Alcohols

HIGHLIGHTS

Often mixtures, usually ethanol & isopropanol

Most common household product 70% isopropyl alcohol

Well absorbed by GI, skin, inhalation

High concentrations can depress CNS leading to coma, death

SIGNS & SYMPTOMS

GI irrigation: gastritis, vomiting

Can be measured in blood and urine

TREATMENT

Support for hypotension, respiratory depression; best in ICU

Glucose if hypoglycemia occurs

Consider hemodialysis

CONTRAINDICATED

Induced emesis

CHAPTER 20

Disinfectants

A wide variety of disinfectant agents are used to destroy microorganisms, and they differ greatly in their toxic effects. However, most can conveniently be grouped into a few categories, some of which are represented in other classes of pesticides. Many of these materials are not registered as pesticides but are registered for medical or medicinal use. This chapter reviews a few of the more common or more toxic disinfectants.

ALCOHOLS

Alcohols have a long history of use as disinfectants. Often disinfectants are mixtures, usually of ethanol and isopropyl alcohol (isopropanol). The alcohol most commonly used in households as a disinfectant is isopropyl alcohol, commonly marketed as a 70% solution. It is a clear, colorless liquid with an odor similar to ethanol.

Toxicology

Isopropyl alcohol is well and rapidly absorbed from the gastrointestinal tract. It is also well absorbed by skin and by inhalation. It is considered to be more toxic to the central nervous system than **ethanol**, with similar effects. Both ingestion and inhalation at high concentrations can result in the rapid onset of CNS depression with subsequent coma and death. Apnea commonly accompanies this CNS depression.[1,2] Similar neurological toxicity has been reported with excessive topical exposure to the umbilicus of a neonate.[3] Irritation of the gastrointestinal tract results in gastritis and severe vomiting. Isopropyl alcohol may also produce mild hepatic injury following acute exposure. Acute tubular necrosis has been reported with this agent,[1] but the renal toxicity is not as great as with methanol poisonings. Ketosis without metabolic acidosis can occur, but prominent hypoglycemia is common.[2,3] This ketosis is the result of direct metabolism of this compound to acetone.[1,3] Monitoring of isopropyl levels is useful, when available. In addition, blood levels of acetone and glucose should be determined to aid in management.

Confirmation of Poisoning

Isopropyl alcohol can be measured in the blood and urine. Serum acetone can also be measured. Blood isopropyl alcohol levels of 128-200 mg/dL have been associated with death.

Treatment of Isopropyl Alcohol Toxicosis

1. Do not induce emesis, since the onset of coma is often rapid with this poisoning. Spontaneous vomiting, however, often occurs.

2. Provide supportive care for hypotension and respiratory depression. This is critical to survival and should be administered whenever possible in an intensive care setting.

3. If hypoglycemia occurs, administer glucose.

4. Consider hemodialysis, which has been reported to be beneficial in patients with severe poisoning who are unresponsive to standard supportive therapy.[1,4]

ALDEHYDES

The two aldehydes most commonly used as disinfectants are **formaldehyde** and **glutaraldehyde**. Formaldehyde is discussed in **Chapter 17, *Fumigants***. Glutaraldehyde is very similar to formaldehyde in its toxicity and treatment, although it is slightly less toxic. Glutaraldehyde is commonly prepared as an aqueous solution at a 2% concentration and is slightly alkaline in this solution. It has been reported to cause respiratory irritation, resulting in rhinitis[5,6] and occupational asthma.[5,7,8] It has also resulted rarely in palpitations and tachycardia in human subjects. At high dosage, given orally, it results in gastrointestinal irritation with diarrhea, which may be hemorrhagic.[9,10,11] Because of the irritant effects of glutaraldehyde, Occupational Safety and Health Administration (OSHA) standards may apply for wearing personal protective equipment to protect the skin (29 CFR 1910.132) and eyes (29 CFR 1910.133). OSHA standards may also require the use of appropriate respirators by employees who may be exposed to glutaraldehyde during routine or emergency work procedures (29 CRF 1910.134).[12]

Treatment of Aldehyde Toxicosis

1. If patient has been in an area with a strong odor of glutaraldehyde due to vaporization, move to fresh air and administer oxygen as needed.

