Introduction

Gastroesophageal reflux (GER) is defined as the retrograde movement of gastric contents into the esophagus; it is a physiologic process that occurs in everyone, young and old, particularly after meals. The most useful classification of GER divides the spectrum of reflux into 3 categories: functional GER, pathogenic GER (or Gastroesophageal Reflux Disease), and secondary GER.

Functional GER represents a benign condition that does not require evaluation or treatment. This does not cause inflammation, lead to long term complications, or affect growth and development. This category ranges from passage of refluxate into the distal esophagus to frequent regurgitation of gastric contents out of the mouth typically seen in infants.

GERD, in contrast, necessitates intervention. This class represents GER with associated complications, either typical (e.g., failure to thrive, anemia, esophagitis, Barrett’s esophagus) or atypical (e.g., wheezing, apnea, pneumonia, chronic sinusitis).

In Secondary GER, some underlying condition causes the retrograde movement of gastric contents. The appropriate treatment may be to address the underlying cause directly (e.g., pyloric stenosis), or to obtain control of GER (e.g., neurologic impairment). Other examples of conditions associated with secondary GER include metabolic defects, food allergy, infection, and nasogastric tubes.

The prevalence of GER depends on age. Approximately 50% of 0-3 month olds have at least 1 episode of regurgitation per day. This increases to a peak of 67% of infants at 4 months of age. This number drops to approximately 5% of 10-12 month olds. The sharpest drop occurs around age 6 months, which is associated with the development of improved neuromuscular control and the infant sitting up. However, only 10% of infants (i.e., <12 months old) with GER develop significant complications. Thus, the incidence of GERD is similar in infants, older children, and adults, as suggested by the GER iceberg.

Other numbers frequently cited in the literature regarding pediatric GER: GER spontaneously resolves in 55% of infants by age 10 months, and 81% by age 18 months.
In older children and adults GERD waxes and wanes, but is felt to be more resistant to complete resolution.

GER and reflux associated complications are more common in neurologically impaired children and premature infants.

Pathophysiology

Boix-Ochoa has identified 6 factors which comprise the antireflux barrier. All of these operate over an area from 3 to 7 cm and represent what is generally referred to as the Lower Esophageal Sphincter (LES):

1. **Pinch-Cock Action** – The right crus of the diaphragm forms a sling-shaped orifice around the esophagus. During deep inspiration, this pulls the esophagus to the right and downward with deep inspiration, thus narrowing the esophageal lumen. This is affected by paralysis of the diaphragm.

2. **Intraabdominal Esophagus** – Felt by Boix-Ochoa to be the key to the LES, and to successful fundoplication. It determines the length of esophagus exposed to intraabdominal pressure and, therefore, the length of “esophageal valve” holding back potential refluxate. A sufficient segment of abdominal esophagus is considered >2 cm; a mechanically incompetent LES has an abdominal length <1 cm, or an overall sphincter length <2 cm. At birth, the total length of the LES ranges from 0.5-1 cm and increases to 2.5-3 cm by 3 months of age. This will be shortened in hiatal hernia, short esophagus, gastrostomy, and esophageal atresia.

3. **Angle of His** – at the junction of the esophagus and the stomach. In a child with a normal-sized intraabdominal esophagus, this angle is acute. With an acute angle, when a child vomits, more contents strike the fundus than escape via the esophagus. The resulting pressure in the fundus further increases the acuity of the angle, shutting off the esophagus. In conditions such as short esophagus, hiatal hernia, and esophageal atresia the angle is obtuse, and the fundus actually acts as a funnel into the esophagus, allowing reflux at much lower intragastric pressures.

4. **Mucosal Rosette** – signifies redundant folds of mucosa at the gastroesophageal junction, present only when a normal angle of His is present. With increased intragastric pressure or negative intrathoracic pressure, these folds squeeze together to form a weak antireflux valve.

