INTRODUCTION AND EPIDEMIOLOGY

Caustics are substances that cause both functional and histologic damage on contact with body surfaces. Many household and industrial chemicals have caustic potential. Caustics are broadly classified as alkalis (pH >7) or acids (pH <7). In developed nations, increased education and product regulation (especially of acids) have decreased morbidity and mortality from caustic exposures in both adults and children. However, in underdeveloped parts of the world, exposure to caustics remains a significant problem.1,2,3,4 The challenges to exposure prevention and patient care include relative lack of childproof containers, easy and unregulated access to highly corrosive substances, cultural-specific propensities to ingest caustics in suicide attempts, sheer high volume of cases, delays to care in rural settings, malnutrition, financial resources of hospitals and families to provide the services needed, and poor follow-up.4 Alkaline ingestions predominate in the developed world,5 whereas acid ingestions are more common in developing countries.6

Caustic exposures tend to fall into three distinct groups: (1) intentional teen or adult ingestions with suicidal ideation;7 (2) unintentional ingestions (the majority of which are by curious children in the toddler age group);8 and (3) other incidental, often occupational or industrial contact exposures. The majority of reported exposures are unintentional or accidental, but although less frequent, intentional ingestions account for the majority of serious injuries.1 The geographic variation in caustic ingestion circumstances, such as involved substances, intention, age of the patient, and extent of evaluation, make it difficult to create encompassing recommendations or a consensus approach.4,9,10

Many chemicals used in industry have caustic potential (Table 200-1). Household caustics are often less concentrated forms of industrial strength cleansers.
## TABLE 200-1

**Common Caustic Compounds**

<table>
<thead>
<tr>
<th>Alkali</th>
<th>Found in</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium hydroxide</td>
<td>Industrial chemicals, drain openers, oven cleaners</td>
</tr>
<tr>
<td>Potassium hydroxide</td>
<td>Drain openers, batteries</td>
</tr>
<tr>
<td>Calcium hydroxide</td>
<td>Cement, hair relaxers, and perm products</td>
</tr>
<tr>
<td>Ammonium hydroxide</td>
<td>Hair relaxers and perm products, dermal peeling/exfoliation, toilet bowl cleaners, glass cleaners, fertilizers</td>
</tr>
<tr>
<td>Lithium hydroxide</td>
<td>photographic developer, batteries</td>
</tr>
<tr>
<td>Sodium tripolyphosphate</td>
<td>Detergents</td>
</tr>
<tr>
<td>Sodium hypochlorite</td>
<td>Bleach</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Acids</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfuric acid</td>
<td>Automobile batteries, drain openers, explosives, fertilizer</td>
</tr>
<tr>
<td><em>Acetic acid</em></td>
<td>Printing and photography, disinfectants, hair perm neutralizer</td>
</tr>
<tr>
<td>Hydrochloric acid</td>
<td>Cleaning agents, metal cleaning, chemical production, swimming pool products</td>
</tr>
<tr>
<td>Hydrofluoric acids</td>
<td>Rust remover, petroleum industry, glass and microchip etching, jewelry cleaners</td>
</tr>
<tr>
<td>Formic acid</td>
<td>Model glue, leather and textile manufacturing, tissue preservation</td>
</tr>
<tr>
<td>Chromic acid</td>
<td>Metal plating, photography</td>
</tr>
<tr>
<td>Nitric acid</td>
<td>Fertilizer, engraving, electroplating</td>
</tr>
<tr>
<td>Phosphoric acid</td>
<td>Rust proofing, metal cleaners, disinfectants</td>
</tr>
</tbody>
</table>
PATHOPHYSIOLOGY

The degree to which a caustic substance produces tissue injury is determined by a number of factors: pH, concentration, duration of contact, volume present, and titratable acid or alkaline reserve. Acids tend to cause significant injuries at a pH <3 and alkalis at a pH >11. The physical properties of the product formulation (i.e., liquid, gel, granular, or solid) can influence the nature of the contact with the tissue. Following ingestion, solid or granular caustics often injure the oropharynx and proximal esophagus, whereas liquid alkali ingestions are characterized by more extensive esophageal and gastric injuries. Titratable acid or alkaline reserve refers to the amount of acid or base required to neutralize the agent; the greater this value, the greater is the potential for tissue injury.

