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5th stage

ANTISEPTICS, DISINFECTANTS, AND STERILANTS

Antiseptics, disinfectants, and sterilants are a diverse group of antimicrobials used to prevent infection. Although these terms are sometimes used interchangeably and some of these xenobiotics are used for both antisepsis and disinfection, the distinguishing characteristics between the groups are important to emphasize. An *antiseptic* is a chemical that is applied to living tissue to kill or inhibit microorganisms. Iodophors, chlorhexidine, and the alcohols (ethanol and isopropanol) are commonly used antiseptics. A *disinfectant* is a chemical or physical agent that is applied to inanimate objects to kill microorganisms. Bleach (sodium hypochlorite), phenolic compounds, and formaldehyde are examples of currently used disinfectants. Neither antiseptics nor disinfectants have complete sporicidal activity. A *sterilant* is a chemical or physical process that is applied to inanimate objects to kill all microorganisms as well as spores. Ethylene oxide and glutaraldehyde are examples of sterilants. Not unexpectedly many of the xenobiotics used to kill microorganisms also demonstrate considerable human toxicity.

ANTISEPTICS

CHLORHEXIDINE

This cationic biguanide has been in use as an antiseptic since the early 1950s. It is found in a variety of skin cleansers, usually as a 4% emulsion (eg, Hibiclens), and may also be found in mouthwash. Chlorhexidine is reported to have low toxicity.

Clinical Effects:
Few cases of deliberate ingestion of chlorhexidine are reported. Symptoms are usually mild and gastrointestinal irritation is the most likely effect after oral ingestion. Chlorhexidine has poor enteral absorption. In one case, ingestion of 150 mL of a 20% chlorhexidine gluconate solution resulted in oral cavity edema and significant irritant injury of the esophagus.

An 80-year-old woman with dementia ingested 200 mL of a 5% chlorhexidine solution and subsequently aspirated. She rapidly developed hypotension, respiratory distress, coma, and died 12 hours following ingestion.

Management:
Treatment guidelines for chlorhexidine exposure are similar to those for other potential caustics. Patients with significant symptoms may require endoscopy, but the need for such extensive evaluation is quite uncommon.

HYDROGEN PEROXIDE

Hydrogen peroxide, an oxidizer with weak antiseptic properties, has been used for many years as an antiseptic and a disinfectant. This oxidizer is generally available in two strengths: dilute hydrogen peroxide, with a concentration of 3%–9% by weight (usually 3%), sold for home use, and concentrated hydrogen peroxide, with a concentration greater than 10%, used primarily for industrial purposes. Commercial-strength hydrogen peroxide is commonly found in solutions varying from 27.5%–70%. Home uses for dilute hydrogen peroxide include ear cerumen removal, mouth gargle, vaginal douche, enema, and hair bleaching. Dilute hydrogen peroxide is also sometimes used as a veterinary emetic.

Toxicity from hydrogen peroxide may occur after ingestion or wound irrigation. Hydrogen peroxide has two main mechanisms of toxicity: local tissue injury and gas formation. The extent of local tissue injury and amount of gas formation is determined by the concentration of the hydrogen peroxide. Dilute hydrogen peroxide is an irritant and concentrated hydrogen peroxide is a caustic. Gas formation results when hydrogen peroxide interacts with tissue catalase, liberating molecular oxygen, and water. At standard temperature and pressure, 1 mL of 3% hydrogen peroxide liberates 10 mL of oxygen, whereas 1 mL of the more concentrated 35% hydrogen peroxide liberates more than 100 mL of oxygen. Gas formation can result in life-threatening embolization.

Clinical Effects:

Airway compromise manifested by stridor, drooling, apnea, and radiographic evidence of subepiglottic narrowing may occur. The combination of local tissue injury and gas formation from the ingestion of concentrated hydrogen peroxide may cause abdominal bloating, abdominal pain, vomiting, and hematemesis. Endoscopy may show esophageal edema and erythema and significant gastric mucosal erosions.

Symptoms consistent with sudden oxygen embolization include rapid deterioration in mental status, cyanosis, respiratory failure, seizures, ischemic ECG changes, and acute paraplegia

Encephalopathy with cortical visual impairment and bilateral hemispheric infarctions detected by MRI imaging may occur after ingestion of concentrated hydrogen peroxide. In a case of acute paraplegia after the ingestion of 50% hydrogen peroxide, MRI revealed discrete segmental embolic infacts of the cervical and thoracic spinal cord as well as both cerebral hemispheres and left cerebellar hemisphere.