2. If skin irritation is noted, decontaminate. Systemic toxicity from skin exposure is unlikely.

CATIONIC DETERGENTS

Several cationic detergents are used as disinfectants. All share the capacity, in sufficient concentration, to cause rather severe caustic burns. Concentrations greater than approximately 7.5% appear necessary to produce significant caustic injuries. However, experience with human exposures to these compounds is very limited. The three agents most commonly used as detergent disinfectants are benzalkonium chloride, cetrimide and cetylpyridium chloride.

No cetrimide preparations are available in the United States; several are available in European Union countries. Concentrated solutions are usually only available in industrial settings, such as production of consumer products, or for use in hospitals for disinfectant purposes. Therefore, acute poisonings are uncommon.

Toxicology

In low concentration solutions, **cationic detergents** have been reported to cause eye discomfort, as well as skin rashes and irritation. A severe contact dermatitis has been reported with a bath oil containing **benzalkonium chloride** and **triclosan**.[13]

Aldehydes

HIGHLIGHTS

Formaldehyde also discussed in Ch. 17

Glutaraldehyde similar but less toxic

SIGNS & SYMPTOMS

Respiratory irritation

GI irritation, diarrhea, possible hemorrhage if oral

TREATMENT

Move to fresh air, administer oxygen as needed

Decontaminate skin if irritated

Cationic Detergent
HIGHLIGHTS

Caustic agents capable of causing burns

Most common: benzalkonium chloride, cetrimide, cetylpyridium chloride (no cetrimide in U.S.)

Acute poisonings are uncommon

SIGNS & SYMPTOMS

Eye, skin, GI irritant

Severe exposures: CNS, liver, pulmonary impacts

TREATMENT

Wash eyes, examine/treat corneas for burns

Endoscopy within 24 hours if indicated

Treat CNS, pulmonary, other systemic effects

CONTRAINDICATED

GI decontamination

In stronger concentrations, they can cause severe corneal and skin burns. Likewise, strong concentrations will result in caustic burns to lips, oral mucosa, esophagus and stomach.[14,15] Vomiting, diarrhea and abdominal pain have been reported.[16] Necrosis of the gut, with peritonitis, has also been reported.[17] In severe exposures, there are also reports of CNS depression, liver injury and pulmonary edema.[14,16]

Treatment of Cationic Detergent Toxicosis

1. If a high concentration solution is in contact with the eyes, wash the eyes profusely and then carefully examine the corneas. If burns have occurred, obtain ophthalmologic care.

2. Do not use any method of gastrointestinal decontamination, including gastric emptying. They are contraindicated in these poisonings. Some experts recommend cautious dilution with small amounts of milk or water.[14,18] Acidic solutions, such as juices, should never be offered for dilution.

3. Conduct an endoscopy if a highly concentrated solution was ingested or oral burns are noted. The patient needs urgent endoscopy for grading of the caustic injury. The endoscopy should be performed within 24 hours to minimize the risk of perforation.[17] A competent surgeon or gastroenterologist should provide subsequent care.

4. Treat CNS, pulmonary and other systemic effects symptomatically, consistent with sound medical practice.

Although corticosteroids are commonly used to treat these burns, their use remains controversial. Use of other agents, such as H_2 antagonists and sulcralfate, has been reported, but also remains controversial at this time.

CHLORHEXIDINE

Chlorhexidine is a cationic biguanide, available in concentrations up to 4% as a topical agent used as a skin cleanser and mouthwash. Skin preparations of 0.5%-4% are marketed under the trade names Hibiclens and Hibistat. It is also marketed as a mouthwash in a 0.12% solution under the trade name Peridex. There is very little human experience with poisonings, as these concentrations do not appear to be significantly toxic.

Toxicology

Chlorhexidine is poorly absorbed from skin or the gastrointestinal tract. Therefore, most effects noted have been primarily local. Low concentration solution ingested or applied to the skin can cause mild local irritation. Contact dermatitis, urticaria and anaphylaxis have followed repeated skin exposures to this agent.[19,20,21] Corneal injuries have been described in several cases after inadvertent exposure of the eyes to the 4% concentration. These injuries have resulted in permanent corneal scarring.[22] Esophageal burns have been reported in a single case after ingestion of a large quantity of a 20% solution of this agent.[23] Ulcerative colitis has been described after an enema of the 4% solution mixed with tap water (10 mL in 2 liters water).[24] Liver toxicity can occur with large exposures.[23]

Treatment of Chlorhexidine Toxicosis

1. If a highly concentrated solution is ingested, manage as a caustic ingestion as described in the preceding *Treatment of Cationic Detergent Toxicosis* subsection, without gastrointestinal decontamination.
2. Perform liver injury panel with large ingestions.
3. If a high concentration solution is in contact with the eyes, wash eyes profusely and examine the corneas carefully. If burns have occurred, obtain ophthalmologic care.

