# CHAPTER 51
# Corrosive Ingestion and Oesophageal Replacement

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## Introduction

Caustic ingestion can produce a progressive and devastating injury to the oesophagus and stomach. Caustic material ingestion is most frequently encountered in children who accidentally swallowed caustic materials or in adults who ingested caustic materials for suicidal purposes.[1,2] Alkaline caustics and acids are the commonest chemicals implicated in caustic burns. Stricture formation with inability to swallow food after the injury is inevitable in some cases. Many different therapies have been recommended. The literature regarding the treatment of these patients is quite controversial and inconclusive. Repeated dilatations to maintain an adequate lumen diameter were given in patients with chronic strictures. In more severe strictures, due to the complications and ineffectiveness of the dilatation, surgical replacement of the oesophagus may be required.

The causative caustic agent is either acid or alkali with different reactions and sequelae. The concentration and amount of the ingested material have an important impact on the injury. Lye is a broad term for a strong alkali used in cleansing agents.[3] For example, sodium and potassium hydroxides in granular, paste, and liquid forms are used in drain and oven cleansers as well as washing detergents. Also, button batteries, which contain high concentrations of sodium and potassium hydroxides, can cause severe injuries. Acids are commonly available in toilet bowel cleansers (sulfuric, hydrochloric); battery fluids (sulfuric); and swimming pool and slate cleansers (hydrochloric).[3] The majority of cases in Egypt are due to caustic potash, with three to five new cases every month and an overall ratio of eight alkali cases for every acid case.

## Prevention

A public health drive is required to educate the population to the dangers of these corrosive products. Safe storage of corrosive liquids, out of reach from children, is needed. Also, corrosive products must be stored in containers with hazard labels rather than in soft drink bottles to reduce the accidental ingestion of corrosive material.

Directives to change the chemical composition of these products and institute safer preparation of button batteries should be given to commercial institutions.

## Pathogenesis and Pathology

The extent of damage to the gastrointestinal tract depends on the agent, its concentration, amount, physical state, and the duration of exposure.[3,4] Acidic solutions usually cause immediate pain, and—unless ingestion is intentional—the agent is rapidly expelled. Alkali solutions, however, are often tasteless and odorless and are swallowed before protective reflexes can be evoked.[1]

Caustic agents in solid form and granules often adhere to the mucous membranes of the mouth, thereby preventing further movement of lye into the oesophagus. The most severe caustic injury generally occurs in the narrowest portion of the oesophagus, usually the midoesophagus in the region of the aortic arch.[5,6]

The primary difference between alkaline and acidic injury is rapid penetration into the tissue by alkali. Alkali has a potent solvent action on the lipoprotein lining, producing a liquefaction necrosis. Thrombosis of adjacent vessels results in further necrosis and bacterial colonisation. Granular agents often produce focal injury to the oropharyngeal and proximal oesophageal mucosa. Liquid alkaline solutions can cause extensive damage to the entire oesophagus and stomach.

Acidic agents produce a coagulation necrosis, resulting in a firm protective eschar that delays injury and limits penetration. The naturally alkaline environment, low viscosity, and rapid transit limit injury of the oropharynx and oesophagus by the acid compounds. Accordingly, acidic agents were thought to spare the oesophagus and injure the stomach. However, ingestion of highly concentrated sulfuric or hydrochloric acid penetrates the oesophageal mucosa and produces severe injury. When the stomach is empty, caustic acids will affect the gastric mucosa along the lesser curvature to the antrum, and when the stomach is full, the acidic agents cause diffuse injury.

Caustic injuries to the gastrointestinal tract are classified pathologically into three degrees according to the depth of injury,[1] as shown in Table 51.1.

