HISTORY AND EPIDEMIOLOGY

As early as 1927, legislation in the United States governing the packaging of lye- and acid-containing products mandated that warning labels be placed on these products. In response to the recognition that caustic exposures were more frequent in children, the Federal Hazardous Substances Act and Poison Prevention Packaging Act were passed in 1970; these acts mandated that all caustics with a concentration greater than 10% be sold in child resistant containers. By 1973, the household concentration for child-resistant packaging was lowered to 2%. In addition, the subsequent development of poison prevention education dramatically decreased the incidence of unintentional caustic injuries in children in the United States. The positive impact of both regulatory legislation and public health intervention is evident when observing the decreasing number of significant exposures in the United States compared to the number of exposures in developing nations that lack these policies.

In the United States, even though legislation limiting the concentration of caustics has existed since the early 20th century, exposures to both acids and alkanis continue to be significant. Data collected by the American Association of Poison Control Centers from 2007 through 2010 revealed 29,748 acid exposures and 13,800 alkali exposures. Of these, 4273 (14.4%) acid exposures and 2645 (19.2%) alkali exposures resulted in moderate to major outcomes and a total of 25 deaths occurred (Chap. 136).

In children, exposures usually consist of household products and occur in an unsupervised setting. In adults, exposures to household or industrial products may result from occupational exposure, suicide attempts, and assaults. Exposure to caustics may occur via the dermal, ocular, respiratory, and gastrointestinal routes.

Caustics cause diverse histologic and functional damage on contact with tissues depending on the tissue and caustic involved. Table 106–1 lists common caustics and the commercial products that contain them. Many are available for home use, in both solid and liquid forms, with variations in viscosity, concentration, and pH.

TABLE 106–1. Sources of Common Caustics

Morbidity and mortality from exposures to caustics is a worldwide problem. One study from India that described outcomes in patients with acid ingestions found that acute complications occurred in 39.1% of cases and death in 12.2%.

Chap. 136
Although less frequent, intentional exposures by adults are invariably more significant. One study noted that while children comprised 39% of admissions for caustic ingestions, adults comprised 81% of patients requiring treatment. The severity of a caustic injury may not be immediately evident in patients who present shortly after exposure. Predicting which patients will require immediate interventions to prevent morbidity and mortality requires the determination and evaluation of multiple clinical and laboratory parameters. This chapter reviews the pathophysiology and approach to patients with potentially serious exposures.

PATHOPHYSIOLOGY

A caustic is a xenobiotic that causes both functional and histologic damage on contact with tissue surfaces. Although there are many ways to categorize caustics, they are most typically classified as acids or alkalis. An acid is a proton donator and causes significant injury, generally at a pH below 3. An alkali is a proton acceptor and causes significant injury, generally at a pH above 11. Chapter 12 contains a more detailed discussion of the chemistry of acids and bases. The extent of injury is modulated by duration of contact; ability of the caustic to penetrate tissues; volume, pH, and concentration; the presence or absence of food in the stomach; and a property known as titratable acid/alkaline reserve (TAR). TAR quantifies the amount of neutralization needed to bring the pH of a caustic to that of physiologic tissues. Neutralization of caustics takes place at the expense of the tissues, resulting in the release of thermal energy, producing burns. Generally, as the TAR of a caustic increases, so does the ability to produce tissue damage. Some xenobiotics, such as zinc chloride and phenol, have a high TAR and are capable of producing severe burns even though their pH is near physiologic.

Alkalis

Following exposure to an alkaline xenobiotic, dissociated hydroxide (OH−) ions penetrate tissue surfaces, producing a histologic pattern of liquefactive necrosis (Figs. 106–1 and 106–2). This process includes protein dissolution, collagen destruction, fat saponification, cell membrane emulsification, transmural thrombosis, and cell death. Animal studies following alkali exposure to the eye demonstrate rapid formation of corneal epithelial defects with eventual deep penetration that may lead to perforation. Similarly, animal studies of the esophagus demonstrate that erythema and edema of the mucosa occur within seconds followed by an inflammatory reaction extending to the submucosa and muscular layers. The alkali, such as sodium hydroxide (“liquid lye”), then continues to penetrate until the OH− concentration is sufficiently neutralized by the tissues.

FIGURE 106–1.
Photograph demonstrating burns to the lips and tongue of a 20 year-old man following ingestion of sodium hydroxide. (Used with permission of The New York City Poison Center Toxicology Fellowship Program.)
Although federal regulations have lowered the maximal available household concentration of many caustics, two industrial strength products seem to be readily available and therefore warrant special mention: ammonium hydroxide and sodium hypochlorite. Ammonia (ammonium hydroxide) products are weak bases—partially dissociated in water—that can cause significant esophageal burns, depending on the concentration and volume ingested. Household ammonium hydroxide ranges in concentration from 3% to 10%. Strictures have formed in patients who ingested 28% solutions. Sodium hypochlorite is the major component in most industrial and household bleaches. Severe injuries typically only occur in patients with large-volume ingestions of concentrated products and most other patients do well with supportive care. A series of 393 patients with household bleach ingestions demonstrated no stricture formation. Likewise, a canine model found that although vomiting was commonly associated with bleach ingestion, no esophageal lesions were noted, and perforation occurred only following prolonged contact.

