Voice, Swallowing and Reflux diagnostic and practice guidelines for the SLP

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I have no professional or monetary connection to Restech. However, I DO depend on St. Cloud Ear, Nose & Throat for my livelihood!
Learning Objectives – Part I

1. Participant will be able to discriminate between the two major classifications of reflux as defined by location – GERD and LPR.

2. Participant will be able to identify signs/symptoms of reflux pertinent to each type of reflux – differential diagnosis.

3. Participant will identify and interpret established screening questionnaires with regard to reflux per patient report.

4. Participant will be able to delineate main signs of reflux tissue changes using videolaryngoscopy.
What is reflux?

- Reflux refers to back flow or retrograde flow of gastric contents through the LES into the esophagus (GER) and possibly through the UES into the pharynx/larynx (LPR). When the situation becomes chronic, it is typically associated with disease (GERD). (Vaezi, 2012)

- What can flow back?
  - Food, gastric digestive acids, pepsin, trypsin, mucus, bile salts, air
  - Gastric acid (hydrochloric acid) is typically at a pH level of 1.5-3.5. It's role is to activate digestive enzymes which break down protein.
  - Reflux of pepsin and bile as well as mucus are considered nonacidic (alkaline) reflux though may be included with gastric (acidic) refluxate.
What is gastric (hydrochloric) acid?

Gastric acid is one of the main secretions of the stomach. It consists mainly of hydrochloric acid and acidifies the stomach content to a pH of 1 to 2.

Chloride (Cl\(^{-}\)) and hydrogen (H\(^{+}\)) ions are secreted separately in the stomach fundus region at the top of the stomach by parietal cells of the gastric mucosa into a secretory network called canaliculi before it enters the stomach lumen. (cited in Wikipedia – see below)


What function does gastric acid serve?

Gastric acid acts as a barrier against microorganisms to prevent infections and is important for the digestion of food. Its low pH denatures proteins and thereby makes them susceptible to degradation by digestive enzymes such as pepsin. The low pH activates the enzyme precursor pepsinogen into the active enzyme pepsin by self-cleavage. After leaving the stomach, the hydrochloric acid of the chyme [semi-fluid digested food] is neutralized in the duodenum by sodium bicarbonate.

The stomach itself is protected from the strong acid by the secretion of a thick, protective mucus layer, and by secretin induced buffering with sodium bicarbonate. Heartburn or peptic ulcers can develop when these mechanisms fail.
Why do we reflux?

- The most common cause of gastroesophageal reflux disease involves dysfunction of the lower esophageal sphincter (LES), a 3-4 cm high-pressure zone of muscular activity in the distal esophagus. (Mitre & Katzka, 2004)

  - A higher muscle tone to maintain tonic contraction is the result of higher intracellular calcium levels in the resting LES compared to the proximal esophagus.

  - Mitre and Katzka (2004) identified three main defects leading to LES incompetence: 1) transient LES relaxations (the most common cause of reflux in GERD (63-74%) patients and normal subjects (70-100%); 2) a chronically hypotensive lower esophageal sphincter, and 3) the effects of a hiatal hernia on sphincteric function.
Transient LES Relaxation

- LES relaxation is normal following a pharyngeal swallow as a response to primary esophageal peristalsis (relaxing for 5-10 seconds) and as a reflex response to secondary peristalsis with the distention of both esophageal striated and smooth muscle.

- Transient LES relaxations can occur following a normal swallow-induced relaxation or spontaneously on a background of stable LES pressure and can last longer than 10 seconds.

- LES competence is further diminished with the inhibition of 1) the crural diaphragm; 2) primary peristalsis, and 3) the gastric fundus.

- LES relaxation can be a physiologic response to gastric distention leading to gastric-gas venting and avoidance of gastrointestinal bloating – may occur with belching, vomiting, rumination.

- Primary cause of post-prandial (after meal) reflux; less common in supine position or with sleep.

- GERD patients have greater amount of reflux during transient LES relaxations (60-70%) compared to norms (40-50%) (Mitre & Katzka, 2004)
The lower esophageal sphincter keeps closed (except opening transiently during swallowing to allow swallowed food to pass into the stomach) to prevent reflux (backflow) of stomach acid into the esophagus. Apart from the lower sphincter, several other mechanisms including the crural diaphragm form the antireflux barriers. Although some reflux is normal, excessive reflux of stomach acid damages the esophagus and the throat structures producing gastroesophageal reflux disease (GERD). (Source: Mittal and Balaban. Copyright © 1997, Massachusetts Medical Society.) From: GI Motility online (2006) doi: 10, 1038/gimo 14.
Transient LES Relaxation and Its Relationship to GERD

