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. 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.\textsuperscript{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\textsuperscript{5,6} and occupational asthma.\textsuperscript{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.\textsuperscript{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).\textsuperscript{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 cetlypyridinium 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.\textsuperscript{13}
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. Vomiting, diarrhea and abdominal pain have been reported. Necrosis of the gut, with peritonitis, has also been reported. In severe exposures, there are also reports of CNS depression, liver injury and pulmonary edema.

**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. 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. 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 H2 antagonists and sulcrafate, 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. 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. Esophageal burns have been reported in a single case after ingestion of a large quantity of a 20% solution of this agent. Ulcerative colitis has been described after an enema of the 4% solution mixed with tap water (10 mL in 2 liters water). Liver toxicity can occur with large exposures.
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 of 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.
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

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**IODINE**

The most common iodine-containing disinfectant is povidone-iodine. A trade name often associated with this agent is Betadine (7.5%-10% solution). Povidone-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.36
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. 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. 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. Symptoms usually include nausea, vomiting and diarrhea. Hypotension, myocardial failure, pulmonary edema, neurological changes may also occur. Liver and renal toxicity, methemoglobinemia and hemolysis have all been reported. 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. 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.

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Hexachlorophene is a potent neurotoxicant. It causes brain edema and spongy degeneration of white matter. 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. Blindness and optic atrophy have also been seen following exposure to hexachlorophene.

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

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.

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. 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. 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. 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. 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.
References


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.


CHAPTER 20
Disinfectants