HYPOCHLORITES

Hypochlorites are implicated in a large proportion of the disinfectant exposures reported to poison control centers in the United States, with more than 30,000 reports in 2009.[25] Most are solutions of **sodium** or **calcium hypochlorite**. **Chloramine**, a disinfectant used in many municipal water supplies, is an infrequent cause of acute poisonings. Sodium and calcium hypochlorite solutions are of relatively low toxicity. They are mildly corrosive to eyes,[26] and mucous membrane burns have been reported.[27] Despite the large number of reports to poison control, significant poisonings are very infrequent with these agents in solution.[25,28]

When hypochlorite solutions are mixed with acids or ammonia solutions, chlorine or chloramine gas is produced, resulting in an irritant with pulmonary toxicity. Many brief exposures have led to transient symptoms requiring limited emergency department management.[29] Prolonged exposure or exposure to high concentrations carries the potential of severe toxic pneumonitis.[30] Great efforts should be made to discourage mixing of these materials with acid or ammonia.

Treatment of Hypochlorite Toxicosis

1. After oral exposures, do not use gastric emptying. If a granular material is ingested and the patient has symptomatic mucosal burns, refer patient to a surgeon or gastroenterologist for consideration of endoscopy and management.
2. If vomiting has not occurred, give patient water or milk for dilution, not to exceed approximately 15 mL/kg in a child or 120-240 mL in an adult. Administration of acids is contraindicated, because of the risk or increasing generation of chlorine gas.
3. If a high concentration solution is in contact with the eyes, wash eyes profusely and examine corneas carefully. If burns have occurred, obtain ophthalmologic care.
4. Manage skin exposure with copious water dilutions.
5. If exposure to vapors or chlorine or chloramine gas has occurred, move patient immediately to fresh air. If symptoms occur or persist, oxygenation should be assessed and oxygen administered as needed. If persistent symptoms occur, obtain a chest film and consider hospitalization. Intensive care may be appropriate in severe inhalations.

Chlorhexidine

HIGHLIGHTS

Used as skin cleanser, mouthwash

Poorly skin, gut absorption

SIGNS & SYMPTOMS

Mild skin irritant, worse if repeat exposures

Corneal, esophageal injuries possible

TREATMENT

For highly concentrated/ large doses

- Ingested: manage as caustic ingestion, perform liver panel
- Eye contact: wash, examine, treat corneas

Hypochlorite

HIGHLIGHTS

Chloramine, sodium/calcium hypochlorite

Many exposures reported; significant poisonings few

SIGNS & SYMPTOMS

Pulmonary irritation, toxicity when mixed with acids or ammonia solutions

GI, eye, skin, pulmonary impacts

TREATMENT

If ingestions result in mucosal burns, refer to surgeon/ gastroenterologist

Decontaminate eyes, skin

Examine/treat corneas

If vapor exposure, move to fresh air and consider oxygen administration, chest film, hospitalization

CONTRAINDICATED

Administration of acids

Gastric emptying

Iodine

HIGHLIGHTS

Most common: 7.5%-10% povidone-iodine solution

Betadine is an example

At standard dilutions, poorly absorbed from GI, skin

Symptomatic poisonings possible on burned skin, wounds

SIGNS & SYMPTOMS

Initial: headache, dizziness, delirium, hallucinations, seizures

Severe: hypotension, arrhythmias, cyanosis, metabolic acidosis, shock, renal failure

TREATMENT

Decontaminate skin

Osmotic agents or diuretics if indicated

Treat seizures

Monitor thyroid

IODINE

The most common iodine-containing disinfectant is povidone-iodine. A trade name often associated with this agent is Betadine (7.5%-10% solution). Povidine-iodine is described as an iodophor, which is a complex of iodine and polyvinylpyrrolidone, a solubilizing agent. It is intended to liberate free iodine in solution for its effect. Although reported concentrations of iodine in these solutions is only 80-120 µg/dL, the total available iodine is approximately 10% of the povidone-iodine. Therefore, a 10% solution will have in the range of 1% total available iodine.