5. **High Pressure Zone** – an area of increased muscular thickness near the gastroesophageal junction, it is described as a manometric sphincter. The basal tone of the HPZ increases until 45 days of age, at which time maturation is complete. Of note, this tone reaches maturity at the same time regardless of gestational age; thus, an immature HPZ CANNOT explain the increased incidence of GER in preemies.

6. **Abdominal Pressure** – Intraabdominal pressure between 6-8 cm H2O is necessary to maintain a competent LES by collapsing the intraabdominal segment. Conditions where intraabdominal pressure is lacking (e.g., omphalocele, gastroschisis, muscular weakness) will result in GER. Under normal circumstances, elevated intraabdominal pressure will NOT cause reflux (not to be confused with intragastric pressure, which will cause reflux when elevated).
Maturation of the LES and gastroesophageal competence are achieved between 5-7 weeks of life, regardless of gestational age or birthweight. Thus, reflux is physiologic in infants less than 6 weeks old, and fundoplication is contraindicated prior to age 6 weeks.

The other factors important in GER relate to opening/relaxation of the LES. These include:
1. Increased intraabdominal pressure, felt to result in reflux only when the LES is relaxed, at least momentarily.
2. Failure to link increased LES pressure with gastric contractions, which can result in reflux while fasting.
3. Increased gastric volume, which is aggravated by delayed gastric emptying.
4. Lack of coordination between gastric peristalsis and opening of the pylorus, which results in delayed gastric emptying.
5. Increased tension of the gastric wall/gastric distention, which shortens the length of the intraabdominal esophagus.

Transient LES relaxations (TLESRs) are currently felt to be the primary explanation for GER events. TLESRs last from 5-30 seconds, and likely represent a mechanism for venting air from the stomach in response to gastric distention in normal individuals. These relaxations are mediated by a vagal reflex pathway that will result in GER when neural control is deranged. There is good evidence that esophagitis disinhibits TLESRs resulting in a vicious cycle of GER and esophagitis.

Esophagitis is the most common complication of GER. Acid and pepsin (low pH) are the primary offenders. Bile acids and trypsin (high pH), present in refluxate as a result of duodenogastric reflux, disrupt the mucosal barrier of the esophagus and increase permeability to hydrogen ions. Of note, gastroesophageal refluxate in infants is frequently of a neutral pH, with these alkaline compounds likely playing a significant role in infant esophagitis.

Factors protecting against esophagitis include:
1. Gravity – plays a very minor protective role unless esophageal peristalsis is defective. In fact, elevating the head of the crib is NOT felt to decrease GER in infants.
2. Local esophageal mucosal factors – e.g., mucus, prostaglandins.
3. Reflux Peristalsis – is the single most important factor in esophageal protection by returning refluxate to the stomach. Of note, esophagitis results in esophageal dysmotility, resulting in a vicious cycle which can be broken with treatment. Premature infants tend to have esophageal dysmotility and poorly coordinated swallow. It is for this reason that GER is thought to be more common in Preemies.
4. Saliva – Swallowed saliva, rich in bicarbonate, neutralizes gastric acid. Of note, GER associated wheezing is thought to be more common at night because, first, swallows virtually cease during sleep and, second, the rate of esophageal peristalsis is slowed.

Important factors that contribute to GER are psychomotor retardation – associated with dysmotility of the upper digestive tract in general, and prematurity.

In addition, several medications and foods can contribute to GER: theophylline, Beta agonists, calcium channel blockers, narcotics, benzodiazepines, anticholinergics, chocolate, fats, caffeine, licorice, carbonated beverages, alcohol, and nicotine.
### Presentation of GERD and Associated Complications

