President of the Ohio Voice Association (OVA), and has organized three recent state-wide conventions. His scope of activities includes voice diagnostics and rehabilitation, with a specialty in professional voice. He received his vocology track MA from the University of Iowa under the mentorship of Ingo Titze. Articles include “Hybrid Voice Therapy” in...
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Journal of Singing, "An Investigation of a Modal-Falsetto Register Transition Hypothesis Using Helox Gas" in the Journal of Voice, and "Foibles of the Singing Voice" in Perspectives on Voice and Voice Disorders. Amongst various conference papers have been several concerning the Voice Category Template and Ratio system (VCTR) at the annual Voice Symposium and the Second International Conference on Physiology and Acoustics of the Singing Voice. Martin has lectured on vocology, vocal health, rehabilitation of the singing voice, and voice clinic activity in many forums, including university medical and singing programs, state conventions, and Opera Columbus.

Martin was a professional singer for twenty years prior to graduate studies in speech pathology, and has performed to critical acclaim with the Canadian Opera Company, Toronto Symphony, Sadler's Wells Opera, Stratford Festival, Elmer Isler Singers, and Tafelmusik. In Toronto, he was a professor in Humber College's Jazz Studies program and organized create-an-opera programs for the COC.

Other educational venues he attended include the University of Toronto (music history and basic sciences), Banff School of Fine Arts (two years in the Music Theater Studio Ensemble), and the Britten-Pears School for Advanced Musical Study in Aldeburgh, England (where he studied with Sir Peter Pears).

Martin is now an avid barbershopper with the renowned Singing Buckeyes; he also sings lead in the quartet, Rhymetime.

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