Esophageal mucosal burns from caustic ingestions are classified by a visual endoscopic grading system: grade 1 burns involve tissue edema and hyperemia; grade 2 burns include ulcerations, blisters, and whitish exudates, which are subdivided into grade 2A (noncircumferential) and 2B (deeper or circumferential) lesions; and grade 3 burns are defined by deep ulcerations and necrotic lesions. Following the initial mucosal injury, tissue remodeling occurs over roughly 2 months. In mild cases, normal esophageal function is restored, but in severe cases, dense scar tissue forms, resulting in stricture formation. Esophageal strictures are a source of significant morbidity and may require long-term treatments with dilations, stenting, or surgery. Early phases of remodeling, particularly days 2 to 14, are associated with increased tissue friability and higher risk of perforation, both spontaneous and iatrogenic.

ALKALI INJURIES

Following caustic alkali exposures, the hydroxide ion easily penetrates tissues, causing immediate cellular destruction via protein denaturation and lipid saponification. This is followed by thrombosis of local microvasculature that leads to further tissue necrosis. Alkali injuries induce a deep tissue injury called liquefaction necrosis. Severe intentional alkali ingestion may cause deep penetration into surrounding tissues with resultant multisystem organ injuries, including esophageal injury, gastric perforation, and necrosis of abdominal and mediastinal structures. Severe injuries to the pancreas, gallbladder, small intestine, and mediastinum after intentional ingestion have been reported. Solid alkali ingestions, such as some lye preparations, have a greater potential for oropharyngeal and proximal esophageal tract injury and less for distal injury.

The most common household alkali is bleach, a 3% to 6% sodium hypochlorite solution with a pH of approximately 11. Household liquid bleach is minimally corrosive to the esophagus and rarely causes significant injury beyond grade 1 esophageal burns. Esophageal stricture was not observed as a complication of household bleach ingestion in a series involving almost 400 patients. However, ingestion of industrial strength bleach containing much higher concentrations of sodium hypochlorite may result in gastric and esophageal necrosis. Bleach ingestion may cause emesis secondary to gastric irritation and/or pneumonitis after aspiration. Pulmonary irritation related to chlorine gas production in the stomach or when mixed with
other substances may also occur.\textsuperscript{12,13} A common reaction is the production of the highly irritating chloramine gas when bleach and ammonia household cleaners are combined.\textsuperscript{14}

**ACIDS**

Injuries by strong acids produce \textit{coagulation necrosis}. Dissociated hydrogen ions and their associated anions penetrate tissues, leading to cell death and eschar formation. This process is believed to limit continued hydrogen ion penetration and protect against deeper injury. When ingested acids settle in the stomach, gastric necrosis, perforation, and hemorrhage may result. Although it was previously thought that acids were esophagus-sparing with most tissue injury concentrated in the stomach, endoscopy following an acid ingestion finds a similar incidence of gastric and esophageal injury.\textsuperscript{15,16,17} Despite relatively less tissue destruction, strong acid ingestion results in high-grade gastric injuries (secondary to pylorospasm and pooling) and a higher mortality rate compared with strong alkali ingestions. Acid ingestion is sometimes complicated by systemic absorption of acid with associated metabolic acidosis, hemolysis, and renal failure.\textsuperscript{18} Hydrofluoric acid is a unique ingestion discussed in chapter 217, Chemical Burns (see Hydrofluoric Acid).

**CLINICAL FEATURES**

The cardinal features of caustic ingestion are a chemical burn to the oral mucosa,\textsuperscript{19} sometimes associated with chemical burns to the skin or eyes from splashes or dribbling (see chapters 217, Chemical Burns and 241, Eye Emergencies). Pharyngeal burns from ingestion produce pain, odynophagia, drooling, and vocal hoarseness. Dyspnea may be caused by edema of the upper airway, aspiration of the caustic substance into the tracheobronchial tree, or inhalation of fumes, particularly acids. Esophageal burns produce dysphagia, odynophagia, and chest pain. Ocular burns are painful, reduce visual acuity, and produce visible damage to the anterior structures of the eye.

