# Gastroesophageal Reflux

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**Gastroesophageal refux is common in infants and generally resolves spontaneously within the first year of life as the lower esophageal sphincter mechanism matures. The reflux is only considered a "disease" (GERD) when it becomes symptomatic or causes pathological consequences. GERD is commonly associated with esophageal atresia and there is a high incidence in neurologicaly impaired children; in both groups conservative treatment is notoriously ineffective. The diagnosis of GER is made on upper gastrointestinal contrast studies, endoscopy and pH monitoring. Medical management comprises antacids, reduction of gastric acid production and prokinetic agents. The indications for anti-reflux surgery include an established esophageal stricture, associated anatomical defect and failure of medical therapy. Apnoeic episodes secondary to documented GER in the infant, constitute an absolute indication for early surgery.**


GASTROESOPHAGEAL REFLUX is defined as the involuntary regurgitation of gastric content into the esophagus. It is a normal physiological process and it is only when it produces symptoms or pathological consequences is it considered a disease (GERD).

## MECHANISMS FOR CONTROLLING GASTROESOPHAGEAL REFLUX

1. Anatomical
   a) lower esophageal sphincter (>10–30 mm Hg)
   b) the length of the intraabdominal segment of the esophagus
   c) the acute angle of His
   d) the mucosal rosette in the lower esophagus
   e) the phrenoesophageal membrane
   f) the pinch-cock effect exerted by the diaphragmatic crura.
2. Physiological
   a) effective peristalsis in the distal esophagus to rapidly clear refluxate from the stomach
   b) prompt and effective gastric emptying.

In infancy, many of the above mechanisms are poorly developed, which explains the high incidence of gastroesophageal reflux in the first year of life. The intraabdominal esophagus measures only 1 cm at birth, compared with 3 cm at 3 months, the angle of His is obtuse in the neonate and only decreases as the child grows, while the pressure in the lower esophageal sphincter increases during the first few months of life.[1] Newell demonstrated a four-fold increase in lower esophageal pressure between 27 and 40 weeks postconceptial age but could not demonstrate a relationship between effective sphincter pressure and postnatal age.

The vast majority of infants with symptomatic reflux will improve steadily during the first year of life and only approximately 5% of infants will require surgical intervention.

The importance of transient lower esophageal sphincter relaxation unassociated with swallowing in the genesis of pathological gastroesophageal reflux has only recently been appreciated.[3]

## PATHOGENESIS

Acid-pepsin reflux into the lower esophagus results in a chemical inflammation of the squamous mucosa, which is ill-equipped to resist the digestive enzymes. In the early stages there is an inflammatory cell infiltration with erythema of the mucosa. With continuing reflux the mucosa becomes friable and bleeds on contact. Later, ulceration develops, which may proceed to stricture formation as fibrous tissue is laid down as a consequence of transmural damage.

## CLINICAL PRESENTATION

### *Early Infancy*

The infant presents with recurrent vomiting, which may be projectile in nature and may even mimic pyloric stenosis. The vomitus generally contains ingested milk only, but hematemesis in the form of fresh blood or of "coffee-grounds" may occur from ulcerative esophagitis. With repeated vomiting the infant fails to thrive. Presentation with recurrent respiratory infections or apneic attacks will be discussed in the section on aspiration syndromes.

### *Later Childhood*

Persistent vomiting is still the major symptom in older children, but the problem may only manifest in the form or night vomiting of mucus. Heartburn caused by esophagitis and dysphagia from ulcerative esophagitis or stricture formation assumes more prominence at this age. Hypochromic microcytic anemia may occur as a consequence of persistent blood loss from ulcerative esophagitis. Asthma or frequent respiratory infections may

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*1055-8586/03/1204-0004$30.00/0*
*doi:10.1053/j.sempedsurg.2003.08.004*

develop as a complication of recurrent aspiration of gastric contents.