**SIGNS AND SYMPTOMS OF GASTROESOPHAGEAL REFLUX IN INFANTS**

<table>
<thead>
<tr>
<th>Typical</th>
<th>Atypical</th>
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<tr>
<td>Crying/Irritability</td>
<td>Apnea/Bradycardia (particularly preemies)</td>
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<tr>
<td>Poor appetite/Feeding resistance</td>
<td>Apparent Life-Threatening Events</td>
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<td>Regurgitation (vomiting less so)</td>
<td>Wheezing/Asthma</td>
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<td>Abdominal/chest pain (heartburn)</td>
<td>Stridor (subglottic stenosis)</td>
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<td>Failure to thrive/weight loss</td>
<td>Recurrent pneumonia</td>
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<td>Sore throat</td>
<td>Chronic cough</td>
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<tr>
<td>Waterbrash</td>
<td>Sandifer’s Syndrome</td>
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<tr>
<td>Hematemesis</td>
<td>Hoarseness/Laryngitis</td>
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<tr>
<td>Anemia</td>
<td>Otalgia</td>
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<td>Sinusitis</td>
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Based on data from the 1950’s, the course of untreated GERD in infants and children has been well documented. GERD resolved by 2 years of age in 60% of patients, but persisted until 4 years of age in 30%. Of those, ½ developed esophageal strictures, and the other ½ died from malnutrition.

That said, chronic complications of GERD typically seen in adults are relatively uncommon in children:

- **Esophageal stricture** – The incidence due to GERD in children is unknown; it usually presents as dysphagia to solids and is often accompanied by Barrett’s esophagus. Typically, these pt’s do not complain of reflux sx, and are thought to have experienced silent reflux. In children, stricture formation may take only a few weeks after the onset of reflux symptoms, so prompt tx is important.

- **Barrett’s Esophagus** – Again, the incidence in children is unknown. However, metaplasia that develops in childhood likely carries over into adulthood, continuing to increase in density until about age 40.

- **Adenocarcinoma** – Exceedingly rare. Fewer than a dozen cases of esophageal adenocarcinoma have been reported worldwide in children.

Significant crossover exists between the atypical presentations of GERD and the otolaryngologic manifestations of GERD. Much of this relates to gastronasal reflux and laryngotracheal reflux. These phenomena have been measured for various studies using dual pH probes with one probe in the distal esophagus and a second probe in the proximal esophagus or pharynx. No normative data exists in children for the more proximal probes, though many feel that ANY episode of pH<4 is abnormal; the nasopharynx and larynx do not enjoy the same protective mechanisms as the esophagus and, thus, are highly susceptible to injury from exposure to gastric contents. This more proximal reflux has been implicated in ALTEs, asthma, subglottic stenosis, etc. Treatment of GER has been reported to solve major therapeutic dilemmas in choanal atresia repair and pediatric chronic sinusitis.

#### 1. Chronic Sinusitis

At least 2 studies have explored the relationship between GER and chronic sinusitis in children. In the first (22), the author found that 63% of his pediatric patients (age 2-18) with chronic sinusitis had GER. 79% of patients with...
GERD and chronic sinusitis experienced significant improvement in their sinusitis sx with treatment for GER. In the second study (4), the author referred 28 consecutive pediatric FESS candidates to a pediatric gastroenterologist prior to surgery; all received medical tx for GER. 68% of those treated experienced resolution of their sinusitis sx. Between medical tx and fundoplication, 89% of the patients showed enough improvement to avoid FESS. In both of these studies, the pt’s had a high incidence of gastronasal reflux as documented by a dual pH probe study; however, sinusitis sx also improved in those pt’s with no documented gastronasal reflux – so a dual pH probe study is not needed for these patients. Thus, GER should be considered in all children with chronic sinusitis, and it may be advisable to treat all of these children empirically for GER before performing FESS. 2 months has been cited as a reasonable amount of time to judge the efficacy of GER tx in this setting.

2. Chronic Cough – In children with normal CXR’s and a cough of at least 1 month’s duration, GER was found to be the third most common etiology (15%) behind cough variant asthma and chronic sinusitis, both of which are frequently associated with GER. Of note, the most common cause in children <18 months of age was an aberrant inominate artery.


4. Globus Pharyngeus – a “lump in the throat”. Frequently associated with GER, the differential diagnosis is important in children: osteophytes of the spine, foreign body, cricopharyngeal hyperactivity, tonsillar hypertrophy, goiter, postcricoid web, cervical lymphadenopathy, or a mass in the pharynx or esophagus.