*Table 51.1: Degree of oesophageal burns*

| Degree of burn | Oesophageal depth |
|---|---|
| First-degree | Superficial, confined to mucosa, heal without stricture formation |
| Second-degree | Penetration into muscularis layer |
| Third-degree | Entire wall of gastrointestinal tract, with or without perforation |

## Clinical Features

The clinical picture on presentations depends mainly on the site and depth of injury caused by the caustic agent. Early manifestations include persistent salivation, dysphagia, hoarseness of voice and stridor, retrosternal chest pain, and hematemesis. Severe gastric injury may present as epigastric pain; retching; or emesis of tissue, blood, or coffee-ground material. Fever, shock, dyspnoea, and acute abdomen strongly indicate oesophageal or gastric perforation.[7]

Late complications of caustic injuries include dysphagia due to the establishment of oesophageal stricture within 1 to 2 months. Early satiety, weight loss, and progressive emesis suggest gastric outlet obstruction. Repeated chest infections may indicate acquired tracheo-oesophageal stricture.

## Diagnosis

Due to the poor correlation between signs and symptoms and the degree of injury, endoscopic examination of the upper gastrointestinal tract is essential in most patients with a history of caustic ingestion. There is great controversy regarding the proper timing of the endoscopy. Many centres have performed the procedure in the first 24 to 48 hours after ingestion with excellent results, and have found the procedure to be safe and accurate. The endoscopic grading of the injury can predict the treatment and outcome, and unnecessary treatment is avoided when oesopha-

geal injury is excluded by endoscopy. In the presence of stridor and respiratory problems, however, early endoscopy is hazardous because it may aggravate the airway obstruction.

Endoscopic grading of corrosive oesophageal and gastric burns is shown in Table 51.2. Grade I and grade IIA injuries do not result in strictures, whereas 70% to 100% of circumferential burns (grade IIB) and grade III lesions result in strictures.[5]

It has been suggested that most patients with severe injury have one or more clinical signs or symptoms (drooling, dysphagia, vomiting, and abdominal pain), and that 50% or more of patients who present with vomiting, stridor, and drooling have associated oesophageal injury. Furthermore, asymptomatic patients are deemed to be unlikely to have lesions that progress to stricture or perforation. Based on these data and on the absence of proven therapy to prevent stricture, the suggestion is that patients require endoscopy only if they are symptomatic.[3,7] Most authorities recommend performing upper endoscopy as soon as the patient is stable.[3,8]

The practice in Egypt is that endoscopy is not done in the acute stage but postponed for 6 weeks and performed only for symptomatic patients.

*Table 51.2: Endoscopic grading of corrosive oesophageal burns.*

| Grade | Finding |
|---|---|
| Grade I | Oedema and erythema |
| Grade IIA | Haemorrhages, erosions, blisters, superficial ulcer, exudate (patchy or linear) |
| Grade IIB | Circumferential lesions |
| Grade III | Multiple deep brownish-black or gray ulcers |
| Grade IV | Perforation |

## Radiologic Studies

### X-ray

Plain chest and abdominal x-rays should be performed in the acute phase of caustic injury. This may reveal evidence of perforation such as pneumothorax, pleural effusion, or air under the diaphragm. If perforation is still suspected despite negative plain films, a study with the use of water-soluble contrast material may reveal extra luminal contrast.[3–7]

### Computed Tomography

Computed tomography (CT) of the oesophagus and stomach with orally administered contrast is the most sensitive method of detecting early perforation.[3] With this approach, life-threatening injuries can be identified and treated at an early stage.[9] In the chronic stages of the illness, maximal wall thickness of oesophageal stricture can be measured with a contrast-enhanced CT scan.[3]

Contrast studies (see Figures 51.1 and 51.2) are most useful in evaluating the inlet of the upper gastrointestinal tract, the oesophageal body, and the stomach outlet at approximately 3 weeks after injury.[3] Oesophageal body strictures can be of variable length, shape, and number. Most strictures are at the region of the aortic arch.[7] It is of utmost importance to study the inlet of the gastrointestinal tract before surgery to plan for the site of the proximal oesophagocolic anastomosis, and to study the outlet to detect any antral stenosis. Any missed strictures at the inlet or outlet after substitute organ replacement of the oesophagus may affect the success of the surgery.