Toxicology

This compound is very poorly absorbed from the gastrointestinal tract, because of the rapid conversion of free iodine to iodide in the stomach. Though highly concentrated iodine solutions or iodine salts are corrosive to the gastrointestinal tract,[31] solutions of **povidone-iodine** have little caustic potential. It is likewise poorly absorbed from intact skin. All symptomatic poisonings reported have occurred either after repeated exposure to burned skin or following irrigation of wounds, joints or serosal surfaces, such as the mediastinum.[32,33,34,35] The one exception was an infant who received an enema of povidone-iodine in a polyethylene glycol solution, followed by whole bowel irrigation with polyethylene glycol mixed with povidone-iodine. This child died with severe hyperglycemia and very high iodine levels.[31]

In povidone-iodine exposures by these routes, the primary symptoms initially appear to be neurological, with headache, dizziness, delirium, hallucinations and seizures.[35] Hypotension, arrhythmias, cyanosis, metabolic acidosis, shock and acute renal failure occur in severe cases.[32,33,34] Hepatic injury, manifested by elevated serum transaminase levels, has also been reported with very high level exposures.[34] Hyperkalemia has occurred, and the serum chloride may be falsely elevated due to the presence of a second halide.[33]

Treatment of Iodine Toxicosis

1. Remove skin contamination by vigorous washing with soap and water.
2. Use osmotic agents or diuretics in symptomatic poisonings, since iodine clearance is apparently enhanced by procedures that enhance chloride excretion.
3. Treat seizures with anticonvulsants, as outlined in **Chapter 3, *General Principles***.
4. Monitor thyroid function following recovery to confirm euthyroid state.

MERCURIALS

A wide variety of organic mercurials have been used as disinfectants and as preservatives. These included **phenylmercuric acetate, phenylmercuric nitrate, nitromersol, thimerosol, mercurochrome** and **mercurobutol**. None is currently registered with the U.S. Environmental Protection Agency. The toxicity and treatment of exposure to these compounds is described in detail in **Chapter 16, *Fungicides*** under the subsection *Organomercury Compounds*.

PHENOLS

Several phenols are used as disinfectants, including cresol, phenol, thymol, hexachlorophene, o-phenylphenol, 4-tert-amylphenol, 2-benzyl-4-chlorophenol and triclosan. Cresol and thymol are alkyl derivatives of phenol, while hexachlorophene and triclosan are chlorinated phenols. Common trade names for commercial products are provided in the margin. One survey found that **triclosan** or a similar agent, **triclocarban**, was found in 45% of liquid and bar soaps available in consumer outlets.[36] However, no episodes of acute toxicity from triclosan have been reported, so the concerns with this agent relate to chronic effects, the development of triclosan resistance in microbial organisms, and reports of contact dermatitis caused by exposure to triclosan.[13,37,38] Cresols and hexachlorophene will be discussed individually; these compounds are familiar and some human data are available.

Toxicology of Cresols

Cresols, in common with phenol and other phenolic compounds, are highly corrosive. Ingestion of concentrated forms causes severe corrosive injury to the mouth and upper gastrointestinal tract. Likewise, severe eye and skin caustic injuries can occur with cresol exposure.[39] Symptoms usually include nausea, vomiting and diarrhea. Hypotension, myocardial failure, pulmonary edema, neurological changes may also occur.[40] Liver and renal toxicity, methemoglobinemia and hemolysis have all been reported.[40,41] After long-term, repeated exposure, contact dermatitis may complicate these exposures. These compounds are well absorbed from the gastrointestinal tract and are also significantly absorbed from the skin and by inhalation.

Treatment of Cresol Toxicosis

1. Do not attempt gastrointestinal decontamination because of the corrosive nature of these compounds. Consider dilution with milk or water if vomiting has not occurred.

2. If a corrosive injury has occurred with burns to the mouth, or if there is a clear history of gastrointestinal exposure, consider endoscopy and consult a gastroenterologist or surgeon for diagnosis and management.