- Patients with GERD experience:
  - a higher frequency of liquid reflux events,
  - a qualitative difference in reflux content (more acidic),
  - a greater cross-sectional opening of the esophagogastric junction,
  - higher liquid flow rates across the junction, and perhaps higher numbers of relaxations, particularly postprandially when compared to controls.
Hypotensive Lower Esophageal Sphincter

- Primarily neurogenic and myogenic failure of the LES muscle.
- An increase in inhibitory mediators including nitric oxide and a decrease in calcium release from intracellular storage (calcium channel blockers) can lead to LES hypotension.
- If the LES is hypotense, reflux can be associated with 1) stress (exercise, coughing/sneezing, pregnancy) overwhelming the LES, and 2) free reflux – travel of refluxate across the LES without restraint.
- Free reflux can be transient or, more rarely, constant and can occur in response to a myriad of foods or drugs.
- Scleroderma can result in severe LES hypotension as well as ineffective esophageal peristalsis – both leading to GERD, regurgitation and dysphagia as well as Barrett's esophagus. (Mitre & Katzka, 2004)
Factors Decreasing LES Pressure (Mitre and Katzka, 2004)

<table>
<thead>
<tr>
<th>Foods</th>
<th>Neural Agents</th>
<th>Other</th>
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<tbody>
<tr>
<td>Chocolate</td>
<td>β-Adrenergic agonists</td>
<td>Theophylline (used for asthma, COPD)</td>
</tr>
<tr>
<td>Ethanol</td>
<td>α-Adrenergic antagonists</td>
<td>Smoking</td>
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<tr>
<td>Peppermint</td>
<td>Anticholinergic agents</td>
<td>Morphine, meperidine</td>
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<td>Caffeine</td>
<td></td>
<td>Calcium-blocking agents</td>
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<td></td>
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<td>Diazepam (used for anxiety, seizures)</td>
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<td>Hormones</td>
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<td>Dopamine</td>
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<tr>
<td>Cholecystokinin</td>
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<td>Vasoactive intestinal polypeptide</td>
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<td>Somatostatin gastric inhibitory polypeptide</td>
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<td>Progesterone</td>
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Tobacco and Alcohol

“Tobacco and alcohol adversely influence almost all of the body's antireflux mechanisms. They delay gastric emptying, decrease both upper and lower esophageal sphincter pressures and esophageal motility, decrease mucosal resistance, and increase gastric acid secretion, and thus, strongly predispose one to reflux.”

In consequence, some physicians recommend antireflux treatment for all patients with laryngeal neoplasia, with or without other risk factors. (Postma & Halum, 2006)
Associated GERD with Hiatal Hernia [1]

There are two major kinds of hiatus hernia:

- The most common (95%) is the sliding hiatus hernia, where the gastroesophageal junction moves above the diaphragm together with some of the stomach.

- The second kind is rolling (or paraesophageal) hiatus hernia, when a part of the stomach herniates through the esophageal hiatus and lies beside the esophagus, without movement of the gastroesophageal junction. It accounts for the remaining 5% of hiatus hernias. [2]

A third kind is also sometimes described, and is a combination of the first and second kinds.

Associated GERD with Hiatal Hernia

From: Merck Manual Home Health Handbook
Sliding Hiatal Hernia and its Relationship to GERD

- Most patients with a hiatal hernia do not have GERD
- Size of a hiatal hernia (>3cm) is associated with increased acid exposure
- Majority of severe esophagitis and Barrett's esophagus patients have a hiatus hernia of 2cm or greater
- A sliding hiatal hernia can disrupt the alignment of the crural diaphragm and lead to reflux by three mechanisms:
  - 1) disruption of the “pinchcock effect” of the crural contraction
  - 2) lowering LES pressure, and
  - 3) creation of a wider cross sectional opening of the esophagogastric junction  (Mitre & Katzka, 2004)
Highlights

Transient LES relaxation
- Most common cause of reflux
- Triggered by proximal gastric distention
- Associated with mild GERD

Hypotensive LES
- Low resting sphincter pressure (<10 mmHg)
- Associated with stress and free reflux
- Related to severe GERD in patients with scleroderma

Sliding hiatal hernia
- Pathophysiologic factor in at least 50% GERD cases
- Increases likelihood of reflux from low LES pressure, deglutitive relaxation, and abdominal muscle straining
- GERD severity correlates to hernia size (Mitre and Katzka, 2004)
GERD – Symptoms and Complications

- Typical symptoms include heartburn and regurgitation, less typically chest pain, dysphagia
- Esophageal GERD-related complications can include:
  - Peptic stricture/mucosal Schatzki Ring with possible esophageal shortening (implicated in dysphagia, weight loss, odynophagia, heartburn, regurgitation, globus and chest pain)
  - Esophagitis and Esophageal ulcers
  - Barrett's esophagus (roughly 10% of esophagitis patients) with possible additional intestinal metaplasia
  - Adenocarcinoma – cancer of esophageal epithelium
  - Asthma (vagally mediated)
Laryngopharyngeal Reflux (LPR) – Symptoms