5. Oropharyngeal Dysphagia – i.e., impaired passage of food from the mouth to the upper esophagus. Some people have an acid-sensitive esophagus, i.e., vagal-mediated reflexes are triggered by the stimulation of esophageal chemoreceptors. This can trigger esophageal spasm/dysmotility, which is relieved by antacids. A similar mechanism has been used to explain bronchoconstriction in GER-induced asthma and GER-induced laryngospasm.

6. Chronic Sore Throat – Usually worse in the morning. It is unresponsive to abx, and throat cultures are negative.

7. Otalgia/Otitis Media – Chronic otalgia has been associated with GER, and often improves with GER tx. Anecdotal reports of chronic or recurrent otitis media secondary to GER have been published, but GER is not generally accepted as a cause of OM.

8. Vocal Cord Granulomas and Ulcers – The larynx is very sensitive to acid. Vocal cord granulomas will recur after surgical removal unless the underlying GER is treated.

9. Obstructive and Central Apneas/ALTEs – An Apparent Life Threatening Event (ALTE) is defined as an episode of apnea, color change, choking, gagging, or marked change in muscle tone in an attack frightening to the caregiver. 7-15% of children with ALTEs die secondary to SIDS. The most common immediate causes of ALTEs are central apnea, obstructive apnea, cardiac arrhythmia, and seizure disorder. In one study (15), 53% of children who had experienced ALTEs were found to have GER, and only ¾ of those children were evaluated for GER; it was, by far, the most common association. Other findings included laryngeal edema, subglottic stenosis,
and tracheomalacia, all of which can be associated with GER. Two mechanisms have been characterized to explain GER-induced apnea:

A) **Esophagolaryngeal Adductor Reflex** – vagally mediated. GER stimulates esophageal chemoreceptors, resulting in laryngospasm (Obstructive Apnea) accompanied by increased secretions, bronchoconstriction, tachycardia, and hypertension.

B) **Laryngeal Chemoreflex** – GER into the larynx directly irritates laryngeal mucosa, resulting in Central Apnea, bradycardia, and hypertension, followed by hypotension.

Of note, children who experience ALTEs are known to have vagal overactivity. One paper from Israel (26) appreciated the frequency with which apneic episodes were attributed to reflux because vomitus/refluxate was found at the mouth of the child. However, seizures (particularly those originating in the right hemisphere) can cause apnea in addition to vomiting via direct vagal relaxation of the LES and a sudden increase in intraabdominal pressure. Thus, the best study to obtain in an ALTE pt may be a simultaneous EEG and pH probe study.

10. **Recurrent Croup/Spasmodic Croup** – One theory: patients with baseline airway inflammation secondary to GER, when challenged with a viral URI, have additional edema resulting in stridor.

11. **Laryngomalacia** – a 50-80% association with GER. Patients with laryngomalacia and a history of cyanotic episodes should undergo rigid bronchoscopy in light of the presence of a synchronous airway lesion in 17% of patients. While performing bronchoscopy, BAL can be performed easily to evaluate for lipid laden macrophages. Rigid esophagoscopy with biopsy should be performed at this time to dx GER. Blind suction biopsy by gastroenterology is a less invasive alternative to rigid esophagoscopy (28).

12. **Pseudolaryngomalacia** – Refers specifically to GER-induced stridor (27). In laryngomalacia, stridor is usually consistent and tends to improve with prone positioning and cessation of crying. In pseudolaryngomalacia, stridor tends to be intermittent and independent of positioning.

13. **Subglottic Stenosis** – In a study by Halstead, 32 of 35 patients with known subglottic stenosis (and 1 borderline patient) had positive pH probes. She treated a group of 25 children with subglottic stenosis aggressively with omeprazole and cisapride, with nonsurgical resolution of the stenosis in 35% of them. The remainder underwent endoscopic repair of their stenoses while receiving tx for GER, and only 1 of them went on to require a trach. This represents a huge improvement over the traditional failure rate of 1/5.7 traditionally associated with the endoscopic repair of subglottic stenosis, and argues strongly for aggressive medical tx of GER in children with subglottic stenosis. Of note, the patients were tapered from ppi’s to H2 blockers once their airways improved.