Ingestion of button batteries were once considered a unique caustic exposure. Composed of metal salts and a variety of alkaline xenobiotics, such as sodium and potassium hydroxide, leakage of battery contents was a legitimate concern. In recent years, however, new techniques used in the production of button batteries that effectively prevent leakage have shifted the concern following their ingestion from caustic to foreign body exposure with the potential for electrical injury. For a more in-depth review of the management of button battery ingestion, the reader is referred to the previous editions of this text and one recent study that describes the electrical injuries that follow ingestion of large-diameter lithium cells lodged in the esophagus for longer than 2 hours.

Household detergents, such as laundry powders, laundry pods, and dishwasher detergents, contain silicates, carbonates, and phosphates, and have the potential to induce caustic burns and strictures, even when ingested unintentionally. Airway compromise also may occur, but the majority of exposures result in only minor toxicity.

Cationic detergents include quinolinium compounds, pyridinium compounds, and quaternary ammonium salts. These are frequently found in products for industrial use, as well as household fabric softeners. A concentration greater than 7.5% can cause severe burns.

**Acids**

In contrast to alkaline exposures, following exposure to an acid, hydrogen (H+) ions desiccate epithelial cells, producing an eschar and resulting in a histologic pattern of coagulation necrosis. This
process leads to edema, erythema, mucosal sloughing, ulceration, and necrosis of tissues. Dissociated anions of the acid (Cl\(^-\), SO\(_4^{2-}\), PO\(_4^{3-}\)) also act as reducing agents, further injuring tissue. Ophthalmic exposure to acids results in coagulative necrosis that tends to prevent further penetration into deeper layers of the eye.

In most series, following an acid ingestion, both the gastric and esophageal mucosa are equally affected.\(^{19,106}\) On occasion, the esophagus may be spared damage while severe injury is noted in the stomach (\textit{Fig. 106–3}). This result tends to be a rarer finding than concomitant injury to both stomach and esophagus and is probably related to the rapid transit time of liquid acids through the upper gastrointestinal tract. Skip lesions from acid ingestions may be a function of viscosity and contact time.\(^{27,36}\) Additionally, acid-induced pylorospasm may lead to gastric outlet obstruction, antral pooling, and perforation.\(^{19,105}\) A cat model of the effects of sulfuric acid on the esophagus revealed a coagulative necrosis of the mucosa with whitish discoloration of the tissues and underlying smooth muscle spasm.\(^5\) Other animal models demonstrate esophageal motility dysfunction and shortening.\(^{36}\)

\textbf{FIGURE 106–3.}
Postmortem specimen from a man with an intentional ingestion of a mixture of phosphoric and hydrochloric acid that was used as a brick cleaner. Note the relative sparing of the esophagus in contrast to full-thickness injury with perforation of the stomach. (\textit{Used with permission of The New York City Poison Center Toxicology Fellowship Program.})

\textbf{Chapters 98 and 107} contain a more detailed discussion of mercury and hydrofluoric acid, respectively, each a unique caustic, and the management specific to their exposure.

\textbf{Classification and Progression of Caustic Injury}
Esophageal burns, secondary to both alkali and acid exposures, are classified based on endoscopic visualization that employs a grading system similar to that used with burns of the skin. Grade I burns are generally defined by hyperemia or edema of the mucosa without evidence of ulcer formation.\(^{17,105}\) Grade II burns include submucosal lesions, ulcerations, and exudates. Some authors further divide grade II lesions into grade IIA, noncircumferential lesions, and grade IIB, near-circumferential injuries.\(^{13}\) Grade III burns are defined as deep ulcers and necrosis into the periesophageal tissues (\textbf{Table 106–2}).\(^{26,36}\)

\textbf{TABLE 106–2. Evaluation of Caustic Injuries and Management}

| View Large | Favorite Table |
Human case reports, postmortem studies, histologic inspection of surgical specimens, and experimental animal models reveal a consistent pattern of injury and repair following caustic injury. As wound healing of gastrointestinal tract tissue occurs, neovascularization and fibroblast proliferation take place, laying down new collagen and replacing the damaged tissue with granulation tissue. A similar pattern of repair occurs following caustic injuries of the eye.

Burns of the esophagus may persist for up to 8 weeks as remodeling takes place and may be followed by esophageal shortening. If the initial injury penetrates deeply enough, there is progressive narrowing of the esophageal lumen. The dense scar formation presents clinically as a stricture. Strictures can evolve over a period of weeks to months, leading to dysphagia and significant nutritional deficits. Grade I burns carry no risk of stricture formation. Grade II circumferential burns lead to stricture formation in approximately 75% of cases. Grade III burns invariably progress to stricture formation and are also at a high risk of perforation.

**CLINICAL MANIFESTATIONS**

The gastrointestinal tract, respiratory tract, eyes, and skin of a patient can be sites of caustic injury. Caustics may produce severe pain on contact with any of these tissues. By far, the majority of long-term morbidity and mortality from caustic exposure results from ingestion.