### Endoscopic Ultrasound

It is likely that endoscopic ultrasound (EUS) provides better determination of the depth of injury and may prove to be adjunctive or even superior to endoscopy in staging caustic oesophageal injury.[7]

## Treatment Options

The goals of therapy are to prevent and treat perforation as early as possible, to avoid strictures of the oesophagus and stomach, and to replace or bypass the damaged organ to allow normal swallowing of food.

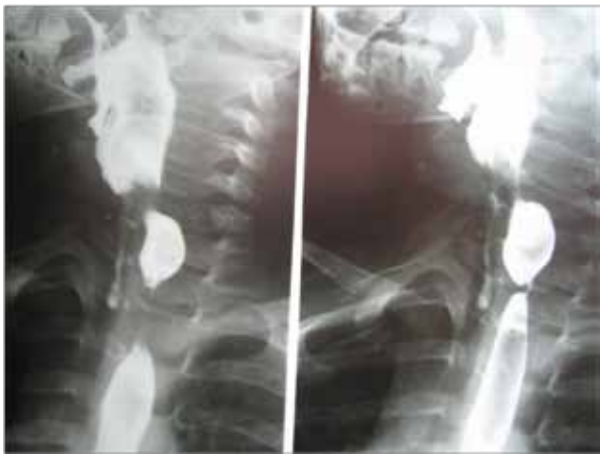

*Figure 51.1: Multiple oesophageal strictures.*

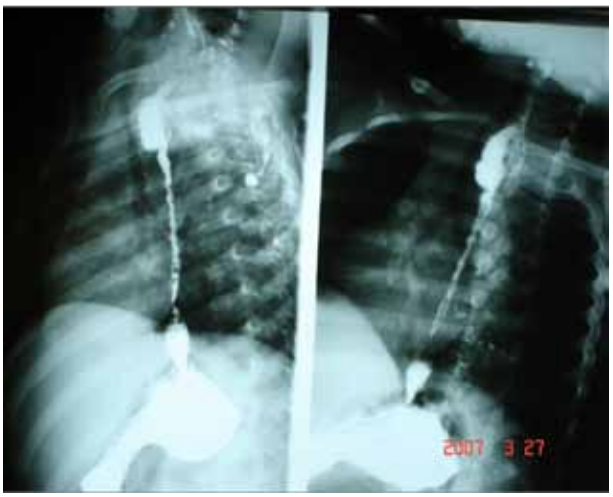

*Figure 51.2: Long oesophageal strictures.*

### Surgery

Although emergency surgery is indicated in cases of perforation, it is difficult initially to predict which patients will develop this complication.[3] Early surgery is essential to improve the prognosis in cases of oesophageal or gastric perforation.[10] Although most investigators state that in selected cases, early surgery would be prudent, the criteria on which to base selection of surgical cases are not well defined.[3] The reduced mortality achieved through early detection of impending or actual perforations outweighs the morbidity and mortality rate associated with surgical exploration in patients with endoscopically diagnosed second-degree burns. However, many surgeons have condemned early surgery because the extent of the injury often cannot be delineated, leaks at anastomotic sites can occur, and surgery will not be needed in the majority of patients.

### Neutralisation or Flushing

To be effective, neutralisation of caustics must be done within the first hour after ingestion of the caustic agent. Lye or other alkali can be neutralised with half-strength vinegar, lemon juice, or orange juice. Acids can be neutralised with milk, egg, or antacids.

Sodium bicarbonate is not used because it generates $CO_2$, which might increase the danger of perforation.[3–5] Water is used only to wash the mouth and not for dilution because it will take the remnant to the rest of the gastrointestinal tract (GIT). Emetics are contraindicated because vomiting renews the contact of the caustic substance with the oesophagus and can contribute to aspiration or perforation if it is too forceful.[3–5]

### Collagen Synthesis Inhibitors

In experimental animals, collagen synthesis inhibitors, such as aminoproprionitrile, penicillamine, N-acetylcysteine, and colchicine, have been shown to prevent alkali-induced oesophageal strictures. These compounds impair synthesis of collagen by interfering with the covalent crosslink. However, no clinical studies have been performed with these agents.[3]