14. **Sandifer’s Syndrome** – abnormal posturing and head tilt related to GER.

**Asthma** – and GER are thought to feed off each other. Asthma theoretically contributes to GER by increasing transdiaphragmatic pressure. There are 3 proposed mechanisms to explain how GER may cause asthma:
A) Macroaspiration of gastric contents into the lungs resulting in irritational bronchospasm (least support)
B) Microaspiration of gastric contents into the upper airway leading to stimulation of upper airway receptors and bronchospasm (some support)
C) Stimulation of esophageal chemoreceptors resulting in vagally mediated bronchoconstriction (most support). This seems to occur only in patients with esophagitis, so resolution of the esophagitis should cure asthma in this model.

In children, asthmatic sx improved in 69% of children with asthma and GER after receiving medical tx for GER. Of note, asthmatics <3 years old are far more likely than older children to respond to medical GER tx. Fundoplication led to asthma improvement in 88% of children with respect to # of hospital visits, # of asthma attacks, and asthma medication/corticosteroid requirements.

**Diagnostic Evaluation**

The **differential diagnosis** for GERD is particularly important in infants and young children. In that age group, GERD is suggested by chronic regurgitation. In that age group, other disorders with a similar presentation include food allergy (especially to cow milk), infection, increased intracerebral pressure (e.g., brain tumor), hydronephrosis, intestinal obstruction, and metabolic disorders (e.g., uremia).

In the **History** it is important to elicit provocative factors such as overfeeding, seated and supine position, and tobacco smoke exposure.

**Physical Exam** should include, in addition to a complete head and neck exam, pulmonary auscultation and neurologic assessment with special attention paid to signs of neurologic impairment. For example, on Laryngoscopy, one would want to look for pharyngeal hypotonia and incoordinate swallowing/pooling of secretions in the hypopharynx, among other things. Empiric therapy is indicated based upon a suggestive history and physical examination, alone. Failure of empiric therapy should prompt further workup.

Of note, NONE of the diagnostic studies used in GER are exceptionally sensitive. The false negative rate for the 4 most commonly used studies (upper GI, gastric scintiscan, 24-hour pH, and esophageal biopsy) range from 59-65% in 1 report. Thus, many authors recommend obtaining 2 studies before ruling out reflux.

**Diagnostic Studies (7):**

1. **Upper GI** – Typically the first study obtained, it is by far the most commonly ordered study in the pre-operative workup of GER patients. It is NOT a sensitive test for the diagnosis of GERD. It is most important in ruling out anatomic abnormalities that may mimic GERD. It will also show hiatal hernia and esophageal dysmotility.

2. **Esophagoscopy and esophageal biopsy** – There are 2 categories of mucosal changes histologically suggestive of esophagitis: reactive epithelial changes, including basal hyperplasia and papillary elongation; and inflammatory changes, including neutrophils, lymphocytes, and eosinophils (in children). Eosinophils in the proximal esophagus are suggestive of food allergy. Early indicators of esophagitis include balloon cells and vascular lakes (which correspond to mucosal red streaks seen grossly on esophagoscopy). Biopsy specimens must be taken from at least 3 cm proximal to the GE junction, otherwise one will obtain false positives.
3. **Direct Laryngoscopy and Bronchoscopy** – Direct Laryngoscopy and Bronchoscopy were performed on a large number of children known to have GERD, and 90% were noted to have at least 1 laryngotracheal abnormality. The most common findings were postglottic edema (69%), tracheal cobblestoning (33%), arytenoid edema (30%), large lingual tonsil (16%), blunting of carina (12.5%), vocal cord edema (12%), vocal cord nodule (12%), and subglottic stenosis (11%).

While performing Bronchoscopy, a BAL is easily performed. It is subsequently examined microscopically for the presence of lipid laden macrophages (LLM), which are diagnostic of aspiration. Trach secretions may also be analyzed for LLM. Of note, the LLM score does NOT distinguish between aspiration from the stomach and aspiration from the mouth/esophagus.

