Acids work through coagulation necrosis (desiccatation or denaturation of superficial tissue proteins), with formation of an eschar by the reaction. This coagulum actually serves to protect the tissue by limiting deeper penetration of the acid, so acidic burns tend to be less significant in general than alkali burns. The squamous epithelium of the pharynx and esophagus are relatively resistant to this type of injury. The esophagus is involved in 6-20% of acid ingestions; the stomach in the most commonly involved organ. The natural acidic pH in the stomach unfortunately often increases the injury and thus there is a relatively high incidence of gastric complications. Erosions and perforation of the stomach tend to occur in the antrum due to dependent pooling here. In addition, the acidic pH of the ingested agent can cause pyloric spasm, preventing gastric emptying, and prolonging exposure. The bitterness of acidic agents can cause choking, sputtering, and vomiting, often resulting in a chemical epiglottitis. Perforation of injured tissue may occur 3-4 days after exposure as the eschar sloughs. A gastric outlet obstruction may develop as scar tissue contracts over a 2-4 week period. Acute complications include gastric and intestinal perforation and upper gastrointestinal hemorrhage. Significant exposures may cause metabolic acidosis, hemolysis, acute renal failure, and death.

Bases include lye (NaOH, KOH), which is found in drain cleaners, ammonia, and electric dishwasher soap. Alkaline ingestions cause tissue injury by liquefactive necrosis (saponification of fats and solubilization of proteins). Cell death occurs from emulsification and disruption of cellular membranes. The hydroxide ion of the base reacts with tissue collagen and causes it to swell and shorten. Small vessel thrombosis and heat production occurs. Tissue injury occurs rapidly, within minutes of contact. Since bases are soluble once they form soaps with fat, there is deep diffusion of bases into exposed tissues. Only neutralization of the substance by the tissue itself stops the reaction. With alkali injuries, the most severely injured tissues are the squamous epithelial cells of the oropharynx, hypopharynx, and esophagus (the most commonly involved organ). The stomach is involved in only 20% of all alkaline ingestions. Tissue edema occurs immediately and may persist for 48 hours, eventually progressing to airway obstruction. Over time, granulation tissue and then scar replace necrotic tissues. The incidence of esophageal stricture formation depends upon the depth of the injury. Superficial burns result in strictures in fewer than 1% of cases; full thickness burns result in strictures in nearly 100% of cases. Severe burns may also be associated with esophageal perforation.

**Role of esophageal endoscopy in caustic ingestion.**

Esophageal stricture and perforation are risks associated with caustic ingestion. Because signs and symptoms (including presence/absence of oral injury) are not predictive of esophageal injury, esophagoscopy should be performed in most patients who are suspected of caustic ingestion. The timing of esophagoscopy is important, although there is some controversy over the ideal time window. Generally, esophagoscopy is carried out within 24 to 48 hours (some authors recommend 72 hours) of ingestion. The 24-hour delay provides time for the injuries in the esophagus and stomach to demarcate, allowing for a more meaningful evaluation of the degree of injury. Esophagoscopy 48-72 hours after ingestion increases the chance of iatrogenic perforation due to structural weakness in the esophageal wall. Rigid versus flexible esophagoscopy is also debatable but most otolaryngologists prefer rigid esophagoscopy. The primary difference is that flexible esophagoscopy allows for examination of the stomach. Esophagoscopy should be continued to the upper limit of a severe (full-thickness) burn. Advancing beyond this point can increase the chance of perforation. The main purpose of endoscopy is to delineate injury location and severity in order to guide subsequent treatment. It should also be noted whether the injury is circumferential.

Degree of esophageal injury:
- Grade I: superficial injury, erythema
- Grade II: transmucosal non-circumferential
- Grade III: transmural, circumferential

Bleaches are approximately 5-6% sodium hypochlorite and produce an ulceration that does not usually result in stricture. An esophagoscopy should be performed if an oropharyngeal burn is present. Otherwise, a barium swallow is obtained in three weeks.