### Nutrition

Intravenous (IV) nutrition is essential for patients in whom perforation has occurred or enteral feeding cannot be maintained. However, in some African institutions where IV nutrition is not available, patients may be started on oral alimentation or a nasogastric feeding tube. In many cases, a feeding gastrostomy or jejunostomy may provide the patient with the necessary nutritional requirement until surgical correction can be performed. A feeding gastrostomy can be used as a route for retrograde dilatation.[3]

### Early Oesophageal Dilatation

Some investigators recommend eosophageal dilatation immediately after injury. Dilatation is performed at frequent intervals until healing occurs.[3] This approach, however, is controversial in that dilatations can traumatise the oesophagus, predisposing to bleeding and perforation, and same data indicate that excessive dilatations cause increased fibrosis.[5] Dilatation is recommended by some only when stricture formation develops. Others pass a string on the nasogastric tube as part of the initial therapy to maintain the oesophageal lumen.[3]

### Corticosteroids

Studies by some investigators in animals have shown that corticosteroids given within 24 hours after alkali injury inhibits granulation and fibroblastic tissue reaction and decreases the incidence of oesophageal stricture.[3,11] Others, however, believe that corticosteroids may obscure evidence of peritonitis and mediastinitis and fail to reduce the incidence of stricture formation. Corticosteroid injections are used in localised strictures. Intralesional triamcinolone injections augment the effects of endoscopic dilatation.[12–14] Several studies have shown that local steroids improve and increase the intervals between dilatation but not the need for replacement.[15]

### Oesophageal Stents

Some investigators have placed intraluminal silastic stents under endoscopic guidance in patients with deep circumferential burns. Unfortunately, the majority of these patients required oesophageal dilatation later.[3,16]

### Endoscopic Dilatation

Treatment of strictures is endoscopic dilatation. Gradual dilatation is essential.[3] Dilatation can be done on a weekly or biweekly basis by using Savary-Gilliard® bougies, and is considered adequate if the oesophageal lumen could be dilated to 11 mm with complete relief of dysphagia. The Savary-Gilliard method is adequate for oesophageal dilatations in the paediatric population.[17,18,15] Balloon dilatation under endoscopic guidance and radiological screening of the oesophageal stricture is also successful (see Figure 51.3). The advantages of this method may be that its forces are exerted radially and the procedure may be performed under better control.[19] Perforation, bleeding, sepsis, and brain abscess may complicate dilatation, however.[3–20] Mitomycin C antibiotic has been recently used with promising results. After endoscopic dilatation, mitomycin can be applied onto the dilatation wound by using a rigid endoscopy.[21]

An adequate lumen should be reestablished within 6 months to 1 year, with progressively longer intervals between dilatations. If, during the course of treatment, an adequate lumen cannot be established or maintained, surgery should be considered. Surgical intervention is indicated in the following cases:

- Patients with failure to swallow solid food, which may lead to deformities of the mandible, temporomandibular joint, and teeth.

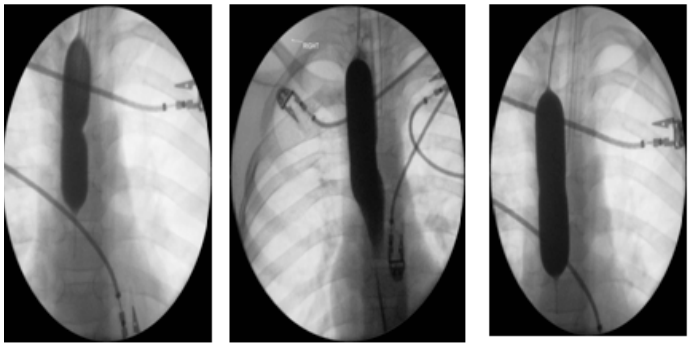

*Figure 51.3: Balloon dilatation.*

- Patients with complete stenosis, in which all attempts have failed to establish a lumen.
- Patients with multiple, tortuous, or very long (more than 5 cm) strictures.
- Patients with severe peri-oesophageal reaction or mediastinitis, and in the development of tracheo-oesophageal fistula.
- Patients who are unwilling or unable to undergo prolonged periods of dilatation.