In general, patients who have ingested either alkalis or acids have similar initial presentations. Depending on the type, amount, and formulation (solid vs. liquid) as well as the percent of tissue exposed, ingestion may lead to the development of severe pain of the lips, mouth, throat, chest, or abdomen. Oropharyngeal edema and burns may lead to drooling and rapid airway compromise. Symptoms of esophageal involvement include dysphagia and odynophagia, whereas epigastric pain and hematemesis may be symptoms of gastric involvement.

Respiratory tract damage may occur through direct inhalation or aspiration of vomitus, leading to the clinical manifestations of hoarseness, stridor, and respiratory distress. Injury may result in epiglottitis, laryngeal edema and ulceration, pneumonitis, and impaired gas exchange. Patients may also be tachypneic or hyperpneic as a compensatory response to the metabolic acidosis, with elevated lactate concentrations from necrotic tissue or hemodynamic compromise.

**Predictors of Injury**

Many attempts have been made to define a method for clinical identification of patients with grade II or III esophageal injuries as these injuries typically progress to severe complications. Various studies, mostly involving alkaline xenobiotics, examine the predictive value of stridor, oropharyngeal burns, drooling, vomiting, and abdominal pain. A retrospective study of 378 children admitted for a caustic injury found that signs or symptoms could not be used to predict significant esophageal injury. However, one prospective study of 79 children evaluated for vomiting, drooling, and stridor found that a combination of two or more of these signs were predictive of significant esophageal injury as visualized on endoscopy. Another study found that drooling, buccal mucosal burns, and white blood cell count were significant independent predictors of severe gastrointestinal tract injury.
following acid ingestions. Studies evaluating the presence or absence of oropharyngeal burns as a predictor of distal esophagogastric injury have repeatedly found this finding to be poorly predictive. In one study esophageal injury was present 51.5% of the time in the absence of oropharyngeal lesions, and 22.2% of these were second- and third-degree burns. A prospective study of alkali ingestions in both adults and children found that stridor was 100% specific for significant esophageal injury, but this was based on only three patients with this sign.

Based on these findings, endoscopy, a standard diagnostic tool used in the management of caustic ingestions, is recommended in all patients with intentional ingestions. Endoscopy should also be performed in any patient with an unintentional ingestion in the presence of stridor and in any patient with two or more of the following findings: pain, vomiting, and drooling. Children with unintentional caustic ingestions who remain completely asymptomatic and tolerate liquids after a few hours of observation probably require no further medical care.

The abdominal examination is likewise an unreliable indicator of the severity of injury. The presence of abdominal pain suggests tissue injury, but the absence of pain or findings on abdominal examination does not preclude life-threatening gastrointestinal damage. Esophageal perforations result in mediastinitis and are commonly associated with fever, dyspnea, chest pain, and subcutaneous emphysema of the neck and chest. Although indicative of viscus perforation, abdominal peritoneal signs are late findings.

In addition to the direct effects that occur with tissue contact, systemic absorption of acids may result in damage to the spleen, liver, biliary tract, pancreas, and kidneys. This may also produce a metabolic acidosis, hemolysis, and, ultimately, death.

Significant complications can occur at various stages of wound recovery. Most importantly, these include airway compromise, hemodynamic instability secondary to hemorrhage from vascular erosion or septic shock, perforations of the gastrointestinal tract with the development of mediastinitis or peritonitis, and other overwhelming infections from bacteria residing in the oropharynx. A patient who survives acute injury with an acid or an alkali may also subsequently develop stricture formation, gastric atony, decreased acid secretion, pseudodiverticula, and gastric outlet obstruction.

Other complications include dysmotility of the pharynx and esophagus, formation of aorto- and trachea-esophageal fistulas, delayed massive hemorrhage from erosion into a great vessel, and pulmonary thrombosis. Those patients surviving a few weeks after a grade II or III injury may subsequently present with dysphagia and vomiting from stricture formation. Injury involving the entire length of the esophagus as well as hematemesis and increased serum lactic dehydrogenase were useful indicators for the development of strictures in one study. Strictures may also present with esophageal motility disorders caused by impaired smooth muscle reactivity. The early assessment and long-term prognosis may be better defined by manometric studies of the esophagus, which provide precise information about the severity of the initial injury and aid in long-term prognosis.
Long-term survivors of moderate and severe injury of the esophagus have a risk of esophageal carcinoma that is estimated to be 1000 times higher than that of the general population and appears to present with a latency of up to 40 years.1

**DIAGNOSTIC TESTING**

**Laboratory**
All patients with presumed serious caustic ingestion should have an evaluation of serum pH, blood type and cross-match, complete blood count, coagulation parameters, electrolytes, and urinalysis. Elevated prothrombin time (PT) and elevated partial thromboplastin time (PTT),104 as well as an arterial pH lower than 7.22,11 are associated with severe caustic injury.