**Staging and treatment of esophageal burns in caustic ingestion (pharmacologic and mechanical).**

**Most common complication of esophageal burn and management.**

**Complications of caustic esophageal burns:**
- 1<sup>st</sup> degree: heal uneventfully
- 2<sup>nd</sup> degree: occasionally form strictures
- 3<sup>rd</sup> degree: always form strictures
- 4<sup>th</sup> degree: additional risk of sepsis or mediastinitis

*Also, an estimated 1000-fold increased risk of carcinoma

*Note: due to difficulty in identifying depth of burn, there is potential for 3<sup>rd</sup> degree burns to develop into a 4<sup>th</sup> degree burn.
Several days after caustic injury, necrotic tissue sloughs leaving areas of injury vulnerable to perforation. Thus all 3rd burns should be closely observed in hospital for several days and receive antibiotics. This is also the reason esophagoscopy should be performed in the first 24 hours.

Treatment – depends on degree of burn
1st degree: no treatment needed
2nd degree: pharmacologic treatment to maintain esophageal lumen/prevent strictures with periodic follow-up to assess need for mechanical treatment
3rd degree:
   Focal: pharmacologic & mechanical treatment to maintain esophageal lumen/prevent strictures
   Extensive: thoracotomy or thoracoscopy to assess for transmural necrosis; possible esophagectomy
4th degree: Esophagectomy

Pharmacologic therapy - Controversial
1) PPI – unproven clinically but generally accepted as a good idea
2) Sucralfate – adheres to ulcers forming a protective layer; has shown promise in preventing strictures
3) Corticosteroids and antibiotics – studies show mixed results in reducing stricturing.
4) Lathyrogenic drugs (N-acetylcysteine, penicillamine) – reduce collagen cross bonding; have been used but benefits in humans unproven
5) Mitomycin – alkylating agent that inhibits DNA and protein synthesis, and there for fibroblast formation; benefits in humans unproven

Mechanical therapy
1) Nasogastric tube- placed at initial time of endoscopy. Serves as stent & means of nutritional support for 3rd degree burns with dysphagia. Ideally should remain in place long enough to allow re-epithelialization (2-3 weeks).
2) Prograde dilation- through an esophagoscope with filiform dilators
3) Retrograde dilation- endless loop of silk passed through gastrostomy & exits mouth. Dilation with serial increases in caliber while maintaining a secure position within the narrowed lumen. Useful for lengthy third-degree burns.

If repetitive dilations fail, surgery is indicated to reconstruct conduit from hypopharynx to stomach.

5. (Amy) Risk factors for foreign body ingestion (FB) and most common type of FB in airway vs. esophageal.

   Most foreign body ingestions occur in children. The incidence is greatest in young children (age 6 months to 4 years), who tend to place objects in their mouths as they are exploring their surroundings. Young children also lack molars for proper grinding of food, tend to be running or playing at the time of aspiration, and lack coordination of swallowing and glottic closure. A chaotic home environment is a risk factor for foreign body ingestion, and young children may also be given foreign bodies by older children or abusive adults. In older children or teenagers, foreign body ingestion may be a way of seeking attention or a manifestation of an underlying a mental disturbance or psychiatric problem. For example, an association exists between toothbrush ingestion and bulimia in teenage girls. Foreign body ingestion is also higher in incarcerated individuals. Food-related foreign body ingestion is more common in adults greater than 60, people with esophageal disease, or individuals who have recently consumed CNS depressants (alcohol).

   Although adults most often present to the ED after ingestion of radiolucent foreign bodies, such as food, children usually swallow radiopaque objects, such as coins, pins, screws, button batteries, or toy parts. In children, vegetable matter tends to be the most common airway foreign body in children; peanuts are the most common food item aspirated. The most common esophageal foreign bodies in children are coins, followed by chicken or fish bones, buttons or tacks, marbles or screws, button batteries, and
Sept 13: Aerodigestive Foreign Bodies

straight pins. In adults, the most common esophageal foreign bodies are food boluses, followed by bones (particularly fish bones), coins, fruit pits, straight pins and dentures.

6. (Kathy) Most common location for esophageal vs. airway FB. Airway symptoms. Esophageal symptoms.