- Also referred to as Extraesophageal, Esophagopharyngeal or Supraesophageal Reflux as well as “silent reflux” due to absence of heartburn symptoms

- Associated voice and swallowing symptoms can include:
  - **Voice** (hoarseness, glottal fry, pitch breaks, loss of singing range, laryngitis)
  - **Throat/Airway** (sore or burning throat, cough, increased throat clearing often with increased phlegm and PND, dyspnea, wheezing/stridor)
  - **Dysphagia** (food sticking in throat/increased difficulty clearing food, globus pharyngus (lump in throat sensation both with swallowing of secretions and/or solids/liquids), chest pain (noncardiac), water brash (increased salivation, sometimes in response to nausea)
LPR – Signs and Complications

• Associated or suggested complications can include:
  
  - edema (swelling), erythema/hyperemia (redness) of larynx/vocal folds
  
  - laryngeal pathology (nodules, polyps, contact ulcers, granuloma, interarytenoid changes (pachydermia), posterior glottic stenosis, subglottic stenosis, Reinke's edema, hyperkeratosis/leukoplakia, tumors, laryngeal cancer),

  - chronic cough, chronic bronchitis, pulmonary fibrosis, pneumonitis, laryngospasms/VCD, dental erosions, rhinitis, sinusitis and obstructive sleep apnea

  - dysphagia, including possible implications in fibrosis of UES leading to UES dysfunction, Zenker's diverticulum

  - implications as possible etiology for SIDS, otitis media (from Eustachian tube to middle ear)
Pathophysiologic Mechanism in LPR

- Two proposed mechanisms -
  - Microaspiration (liquid and/or gaseous aerolization) of gastric or duodenal contents
    - Protective structures include: LES and UES, peristaltic actions of the esophagus, acid neutralization by saliva, protective airway reflexes (esophagoglottal closure and cough), mucociliary clearance, release of bicarbonate
  - Stimulation of a vagal reflex arc (Harding, 2006)
    - Associated with LPR, cough, asthma
Caustic Offenders – Response and damage

- Caustic offenders which can impact negatively on laryngeal/pharyngeal structures and mucosa include:
  - Gastric acid
  - Pepsin – a primary cause of mucosal damage or inflammation during LPR, due to activation of pepsin's digestive enzyme activity in the presence of acid (either refluxed or ingested) with subsequent mitochondrial damage and induction of cellular damage (depletes epithelial protective isoenzymes (sodium bicarbonate) (Bock et al., 2011, Blumin et al., 2012)
  - Duodenal Bile acids/salts and trypsin (through the pyloric valve into stomach and on through the LES) (often referred to as nonacidic reflux) (unresponsive to typical acid reflux therapy if acid not present)
Differences Between GERD and LPR

- As delineated by Postma and Halum (2006):
  - It is normal to have a certain amount of reflux into the esophagus – a diagnosis of GERD is not made unless the pt. exhibits over 45 reflux episodes or elevated acid with probe. In contrast, one or two episodes of reflux at or above the proximal (esophageal) probe is accepted as diagnosis for LPR
  - Obesity has a strong association with GERD, not LPR
  - Hoarseness is a pervasive symptom with LPR, not GERD
  - Esophageal acid clearance (motility) is usually normal with LPR, not GERD
  - LPR patients more likely to reflux upright; GERD in supine position
  - Esophagitis is pervasive with GERD; only 12% of LPR patients
GERD, LPR and Children

According to the American Academy of Otolaryngology-Head and Neck Surgery, infant “spitting up” is essentially LPR but is normal in the first year due to 1) the immaturity of both the UES and LES, 2) the shorter distance from the stomach to the throat and 3) greater amount of time infants are lying down. However, associated breathing/feeding problems need an evaluation by a specialist.

- Symptoms in children may include: chronic cough, hoarseness, stridor, croup, asthma, sleep disordered breathing, spit up, feeding difficulty, turning blue (cyanosis), aspiration, pauses in breathing (apnea), apparent life threatening event (ALTE), failure to thrive.

- Complications can include subglottic stenosis, hoarseness, vocal fold nodules, laryngospasm, globus, chronic cough, possible eustachian tube dysfunction with recurrent ear infections or persistent middle ear fluid, possible sinusitis, and tooth enamel erosion.

- Children may deny symptoms if they have had them since birth.

- Using child friendly descriptors (“sour burps”, “mini throw-ups” may be beneficial in initial diagnosis).

(Theis, 2011)