*Associated Anatomic Defects*

Gastroesophageal reflux occurs in over 40%[4] of patients with esophageal atresia and is more common in congenital diaphragmatic hernia and defects of the anterior abdominal wall. Other precipitating causes are malrotation and other causes of gastric outlet obstruction, eg, pyloric stenosis or antral dysmotility.

*Neurological Abnormalities*

The association between gastroesophageal reflux and severe mental retardation and other neurological abnormalities has only recently been appreciated. An estimated 10% of retarded children in institutional care manifest vomiting as a major problem. The tendency is to ascribe the vomiting to psychological causes and this has led to prolonged delay in diagnosis, exposing the child to the development of complications from the reflux. Severe failure to thrive, iron-deficiency anemia, recurrent bouts of pneumonia, and strictures frequently occur in these children.[5,6]

*Aspiration Syndromes*

Recurrent episodes of pneumonia, attacks of asthma, and apneic episodes resulting in near-miss sudden infant death syndrome have been ascribed to aspiration of gastric contents. Acute life-threatening events result from either laryngospasm or reflex bradycardia.

*Unusual Presentations*

Dystomic contortions of the head and neck (Sandifer's syndrome) and protein-losing enteropathy are rare manifestations of ulcerative esophagitis.

## DIAGNOSIS

A range of investigations are available to establish the diagnosis and to define the severity of the reflux.

1. Barium esophagogram will determine the presence of an associated hiatal hernia and the anatomy of the esophagus with reference to ulceration or stricture formation. In addition, abnormal peristaltic activity of the esophagus should be noted as well as malrotation or delayed gastric emptying. The degree of reflux can be graded according to the highest level the refluxate ascends in the esophagus. The sensitivity of contrast studies is only 40% but the specificity is 85%.
2. pH monitoring is currently regarded as the most sensitive (90%) and specific test (100%) for diagnosing GER. Significant reflux occurs when the pH falls below 4 and the results can be used to quantitate the reflux and provide information about the number and duration of each reflux episode. The pH study may show a correlation between symptoms and episodes of reflux but cannot predict the severity of an episode. Thus, for example, its use in diagnosing acute life-threatening episodes is very limited. The use of double probes (in the distal esophagus and in the stomach) and combination probes (combined with manometry) may improve its usefulness.
3. Upper gastrointestinal endoscopy has a high specificity (95%) but a lower sensitivity (70%) for diagnosis GER. Over 50% with GERD are "endoscopy negative" but the examination is important for children with ulcerative esophagitis or strictures. Biopsy is essential for suspected areas of Barratt's esophagitis and may show eosinophilic infiltrate in otherwise normal-appearing mucosa.
4. Esophageal manometry use is limited in children due to the difficulty in performing this study particularly in small children. Manometry was originally proposed in adults as a predictive test for possible postoperative dysphagia after fundoplication but recent randomized controlled trials have shown that manometry has no value in predicting postoperative symptoms.[7,8]
5. Gastric isotope scintiscan. A radionuclide technetium sulphur colloid is used to assess gastric emptying with 50% of the isotope expected to leave the stomach within 60 minutes. The clinical value of the test in assessing the needs for a pyloroplasty is controversial. Maxson et al[9] found no significant difference in postoperative results in children with abnormal compared with those with normal gastric emptying studies.
6. Other investigations. Videofluoroscopic assessment of swallowing is important in the neurologically impaired group to diagnose overt or silent aspiration on swallowing. The detection of lipid-laden macrophages in tracheal aspirate may indicate aspiration during GER but the sensitivity and specificity is low at 38% and 59% respectively.[10]

## TREATMENT

1. Medical management. Traditional measures including positioning maneuvers and small volume, frequent thickened feeds have no proven efficacy in reducing GER.[11,12] Drug therapy aims at decreasing the acid content of gastric secretion and improving esophageal and gastric motility. The former includes antacids, eg., Gaviscon and $H_2$-blocking agents (ranitidine) or proton pump inhibitors (omeprazole). Prokinetic agents of value are dopamine antagonists (metoclopramide) and domperidone.