Absorption of nonionized acid from the stomach mucosa may result in acidemia. Following ingestion of hydrochloric acid, hydrogen and chloride ions (both of which are accounted for in the measurement of the anion gap) dissociate in the serum resulting in a hyperchloremic normal anion gap metabolic acidosis. Other acids, such as sulfuric acid, result in an elevated anion gap metabolic acidosis because the sulfate anion (SO$_4^{2-}$) is not measured in the calculation of the anion gap. Although alkalis are not absorbed systemically, necrosis of tissue may result in a metabolic acidosis with an elevated lactate concentration.

A gastric pH greater than 7.30 correlated retrospectively with severe alkaline injury. The prospective usefulness of this information is limited, as obtaining gastric secretions without direct visualization is dangerous. One prospective study in children also found an increase in uric acid and decreases in phosphate and alkaline phosphatase concentrations to be useful in predicting the presence of esophageal injuries.75

**Radiology**
Chest and abdominal radiographs are useful in the initial stages of assessment to detect gross signs of esophageal or gastric perforation. Signs of alimentary tract perforation that may be present on plain radiographs include pneumomediastinum, pneumoperitoneum, and pleural effusion. However, these studies have a limited sensitivity, and an absence of findings does not preclude perforation.105 Free intraperitoneal air is best visualized on an upright chest radiograph. Occasionally, free air may only be visible on the lateral view. In patients too ill to obtain an upright chest radiograph, an abdominal radiograph obtained with the patient in a left-side-down position may reveal free intraperitoneal air adjacent to the liver. Additionally, bedside ultrasound may be useful in the diagnosis of free air and is based entirely on the lack of visualization of the usual intraperitoneal structures.10 124 Computed tomography (CT) scanning is considerably more sensitive than both radiography and ultrasound for detecting viscus perforation and should be obtained in patients with potentially serious caustic ingestions as soon as is feasible.23 101

A contrast esophagram is useful for defining the extent of esophageal injury (Fig. 106–4). Late after the ingestion, it can detect stricture formation. In patients for whom there is a high suspicion for esophageal perforation and in whom adequate visualization of the upper gastrointestinal tract by
endoscopy is not possible (grade IIb circumferential burns or grade III burns), an enteric contrast study (esophagram and upper gastrointestinal series) can be obtained 24 hours after the ingestion. Extravasation of contrast outside of the gastrointestinal tract is diagnostic of perforation. Water-soluble contrast should be used when perforation is suspected as it is less irritating than barium contrast agents to mediastinal and peritoneal tissues if extravasated. However, barium contrast agents are more radiopaque than water-soluble agents and offer greater radiographic detail. Consequently, some authors recommend barium swallow if the water-soluble contrast study is nondiagnostic but demonstrates no leak. In addition, if there is risk of aspiration, barium is preferred because water-soluble contrast material can cause a severe chemical pneumonitis. Significant necrosis with impending perforation may be suspected on enteric contrast studies when there is esophageal dilation, displacement of the pleural reflection, and widening of the pleuroesophageal line. Enteric contrast studies may fail to detect perforation and therefore must be interpreted within the context of the patient’s clinical status.

FIGURE 106–4.

(A) Barium swallow several days after ingestion of liquid lye shows the esophagus to be atonic. There is poor coating of the esophagus, suggesting edema and intramural penetration. Note that the initial evaluation immediately following a caustic ingestion to assess the extent of injury is esophagoscopy, rather than a contrast esophagram. (B) Four months later, a repeat barium esophagram shows a severe stricture below the middle third of the esophagus. The barium barely passes the stricture, and the remainder of the esophagus is pencil thin. (Used with permission of Emil J. Balthazar, MD, Professor of Radiology, New York University.)

The role for CT scans in caustic ingestions has not been prospectively investigated. In the acute stage, CT has great sensitivity at detecting extraluminal air in the mediastinum or peritoneal cavity as a sign of perforation. In addition, CT can visualize the esophagus and stomach distal to severe caustic burns that cannot be safely seen using endoscopy or an esophagram. CT may therefore replace enteric contrast radiography for detection of perforation within 24 hours of a caustic ingestion. Additionally, one retrospective study suggests that CT grading of esophageal injuries may be superior to endoscopy for prediction of the degree of esophageal damage and the development of stricture formation. These results suggest a promising future role for this noninvasive study following caustic ingestions. Other imaging modalities have been proposed for assessing esophageal injury after ingestion of caustic substances, including technetium 99m-labeled sucralfate swallow for the presence of injury and esophageal ultrasonography for determining the depth of injury.

Another use of radiographic imaging is to noninvasively follow the patient after initial evaluation and stabilization. For example, contrast radiography is routinely used in the weeks or months following a caustic ingestion to detect esophageal narrowing representing stricture formation. Chest CT may also be useful to determine the response of strictures to dilation procedures.
Endoscopy

Endoscopy should be performed within 12 hours and generally not later than 24 hours postingestion. Numerous case series demonstrate that the procedure is safe during this period. Early endoscopy serves multiple purposes in that it allows patients with minimal or no evidence of gastrointestinal injury to be discharged. It also offers a rapid means of obtaining diagnostic and prognostic information while shortening the period of time that patients forego nutritional support, permitting more precise treatment regimens. The use of endoscopic assessment from the second or third day postingestion is discouraged and should be avoided between 5 days and 2 weeks postingestion; at this time, wound strength is least and the risk of perforation is greatest.