**HISTORY**

The key priority is rapid airway assessment and stabilization. Following that, obtain a directed history to determine the type and amount of caustic ingested and the presence of coingestants. Determine if the ingestion was intentional or unintentional.

**PHYSICAL EXAMINATION**

Look for signs of respiratory distress or circulatory shock. With ingestions, look for signs of pharyngeal injury (mucosal burns, drooling), respiratory injury (dysphonia, coughing, stridor, wheezing), and gastric injury (vomiting, epigastric tenderness) (\textit{Figure 200-1}).\textsuperscript{5,19,20,21,22,23,24} Streaks of caustic burns on the face or chest are called "dribble burns" (\textit{Figure 200-2}).

\textbf{FIGURE 200-1.}
Acid ingestion. A. Moderate intraoral burns on buccal mucosa and tongue. B. Lingual burns.

Acid ingestion. Dermal dribble burns on upper chest.
DIAGNOSIS

Conflicting data exist on the reliability of presenting signs and symptoms to predict upper GI injuries.\textsuperscript{25,26,27,28,29} Intentional ingestions are associated with higher grades of GI tract injury, with or without clinically obvious signs.\textsuperscript{30} The incidence of serious GI injury after pediatric unintentional ingestions has been the focus of many studies.\textsuperscript{5,25-27,29,31,32} Although serious esophageal injury can occur in the absence of oral burns, essentially all children with serious esophageal injuries (grade 2 or 3) after accidental caustic ingestion have some initial sign or symptom, such as stridor, drooling, or vomiting.\textsuperscript{26,27,29} Pain alone is an inconsistent predictor of severity of injury, and pain may be absent initially and early in the clinical course.

Assess for hemodynamic instability. Causes of shock include GI bleeding, complications of GI perforation, volume depletion, and toxicity from coingestants. Examine for peritoneal signs due to hollow viscous perforation. Consider mediastinitis in patients complaining of chest discomfort, and palpate the chest wall and neck for signs of subcutaneous emphysema. Inspect the eyes for ocular burns and the skin for splash and dribble burns.

LABORATORY TESTING

For children who accidentally ingest common household alkalis (e.g., bleach) or acids (e.g., toilet bowl cleaner), the need for ancillary testing is only necessary in patients with signs or symptoms of significant injury: drooling, respiratory distress, or vomiting. For an intentional ingestion, or one from a strong acid or
alkali, laboratory evaluation should include a venous or arterial blood gas, electrolyte panel, hepatic profile, complete blood count, coagulation profile, lactate, and blood type and screen. Caustic ingestions can cause an anion gap acidosis based on lactate production due to direct tissue injury or shock. Strong acid ingestions may be associated with both severe anion gap (e.g., sulfuric acid) and nongap acidoses (e.g., hydrochloric acid). Obtain acetaminophen and salicylate levels to screen for potential coingestants in suicidal patients. An ECG is indicated following a hydrofluoric acid exposure to check for QT-interval prolongation from hypocalcemia.

**IMAGING**

Obtain a chest radiograph in patients with chest pain, dyspnea, or vomiting to check for peritoneal and mediastinal air. Thoracoabdominal CT scanning is useful to assess for esophageal injury after ingestion of strong caustics or if intra-abdominal perforated viscus is suspected. Endoscopy is the gold standard for identifying the severity of esophageal injury. IV and oral contrast-enhanced CT scanning is an alternative when endoscopy is not readily available or during the postingestion time period when the risk of esophageal perforation by endoscopy is increased (see *Endoscopy* section). Strong acid ingestions may be associated with both severe anion gap (e.g., sulfuric acid) and nongap acidoses (e.g., hydrochloric acid). Obtain acetaminophen and salicylate levels to screen for potential coingestants in suicidal patients. An ECG is indicated following a hydrofluoric acid exposure to check for QT-interval prolongation from hypocalcemia.