4. **pH Probe** – 24-hour pH monitoring is considered the gold standard in the diagnosis of GER. However, refluxate in infants is often not significantly acidic, and this test only measures acid reflux. Standard milk formula has a pH>6, so apple juice (pH=3.5) is frequently alternated with formula to enhance the test’s sensitivity. The most useful measure from this study is the reflux index, which represents the percent of study time with a pH <4.

5. **Scintigraphy** – This involves continuous imaging after feeding the patient a technetium 99m-radiolabeled meal. It is most commonly used to evaluate gastric emptying. It will also show non-acid reflux, and it is also specific (but not sensitive) for the detection of aspiration of gastric contents.

6. **Modified Bernstein Test** – This is the only study that shows a cause and effect relationship between esophageal acidification and symptomatology. It involves the infusion of HCl into the distal esophagus alternating with a saline control. If the symptoms are elicited, it is considered a positive test. This is most useful in patients with stridor or apneas thought to be related to reflux.

7. **Esophageal Manometry** – More or less limited to research.

**Conservative Treatment**

All GER treatment should start with **conservative measures**:

A) Avoid inappropriate feedings of infants – no huge feedings, and minimize air feedings/jostling during feeds.

B) No feeds 2 hours before bedtime, when possible.

C) Eliminate caffeine and chocolate from the diet, and avoid tobacco exposure.

D) Positioning – prone positioning is the best position for infants with GERD. However, prone positioning is also associated with SIDS. Prone positioning is still advocated in infants with GERD if the parents are carefully instructed to eliminate all puffy bedding materials. Elevation of the head of the crib is NOT of clear benefit in infants, though elevation of the head is recommended for older children. The seated position actually worsens GER.

E) Thickening of Feeds – by adding 1 tablespoon of dry rice cereal per ounce of milk formula. This increases caloric density from 20 to 30 kcal/ounce, decreases episodes of emesis, increases sleeping time, and decreases crying time. Alternatively, one may purchase prethickened formula in powder form.
F) Small, frequent feeds – Clearly indicated in infants who consume excessively large meals; of questionable benefit in everybody else. The theory behind this approach is to decrease intragastric volume and pressure that may lead to TLESRs.

G) Continuous feeding via an NGT – for 7-10 days to treat children with FTT related to GER. Children who don’t gain weight adequately with NG feeds should be given a trial of nasoduodenal feeds in the event that delayed gastric emptying is playing a role. However, NG tubes size 12 French or larger have actually been associated with increased reflux, so select a small tube (19).

Medical Treatment
Prokinetic pharmacotherapy is the first line in young children (<2 years old) because GER is primarily a problem of motility in this age group. Acid suppression is added when esophagitis is suspected.

Prokinetics
A) Cisapride was once the first line medication in the treatment of GER in children. Several studies showed it was more effective than other prokinetic agents in the treatment of GER. Cisapride has minimal side effects with the exception that it is known to predispose to prolongation of the QT interval, and there have been anecdotal reports of deadly cardiac arrhythmias (namely, torsades de pointes and heart block). Known risk factors for the development of these arrhythmias include preterm and newborn age, heart disease, electrolyte abnormalities (hypokalemia, hypomagnesemia), and genetic syndromes with prolonged QT. QT prolongation is exceptionally rare in children without these risk factors taking an appropriate dose of cisapride. In fact, of the 12.6 million children who used cisapride in England, not a single death was reported in previously healthy children taking an appropriate dose of cisapride; the conservative estimate for risk of death is 1/250,000, as compared to 0.07% in previously healthy children undergoing fundoplication. If cisapride is used, a baseline EKG, and a followup EKG 5 days after the initiation of therapy should be obtained. Furthermore, any compounds known to inhibit cytochrome P450 3A4 must be avoided while taking cisapride. These include macrolides (except azithromycin); azoles; nefazodone, fluoxetine, and fluvoxamine (antidepressants); HIV protease inhibitors; Delavirdine; amiodarone; cimetidine; diltiazem; verapamil; isoniazid; metronidazole; quinine; zileuton; quinupristin/dalfopristin; and, finally, grapefruit juice.