3. If a high concentration solution is in contact with the eyes, wash eyes with profuse amounts of water and follow with a careful exam of the corneas. If burns have occurred, provide ophthalmologic care. Given the corrosive nature of the substance, referral to an ophthalmologist should be considered.

4. Provide respiratory and circulatory support in accordance with sound medical management. If severe systemic symptoms persist, the patient should be treated in an intensive care unit, if possible.

Toxicology of Hexachlorophene

Hexachlorophene is well absorbed via the oral and dermal routes. Dermal exposures have led to severe toxicity and death in neonates, due to application to damaged skin or repeated or high-concentration skin exposures.[42] It should never be used as a disinfectant on open wounds or abraded or inflamed skin surfaces. It is not significantly caustic, however, and exposure does not result in the severe caustic injuries seen with other phenolic chemicals.

Phenols

COMMERCIAL PRODUCTS

Triclosan: many consumer soap products

Mixed cresols in soap: Lysol

Hexachlorophene: Phisohex, Bilevon, Dermaadex, Exofene, Gamophen, Texosan, Surgi-Cen, Surofene, various soap bars and cosmetics

HIGHLIGHTS

Triclosan

- Very common, no acute toxicity reports
- Contact dermatitis

Cresols

- Highly corrosive
- Can cause severe mouth, GI, eye, skin injury
- Well absorbed from GI, skin, inhalation

Hexachlorophene

- Well absorbed via skin, gut
- Not as caustic as other phenolic compounds
- Potent neurotoxicant

continued next page

Phenols, cont.

SIGNS & SYMPTOMS

Cresols

Nausea, vomiting, diarrhea

Caustic skin, eye injuries

Hexachlorophene

Complex CNS effects

Lethargy, muscle weakness/fasciculation, irritability, cerebral edema, paralysis

Vomiting, diarrhea, anorexia

Skin rash

TREATMENT

Cresols

Consider GI dilution with water/milk

Consider endoscopy with gastroenterologist consult

Decontaminate eyes, examine/treat corneas

Respiratory, circulatory support

Hexachlorophene

Consider activated charcoal

Decontaminate skin with soap and water

Control seizures

Support cardiovascular, respiratory systems

CONTRAINDICATED

Cresols

GI decontamination

Hexachlorophene is a potent neurotoxicant. It causes brain edema and spongy degeneration of white matter.[43] This neurotoxicity can be seen after acute or chronic exposures, either by skin absorption or ingestion. The nervous system symptoms are complex. Lethargy is an early manifestation, followed by muscular weakness, muscular fasciculation, irritability, cerebral edema and paralysis, leading to coma and death. Seizures commonly occur in more severe cases.[42,44] Blindness and optic atrophy have also been seen following exposure to hexachlorophene.[45]

In addition to the neurological effects, common early symptoms of poisoning are vomiting, diarrhea and anorexia.[44] These findings have been accompanied in animals by significant hepatotoxicity.[46] With skin exposure, an erythematous, desquamative rash is often noted at the site of exposure.[44] With chronic exposure, contact dermatitis may be noted. In severe poisonings, cardiovascular symptoms, including hypotension and bradycardia have been noted.[47] In a single case, repeated exposure to this compound led to asthma in a pediatric nurse.[48]

Treatment of Hexachlorophene Toxicosis

1. Although this compound is quite toxic systemically and enhanced clearance methods would appear beneficial, there is no evidence to support efficacy of hemodialysis, peritoneal dialysis, hemoperfusion or exchange transfusion.[47]

2. Consider using activated charcoal. Since hexachlorophene is thought to have an enterohepatic recirculation, it is possible that repeated dosing of activated charcoal, as outlined in the **Chapter 3, *General Principles***, will enhance clearance of this compound although hexachlorophene does not bind well to charcoal and there are no clinical trials of this therapy for this agent.

3. If exposure has occurred through the skin, wash skin aggressively with soap or detergent and water to remove any residues still on the skin. Since hexachlorophene is not soluble in water, washing with water alone will not provide significant benefit.

4. Perform neurological support and seizure control, as these are critical to survival. When possible, perform in an intensive care setting. Seizure control should be in accordance with recommendations in **Chapter 3.**

5. Provide cardiovascular and respiratory support, which are also very important to success in treating severe poisonings with this agent. This care should be provided in an intensive care unit in accordance with accepted medical practice.