**ENDOSCOPY**

Endoscopy is the gold standard for evaluating the location and severity of injury to the esophagus, stomach, and duodenum after caustic ingestion. The controversy has been who needs endoscopy and when should it be done. Patients with intentional caustic ingestions should undergo early endoscopy because ingestions with suicidal intent carry the highest risk of clinically important injury. In unintentional ingestions, particularly by children, the decision to perform endoscopy is not clear cut. Most children with serious caustic esophageal injury will be symptomatic, and although there is a correlation between clinical findings and corrosive severity, lack of symptoms is judged by some authors not to be an adequate predictor of no injury. Early endoscopy is recommended after unintentional caustic ingestions in adults and children with signs or symptoms of serious injury such as stridor or significant oropharyngeal burns and/or vomiting, drooling, or food refusal, with or without oropharyngeal burns.

The general purpose of endoscopy is diagnosis. Early endoscopy permits grading of injuries, helps guide treatment, and predicts future morbidity (Table 200-2). Early endoscopy is safe and may decrease the time that patients will be without nutritional support. This is particularly important in children with low glycogen stores and in high-grade injuries, where caloric requirements will be high.
TABLE 200-2

Correlation of Esophageal Injury Grade with Morbidity and Interventions

<table>
<thead>
<tr>
<th>Endoscopic Injury Grade</th>
<th>Grade 1 Esophageal Injury</th>
<th>Grade 2A Esophageal Injury</th>
<th>Grade 2B and 3 Esophageal Injuries</th>
</tr>
</thead>
<tbody>
<tr>
<td>Future morbidity</td>
<td>No risk of strictures or carcinoma</td>
<td>Strictures tend not to occur</td>
<td>At risk for hemorrhage and perforation (early), strictures (delayed), and carcinoma (late)</td>
</tr>
<tr>
<td>Nutritional support</td>
<td>Diet as tolerated</td>
<td>If unable to tolerate PO, provide nutritional support via nasogastric, orogastric, or percutaneous feeding tube or Total parenteral nutrition</td>
<td>Initiate early percutaneous feeding tube or Total parenteral nutrition</td>
</tr>
<tr>
<td>General interventions</td>
<td>Supportive care</td>
<td>Admission recommended, supportive care</td>
<td>Intensive care unit admission recommended; may require additional imaging or surgical exploration for gastric injuries</td>
</tr>
</tbody>
</table>

Tissue friability after a caustic burn increases significantly at 24 to 48 hours after injury and is maximal between days 5 and 14. **Most experts agree that endoscopy should be performed early after ingestion, ideally <12 hours and not >24 hours after ingestion to avoid iatrogenic perforation.**\(^{36,47}\) Traditionally, endoscopists have terminated their examination at the first sign of severe esophageal injury (grade 2B or 3). Experienced operators using smaller, flexible endoscopes with minimal insufflation of air can decrease the incidence of perforation, permitting more distal visualization documenting all injuries to the esophagus, stomach, and duodenum.

**TREATMENT**

**AIRWAY**

The first priority is airway maintenance. Patients with respiratory distress may have significant oral, pharyngeal, and/or laryngotracheal injuries that require emergent airway management. If emergency airway management precedes patient decontamination, prevent exposure to the ED staff. Caustic airway injuries are
difficult airways. Ideally, patients with potential airway injuries should have fiberoptic evaluation of the airway before intubation to determine the extent of the damage, but this may not always be possible. **Blind nasotracheal intubation is contraindicated** due to the potential for exacerbating airway injuries. **Oral intubation with direct visualization is the first choice for definitive airway management.** For potential airway compromise, establish a secure endotracheal airway early rather than risk greater difficulty later when secondary effects of injury, such as edema, complicate the situation. Cricothyrotomy may be needed if oropharyngeal edema, tissue friability, and bleeding make intubation difficult or impossible.

Avoid laryngeal mask airways, combination tubes with pharyngeal and tracheal balloons, retrograde intubation, and bougies because these devices/techniques can increase tissue damage or cause perforation.

**DECONTAMINATION, NEUTRALIZATION, AND DILUTION**

The ED staff should take precautions to prevent ongoing injury to the patient and staff from continued caustic exposure. ED staff involved should wear protective gowns, gloves, and masks with face shields. Standard decontamination, with removal of soiled or soaked clothing and copious irrigation with towels and soap (as needed), is adequate in most cases. Vomiting may re-expose patient and staff to the caustic agent.