Clinical sequelae from the ingestion of dilute hydrogen peroxide are usually much more benign. Nausea and vomiting are the most common symptoms. A whitish discoloration may be noted in the oral cavity. Gastrointestinal injury is usually limited to superficial mucosal irritation, but multiple gastric and duodenal ulcers, accompanied by hematemesis, and diffuse hemorrhagic gastritis are reported. Portal venous gas embolization may occur as a result of the ingestion of 3% hydrogen peroxide

Gas embolism, resulting in intestinal gangrene, was reported to occur following colonic lavage with 1% hydrogen peroxide during surgical treatment of meconium ileus. Multiple

cases of acute colitis are reported as a complication of administering 3% hydrogen peroxide enemas. The use of 3% hydrogen peroxide as a mouth rinse is associated with the development of oral ulcerations.

Diagnosis:

A careful examination should be performed to detect any evidence of gas formation. A chest radiograph might reveal gas in the cardiac chambers, mediastinum, or pleural space. An abdominal radiograph might show gas in the GI tract or portal system and define the extent of bowel distension. MRI and CT scan might be useful for detecting brain and spinal cord lesions secondary to gas embolism. Endoscopic evaluation might be necessary in patients who ingest concentrated hydrogen peroxide to determine the extent of mucosal injury.

Management:

The treatment of patients with hydrogen peroxide ingestions depends, to a large degree, on whether the patient has ingested a diluted or concentrated solution. Those with ingestions of concentrated solutions require expeditious evaluation. Dilution with milk or water, although unstudied, is unlikely to be helpful. Nasogastric aspiration of hydrogen peroxide might be helpful if the patient presents immediately after ingestion. Induced emesis is contraindicated and activated charcoal offers no antidotal benefit. Patients with abdominal distension from gas formation should be treated with nasogastric suctioning. Those with clinical or radiographic evidence of gas in the heart should be placed in the Trendelenburg position to prevent gas from blocking the right ventricular outflow tract. Careful aspiration of intracardiac air through a central venous line may be attempted in patients in extremis. Case reports suggest that hyperbaric therapy may be useful in cases of life-threatening gas embolization after hydrogen peroxide ingestion. Asymptomatic patients who unintentionally ingest small amounts of 3% hydrogen peroxide can be safely observed at home.

IODINE AND IODOPHORS

Iodine is one of the oldest topical antiseptics. Iodine usually refers to molecular iodine, also known as I_2, free iodine, and elemental iodine which is the active ingredient of iodine-based antiseptics. The use of ethanol as the solvent, such as tincture of iodine, allows substantially more concentrated forms of I_2 to be available. I_2 and tincture of iodine ingestions are much less common than in the past as a result of the change in antiseptic use from iodine to iodophor antiseptics.

Iodophors have molecular iodine compounded to a high-molecular- weight carrier or to a solubilizing agent. Povidone-iodine (Betadine), a commonly used iodophor, consists of iodine linked to polyvinylpyrrolidone (povidone). Iodophors, which limit the release of molecular iodine and are generally less toxic, are the standard iodine-based anti- septic preparations. Iodophor preparations are formulated as solutions, ointments, foams, surgical scrubs, wound-packing gauze, and vaginal preparations. The most common preparation is a 10% povidone-iodine solution that contains 1% “available” iodine, but only 0.001% free iodine

Iodine is used to disinfect medical equipment and drinking water. Iodine is an effective antiseptic against bacteria, viruses, protozoa, and fungi, and is used both prophylactically and therapeutically. Iodine is cytotoxic and an oxidant.

There may be significant systemic absorption of iodine from topical iodine or iodophor preparations. Markedly elevated iodine concentrations do occur in patients who receive topical iodophor treatments to areas of dermal breakdown, such as burn injuries. Significant absorption occurs when iodophors are applied to the vagina, perianal fistulas, umbilical cords, and the skin of low-birth-weight neonates. A fatality following intraoperative irrigation of a hip wound with povidone-iodine is also reported. In this latter case, the postmortem serum iodine concentration was 7000 ug/dL (normal: 5–8 ug/dL).