   Most esophageal FB impactions occur in the cervical esophagus just below the cricopharyngeus muscle. Another 4-5% of esophageal FB become lodged at the mid or distal esophagus, often caused by extraluminal compression by the aortic arch or left mainstem bronchus. Typical symptoms of esophageal FB ingestion include drooling, dysphagia, emesis, and chest pain.

   Most airway foreign bodies become lodged in the bronchi, because their size and configuration allow passage through the larynx and trachea. Bronchial foreign bodies are more common in the right mainstem bronchus, which is thought to be caused by the position of the carina to the left of the midline and its lesser angle of divergence from the tracheal axis. Larger objects may become impacted in the larynx or trachea, at times causing complete obstruction, an acute emergency. Typical signs and symptoms of airway FB include cough, wheezing, stridor, cyanosis, or asymmetric breath sounds. Esophageal FB may also cause respiratory symptoms in a young child. A high index of suspicion should be maintained when evaluating children presenting with recurrent croup, asthma, or pneumonia without the expected response to treatment.

7. (Kathy) Role of radiographs for both esophageal and airway FB.

   Posteroanterior and lateral x-rays of the neck and chest are the imaging studies of Choice. Radiopaque foreign bodies should be straightforward to diagnose, whereas other foreign bodies may be more difficult. Flat objects in the esophagus (coins) are oriented in the coronal plane, while in the airway it is oriented in the sagittal plane. Even if no foreign body is visualized, localized atelectasis or infiltrates, unilateral hyperinflation, lobar collapse, or mediastinal shift may be present on plain film x-rays. However, high clinical suspicion or historical evidence (ie, witnessed ingestion or aspiration) warrants rigid endoscopy even if x-rays are normal. Airway fluoroscopy is sometimes used if plain films are not diagnostic or if the patient cannot cooperate, which has the added advantage of demonstrating a dynamic view of the airway. Barium swallow is generally not indicated and the presence of barium can make esophageal FB extraction more difficult.

8. (Tali) A 2 year old is seen chewing an acorn. He has a coughing episode and the father attempts a finger sweep. After paroxysmal cough, he is improved over 20 minutes. In the ER, he is afebrile comfortable with good saturations. Breath sounds reveal occasional unilateral wheeze. X-rays are unremarkable. How will you manage this patient?

Take to OR for direct laryngoscopy/bronchoscopy

Children more prone to aspiration: lack molar teeth to chew well, talk and run while eating, put objects in mouth, siblings place in baby’s mouth.

Peak age risk: toddler to preschool (<4 years old).

Most common entities: nuts, raisins, sunflower seeds, improperly chewed pieces of meat and small smooth items such as grapes, hot dogs, and sausages. Dried fruits may cause progressive obstruction as they absorb water.

Review of 1068 foreign body aspirations in children: most common location (52%) right main stem, 18% left main bronchus, 13% trachea. In upright child, right-sided airways are direct entry from the trachea. The left main bronchus is smaller than the right main bronchus and is slightly angled.

Mortality: acute aspiration, morbidity: chronic lung and airway damage

Clinical History: Sudden episode coughing or choking with subsequent wheezing, stridor or coughing. Unwitnessed may present with persistent wheezing non-responsive to inhaled b-blockers, recurrent pneumonia, lung abscess, focal bronchiectasis or hemoptysis. If subglottic, may have persistent stridor, recurrent croup or voice changes.

Physical: Wheezing, stridor, decreased breath sounds, often unilateral, not always. Inspiratory sounds if object in extrathoracic trachea. If the lesion is in the intrathoracic trachea, noises are symmetric but sound more prominent in the central airways. These sounds are a coarse wheeze (respiratory stridor) heard with the same intensity all over the chest. Past carina, breath sounds usually asymmetric. May have no physical findings.

Radiography: Most aspirated foreign bodies are radiolucent. CXR: pain: area of focal overinflation or atelectasis depending on degree of obstruction (complete occlusion --> opacification of distal airway, partial → ball valving with overinflation). A/P CXR on expiration: trapped air in the affected portion of the lung. Fluoroscopy: may be helpful in showing focal air trapping and/or paradoxical diaphragmatic motion. CT: may demonstrate the material in the airway, focal airway edema, or focal overinflation not detected in the plain radiographs. Some would forgo CT scanning if the index of suspicion is high and proceed with bronchoscopy.