**Gastric decontamination with activated charcoal is contraindicated if a caustic is the only ingestion.** Charcoal does not adhere well to most caustics and will impede visualization when endoscopy is performed. However, activated charcoal may be considered when coingestants pose a risk for severe systemic toxicity. **Ipecac syrup is contraindicated,** because vomiting will result in repeat exposure of the airway and GI mucosa to the caustic agent and could precipitate perforation.

In general, do not insert nasogastric tubes until after endoscopic evaluation. With high-grade esophageal burns, feeding tubes may be inserted under endoscopic guidance if clinically indicated.

Dilution and neutralization therapy are not recommended in the prehospital setting or ED because there is no proven human benefit and potential risk of gastric distension, vomiting, and perforation.

**FLUID RESUSCITATION**

Establish large-bore IV access and resuscitate with crystalloids. Coingestants, bleeding, and third spacing, as well as metabolic disarray from acid-base derangements, can lead to shock. Central venous access may be required for monitoring of resuscitation.

**SYSTEMIC STEROIDS AND PROPHYLACTIC ANTIBIOTICS**

**There is currently no evidence of consistent benefit from systemic steroids, so steroids are not recommended as part of ED treatment.**

The ability of steroids to inhibit the inflammatory response led to the hypothesis that steroids may decrease stricture formation after caustic ingestion, and animal models have suggested benefit. However, individual
human trials and pooled meta-analysis have not shown benefit for injury,\textsuperscript{54,55,56,57,58,59,60} and steroids may increase the risk of infection, perforation, and hemorrhage.\textsuperscript{56}

One criticism of pooled meta-analysis data is that the individual studies did not clearly distinguish between grade 1 and 2A lesions, which do not typically lead to strictures, and grade 2B and 3 injuries, which might theoretically benefit from steroids.\textsuperscript{59}

There is no current evidence to support the ED administration of prophylactic antibiotics after caustic ingestions in humans. However, in protocols in which steroids are used or in grade 2B or 3 injury, addition of penicillin or another antibiotic that covers oral flora has been part of treatment regimen.\textsuperscript{56,61} Because steroids have largely fallen out of favor, the need for antibiotics in the ED is rare.

**SURGERY AND ESOPHAGEAL STENTING AND DILATION**

Major ingestions of caustic agents may result in perforation of the GI tract or extensive tissue necrosis requiring emergency surgery.\textsuperscript{62,63,64,65,66,67} Laparotomy is generally preferred over laparoscopic evaluation for posterior gastric visualization. The indications for emergency laparotomy include esophageal perforation, peritoneal signs, or free intraperitoneal air. Large-volume ingestions (>150 mL), signs of shock, respiratory distress, persistent lactic acidosis, ascites, and pleural fluid may be other indications for surgical exploration.\textsuperscript{62}

For grade 2B and 3 injuries without obvious perforation, recommendations include a period of esophageal rest,\textsuperscript{68} early gastrostomy for enteral feeding,\textsuperscript{38,69} and dilation therapy (in the first 3 weeks) with or without stenting.\textsuperscript{70,71,72,73} Once strictures form, they may be difficult to treat and require stenting and/or multiple balloon dilatations or bouginage.\textsuperscript{9} Controversy exists about the most appropriate treatment for esophageal stricture (i.e., long-term repetitive dilation therapy versus surgery).\textsuperscript{9,72,73,74}

**TREATMENT OF SYSTEMIC TOXICITY**

Morbidity or death from alkali injuries usually results from the complications of direct tissue necrosis, but acid ingestions may result in additional systemic toxicity from absorption of the acid.\textsuperscript{18} Acid-base disorders (increased anion gap or normal anion gap acidosis depending on acid ingested), hemolysis, coagulopathy, and renal failure may result. In cases of systemic toxicity, traditional critical-care principles should be applied to optimize the patient's hemodynamics. Acute lung injury (noncardiogenic pulmonary edema) may follow caustic ingestions as a complication of local or systemic effects.