Clinical Effects:

Problems associated with the use of iodine include unpleasant odor, skin irritation, allergic reactions and clothes staining. Ingestion of iodine may cause abdominal pain, vomiting, diarrhea, GI bleeding, delirium, hypovolemia, anuria, and circulatory collapse. Severe caustic injury of the GI tract may occur. The ingestion of approximately 45 mL of a 10% iodine solution resulted in death from multisystem failure 67 hours after ingestion.

Acid–base disturbances are among the most significant abnormalities associated with iodine and iodophors. Metabolic acidosis occurred in several burn patients after receiving multiple applications of povidone-iodine ointment. These patients had elevated serum iodine concentrations and normal lactate concentrations. The exact etiology of the acidosis remains unclear. Postulated mechanisms for the acidosis include the povidone-iodine itself (pH 2.43), bicarbonate consumption from the conversions of I_2 to NaI, and decreased renal elimination of H^+ as a consequence of iodine toxicity. Metabolic acidosis associated with a high lactate level after iodine ingestion likely reflects tissue destruction.

Electrolyte abnormalities also may occur following the absorption of iodine. A patient with decubitus ulcers who received prolonged wound care with povidone-iodine–soaked gauze developed hypernatremia, hyperchloremia, metabolic acidosis, and renal failure.

Management

The patient who ingests an iodine preparation requires expeditious evaluation, stabilization, and decontamination. Careful nasogastric aspiration and lavage may be performed to limit the caustic effect of the iodine if signs of perforation are absent. Irrigation with a starch solution will convert iodine to the much less toxic iodide and, in the process, turn the gastric effluent dark blue-purple. This change in color may serve as a useful guide in determining when lavage can be terminated. If starch is not available, milk may be a useful alternative. Instillation of 100 mL of a solution of 1%–3% sodium thiosulfate can also be used to convert any remaining iodine to iodide. Activated charcoal binds iodine and may be useful. Early endoscopy may help assess the extent of the gastrointestinal injury.

Most patients with iodophor ingestion require only supportive management. The use of starch or sodium thiosulfate may be considered in symptomatic patients. Hemodialysis and

continuous venovenous hemodiafiltration was used successfully to enhance elimination of iodine in patient with renal insufficiency who had become iodine toxic after undergoing continuous mediastinal irrigation with povidone-iodine.

POTASSIUM PERMANGANATE

Potassium permanganate ($KMnO_4$) is a violet water-soluble xenobiotic that is usually sold as crystals or tablets or as a 0.01% dilute solution. Potassium permanganate is a strong oxidizer and poisoning may result in local and systemic toxicity. Upon contact with mucous membranes, potassium permanganate reacts with water to form manganese dioxide, potassium hydroxide, and molecular oxygen. Local tissue injury is the result of contact with the nascent oxygen, as well as the caustic effect of potassium hydroxide. A brown-black staining of the tissues occurs from the manganese dioxide. Systemic toxicity may occur from free radicals generated by absorbed permanganate ions.

Clinical Effects:
Following ingestion, initial symptoms include nausea and vomiting. Laryngeal edema and ulceration of the mouth, esophagus, and, to a lesser extent, the stomach, may result from the caustic effects. Airway obstruction and fatal gastrointestinal perforation and hemorrhage may occur. Esophageal strictures and pyloric stenosis are potential late complications.

Although potassium permanganate is not well absorbed from the GI tract, systemic absorption may occur, resulting in life-threatening toxicity. Systemic effects include hepatotoxicity, renal damage, methemoglobinemia, hemolysis, hemorrhagic pancreatitis, airway obstruction, acute respiratory distress syndrome, disseminated intravascular coagulation, and cardiovascular collapse. Elevation in blood or serum manganese concentration may also occur, confirming systemic absorption (normal concentrations blood manganese 3.9–15.0 ug/L; serum manganese 0.9–2.9 ug/L).

Chronic ingestion of potassium permanganate may result in classic manganese poisoning (manganism) characterized by behavioral changes, hallucinations, and delayed onset of parkinsonian-like symptoms. A 66-year-old man who mistakenly ingested 10 g of potassium permanganate over a 4-week period (because of medication mislabeling) developed impaired concentration and autonomic and visual symptoms. He also developed abdominal pain, gastric ulceration, and alopecia. Serum manganese concentration was elevated. Nine months later, the patient's neurologic examination displayed extrapyramidal signs consistent with parkinsonism.