The choice of rigid versus flexible endoscopy is dependent on the comfort and experience of the endoscopist. The flexible endoscope has a smaller diameter but may require gentle insufflation of air to achieve or enhance visualization. A prospective evaluation of the role of fiberoptic endoscopy in the management of caustic ingestions recommended the following guidelines: (a) direct visualization of the esophagus prior to advancing the instrument, (b) minimal insufflation of air, (c) passage into the stomach unless there is a severe (particularly circumferential) esophageal burn, and (d) avoidance of retroversion or retroflexion of the instrument within the esophagus. Provided that the patient is hemodynamically stable and endoscopy is indicated, every attempt should be made to visualize the esophagus, stomach, and duodenum as soon as possible after a caustic ingestion. The absence of burns in the esophagus does not imply that severe necrosis and ulcerations do not exist in the stomach and duodenum. In the case of termination of endoscopy because of grade IIb or grade III esophageal burns, barium studies, CT scan, or consideration of surgical exploration should be undertaken to visualize remaining structures.

Endoscopy permits limited evaluation of gastrointestinal injury. For example, the endoscopist is able to appreciate only the mucosal surface of tissues, not the serosal side. This is especially evident in stomach ulcerations, which may appear black and necrotic from a true burn through the layers of the stomach or from the effect of stomach acid on the blood exposed from a shallow lesion. As mentioned above, in these cases, endoscopic ultrasonography during endoscopy may improve assessment of injury depth. Often, however, only direct visualization of serosal and mucosal tissues with laparoscopy or laparotomy allows for definitive evaluation.

Most cases of perforation clearly linked to endoscopy have occurred when the endoscope was advanced through an esophagus with severe circumferential lesions—a violation of current endoscopic standards. In addition, perforations are also more likely to occur when rigid instruments are used in children or in uncooperative patients. Thus the use of the flexible endoscope and adequate procedural sedation has decreased the complications from endoscopic evaluation. Some authors advocate the presence of a surgeon during endoscopy to assist in the assessment for potential surgical intervention.

MANAGEMENT
Acute Management
As in the case of any patient presenting with a toxicologic emergency, the health care provider must adhere to universal precautions utilizing early decontamination as described in the following section. Initial stabilization should include airway inspection and protection, basic resuscitation principles, and decontamination. Examination of the oropharynx for signs of injury, drooling, and vomitus, as well as careful auscultation of the neck and chest for stridor, may reveal signs of airway edema that should prompt immediate airway protection. Careful and constant attention to signs and symptoms of respiratory distress and airway edema, such as a change in voice, are essential and should prompt early intubation as airway edema may rapidly progress over minutes to hours.

If airway involvement is significant enough to warrant intubation, it is best to mobilize a team of the most skilled physicians early in case of unforeseen complications. A delay in prophylactic airway protection may make subsequent attempts at intubation or bag-valve-mask ventilation difficult or impossible. Direct visual inspection of the vocal cords with a fiberoptic laryngoscope may also reveal signs of impending airway compromise. Patients necessitating intubation are best served by direct visualization of the airway either via direct laryngoscopy or fiberoptic endoscopy, as perforation of edematous tissues of the pharynx and larynx is a grave complication that may occur during blind nasotracheal intubation attempts. Neuromuscular blockers should be avoided for induction of intubation as airway edema and bleeding may distort the anatomy limiting the ability to successfully ventilate via bag-valve-mask should intubation be unsuccessful.

Nonsurgical airway placement is recommended whenever possible as both cricothyrotomy and tracheostomy may interfere with the surgical field if esophageal repair is required. Some patients with significant ingestions, however, may require emergent surgical airway intervention. The decision to perform a surgical airway is dependent on the status of the patient, the ability to orotracheally or nasotracheally intubate via a fiberoptic endoscope, and the comfort of the physician performing the procedure.

Following control of the airway, large-bore intravenous access should be secured and volume resuscitation initiated. Although not studied, most clinicians agree that patients with signs of caustic-induced airway edema benefit from dexamethasone 10 mg (intravenous) in adults and 0.6 mg/kg up to a total dose of 10 mg in children. Both acid and alkali ingestions cause “third spacing” of intravascular fluid to the interstitial space, which can result in hypotension. Empiric rehydration with clinical assessment of central venous pressures should be used to guide individual fluid requirements. Serial physical examinations and constant monitoring of the vital signs and urine output may provide information on the severity of the exposure and the progression in clinical status.

Decontamination, Dilution, and Neutralization
Decontamination should begin with careful, copious irrigation of the patient’s skin and eyes when indicated to remove any residual caustic and to prevent contamination of other patients, staff, and equipment.
Gastrointestinal decontamination is usually limited in patients with a caustic ingestion. Induced emesis is contraindicated, as it may cause reintroduction of the caustic to the upper gastrointestinal tract and airway. Activated charcoal is also contraindicated, as it will interfere with tissue evaluation by endoscopy and preclude a subsequent management plan. Additionally, most caustics are not adsorbed to activated charcoal.