## Treatment Recommendations

During the acute phase, we give oral feeding as tolerated; otherwise, the patients are kept on IV fluid until oedema subsides; if dysphagia persists, we prefer to perform gastrostomy to keep the general condition of the patient maintained until an upper endoscopy is performed after 6 weeks. In Egypt, we are performing the gastrostomy by laparoscopy, with better results and fewer adhesions (see Figure 51.4). Our protocol for oesophageal dilatation starts after 6 weeks and is repeated every 2 weeks for 6 months by using the Savary-Gilliard dilator.

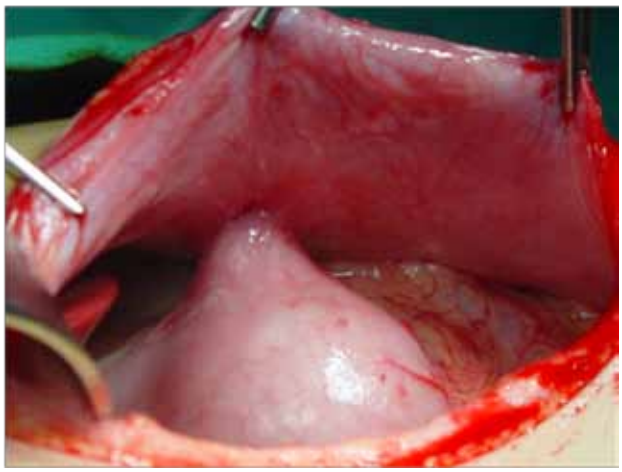

*Figure 51.4: Appearance of the stomach after laparoscopic gastrostomy (no adhesions).*

## Surgical Interventions

The variety of abnormalities seen requires that creativity be used when considering oesophageal reconstruction. Skin tube oesophagoplasties are now used much less frequently and are mainly of historical interest. Currently, the stomach, jejunum, and colon are the organs used to replace the oesophagus through either the posterior mediastinum or the retrosternal route. A retrosternal route is chosen when there has been a previous oesophagectomy or if there is extensive fibrosis in the posterior mediastinum. When all factors are considered, the order of preference for oesophageal substitution depends on the experience of the surgeon and the practice in the institution.[5]

