

HIGHLIGHTS

- Compounds are registered for medical or medicinal use rather than as pesticides
- Several are among the most frequently reported human poisonings in the U.S.
- Iodine is well absorbed through abraded or burned skin

Signs and Symptoms:

- Highly variable based on agent
- Many are irritants and corrosives
- Iodine causes neurological symptoms, shock, renal failure, and hyperkalemia
- Pine oil can cause aspiration pneumonia

Treatment:

Follow general principles of decontamination and supportive care

Contraindicated:

- Gastric emptying and decontamination procedures are contraindicated in poisonings due to corrosive agents and pine oil

Disinfectants

A wide variety of disinfectant agents are used to destroy microorganisms and they differ greatly in their toxic effects. Most disinfectants can conveniently be grouped into a few categories, some of which are also 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 severely 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 of Isopropyl Alcohol

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 with acute exposures. 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 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: Isopropyl Alcohol

1. Gastrointestinal decontamination. Since the onset of coma is often rapid with this poisoning, induced emesis is contraindicated, though spontaneous vomiting often occurs. If the patient has ingested a large amount, has not vomited, and is seen within one hour of exposure, consideration should be given to gastric emptying by lavage as outlined in Chapter 2.

Isopropyl alcohol is well adsorbed to charcoal, so activated charcoal should probably be administered, as outlined in Chapter 2.

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

3. If hypoglycemia occurs, glucose administration is indicated in order to maintain normoglycemia.

4. Hemodialysis has been reported to be beneficial in patients with severe poisoning 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 probably 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.^{6,7,8} 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. Due to the irritant effects of glutaraldehyde, the wearing of personal protective equipment is required for the protection of skin (29 CFR 1910.132), and eyes (29 CFR 1910.133). OSHA standards require the use of appropriate respirators by employees that may be exposed to glutaraldehyde during routine or emergency work procedures (29 CFR 1910.134).

Commercial Products

ALCOHOLS

Isopropyl alcohol

ALDEHYDES

formaldehyde

glutaraldehyde

CATIONIC DETERGENTS

benzalkonium chloride

cetrimide

cetylpyridium chloride

CHLORHEXIDINE

Hibiclens

Hibistat

Peridex

HYPOCHLORITES

calcium hypochlorite

sodium hypochlorite

IODINES

povidone-iodine

Betadine

Ioprep

Pharmadine

MERCURIALS

mercurobutol

mercurochrome

merthiolate

nitromersol

phenylmercuric acetate

phenylmercuric nitrate

thimerosal

PHENOLS

2-benzyl-4-chlorophenol

cresol

Lysol

hexachlorophene

Bilevon

Dermaadex

Exofene

Gamophen

Phisohex

Surgi-Cen

Surofene

Texosan

o-phenylphenol

phenol

4-tert-amyphenol

thymol

triclosan

PINE OIL

Treatment: Glutaraldehyde

1. Gastrointestinal decontamination. If a large amount has been ingested and retained, and the patient is seen within one hour of exposure, consider gastric emptying as described in Chapter 2. Administration of activated charcoal should be considered, as described in Chapter 2.

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

3. Skin decontamination. If skin irritation is noted, vigorous skin decontamination is indicated. However, systemic toxicity from skin exposure appears unlikely.

CATIONIC DETERGENTS

Several cationic detergents are used as disinfectants. All share the capacity, in sufficient concentration, to behave as caustic agents, capable of causing rather severe, caustic burns. It appears that concentrations greater than approximately 7.5% are 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.

Though there are no cetrimide preparations available in the U.S., 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, these agents have been reported to cause eye discomfort as well as skin rashes and irritation. 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.^{9,10} Vomiting, diarrhea, and abdominal pain have been reported.¹¹ Necrosis of the gut, with peritonitis, has also been reported.¹² In severe exposures, there are also reports of CNS depression, liver injury, and pulmonary edema.^{9,11}

Treatment

1. Skin decontamination. If a high-concentration solution has been applied to skin, aggressive skin contamination and treatment of burns is appropriate. If

a high concentration solution is in contact with the eyes, profuse washing of the eyes is indicated followed by a careful exam of the corneas. If burns have occurred, appropriate ophthalmologic care should be provided.

2. Gastrointestinal decontamination. Gastric emptying and other methods of gastrointestinal decontamination are **contraindicated** in these poisonings. Some experts recommend cautious dilution with small amounts of milk or water.^{9,13} Acidic solutions such as juices should never be offered for dilution.

3. 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 from the procedure.¹² A competent surgeon or gastroenterologist should provide subsequent care.

4. Other agents. Although corticosteroids are commonly used to treat these burns, their use remains controversial. Use of other agents, such as H₂ antagonists and sulcralfate, has been reported but remains controversial at this time.