2. Indications for surgery:
   a) the presence of an established esophageal stricture. Resolution of the stricture will not occur until the reflux has been controlled.
   b) an associated anatomical defect, such as a large hiatal hernia or intestinal malrotation.
   c) failure of effective medical treatment:
      i) in esophageal atresia with or without anastomotic stricture
      ii) in children with severe learning disability.[13] Their response to medical measures is notoriously poor while the additional nursing/caring burden imposed by repeated vomiting adds significantly to the social stress of the family (see below)
      iii) continuing failure to thrive after 3-6 months trial of medical therapy.
   d) apneic episodes, near-miss sudden infant death and recurrent respiratory infections indicate early surgery rather than prolonged exposure to medical treatment.

A number of factors contribute to the increased incidence of reflux in the neurologically impaired child. These include reduced lower esophageal sphincter pressure, especially in the presence of severe esophagitis, dysmotility of the esophagus reducing acid clearance, the raised intraabdominal pressure due to spasticity, scoliosis and recurrent seizures, and the prolonged period of time spent in the supine position.

## SURGERY

The Nissen fundoplication is the procedure of choice. The procedure aims to establish a high-pressure zone in the distal esophagus by accentuating the angle of His, by lengthening the intraabdominal esophagus, by creating a flutter valve at the esophagogastric junction and by repairing the hiatus by approximating the crura of the diaphragm. The procedure comprises a 360°, short (2-3 cm), floppy fundoplication. A gastrostomy is added particularly to the neurologically impaired child if there are significant swallowing problems or aspiration on swallowing detected on videofluoroscopy.

The procedure can be equally performed by open surgery or by laparoscopy. The latter method has the advantage of better cosmesis and reduced postoperative pain and time to establish enteral nutrition.[14-16]

Recently, two endoscopic techniques have been described for the treatment of GER. The application of endoscopic radiofrequency energy to the gastroesophageal junction claims resolution of symptomatic GER in 87% of patients.[17] Endoscopic gastroplication using the Bard Endo Cinch® device aims at reproducing the mucosa rosette at the gastroesophageal junction.[18] The long-term results of these procedures will determine the ultimate success rate.

## COMPLICATIONS

Complications following fundoplication occur more frequently in children, particularly in infants less than 4 months of age and those with esophageal atresia,[19] and in the neurologically impaired compared with the neurologically normal child.[20,21]

The following complications are encountered:

1. Disruption of the wrap (8%-12%).
2. Dysphagia due to excessively tight wrap (2%-12%), which occurs more frequently in the laparoscopic group.
3. Herniation of the wrap into the posterior mediastinum due to disruption of the crura repair. Excessive retching postoperatively may contribute to the failure of the crura repair.
4. Gas bloating (4%-10%) is more common in the neurologically impaired group. Di Lorenzo and Orenstein[22] have described a number of components of gas bloat syndrome:
   (a) abnormal motility, which is often present before surgery and may be exacerbated by vagal nerve dysfunction, which may be present in up to 20% of patients following surgery
   (b) impaired gastric accommodation secondary to use of the highly distensible posterior fundus in formation of the wrap, with subsequent overdistension of the gastric antrum
   (c) gastric hypersensitivity, a poorly described visceral response to increases in intraluminal pressure, which may also stimulate the emetic reflex and finally
   (d) dumping syndrome, a term which describes a symptom complex that occurs in up to 30% of children after fundoplication and constitutes nausea, retching, diaphoresis, diarrhea, and swings in blood glucose related to discharge of gastric contents into the duodenum.
5. Adhesion intestinal obstruction (2%-10%) is more common if additional procedures such as gastrostomy, Ladd's procedure for malrotation, appendicectomy, are performed.

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