Management:

Because the consequential effects of potassium permanganate ingestion are a result of its liberation of strong alkalis, the initial treatment of such a patient should include assessment for evidence of airway compromise. Dilution with milk or water may be useful. Patients with symptoms consistent with caustic injury should undergo early upper GI endoscopy.

Corticosteroids along with antibiotics may be warranted if laryngeal edema is present. Analysis of liver enzymes, BUN, creatinine, lipase, serum manganese, and methemoglobin concentrations should be performed when systemic toxicity is suspected. Methemoglobinemia, if clinically significant, should be treated with methylene blue. Dermal irrigation with dilute oxalic acid may be successful in removing cutaneous staining. The administration of *N*-acetylcysteine to increase reduced glutathione production, thereby limiting free radical– mediated oxidative injury in cases of systemic potassium permanganate poisoning, has been suggested, but clinical trials have not been performed.

CHLORINE (Cl_2):

Chlorine, one of the first antiseptics, is still used in the treatment of the community water supply and in swimming pools. Chlorine is a potent pulmonary irritant that can cause severe bronchospasm and acute lung injury.

Chlorine gas is a valuable oxidizing agent with various industrial uses, and occupational exposure is common. Chlorine gas was used as a chemical warfare agent by both the French and the Germans in World War I (When released on the battlefield, chlorine forms a yellow-green cloud with a distinct pungent odor detectable at levels that are not immediately dangerous). Although chlorine gas is not generally available for use in the home, domestic exposure to chlorine gas is common. The admixture of an acid to bleach liberates chlorine gas (Figure 1). Because the anionic component of the acid is not involved in the reaction, combining hypochlorite with virtually any acid, such as phosphoric, hydrochloric, or sulfuric acid, may result in the release of chlorine gas. As such, inappropriate mixing of cleaning agents is the cause of most non-occupational exposures. Rarely, patients have intentionally generated chlorine gas in this manner for purportedly "pleasurable" purposes. Concentrated chlorine gas may be generated when aging swimming pool chlorination tablets, such as calcium hypochlorite [$Ca(OCl)_2$] or trichloro-*s*-triazinetrione (TST), decompose or are inadvertently introduced to a swimming pool while swimmers are present. Inadvertent mixture of $Ca(OCl)_2$ and TST results in excessive chlorine gas generation and may also be explosive. Acute chlorine toxicity may occur when there is a failure of the system when compressed chlorine gas is used for direct chlorination of public swimming pools or for drinking water systems. Occasional mass poisoning may occur during scientific, industrial, or transportation incidents.

Figure 1: *Chlorine chemistry. (A) Formation of chlorine gas from the acidification of hypochlorous acid. (B) Dissolution of chlorine in mucosal water to generate both hydrochloric and hypochlorous acids (HCl and HOCl) and oxidants [O].*

The odor threshold for chlorine is low, but distinguishing toxic from permissible air

concentrations may be difficult until toxicity is manifest. The intermediate solubility characteristics of chlorine result in only mild initial symptoms after moderate exposure and permit a substantial time delay, typically several hours, before clinical symptoms develop. Chlorine dissolution in lung water generates HCl and hypochlorous (HOCl) acids. Hypochlorous acid rapidly decomposes into HCl and nascent oxygen (O^{-}). The unpaired nascent oxygen atom produces additional pulmonary damage by initiating a free radical oxidative cascade. Although the majority of life-threatening chlorine poisonings occur after acute, large exposures, patients with chronic, low-concentration exposure or recurrent, moderate concentration poisonings may manifest increased bronchial responsiveness.

Management of patients with acute respiratory tract injury begins with meticulous support of airway patency by limiting bronchial and pulmonary secretions and maintaining oxygenation. Although various theoretical and experimental treatment modalities have been proposed, supportive care remains the mainstay of therapy. Supplemental oxygen, bronchodilators, and airway suctioning should be used if clinically indicated.

There is an interesting report of simultaneous, presumably equivalent chlorine exposure in two sisters, with improved outcome in the sister who received steroid treatment. Most available research evaluates parenterally administered corticosteroids, although animal models demonstrate a beneficial effect of nebulized beclomethasone and nebulized budesonide after acute chlorine poisoning.