B) Metoclopramide – side effects include nervousness, somnolence, and dystonic reactions. Extrapyramidal signs (involuntary limb movements, torticollis, facial grimacing), if present, usually occur 24-48 hours after starting therapy. These side effects are more common in children, and respond quickly to withdrawal of the drug and valium. Benztrapine may also be given.

Acid Suppressants
A) Antacids may be used for symptomatic relief; they should not be given long term or mixed in with infant formula because they can result in Aluminum toxicity and osteomalacia.

B) H-2 blockers (Cimetidine, Ranitidine, Famotidine) are usually adequate to achieve acid suppression in young children and adolescents.
C) Proton Pump Inhibitors (Omeprazole is the only PPI that has really been studied in children) usually required in cases of severe chronic respiratory distress and neurologic disability. It should be given with or just before the first meal of the day. In small children, the granules should be delivered in an acidic medium, e.g., apple sauce or yogurt.

**Surgery**

A national survey involving nearly 7,500 pediatric patients showed that essentially 2 procedures are performed to address GER in children (8): the Nissen fundoplication (2/3) and the Thal-Ashcraft procedure (1/3). At UTMB, the Thal-Ashcraft procedure is more commonly performed.

**Indications** for fundoplication include

- Failure of medical therapy
- Esophagitis with stricture (fundoplication is performed in addition to sequential dilatations)
- Persistent chronic pulmonary disease
- Family cannot tolerate continuous vomiting
- Following repair of TEF with esophageal atresia

Historically, fundoplication was rarely performed in children less than 2 years of age. However, the procedure is indicated in Infants with GER and Neurologic impairment (typically performed with concurrent G-tube placement)

**Indications** for Thal-Ashcraft include:

- Bronchopulmonary Dysplasia/RDS
- Apneic episodes
- Associated congenital anomalies (e.g., diaphragmatic hernia, esophageal atresia)

Fundoplication has been shown to be safer and more effective in children as compared to adults, and has even been shown to be safe in children younger than 3 months of age.

Good outcomes occur in 94% of neurologically normal (NN) and 85% of neurologically impaired (NI) children. The associated complication rates are 4.2% (NN) and 12.8% (NI). Disruption of the fundoplication occurs in a total of 7.1%, with reoperation required in 3.6% (NN) and 11.8% (NI).

The Nissen and Thal represent 2 different conceptual approaches to GERD.

**Nissen** – The concept is to create a tight valve to act as a permanent substitute for an ineffective antireflux mechanism. A 360 degree wrap is formed around the abdominal esophagus using the gastric fundus. This is intended to act as a valve against reflux. Complications include gas bloat syndrome, dumping syndrome, dysphagia, and oral dysfunction. Gastrostomy is frequently performed in conjunction with the Nissen for postoperative feeding and gastric venting to alleviate gas bloat. A history of esophageal atresia repair and substantial middle and upper esophageal dysmotility are contraindications to the Nissen.

**Thal** – The idea is to correct the abnormal anatomy to enable the biological antireflux mechanism via 3 objectives: 1) a long intraabdominal esophagus, 2) fixation of the intraabdominal esophagus, and 3) restoration of the angle of His. To achieve this, the lower esophagus is freed and lifted upward and to the left. A partial, 180 degree anterior
wrap is then constructed using the gastric fundus. The Thal avoids gas bloat syndrome and allows for vomiting.

More recent data from Japan suggests that fundoplication does not function in the way it was intended. It does NOT increase basal LES pressure, nor does it decrease the duration of TLESRs. Its primary effects appear to be a decrease in the frequency of TLESRs and an increase in the nadir pressure of TLESRs (i.e., it causes incomplete TLESRs). A potential explanation is that wraps affect the distensibility of the gastric cardia, which is the most potent area for triggering TLESRs (12).

References