PINE OIL

Toxicology

Exposures to **pine oil** detergent and disinfectant solutions are commonly reported to poison control centers in the United States.[49] Pine oil is an agent commonly contained in a variety of household and commercial cleaners and disinfectants. It is a mixture of monoterpenes derived from the distillation of wood from various pine species, with approximately 57% being alpha-pinene.[50] Its most common side effects in smaller dosage are irritation of mucous membranes, gastrointestinal irritation, mild respiratory and CNS depression and renal toxicity. Larger ingestions can result in severe respiratory distress, cardiovascular collapse, and severe CNS effects. Renal failure and myoglobinuria have also been reported in severe poisonings.[51] Since even small ingestions can result in severe aspiration pneumonia, all ingestions should be considered potentially hazardous.

While many of the reported effects of poisoning with this agent are related to direct irritant effect on mucous membranes, gastrointestinal tract and lungs (by aspiration), some reports suggest significant absorption from oral and rectal exposures. Other reports suggest a lesser rate of absorption.[50] While alpha terpineol can be measured in blood, there are no data relating terpineol levels to degree of toxicity; this measure, therefore, is not considered useful in guiding diagnosis and management.

Treatment of Pine Oil Toxicosis

1. Do not induce emesis. Since there is a high risk of aspiration pneumonia, induced emesis is usually considered contraindicated in these poisonings. However, spontaneous emesis may occur because of direct irritation of the gastric mucosa.
2. If a high concentration solution is in contact with the eyes, flush eyes profusely and carefully examine corneas. If burns have occurred, obtain ophthalmologic care.
3. Observe the patient for at least 6 hours with any significant ingestion in order to observe the onset of any symptoms, particularly pulmonary symptoms.
4. Order chest films and measure oxygenation if any pulmonary symptoms are observed. If pulmonary symptoms occur, hospitalization is appropriate. With severe pulmonary symptoms transfer to an intensive care unit is usually appropriate. With severe aspiration, manage as with any severe aspiration pneumonia, in accordance with accepted medical practice.
5. Treat other severe systemic effects in accordance with accepted medical practice.

There is no evidence that activated charcoal is helpful in these poisonings. Likewise, although a variety of enhanced elimination methods have been proposed and tried, there is no evidence to support their efficacy.