Although the use of neutralizing agents are contraindicated in acid or alkali injury of the GI tract, the large surface area of the lung and the relatively small amount of xenobiotic present allow dissipation of the heat and gas generated during neutralization. Case studies suggest that nebulized 2% sodium bicarbonate may be beneficial in patients poisoned by acid-forming irritant gases.

DISINFECTANTS

■ FORMALDEHYDE

Formaldehyde is a water-soluble, highly reactive gas at room temperature. Formalin consists of an aqueous solution of formaldehyde, usually containing approximately 37% formaldehyde and 12%–15% methanol. Formaldehyde is irritating to the upper airways, and its odor is readily detectable at low concentrations. Lethality in adults may follow ingestion of 30–60 mL of formalin.

Formerly used as a disinfectant and fumigant, its role as a disinfectant is now largely confined to the disinfection of hemodialysis machines. Nonetheless, formaldehyde has many other applications. Healthcare workers are probably most familiar with the use of formaldehyde as a tissue fixative and embalming agent.

Exposure to formaldehyde, a potent caustic, may result in both local and systemic

symptoms, causing coagulation necrosis, protein precipitation, and tissue fixation. Ingestions of formalin may result in significant gastric injury, including hemorrhage, diffuse necrosis, perforation, and stricture. The most extensive damage appears in the stomach, with only occasional involvement of the small intestine and colon. Chemical fixation of the stomach may occur.

The most striking and rapid systemic manifestation of formaldehyde poisoning is metabolic acidosis, resulting both from tissue injury and from the conversion of formaldehyde to formic acid. The patient may present with profound acidemia, accompanied by a large anion gap metabolic acidosis. Although the methanol component of the formalin solution is readily absorbed and has resulted in methanol concentrations as high as 40 mg/dL, the rapid metabolism of formaldehyde to formic acid appears to be responsible for much of the acidosis

Clinical Effects:
Patients presenting after formaldehyde ingestions complain of the rapid onset of severe abdominal pain, which may be accompanied by vomiting and diarrhea. Altered mental status and coma usually follow rapidly. Physical examination may demonstrate epigastric tenderness, hematemesis, cyanosis, hypotension, and tachypnea. Hypotension may be profound with decreased myocardial contractility, as well as hypovolemic shock, contributing to the cardiovascular instability. Early endoscopic findings include ulceration, necrosis, perforation, and hemorrhage of the stomach, with infrequent esophageal involvement. Chemical pneumonitis occurs after significant inhalational exposure. Intravascular hemolysis is described in hemodialysis patients whose dialysis equipment contained residual formaldehyde after undergoing routine cleaning.

Formaldehyde, at concentrations as low as 1 ppm, may cause significant irritation to mucous membranes of the upper respiratory tract and conjunctivae. Formaldehyde is also a potential sensitizer for immune-mediated reversible bronchospasm.

Management:
The immediate management of a patient who has ingested formaldehyde includes dilution with water. Although such an approach may be useful in reducing the caustic effect, strong evidence for a beneficial result is lacking. Gastric aspiration with a small-bore nasogastric tube may limit systemic absorption. The role of activated charcoal is not studied and it probably should not be used if endoscopy is considered likely. Significant acidemia should be treated with sodium bicarbonate and folinic acid. Immediate hemodialysis may remove the accumulating formic acid as well as the parent molecules, formaldehyde, and methanol. Early endoscopy is recommended for all patients with significant GI symptoms to assess the degree of burn injury. Surgical intervention may be required for those with suspected severe burns and/or perforation.

STERILANTS

■ ETHYLENE OXIDE

Ethylene oxide is a gas that is commonly used to sterilize heat-sensitive material in healthcare facilities. Unlike antiseptics and disinfectants, which generally do not exhibit full sporicidal activity, sterilants, such as ethylene oxide, inactivate all organisms. Ethylene oxide is also used in the synthesis of many chemicals, including ethylene glycol, surfactants, rocket propellants, and petroleum demulsifiers, and has been used as a fumigant.

Medical attention regarding ethylene oxide toxicity has centered on its mutagenic and possible carcinogenic effects. Retrospective studies suggest a possible excess incidence of leukemia and gastric cancer in ethylene oxide-exposed workers. These studies are inconclusive, and the carcinogenicity of ethylene oxide remains subject to debate.

