

Laryngopharyngeal reflux: Position statement of the Committee on Speech, Voice, and Swallowing Disorders of the American Academy of Otolaryngology–Head and Neck Surgery

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The term *reflux* (derived from the Latin words *re* [“back”] and *fluere* [“to flow”]) literally means *back-flow*. The term *gastroesophageal reflux* (GER) refers to the backflow of stomach contents into the esophagus. GER may be physiologic, and indeed up to 50 GER episodes a day, occurring mostly after meals, is accepted as being within the normal range.¹⁻³ *Gastroesophageal reflux disease* (GERD) is a clinical term that refers to GER that is excessive and that causes tissue damage (eg, esophagitis) and/or clinical symptoms (eg, heartburn).¹

Laryngopharyngeal reflux (LPR) refers to the backflow of stomach contents into the throat, that is, into the laryngopharynx. There are numerous synonyms for LPR in the medical literature; the most accepted of these terms is *extraesophageal reflux* (Table 1). Most commonly, patients presenting to gastroenterologists have GERD, but most otolaryngologic patients have LPR. LPR differs in many ways from classic GERD.^{1,4-9}

LPR DIFFERS FROM CLASSIC GERD

Patients with LPR appear to have different pathophysiologic mechanisms and patterns of reflux, as

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well as different symptoms, manifestations, and responses to treatment compared with patients with GERD.^{1,4-14} The most significant difference between LPR and GERD is that the majority of patients with LPR do not have esophagitis or its primary symptom, heartburn.^{1,4,6,9} Indeed, in a number of studies, the incidence of heartburn in patients with LPR is <40%, and the incidence of esophagitis is approximately 25%.^{1,4-6} Thus, the vast majority of patients with LPR do not have esophagitis, the diagnostic sine qua non of GERD.^{1,6,9}

It appears that the mechanisms of LPR are different from those of GERD.⁴⁻⁹ Patients with LPR are predominantly upright (daytime) refluxers, whereas GERD patients are predominantly supine (nocturnal) refluxers.¹⁻⁶ There are prolonged periods of acid exposure in GERD but not in LPR.^{1,2} In addition, patients with GERD have dysmotility and prolonged esophageal acid clearance, whereas those with LPR do not.^{1,7} It is believed that the primary defect in GERD is lower esophageal dysfunction, whereas the primary defect in LPR is upper esophageal sphincter dysfunction. It is likely that these differences in mechanisms and patterns account for the differences in symptoms and manifestations of LPR and GERD. It is important to note that although most patients with LPR do not have GERD, some patients do indeed have both LPR and GERD.

SYMPTOMS OF LPR

The symptoms of LPR are protean; however, the most common are hoarseness, globus pharyngeus, dysphagia, cough, chronic throat clearing, and sore throat (Table 2). These symptoms are often intermittent or “chronic-intermittent.”

CLINICAL MANIFESTATIONS

The most common manifestation of LPR is reflux laryngitis with or without granulation or gran-

Table 1. Synonyms for laryngopharyngeal reflux

Reflux laryngitis
Laryngeal reflux
Gastropharyngeal reflux
Pharyngoesophageal reflux
Supraesophageal reflux
Extraesophageal reflux
Atypical reflux

uloma formation.¹¹⁻¹⁷ In addition, reflux has been reported to be associated with subglottic stenosis, laryngeal carcinoma, polypoid degeneration, laryngospasm, paradoxical vocal fold movement, and vocal nodules^{1,4,6,10-24} (Table 2). Other manifestations in the head and neck that have been reported include asthma, sinusitis, and otitis media.²⁵⁻³⁰ In addition, pediatric LPR is now receiving considerable attention.²⁹⁻³⁴

It has been estimated that up to half of patients with laryngeal and voice disorders have reflux.³⁵

Table 2. Symptoms and clinical manifestations reported to be related to laryngopharyngeal reflux

Symptoms	Conditions
Chronic dysphonia	Reflux laryngitis
Intermittent dysphonia	Subglottic stenosis
Vocal fatigue	Carcinoma of the larynx
Voice breaks	Endotracheal intubation injury
Chronic throat clearing	Contact ulcers and granulomas
Excessive throat mucus	Posterior glottic stenosis
"Postnasal drip"	Arytenoid fixation
Chronic cough	Paroxysmal laryngospasm
Dysphagia	Paradoxical vocal fold movement
Globus	Globus pharyngeus
Intermittent airway obstruction	Vocal nodules
Chronic airway obstruction	Polypoid degeneration
Wheezing	Laryngomalacia
	Pachydermia laryngis
	Recurrent leukoplakia
	Sudden infant death syndrome
	Sinusitis
	Otitis media
	Sleep apnea
	Exacerbation of asthma

The prevalence of LPR in other disorders of the head and neck remains unknown.

DIAGNOSIS

The diagnosis of LPR can be made on the basis of the symptoms and laryngeal findings,^{1,4,36} but ambulatory 24-hour double-probe (simultaneous esophageal and pharyngeal) pH monitoring remains the gold standard for the diagnosis of LPR when the diagnosis is in question.^{1-3,37-39} In addition, double-probe pH testing is often used to evaluate drug efficacy. Other diagnostic tests, such as barium esophagography or esophagoscopy, are far less sensitive for LPR.^{1,40} Even though barium studies and esophagoscopy are not usually used to diagnose LPR, it may be advisable to screen the esophagus for related pathology with one of these methods.^{1,9}

TREATMENT

H₂-antagonists and proton pump inhibitors (PPIs) have been used to treat both GERD and LPR.⁴¹⁻⁵³ In general, treatment for LPR needs to be more aggressive and prolonged than that for GERD.^{1,4,14-17,50-55} It has been shown that as few as 3 experimental LPR episodes a week can result in severe laryngeal damage.^{1,20} The larynx is more susceptible to reflux injury than the esophagus, because it lacks both the extrinsic and intrinsic epithelial defenses of the esophagus.^{1,20,56}

The type of treatment is dependent on the symptoms and severity of LPR and on the patient response to treatment. Patients with mild and/or intermittent symptoms can be treated with dietary and lifestyle modifications as well as with H₂-antagonists such as ranitidine.^{1,4,14,17} The majority of patients with LPR, however, require at least twice-daily dosing with PPIs.^{4,36,50-53,57} The reason for the twice-daily dosing is that none of the PPIs exert acid suppression (intra-gastric pH >4) for >16.8 hours.^{51,52} In some patients, it is necessary to treat with both a PPI and an H₂-antagonist.⁵⁰

Within 2 to 3 months of treatment, most patients report significant symptomatic improvement; however, it takes 6 months or longer for the laryngeal findings of LPR to resolve.^{57,58} Thus, twice-daily dose PPI treatment is recommended for a minimum initial treatment for a period of 6 months in many patients with LPR. Some patients

may require prolonged tapering and/or chronic treatment. As an alternative to medical treatment, fundoplication has been shown to be an effective therapy for LPR.^{59,60}

CONCLUSIONS

Patients with LPR have upper aerodigestive manifestations of reflux disease rather than classic GERD (ie, esophagitis and its sequelae). The manifestations and symptoms of LPR are different because the mechanisms and patterns of reflux are different than those of GERD. For most patients with LPR, twice-daily dosing with a PPI is recommended for an initial treatment for a period of no less than 6 months, and lifetime treatment may be required.

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