**NUTRITIONAL SUPPORT**

Nutritional support is often necessary following a severe caustic injury to the esophagus or stomach. Support can be achieved by percutaneous (usually jejunostomy) feeding, nasoenteral feeding, or total parenteral nutrition.\textsuperscript{61,75}
EXPERIMENTAL THERAPIES

Animal experiments have found that drugs affecting collagen deposition, including interferon-α-2b, octreotide, β-aminopropionitrile, colchicine, N-acetylcysteine, and d-penicillamine, can prevent esophageal strictures after caustic alkali ingestion. Pentoxifylline, a local inflammatory and microcirculation mediator, has experimental benefit. Mitomycin C, a fibroblast proliferation inhibitor, has been used topically on strictures with some success. Oral agents to coat and protect the GI tract from insult, including sucralfate, bismuth subsalicylate, and sodium polyacrylate, are beneficial in animal experiments. None of these agents have been evaluated in controlled human clinical trials, and no specific recommendation can be made regarding their use. H₂ blockers and proton pump inhibitors are often used in the treatment protocol, but no evidence supports or refutes their use.

DISEASE COMPLICATIONS

Short-term prognosis is worse with grade 3 GI injury, systemic complications, and age >65 years. Most long-term sequelae from caustic exposure are related to injuries to the GI tract. Acid ingestions may scar the pylorus and result in gastric outlet obstruction. Caustic alkali ingestions may result in esophageal strictures, which may result in dysphagia, odynophagia, and malnutrition. Persistent drooling, reluctance to eat, severe oropharyngeal burns, and persistent fever correlate with the development of esophageal stricture after accidental caustic ingestion in children.

Patients with grade 3 caustic injuries to the esophagus have about a 1000-times increased risk for squamous cell cancer of the esophagus that can occur decades after the initial ingestion and resulting esophageal injury. Because cancer can develop if a portion of the esophagus remains after reconstructive surgery for esophageal stricture, total removal of the esophagus is recommended.

DISPOSITION AND FOLLOW-UP

Admit all patients with symptomatic caustic ingestions. Patients with grade 1 injuries can be discharged from hospital after endoscopy, provided they can tolerate oral fluids and food. Grade 2A injuries warrant hospitalization to ensure that symptoms and injury do not progress. Grade 2B and 3 injuries are significant, require enteral or parenteral nutrition, and have an early risk for bleeding or perforation; admit to an intensive care unit. Organ perforation or extensive necrosis is an indication for surgery. Contact the regional poison control center for data collection purposes and assistance with management.

SPECIAL CONSIDERATIONS

LAUNDRY DETERGENT POD INGESTIONS
Laundry detergent pods, also known as capsules, liquitabs, or sachets, have been available in Europe for over a decade and were introduced to the U.S. market in 2010. Each pod contains concentrated detergent within a dissolvable plastic membrane. An individual pod may have internal chambers that contain a stain remover and brightener separate from the detergent. Exposure to the concentrated preparation in these pods is more likely to produce symptoms than exposure to traditional laundry detergent products.

Dermal or ocular exposure to the contents can produce irritation of the skin, conjunctiva, or cornea. Ingestion by young children can produce serious toxicity with profuse vomiting, respiratory distress, and neurologic depression. The etiology of these systemic symptoms is unknown. Caustic injury to the pharynx and esophagus can produce difficulty swallowing with drooling and aspiration during recovery. Treatment is supportive with airway protection and mechanical ventilation. In severe cases, ventilation may be required for days.

Acknowledgment: The authors wish to thank Dr. G. Richard Bruno for his contributions to this chapter in previous editions.

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**USEFUL WEB RESOURCES**


American Association of Poison Control Centers (AAPC)—[http://www.aapcc.org/DNN](http://www.aapcc.org/DNN)

American Academy of Clinical Toxicology (AACT)—[http://www.clintox.org/index.cfm](http://www.clintox.org/index.cfm)

Asia Pacific Association of Medical Toxicology (APAMT)—[http://www.asiatox.org](http://www.asiatox.org)

European Association of Poisons Centres and Clinical Toxicologists (EAPCCT)—[http://www.eapcct.org](http://www.eapcct.org)
South Asian Clinical Toxicology Research Collaboration (SACTRC)—http://www.sactrc.org

TOXBASE: The primary clinical toxicology database of the National Poisons Information Service—http://www.toxbase.org

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