Clinical Effects:
The acute toxicity of ethylene oxide is mainly the result of its irritant effects. Conjunctival, upper respiratory tract, GI, and dermal irritation may occur. Dermal burns from acute exposure to ethylene oxide are reported. Acute exposure to a broken ethylene oxide ampule by a 43-year-old recovery room nurse resulted in nausea, light- headedness, malaise, syncope, and recurrent seizures. There were no long-term complications.

Chronic exposure to high concentrations of ethylene oxide may cause mild cognitive impairment and motor and sensory neuropathies. The risk of cancer with occupational exposure is low.

Management:
Treatment for patients with ethylene oxide exposure is supportive.

TABLE 102–1. Antiseptics, Disinfectants, Sterilants, and Related Xenobiotics

Xenobiotic	Commercial Product	Use	Toxic Effects	Therapeutics and Evaluation
Acids				
Boric acid	Borax Sodium perborate Dobell solution	Antiseptic Mouthwash Eyewash Roach powder	Blue-green emesis and diarrhea Boiled lobster appearance of skin CNS depression; renal failure	GI decontamination Hemodialysis (rare)
Alcohols				
(Chaps. 77 and 107)				
Ethanol	Rubbing alcohol (70% ethanol)	Antiseptic Disinfectant	CNS depression Respiratory depression Dermal irritant	Supportive
Isopropanol	Rubbing alcohol (70% isopropanol)	Antiseptic Disinfectant	CNS depression Respiratory depression Ketonemia, ketonuria GI irritation/bleeding Hemorrhagic tracheobronchitis	Supportive Hemodialysis (rare)
Aldehydes				
Formaldehyde	Formalin (37% formaldehyde, 12%–15% methanol)	Disinfectant Fixative Urea insulation	Caustic CNS depression Carcinogen	Gastric lavage Hemodialysis Sodium bicarbonate Endoscopy Folinic acid

Chlorinated Compounds				
Chlorhexidine	Hibiclens	Antiseptic	GI irritation	Supportive
Chlorates	Sodium chlorate	Antiseptic	Hemolytic anemia	Exchange transfusion
	Potassium chlorate	Matches	Methemoglobinemia	Hemodialysis
		Herbicide	Renal failure	
Chlorine		Disinfectant	Irritant	Supportive
Chlorophors (sodium hypochlorite)	Household bleach (5% NaOCl)	Disinfectant	Mild GI irritation	Endoscopy (rare)
	Dakin solution (1 part 5% NaOCl, 10 parts H_2O)	Decontaminating solution		
Ethylene Oxide		Sterilant	Irritant	Supportive
		Plasticizer	CNS depression	
			Peripheral neuropathy	
			Carcinogen?	
Mercurials	Merbromin 2% (Mercurochrome)	Antiseptic (obsolete)	CNS	Gastric lavage, activated charcoal dimercaprol, succimer
(Chaps. 55 and 96)	Thimerosal (Merthiolate)		Renal	
Iodinated Compounds				
Iodine	Tincture of iodine (2% iodine, 2% sodium iodide and 50% ethanol)	Antiseptic	Caustic	Milk, starch, sodium thiosulfate
				Endoscopy
	Lugol solution (5% iodine)			
Iodophors	Povidone-iodine (Betadine) (0.01% iodine)	Antiseptic	Limited	Same as iodine

TABLE 102–1. Antiseptics, Disinfectants, Sterilants, and Related Xenobiotics (*Continued*)

Xenobiotic	Commercial Product	Use	Toxic Effects	Therapeutics and Evaluation
Oxidants				
Hydrogen peroxide	H_2O_2 3%–household	Disinfectant	Oxygen emboli	Gastric lavage
	H_2O_2 30%–industrial		GI caustic	Radiographic evaluation
				Endoscopy
Potassium permanganate	Crystals, solution	Antiseptic	Oxidizer, caustic, increased serum manganese	Decontamination
				Endoscopy as needed
Phenols				
Nonsubstituted	Phenol (carbolic acid)	Disinfectant	Caustic	Decontamination: polyethylene glycol or water
			Dermal burns	
			Cutaneous absorption	Endoscopy as needed
			CNS effects	
Substituted	Hexachlorophene	Disinfectant	CNS effects	Supportive
Quaternary Ammonium Compounds				
Benzalkonium chloride	Zephiran	Disinfectant	GI caustic	Consider endoscopy