5. CNS, pulmonary and other systemic effects should be treated symptomatically, consistent with sound medical practice.

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. If a low concentration solution is ingested or applied to the skin, mild local irritation can be seen. Contact dermatitis, urticaria, and anaphylaxis have followed repeated skin exposures to this agent.^{14,15} 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.¹⁶ Esophageal burns have been reported in a single case after ingestion of a large quantity of a 20% solution of this agent.¹⁷ Ulcerative colitis has been described after an enema of the 4%

solution mixed with tap water (10 mL in 2 liters water).¹⁸ Liver toxicity can occur with large exposures.¹⁷

Treatment

1. Gastrointestinal decontamination. If ingestion of a large quantity has occurred within an hour and the patient has not vomited, gastrointestinal decontamination as described in Chapter 2 should be considered. If a highly concentrated solution has been ingested, manage as a caustic ingestion as described in the cationic detergents, without gastrointestinal decontamination.

2. Liver injury panel should be performed with large ingestions.

3. Eye decontamination. If eye exposure has occurred, the eyes should be vigorously irrigated and a careful ophthalmologic exam should be performed for corneal injury. If an injury has occurred, an ophthalmologic consultation should be obtained.

HYPOCHLORITES

Hypochlorites are implicated in a large proportion of the disinfectant poisonings reported to poison control centers in the United States. Most are solutions of sodium or calcium hypochlorite solutions. Chloramine, a disinfectant used by 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 the eyes,¹⁹ and mucous membrane burns have been reported.²⁰ Significant poisonings are very infrequent with these agents in solution.²¹

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.²² However, in cases of prolonged exposure or exposure to high concentrations, there is the potential for severe toxic pneumonitis.²³ While severe injury may be the exception to the rule, great efforts should be made to discourage mixing of these materials with acid or ammonia.

Treatment

1. Gastric decontamination. After oral exposures, gastric emptying is not indicated. If a granular material is ingested, and the patient has symptomatic mucosal burns, referral to a surgeon or gastroenterologist for consideration of endoscopy and management may be appropriate.

2. Dilution with water or milk not to exceed approximately 15 mL/kg in a child or 120-240 mL in an adult is probably appropriate if vomiting has not occurred. Administration of acids is contraindicated, due to the risk of increasing generation of chlorine gas.

3. Eye decontamination. If eyes were exposed, they should be irrigated profusely with water or saline. If corneal burns are detected, referral to an ophthalmologist is appropriate.

4. Skin decontamination. Skin exposure should also be managed by copious water dilutions. See Chapter 2.

5. Fresh air. If exposure to vapors or chlorine or chloramine gas has occurred, patient should immediately be moved to fresh air. If symptoms occur or persist, oxygenation should be assessed and oxygen should be administered as needed. If persistent symptoms occur, a chest film should be obtained and hospital care considered. Intensive care may need to be provided in severe inhalations.

IODINE

The most common iodine-containing disinfectant is povidone-iodine (providone), in 7.5-10% solutions. 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 $\mu\text{g}/\text{dL}$, 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 of Povidone-Iodine

This compound is very poorly absorbed from the gastrointestinal tract, due to the rapid conversion of free iodine to iodide in the stomach. Although highly concentrated iodine solutions or iodine salts are corrosive to the gastrointestinal tract,²⁴ solutions of povidone-iodine have little caustic potential. Likewise, the compound is 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.²⁵⁻²⁸ 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.²⁴

In povidone-iodine exposures by these routes, the primary symptoms initially appear to be neurological, with headache, dizziness, delirium, hallucina-

tions, and seizures.²⁶ Hypotension, arrhythmias, cyanosis, metabolic acidosis, shock, and acute renal failure occur in severe cases.^{25,27,28} Hepatic injury, manifested by elevated serum transaminase levels, has also been reported with very high level exposures.²⁷ Hyperkalemia has occurred, and the serum chloride may be falsely elevated due to the presence of a second halide.²⁵

Treatment: Povidone-Iodine

- 1. Skin decontamination.** Remove skin contamination by vigorous washing with soap and water. See Chapter 2.
- 2. Gastrointestinal decontamination.** If the patient is seen soon after a very large ingestion, and vomiting has not occurred, consider gastrointestinal decontamination, as outlined in Chapter 2. Consider single dose charcoal.
- 3. Iodine clearance** is apparently enhanced by procedures that enhance chloride excretion. Therefore, osmotic or choluretic diuresis is probably indicated in these poisonings, if symptomatic.
- 4. Seizures.** Treat seizures with anticonvulsants, as outlined in Chapter 2.
- 5. Monitor thyroid** function following recovery to confirm euthyroid state.

MERCURIALS

A wide variety of organic mercurials have been used as disinfectants and as preservatives. Nearly all uses have been banned in the United States. The toxicity and treatment of exposure to these compounds is described in detail in Chapter 15, Fungicides, under organomercury compounds and will not be repeated here.

PHENOLS

Several phenols are used as disinfectants. Cresol and thymol are alkyl derivatives of phenol, while hexachlorophene and triclosan are chlorinated phenols. Common commercial preparations are Lysol[®], a 50% solution of mixed cresols in soap, and hexachlorophene, marketed under several trade names in soap bars and some cosmetics. Cresols and hexachlorophene are discussed individually as examples of these compounds that are familiar and for which there are some human data.