Exceptions, such as cationic detergents, that do bind well to activated charcoal have not been evaluated with a large series. For this reason, therapy with activated charcoal following any caustic ingestion cannot be recommended. Gastric emptying via cautious placement of a narrow nasogastric tube with gentle suction may be attempted to remove the remaining acid in the stomach only in patients with large, life-threatening, intentional ingestions of acid who present within 30 minutes. Although this technique has never been studied and carries the risk of perforation, the outcome for this particular group of patients with massive exposure is often grave, and options for treatment are limited. Therefore, preventing absorption of some portion of the ingested acid may have potential benefit in reducing systemic toxicity. Although the procedure has the potential to induce injury, a risk-to-benefit analysis favors gastric emptying following a presumed lethal ingestion.

In contrast, gastric emptying should be avoided with alkaline and unknown caustic ingestions as blind passage of a nasogastric tube carries the risk of perforation of damaged tissues, a risk that outweighs the benefit.

Exceptions to the general rules of gastrointestinal decontamination of caustics exist in the management of zinc chloride (ZnCl₂) and mercuric chloride (HgCl₂). Both are caustics with severe systemic toxicity. Ingestion of these xenobiotics causes life-threatening illness from cationic metal exposure. The local caustic effects, though of great concern, are less consequential than the manifestations of systemic absorption. Therefore, prevention of systemic absorption should be addressed primarily, followed by the direct assessment and management of the local effects of these xenobiotics. Initial management to prevent systemic absorption includes aggressive decontamination with gentle nasogastric tube aspiration and administration of activated charcoal. In vitro data exist to suggest adequate activated charcoal adsorption of Hg₂⁺.

The use of dilutional therapy has been examined using in vitro, ex vivo, and in vivo models in an attempt to assess its efficacy in caustic ingestions. An early in vitro model demonstrated a dramatic increase in temperature when either water or milk was added to a lye containing crystal drain opener (NaOH). Another in vitro model found less consequential increases in temperature despite large volumes of diluent. Results of both studies suggested that dilutional therapy was of limited benefit. Dilutional therapy was also attended by an increase in temperature in an ex vivo study of harvested rat esophagi that examined the histopathologic effects of saline dilution after an alkali injury. Additionally, the usefulness of dilution appeared to be inversely related to the length of time from exposure, with minimal efficacy when delay to initiation was as short as 30 minutes. In contrast, an in vivo canine model of alkaline injury demonstrated that water dilution did not cause an increase in either temperature or intraluminal pressures.
The extrapolation of these variable results to humans with caustic ingestions is limited and suggests that histologic damage can only be attenuated by milk or water when administered within the first seconds to minutes following ingestion.\(^5\)\(^,\)\(^42\)\(^,\)\(^43\)\(^,\)\(^44\)\(^,\)\(^45\)\(^,\)\(^53\) For solid, as opposed to liquid, substances (e.g., crystal lye), there may be some value for delayed dilutional therapy, as tissue contact time is increased with solids and their concentration is usually 100% over a small surface area. Milk may be the best diluent to attenuate the heat generated by a caustic.

Caution should be used in advising patients or family members about the use of diluents. A child who refuses to swallow or take oral liquids should never be forced to do so. In general, dilutional therapy should be limited to the first few minutes after ingestion in patients who have no airway compromise; are not complaining of significant pharyngeal, chest, or abdominal pain; are not vomiting; and are alert. Dilutional therapy should be avoided in patients with nausea, drooling, stridor, or abdominal distension as it may stimulate vomiting and result in reintroduction of the caustic into the upper gastrointestinal tract.\(^87\)

Attempts at neutralization of ingested caustics should likewise be avoided. This technique has the potential to worsen tissue damage by forming gas and generating an exothermic reaction. In vitro and ex vivo models demonstrate that neutralization of caustics generates heat, requires a large volume to attain physiologic pH, and may have limited usefulness in preventing histologic damage if delayed beyond the first several minutes following caustic exposure.\(^41\)\(^,\)\(^87\) In one in vivo canine model, orange juice was used to neutralize sodium hydroxide–induced gastric injury and demonstrated no change in temperature or intraluminal pressure.\(^45\) Despite this study, neutralization is not recommended; there are no other data demonstrating that clinical outcome is improved.

**Surgical Management**

The decision to perform surgery in patients with caustic ingestions is obvious in the presence of either endoscopic or diagnostic imaging evidence of perforation,\(^104\) severe abdominal rigidity, or persistent hypotension. Hypotension is a grave finding and often indicates perforation or significant blood loss. Additionally, elevated PT and PTT,\(^104\) as well as acidemia,\(^11\) are correlated with severe caustic injury.

Many patients will not have an obvious indication for surgical intervention despite impending perforation, necrosis, sepsis, or delayed hemorrhage. Although more challenging to diagnose, all these sequelae are potentially avoidable if surgery is performed early.\(^74\) as morbidity and mortality increase in patients whose surgery is delayed.\(^22\)\(^,\)\(^47\)\(^,\)\(^85\) For this reason, some surgeons advocate surgery for all patients with grades II and III esophageal burns identified on endoscopy.\(^22\)\(^,\)\(^65\) This aggressive approach allows for direct inspection of serosal surfaces and an opportunity for early surgical repair.