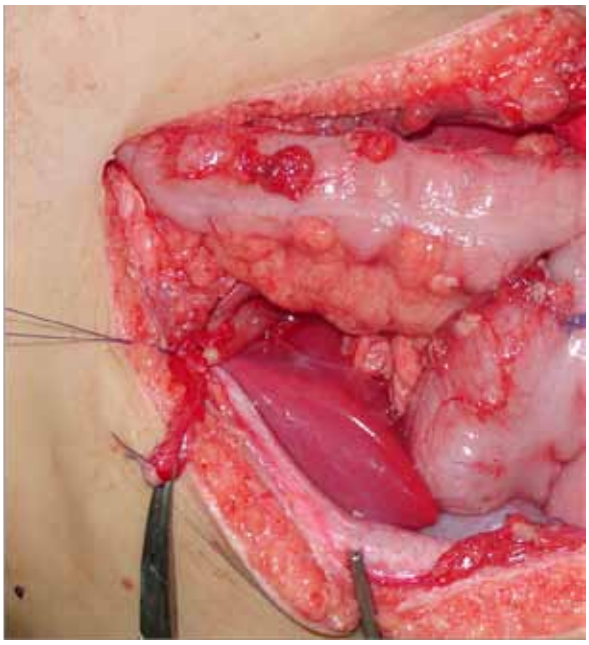

*Figure 51.5: Fixing the round ligament to the sternum.*

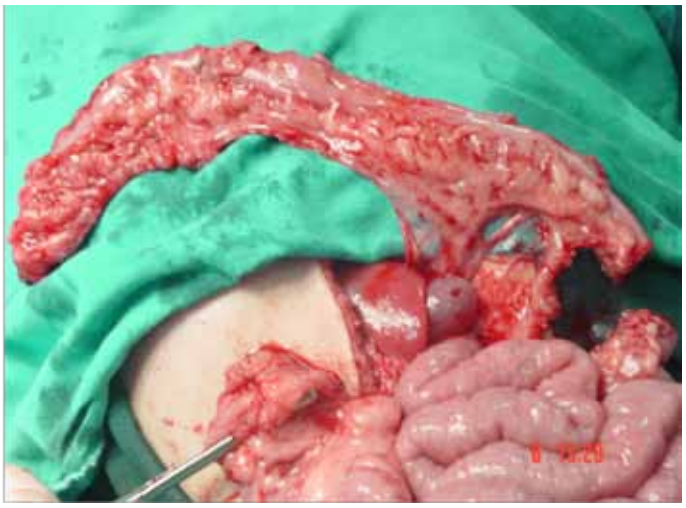

*Figure 51.6: Colonic graft based on the left colic vessels and sigmoid vessels (double blood supply).*

*Figure 51.7: End-to-side oesophagocolic anastomosis.*

## Colon Substitution

The isoperistaltic left colon segment based on the left colic vessels is a very suitable substitute for the oesophagus in children. A sufficient length is available to replace the whole oesophagus and even the lower pharynx if needed. The blood supply from the left colic vessels is robust and rarely prone to anatomic variations. The close relation between the marginal vessels and the border of the viscus results in a straight conduit with little redundancy. The left colon seems to transmit food more easily than the right colon, and has proved to be relatively acid resistant.[22–24]

The transhiatal oesophagectomy with posterior mediastinal isoperistaltic left colon has been the most direct and shortest route for the oesophageal substitute between the neck and abdomen. It permits the removal of the scarred oesophagus, which has a definite increased risk of malignant changes, cyst formation, and empyema if left in place.[22–24]

The retrosternal route is still an ideal route for many surgeons. Retrosternal colon by-pass avoids any thoracic dissection and preserves the vagus nerves. It takes less operative time and avoids injuries to such intrathoracic structures as trachea and major vessels. The postoperative period is less stormy than the transhiatal route, and few patients require postoperative ventilations. Many modifications have been performed to make a straight route out of the anterior mediastinal/retrosternal route. Dividing the strap muscles in the neck and fixing the falciform ligament to the sternum (Figure 51.5) avoids the colon being stretched over the liver when the child is in an erect position.[22–24]

An equal number of each technique were performed at Ain Shams University in Cairo. We prefer using the transverse colon, based on a double blood supply from the left colic vessels (Figure 51.6) with comparable results for retrosternal and transhiatal techniques. Usually, we add an antireflux procedure while performing the cologastric anastomosis by wrapping the lower colon by stomach in the transhiatal approach and creating an angle between the lower colon and the anterior gastric wall. The length of tucking of the colon to the anterior gastric wall to create an antireflux mechanism should be 4–5 cm.

Leakage from the cervical oesophagocolic anastomosis has been reported to be 12–71% and is usually followed by varying degrees of stricture after the cessation of leakage. Performing an end-to-side anastomosis between the unequal diameters of the oesophagus and colon has decreased the incidence of leakage from the neck anastomosis (Figure 51.7). The use of a double blood supply to the colonic graft and the use of a vascularised omental flap to wrap the anastomosis have also reduced the leakage rate.[22–24]

Long-term follow-up for patients with colonic replacement of the oesophagus have shown excellent results. Late complications, such as strictures of the cervical end, may lead to varying degrees of dysphagia. Strictures may be dilated; however, the majority will need revision of the anastomosis. Redundancy of the interposed colonic graft in the chest may lead to stasis and dysphagia due to kinking of the graft (Figure 51.8). Proper measurement of the graft length and avoiding opening the pleura may decrease the incidence of redundancy of the colonic graft.[22–24]