Treatment: Even if the foreign body does not show up on any of the radiographic studies, a foreign body may still be present, and an endoscopy should be performed if the suspicion is high.

If history and physical typical, proceed immediately to rigid bronchoscopy. May use flexible bronchoscopy to detect foreign body. If the child has respiratory distress and is unable to speak or cry, complete airway obstruction is likely present → Heimlich maneuver to dislodge object.
Sept 13: Aerodigestive Foreign Bodies

Bronchodilators and corticosteroids not necessary, no chest PT as may dislodge the material to more dangerous location.
If focal swelling on direct laryngoscopy/bronchoscopy ➔ oral/IV steroids
Unless the airway secretions are infected with organisms present, antibiotics are not necessary.

9. (Caroline) Management of battery ingestion.

ABCs

CXR and AXR to determine location

Remove batteries in esophagus emergently because of the risk of esophageal burns and resultant complications via flexible fiberoptic endoscopy

Can attempt foley balloon catheter technique if can’t perform endoscopically (insert foley, place the patient in the lateral decubitus or Trendelenburg position, inflate balloon, and withdraw catheter under fluoroscopic guidance, remove battery with McGill forceps or expelled by the patient.

Batteries localized beyond the esophagus – Do not need to retrieve unless patient with symptoms of GI tract injury (eg, hematochezia, abdominal pain, tenderness) or a large-diameter battery fails to pass beyond the pylorus. Some suggest that any delay in GI transit (distal to the pylorus) greater than 8 hours requires intervention b/c of the potential for erosive/corrosive complications.

Tips: No ipecac if battery in stomach, stool inspection and weekly x-rays recommended if battery not retrieved surgically, patients younger than 6 years who have ingested a battery with a diameter of 15 mm or greater should have a repeat x-ray in 48 hours if the battery was originally in the stomach. These batteries do not generally pass the pylorus after 48 hours. Endoscopic retrieval may be necessary.

10. (Scott) You are called to the ER for evaluation of a 2-year-old who has recently ingested ½ bottle of Drano. She is in no respiratory distress, but is crying and drooling. What do you do?

5,000-15,000 caustic congestions yearly in US. (50%-80% peds).
High family stress is the most contributing factor. After the liquid products were introduced in 1960s, a large amount of mucosa from the mouth all the way down to the duodenum can be damaged with ingestion.

Classification of burn:
Grade I: nonulcerative esophagitis, mild erythema, no sloughing, and edema of the mucosa.
Grade II: white exudate, erythema, underlying ulceration may be apparent, may extend into the muscle layers.
Grade III: Very dusky mucosa, very black, and transmural tissue is involved. There can be deep ulcerations that extend into the periesophageal space or perforations, and sometimes the lumen may be completely obliterated.

Caustic ingestion fall into three categories: alkali agents with pH > 7; acids, with pH < 7; and bleaches, with pH=7.
The most common ingestions in the United States, about 60% to 80% of the time, are alkali ingestions.

Histologically, a liquefaction necrosis occurs, causing denaturing of proteins, saponification of fat, and blood vessel thrombosis with very early disintegration of the tissues. This allows for very deep penetration and further damage into the muscle layer and beyond. 30% of children ingesting alkali agents end up with esophageal burns, 80% having high grade II and III that do go on oftentimes to develop strictures. Burns tend to be at anatomically narrowing areas. at the cricopharyngeus, the arch of the aorta, etc. If there is significant reflux, the agent can reflux and cause additional damage in the area. Mucosal disintegration from alkali agents actually can occur over a three-day period after the ingestion, and about days 7-21 the esophageal wall is weakest. Superficial mucosal burns may tend to heal without sequelae. Burns that disrupt the submucosal and muscular layers often are complicated by an inflammatory reaction. The body begins to try to heal this area, and fibroblasts move in, laying down collagen matrix. This matrix is haphazard and contracts and allows for adhesive bands to form, and pseudodiverticula can form between these bands. Usually a completely circumferential lesion involving the entire wall leads to significant strictures in a clinical setting.