Multiple studies have attempted to codify the signs and symptoms necessary or sufficient to rapidly identify patients who would benefit from surgery but who lack clear clinical indications. Several retrospective and prospective series of caustic ingestions found that patients with large ingestions (>150 mL), shock, acidemia, or coagulation disorders tended to have severe findings on surgical
exploration. These studies also reinforce that the abdominal examination was frequently unreliable in predicting the need for surgery. It should be noted, again, that patients with severe acid injuries may lack abdominal pain, abdominal tenderness, and have positive findings on diagnostic imaging. One author used a stepwise approach of bronchoscopy, endoscopy, and abdominal ultrasonography to provide additional information regarding extent of injury prior to surgery. Respiratory distress, ascites, pleural fluid, and a serum pH less than 7.2 were used as indications for surgery. A history of a large-volume caustic ingestion (between 40 and 200 mL) should also prompt consideration of early surgical intervention as delay is associated with increased mortality.

Surgical intervention may include laparotomy for tissue visualization, resection, and repair of perforations. Laparoscopy may also be used, although it may not allow inspection of the posterior aspect of the stomach.

**Subacute Management**
The extent of tissue injury dictates the subsequent management and disposition of patients with caustic ingestions.

**Grade I Esophageal Injuries.**
Patients with isolated grade I injuries of the esophagus do not develop strictures and are not at increased risk of carcinoma. Their diet can be resumed as tolerated. No further therapy is required. These patients can be discharged from the emergency department as long as they are able to eat and drink and their psychiatric status is stable.

**Grade IIa Esophageal Injuries.**
If endoscopy reveals grade IIa lesions of the esophagus and sparing of the stomach, a soft diet can be resumed as tolerated or a nasogastric tube can be passed under direct visualization. If oral intake is contraindicated because of the risk of perforation, feeding via gastrostomy, jejunostomy, or total parenteral nutrition should be instituted as rapidly as possible. Providing interim enteral support is imperative as metabolic demands are increased in any patient with a significant burn.

**Grades IIb and III Esophageal Injuries.**
Patients with grades IIb and III lesions must be followed for the complications of perforation, infection, and stricture development. Strictures are a debilitating complication of both acid and alkali ingestions that can evolve over a period of weeks or months. Strictures form as a result of the natural process by which the body repairs injured tissue through the production of collagen with resultant scar formation. Although corticosteroid therapy is theorized to arrest the process of inflammatory repair and potentially prevent stricture formation, there is some evidence that grade III burns, in particular, will progress to stricture formation regardless of therapy. In addition to stricture formation, patients with grade III burns are also at high risk for other complications, including fistula formation, infection, and perforation with associated mediastinitis and peritonitis. The use of corticosteroids in the management of grade III burns may mask infection and make the friable, necrotic esophageal tissue more prone to perforation. For these reasons, corticosteroid
therapy is not a recommended therapy for grade III esophageal burns. When required in these patients for other indications such as caustic-induced airway inflammation, short-term corticosteroids should be administered.

Currently, some controversy exists regarding the use of corticosteroid therapy in the management of grade IIb circumferential esophageal burns. A meta-analysis of studies completed from 1956 to 1991, with a total of 361 patients, evaluated the efficacy of corticosteroid therapy and found that in patients with grades II and III esophageal burns, strictures formed in 19% of the corticosteroid-treated group and in 41% of the untreated group. The usefulness of the results of this study, however, are limited as no distinction was made between grades II and III burns. Another meta-analysis of studies from 1991 to 2003, with a total of 211 patients, was unable to find a benefit in treating patients with corticosteroids with grades II and III esophageal burns. However, no distinction was made between grades II and III burns. A systematic pooled analysis of studies from 1956 to 2006, with a total of 328 patients, attempted to reevaluate the usefulness of corticosteroid therapy in grade II esophageal burns. Although methodologically limited, this study found no benefit in treating patients with steroids with grade II esophageal burns. A major limitation to the clinical usefulness of this study is that no distinction was made between grades IIa and IIb burns. In addition, a multitude of case series also failed to clearly differentiate between grades IIa, IIb, and III lesions, making clinical application of their results difficult.

Two prospective studies attempted to evaluate the efficacy of corticosteroid therapy for caustic injuries to the esophagus. Both these studies failed to show a benefit of corticosteroid therapy, and one even suggested harm. It is imperative that the clinician understands that neither study clearly differentiates between grades IIb and III lesions.

Adequate human data demonstrating the efficacy of corticosteroids with or without antibiotics in the treatment of grade IIb circumferential lesions have yet to be generated. Because of the inherent risks involved in this therapy and the paucity of data supporting their use, corticosteroid therapy in the management of grade IIb esophageal burns can no longer be routinely recommended.

No major outcome studies have investigated the use of antibiotics alone as prophylactic treatment for stricture prevention, but most clinicians would agree that it is probably best to reserve antibiotics for an identified source of infection.