## Gastric Substitution

In gastric substitution, a cervico-abdominal approach is used. The neck incision may be right- or left-sided. The abdominal incision may be upper umbilical midline or transverse. The gastrostomy is carefully taken down and closed in two layers. The vessels on the greater curvature of the stomach are preserved for the gastric transposition or if a carefully constructed gastric tube is used. Where possible, a posterior mediastinal route is preferred to a retrosternal route. The fundal end of the stomach is anastamosed to the oesophageal stump in the neck, securing the neck anastamosis with sutures to the retro clavicular tissue. A wide bore nasogastric tube is introduced into the thoracic stomach to avoid acute gastric distention. Gastrohiatal sutures are applied to prevent herniation of abdominal contents. A pyloroplasty is performed and the pylorus is kept below the diaphragm (Figures 51.9

and 51.10) A feeding jejunostomy rarely is required for caustic injuries. Reported complications from Great Ormond Street Children's Hospital in London are as follows:[25] Mortality, 5.2%; anastamotic leaks, 12%; oesophageal strictures, 38%. All leaks but one closed spontaneously, and all strictures except three responded to dilatation. Transient dumping syndrome and delayed gastric emptying were also noted. Results were excellent in 90% of patients with minimal impact on growth, development, and respiratory function.

### Jejunal Substitution

The jejunum is more commonly used in adults. It is used mainly as a free graft with microvascular anastamosis. Due to the popular use of the colon or stomach as replacement organs with good results, jejunal substitution is used less in the paediatric population.

## Surgical Technique

The most critical point in the planning of the operation is the selection of the site for proximal anastomosis. The site of the upper anastomosis depends on the extent of the pharyngeal and cervical oesophageal damage. When the cervical oesophagus is destroyed and a pyriform sinus remains open, the anastomosis can be made to the hypopharynx. When the pyriform sinus is completely stenosed, a transglottic approach is used to perform an anastomosis to the posterior oropharyngeal wall.

Recovery is long and difficult and may require several endoscopic dilatations and, often, reoperations. Sleeve resections of short strictures are not successful because the extent of damage to the wall of the oesophagus can be greater than realised, and almost invariably the anastomosis is carried out in a diseased area. The management of a bypassed damaged oesophagus after injury is problematic. The extensive dissection necessary to remove the oesophagus, particularly in the presence of marked perioesophagitis, is associated with significant morbidity. Leaving the oesophagus in place preserves the function of the vagus nerves and, in turn, the function of the stomach. Leaving a damaged oesophagus in place, however, can result in multiple blind sacs and subsequent development of mediastinal abscesses years later. Most experienced surgeons recommend that the oesophagus be removed unless the operative risk is unduly high.[5]

Antral stenosis may develop rapidly 3 to 6 weeks after the injury, but in some cases it may appear only after several years.[26] Therefore, long-term follow-up is required even though the initial symptoms of the patients are minimal.[26] Some surgeons perform a Billroth I procedure for severely injured mucosa with complete pyloric obstruction, and pyloroplasty for moderate mucosa injury associated with partially obstructed but still viable pylorus.[27] However, distal gastric resection is usually recommended. Although many patients are initially achlorhydric, vagotomy is usually performed because acid production may return. With extensive injury, subtotal or total gastrectomy or partial oesophagectomy may be necessary.

A strong association exists between caustic injury and squamous cell carcinoma of the oesophagus. From 1% to 7% of patients with carcinoma of the oesophagus have a history of caustic ingestion. A 1000- to 3000-fold increase has been estimated in the expected incidence of oesophageal carcinoma after caustic ingestion.[3–7] Because of the markedly increased incidence of cancer in these patients, many authors have recommended yearly endoscopic surveillance beginning 20 years following caustic oesophageal injury.[7]