There is no reliable relationship between the signs, symptoms, and physical exam. Therefore, this should never stop you from an endoscopic evaluation if your suspicion is high that there actually was ingestion. Hoarseness or stridor could be indicative that there might be some airway compromise, and substernal, back, and abdominal pain can indicate severe injuries. However, you can still have severe injuries without any signs at all. 8%-20% of patients with significant esophageal or gastric injury had no obvious signs or symptoms at all. Lab values are not helpful.
Sept 13: Aerodigestive Foreign Bodies

A lateral neck film sometimes is helpful if you are concerned about airway compromise. Chest x-rays are helpful in the diagnosis of an acute perforation. Barium esophagram has extremely high false negative rates, does not see Grade I/II lesions, and delays endoscopy, which is a definitive procedure. After 48 hours endoscopy is no longer safe due to the increased risk of perforation and the weakness of the esophageal wall, and then you can consider barium esophagram.

The timing of endoscopy remains controversial. If it is less than 24 hours, the full demarcation of the injury has not yet occurred, the total injury may be underestimated. If greater than 48 hours, there is an increased risk of perforation. So, it is generally accepted that endoscopy should occur between 24 and 48 hours, and if a completely circumferential lesion is encountered, it is advisable to stop further endoscopy at that time to decrease the risk of perforation.

Management:
1. Initial treatment is airway management. ABCs.
2. Sometimes diluting agents such as milk or water can be given to the patient. Milk is a neutral buffer, but too much can induce vomiting, further increasing the injury. Charcoal and emetics should not be used because they increase vomiting. Blind passage of a nasogastric tube is contraindicated, and may cause perforations.
3. Steroids are given to reduce stricture formation. In the grade III injury, steroids should not be used because they usually go on to surgery, and the steroids can slow healing.
4. Prophylactic antibiotic use has been controversial. Studies failed to show a decrease in stricture rate or infection rate. Antibiotics should be started if there is any sign of infection.
5. Many recommend antireflux therapy even though there are no proven human studies.
6. CXR/Lateral XR to evaluate perforation/airway compromise.
7. Many recommend esophageal stenting (endoscopically to decrease the risk of perforation) to reduce stricture formation with a NG tube. Theoretically, the stent should remain in place while re-epithelization occurs (2-3 weeks).
8. Esophagoscopy is standard of care at 24-48 hours. If more than 48 hours after injury, then consider barium esophagram.
9. Lathyrogens (N-acetylcysteine and penicillamine) are agents that reduce collagen cross linking and reduce formation of collagen in the laboratory setting.
10. Esophageal dilation is used to avoid stricture formation. Bouginage usually begins 2-3 weeks after the initial injury. Long-term effectiveness is unclear, and with repeated dilations, there is an increased risk of perforation.
11. Complications: nasopharyngeal stenosis, hypopharyngeal stenosis, and laryngeal stenosis, as well as fixation of the tongue and esophageal strictures. Mortality is estimated at between 0% and 20%. There is a 1000 fold increased risk of esophageal cancer 25 years after a caustic injury.

11. **(Vicki)** A child swallows a safety pin that is now lodged in mid-esophagus.

Direct the esophagoscope toward the sternal notch keeping the lumen in view at all times. Usually safety pins that become lodged are open and locating the tip is crucial. The pin usually has to be moved distally first in order to disengage the tip from the surrounding mucosa. Two methods are suggested for removal. 1) Sheath the point with the scope and place the keeper against the outside of the scope. 2) Push the pin into the stomach and under fluoroscopy flip the safety pin point down. Just a note that there is a device called the Clerf-Arrowsmith pin closing forcep. This can get tangled with the pin which makes both impossible to remove. Avoid them. Sometimes the safest thing to do is an open procedure.

Once the object is grasped, advance the esophagoscope toward the object and remove the object, forceps and scope all at once. Reassess the esophagus once the pin has been removed to look for any mucosal damage or additional foreign bodies.