Pine Oil

HIGHLIGHTS

Common household/ commercial cleaning ingredient

Monoterpene wood derivative

Primarily inhalation and ingestion route to poisoning

All ingestions have potential for severe aspiration pneumonia

Oral, rectal exposure routes also possible

SIGNS & SYMPTOMS

Mucous membrane, GI irritation

Mild respiratory, CNS depression

Severe respiratory, cardiovascular, CNS impacts from larger ingestions

TREATMENT

Flush eyes and examine/ treat corneas

Observe for 6 hours post-exposure, esp. for pulmonary symptoms

Pulmonary support

Order chest films and measure oxygenation

Hospitalize, consider ICU

In severe cases, manage as aspiration pneumonia

CONTRAINDICATED

Induced emesis

References

1. Lacouture PG, Wason S, Abrams A, Lovejoy FH, Jr. Acute isopropyl alcohol intoxication. Diagnosis and management. *Am J Med*. Oct 1983;75(4):680-686.
2. Rich J, Scheife RT, Katz N, Caplan LR. Isopropyl alcohol intoxication. *Arch Neurol*. Mar 1990;47(3):322-324.
3. Vivier PM, Lewander WJ, Martin HF, Linakis JG. Isopropyl alcohol intoxication in a neonate through chronic dermal exposure: a complication of a culturally-based umbilical care practice. *Pediatr Emerg Care.* Apr 1994;10(2):91-93.
4. Manring E, Meggs W, Pape G, Ford M. Toxicity of an intravenous infusion of isopropyl alcohol. *J Toxicol Clin Toxicol.* 1997;35:503.
5. Corrado OJ, Osman J, Davies RJ. Asthma and rhinitis after exposure to glutaraldehyde in endoscopy units. *Hum Toxicol*. Sep 1986;5(5):325-328.
6. Norback D. Skin and respiratory symptoms from exposure to alkaline glutaraldehyde in medical services. *Scand J Work Environ Health.* Dec 1988;14(6):366-371.
7. Chan-Yeung M, McMurren T, Catonio-Begley F, Lam S. Occupational asthma in a technologist exposed to glutaraldehyde. *J Allergy Clin Immunol.* May 1993;91(5):974-978.
8. Stenton SC, Beach JR, Dennis JH, Keaney NP, Hendrick DJ. Glutaraldehyde, asthma and work--a cautionary tale. *Occup Med (Lond)*. May 1994;44(2):95-98.
9. Symptoms of irritation associated with exposure to glutaraldehyde--Colorado. *MMWR Morb Mortal Wkly Rep.* Apr 3 1987;36(12):190-191.
10. Fukunaga K, Khatibi A. Glutaraldehyde colitis: a complication of screening flexible sigmoidoscopy in the primary care setting. *Ann Intern Med.* Aug 15 2000;133(4):315.
11. Stonehill AA, Krop S, Borick PM. Buffered glutaraldehyde -- a new chemical sterilization solution. *Am J Hosp Pharm*. 1963;20:458-465.
12. US Department of Labor OSaHA. *Best practices for the safe use of glutaraldehyde in health care.* 2006.
13. Storer E, Koh KJ, Warren L. Severe contact dermatitis as a result of an antiseptic bath oil. *Australas J Dermatol.* Feb 2004;45(1):73-75.
14. Mucklow ES. Accidental feeding of a dilute antiseptic solution (chlorhexidine 0.05% with cetrimide 1%) to five babies. *Hum Toxicol.* Nov 1988;7(6):567-569.
15. Wilson JT, Burr IM. Benzalkonium chloride poisoning in infant twins. *Am J Dis Child*. Oct 1975;129(10):1208-1209.
16. Chan TY. Poisoning due to Savlon (cetrimide) liquid. *Hum Exp Toxicol.* Oct 1994;13(10):681-682.
17. Zargar SA, Kochhar R, Mehta S, Mehta SK. The role of fiberoptic endoscopy in the management of corrosive ingestion and modified endoscopic classification of burns. *Gastrointest Endosc.* Mar-Apr 1991;37(2):165-169.
18. Consensus: POISONDEX Editorial Board consensus opinion poll, irritants/caustics specialty board. 1988.
19. Perrenoud D, Bircher A, Hunziker T, et al. Frequency of sensitization to 13 common preservatives in Switzerland. Swiss Contact Dermatitis Research Group. *Contact Dermatitis.* May 1994;30(5):276-279.
20. Okano M, Nomura M, Hata S, et al. Anaphylactic symptoms due to chlorhexidine gluconate. *Arch Dermatol*. Jan 1989;125(1):50-52.
21. Wong WK, Goh CL, Chan KW. Contact urticaria from chlorhexidine. *Contact Dermatitis.* Jan 1990;22(1):52.
22. Tabor E, Bostwick DC, Evans CC. Corneal damage due to eye contact with chlorhexidine gluconate. *JAMA*. Jan 27 1989;261(4):557-558.

23. Massano G, Ciocatto E, Rosabianca C, Vercelli D, Actis GC, Verme G. Striking aminotransferase rise after chlorhexidine self-poisoning. *Lancet*. Jan 30 1982;1(8266):289.

24. Hardin RD, Tedesco FJ. Colitis after Hibiclens enema. *J Clin Gastroenterol*. Oct 1986;8(5):572-575.

25. Bronstein AC, Spyker DA, Cantilena LR, Jr., Green JL, Rumack BH, Giffin SL. 2009 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 27th Annual Report. *Clin Toxicol (Phila).* Dec 2010;48(10):979-1178.

26. Ingram TA, 3rd. Response of the human eye to accidental exposure to sodium hypochlorite. *J Endod.* May 1990;16(5):235-238.

27. French RJ, Tabb HG, Rutledge LJ. Esophageal stenosis produced by ingestion of bleach: report of two cases. *South Med J*. Oct 1970;63(10):1140-1144.

28. Landau GD, Saunders WH. The Effect of Chlorine Bleach on the Esophagus. *Arch Otolaryngol.* Aug 1964;80:174-176.

29. Mrvos R, Dean BS, Krenzelok EP. Home exposures to chlorine/chloramine gas: review of 216 cases. *South Med J.* Jun 1993;86(6):654-657.