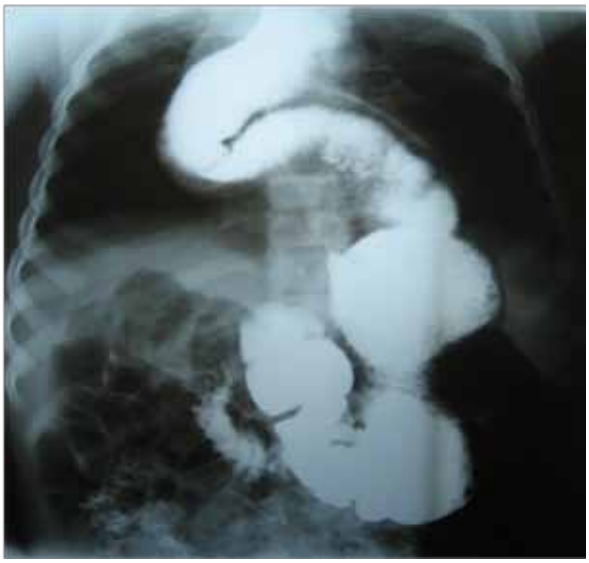

*Figure 51.8: Retrosternal colonic graft.*

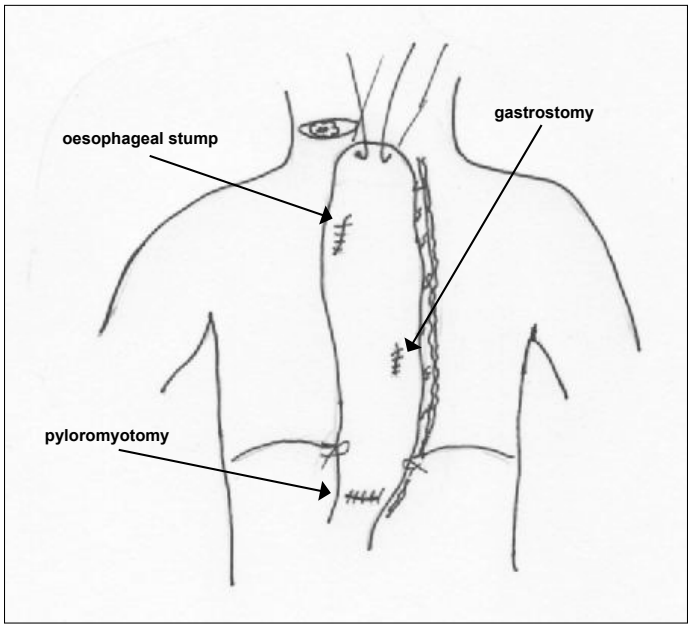


*Figure 51.9: Gastric interposition.*

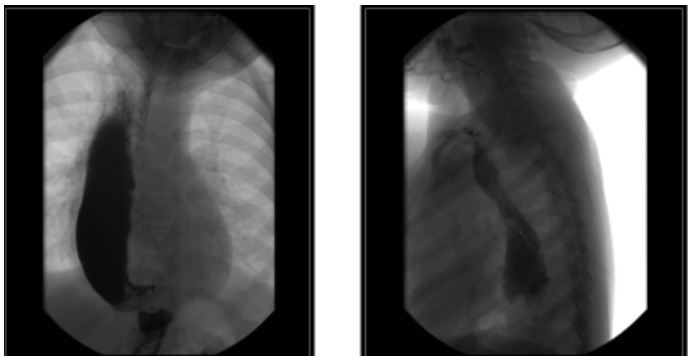

*Figure 51.10: Contrast of gastric interposition.*

## Key Summary Points

1. A preventive scheme is essential to avoid the devastating accidental injuries due to corrosive ingestion.
2. Alkaline injury is more extensive than acid injury due to the delay in the protective reflexes in an acid injury and rapid penetration of alkali into the tissues.
3. The most severe caustic injury generally occurs in the narrowest portion of the oesophagus, usually the midoesophagus in the region of the aortic arch.
4. Early clinical manifestations include persistent salivation, dysphagia, hoarseness of voice and stridor, retrosternal chest pain, and hematemesis.
5. Fever, shock, dyspnoea, and acute abdomen strongly indicate oesophageal or gastric perforation.
6. Dysphagia is the late presentation due to stricture.
7. Diagnosis is by imaging and endoscopy.
8. The goals of therapy are to prevent and treat perforation as early as possible, to avoid strictures of the oesophagus and stomach, and to replace or bypass the damaged organ to allow normal swallowing of food.
9. Therapy is variable.

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