A variety of other management strategies have been used in an attempt to prevent strictures and esophageal obstruction. In both animal models and in human case series, intraluminal stents and nasogastric tubes made of silicone rubber tubing can successfully maintain the patency of the esophageal lumen. For nutritional support, the stents are usually attached to a feeding tube secured in the nasopharynx through which the patient can receive feedings without interfering with esophageal repair. These tubes are left in place for 3 weeks and are often used with concomitant corticosteroid and antibiotic therapy. In animal models, the use of a stent for 3 weeks is superior in maintaining esophageal patency when compared to corticosteroids and antibiotics alone.
Potential disadvantages of esophageal stents include mechanical trauma at the site and increased reflux, both of which may inhibit healing. A feline model of esophageal exposure to sodium hydroxide used stents but reported deaths from aspiration and mediastinitis. One series of 251 humans exposed to caustics who were managed with silicone rubber stents found that the procedure was successful in preventing stricture formation.

Additionally, a plethora of animal models have attempted to identify therapies that attenuate oxidative damage, inhibit synthesis, or stimulate breakdown of collagen and thereby prevent stricture formation. β-Amino propionitrile, penicillamine, N-acetylcysteine, halofuginone, vitamin E, sphingosylphosphorylcholine, colchicine, erythropoietin, mitomycin C, ozone, fibroblast growth factor, 5-fluorouracil, buprofen, and retinoic acid are some of these xenobiotics. As none of these treatments have been adequately studied in humans, they cannot currently be recommended in the routine management of caustic ingestions.

**Chronic Treatment of Strictures**

Commonly, the management of esophageal strictures includes early endoscopic dilation, for which a variety of types of dilators are available. Contrast CT can be used to determine maximal esophageal wall thickness, which can then be used to predict response, as well as the number of sessions required to achieve adequate dilation. Multiple dilations are often necessary. In one study, patients with a maximal esophageal wall thickness of 9 mm or greater required more than seven sessions to achieve adequate dilation. This was significantly higher than in patients with a lesser maximal wall thickness. Measurement of maximal wall thickness may be also be useful in determining long-term follow-up, type of nutritional support, and the potential need for surgical repair as an alternative to dilations. It may also provide an indication for those who should undergo dilation under fluoroscopy to limit the risk of perforation.

The risk of perforation from esophageal dilation is decreased if the initial procedure is delayed beyond 4 weeks postingestion, when healing, remodeling, and potential stricture formation in the esophagus have already taken place. Several series report perforation secondary to esophageal dilation. Following perforation, patients may complain of dyspnea or chest pain with associated subcutaneous emphysema or pneumomediastinum. Diagnostic imaging may identify the perforation and provide information for emergent surgical repair if the diagnosis is unclear.

Patients with stricture formation require long-term endoscopic follow-up for the presence of neoplastic changes of the esophagus that may occur with a delay of several decades.

**Management of Ophthalmic Exposures**

Ophthalmic exposures frequently occur from splash injuries and malicious events as well as from the alkaline byproducts of sodium azide released in automobile air bag deployment and rupture. The mainstay of therapy for these patients is immediate irrigation of the eye for a minimum of 15 minutes with 0.9% sodium chloride, lactated Ringer solution, or tap water, if it is the only therapy immediately available. Several liters of irrigation fluid are recommended. The normal pH of ophthalmic secretions is approximately 6.5 to 7.6. This can be tested colorimetrically by using a urine dipstick, which can
test a range of pH from 5 to 9. Litmus paper can be used in the same fashion. Another useful option in acid exposures is Nitrazine paper, which changes color from yellow to dark blue at a pH above 6.5. These different test strips can be applied to the ophthalmic secretions to test the baseline pH and followed with intermittent evaluations after 15 minutes of lavage to determine the adequacy of irrigation. If these xenobiotics are not readily available, irrigation should not be delayed, as the depth of penetration of the caustic agent will determine outcome. Anterior chamber irrigation may be required and should be performed emergently by an ophthalmologist. A thorough eye examination should be completed, and follow-up should be arranged. Chapter 25 contains a more detailed description of the evaluation and management of toxicologic emergencies of the eye.

**SUMMARY**

- Initial management of all patients with caustic exposures begins with universal precautions in an effort to prevent further contamination of staff, other patients, and equipment.
- In patients with caustic ingestions, airway assessment and stabilization are of primary importance. Airway edema is the only indication for initiation of corticosteroid therapy.
- There is no routine recommendation for induced emesis, lavage, activated charcoal, neutralization, or dilutional therapy.
- Significant caustic injury should be suspected in all patients with intentional ingestions and in patients with unintentional ingestions presenting with stridor; vomiting; drooling; and pain in the oropharynx, chest, or abdomen.
- All patients with suspected significant ingestions should undergo endoscopy or CT emergently so that effective treatment strategies may be initiated expeditiously.
- Surgeons should be involved in the initial assessment of all patients with suspected significant ingestions and those who have an acute abdomen or hypotension so that any surgical intervention deemed necessary may be performed promptly.

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