30. Reisz GR, Gammon RS. Toxic pneumonitis from mixing household cleaners. *Chest*. Jan 1986;89(1):49-52.

31. Kurt TL, Morgan ML, Hnilica V, Bost R, Petty CS. Fatal iatrogenic iodine toxicity in a nine-week old infant. *J Toxicol Clin Toxicol.* 1996;34(2):231-234.

32. Campistol JM, Abad C, Nogue S, Bertran A. Acute renal failure in a patient treated by continuous povidone-iodine mediastinal irrigation. *J Cardiovasc Surg (Torino).* Jul-Aug 1988;29(4):410-412.

33. Means LJ, Rescorla FJ, Grosfeld JL. Iodine toxicity: an unusual cause of cardiovascular collapse during anesthesia in an infant with Hirschsprung's disease. *J Pediatr Surg.* Dec 1990;25(12):1278-1279.

34. Pietsch J, Meakins JL. Complications of povidone-iodine absorption in topically treated burn patients. *Lancet*. Feb 7 1976;1(7954):280-282.

35. Ponn RB. Continuous povidone-iodine irrigation. *Ann Thorac Surg.* Feb 1987;43(2):239.

36. Perencevich EN, Wong MT, Harris AD. National and regional assessment of the antibacterial soap market: a step toward determining the impact of prevalent antibacterial soaps. *Am J Infect Control.* Oct 2001;29(5):281-283.

37. Robertshaw H, Leppard B. Contact dermatitis to triclosan in toothpaste. *Contact Dermatitis*. Dec 2007;57(6):383-384.

38. Wong CS, Beck MH. Allergic contact dermatitis from triclosan in antibacterial handwashes. *Contact Dermatitis.* Nov 2001;45(5):307.

39. Pegg SP, Campbell DC. Children's burns due to cresol. *Burns Incl Therm Inj.* Apr 1985;11(4):294-296.

40. Arthurs GJ, Wise CC, Coles GA. Poisoning by cresol. *Anaesthesia*. Jul-Aug 1977;32(7):642-643.

41. Chan TK, Mak LW, Ng RP. Methemoglobinemia, Heinz bodies, and acute massive intravascular hemolysis in lysol poisoning. *Blood*. Dec 1971;38(6):739-744.

42. Mullick FG. Hexachlorophene toxicity. Human experience at the Armed Forces Institute of Pathology. *Pediatrics*. Feb 1973;51(2):395-399.

43. Anderson JM, Cockburn F, Forfar JO, Harkness RA, Kelly RW, Kilshaw B. Neonatal spongioform myelinopathy after restricted application of hexachlorophane skin disinfectant. *J Clin Pathol*. Jan 1981;34(1):25-29.

44. Martin-Bouyer G, Lebreton R, Toga M, Stolley PD, Lockhart J. Outbreak of accidental hexachlorophene poisoning in France. *Lancet*. Jan 9 1982;1(8263):91-95.

45. Slamovits TL, Burde RM, Klingele TG. Bilateral optic atrophy caused by chronic oral ingestion and topical application of hexachlorophene. *Am J Ophthalmol.* May 1980;89(5):676-679.

46. Prasad GV, Rajendra W, Indira K. Brain ammonia metabolism in hexachlorophene-induced encephalopathy. *Bull Environ Contam Toxicol.* Apr 1987;38(4):561-564.

47. Boehm RM, Jr., Czajka PA. Hexachlorophene poisoning and the ineffectiveness of peritoneal dialysis. *Clin Toxicol.* Mar 1979;14(3):257-262.

48. Nagy L, Orosz M. Occupational asthma due to hexachlorophene. *Thorax*. Aug 1984;39(8):630-631.

49. Bronstein AC, Spyker DA, Cantilena LR, Jr., Green JL, Rumack BH, Giffin SL. 2008 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 26th Annual Report. *Clin Toxicol (Phila).* Dec 2009;47(10):911-1084.

50. Koppel C, Tenczer J, Tonnesmann U, Schirop T, Ibe K. Acute poisoning with pine oil - metabolism of monoterpenes. *Arch Toxicol*. Nov 1981;49(1):73-78.

51. Litovitz TL, Schmitz BF, Matyunas N, Martin TG. 1987 annual report of the American Association of Poison Control Centers National Data Collection System. *Am J Emerg Med.* Sep 1988;6(5):479-515.