Toxicology of Cresols

Cresols, in common with phenol and other phenolic compounds, are highly corrosive to all surfaces. With ingestion of concentrated forms they cause severe corrosive injury to the mouth and upper gastrointestinal tract. Likewise, severe eye and skin caustic injuries can occur with cresol exposure.²⁹ Symptoms usually include nausea, vomiting, and diarrhea. Hypotension, myocardial failure, pulmonary edema, and neurological changes may also occur.³⁰ Liver and renal toxicity, methemoglobinemia, and hemolysis have all been reported.^{30,31} 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: Cresols

1. Gastrointestinal decontamination. Due to the corrosive nature of these compounds, gastrointestinal decontamination should not be attempted. Consideration of dilution with milk or water is appropriate if vomiting has not occurred.

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

3. Skin decontamination. If skin or eye contamination has occurred, copious irrigation should be performed. This should be followed by a careful eye examination for corneal burns. If corneal burns are noted, ophthalmologic consultation should be obtained.

4. Respiratory and circulatory support should be provided 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 orally and dermally. Dermal exposures have led to severe toxicity and death in neonates, due to application to damaged skin, or repeated or high-concentration skin exposures.³² Hexachlorophene should never be used as a disinfectant on open wounds or abraded or inflamed skin surfaces. In distinction to other phenolic compounds, this agent is not significantly caustic and exposure does not result in the severe caustic injuries seen with other phenolic chemicals.

Hexachlorophene is a potent neurotoxicant. It causes brain edema and spongy degeneration of white matter.³³ 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.^{32,34} Blindness and optic atrophy have been reported following exposure to hexachlorophene.³⁵

In addition to the neurological effects, common early symptoms of poisoning are vomiting, diarrhea, and anorexia.³⁴ These findings have been accompanied in animals by significant hepatotoxicity.³⁶ With skin exposure, an erythematous desquamative rash is often noted at the site of exposure.³⁴ With chronic exposure, contact dermatitis may be noted. In severe poisonings, cardiovascular symptoms, including hypotension and bradycardia, have been noted.³⁷ In a single case, repeated exposure to this compound led to asthma in a pediatric nurse.³⁸

Treatment: Hexachlorophene

1. Gastrointestinal decontamination. Since this agent is not generally caustic, consideration should be given to aggressive gastrointestinal decontamination. If the patient has ingested a significant amount and is seen within one hour of exposure, gastric emptying is likely to be useful, as described in Chapter 2.

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

2. Other therapies. Though this compound is quite toxic systemically and enhanced clearance methods would appear beneficial, there is no evidence to support the efficacy of hemodialysis, peritoneal dialysis, hemoperfusion, or exchange transfusion.³⁷

3. Skin decontamination. If exposure has occurred through the skin, aggressive washing of skin with soap or detergent and water is probably beneficial, to remove any residues still on the skin. Since hexachlorophene is not soluble in water, water washing alone will provide no significant benefit. See Chapter 2.

4. Neurological support and control of seizures is critical to survival and should be performed, when possible, in an intensive care setting. Seizure control should be in accordance with recommendations in Chapter 2.

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

PINE OIL

Pine oil detergent and disinfectant solutions are among the most common poisonings reported to poison control centers in the U.S. A relatively high number of these are reported as serious or fatal. Pine oil is found 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.³⁹ 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.⁴⁰ 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 lung (by aspiration), some reports suggest significant absorption from oral and rectal exposures. Other reports suggest a lesser rate of absorption.³⁹ While alpha-terpineol can be measured in blood, there are no data relating levels to degree of toxicity. Consequently, this measure is not considered useful in guiding diagnosis and management.

Treatment

1. Gastrointestinal decontamination. Since there is a high risk of aspiration pneumonia, induced emesis is usually considered contraindicated in these poisonings. However, spontaneous emesis may occur due to direct irritation of the gastric mucosa.

If the patient is seen within an hour of ingestion and a large amount has been ingested, gastric emptying by intubation and lavage may be considered, as described in Chapter 2. However, some studies have suggested greater rates of complications with lavage than with ipecac-induced emesis.⁴⁰

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

2. Eye decontamination. If eye exposure has occurred, copious irrigation of the eyes is appropriate.

3. Pulmonary symptoms. The patient should be observed for at least six hours with any significant ingestion in order to observe the onset of any symptoms, particularly pulmonary symptoms. If any pulmonary symptoms are observed, the patient should have a chest film and measurement of oxygenation,

and hospitalization is appropriate. With severe pulmonary symptoms, transfer to an intensive care unit is usually appropriate. With severe aspiration, management should be handled as in any severe aspiration pneumonia, in accordance with accepted medical practice. Other severe systemic effects should be treated in accordance with accepted medical practice.

References

1. Lacouture PG, Wason S, Abrams A, et al. Acute isopropyl alcohol intoxication: Diagnosis and management. *Am J Med* 1983;75:680-6.
2. Rich J, Scheife RT, Katz N, et al. Isopropyl alcohol intoxication. *Arch Neurol* 1990;47:322-4.
3. Vivier PM, Lewander WJ, Martin HF, et al. Isopropyl alcohol intoxication in a neonate through chronic dermal exposure: A complication of a culturally-based umbilical care practice. *Pediatr Emerg Care* 1994;10:91-3.
4. Manring E, Meggs W, Pape G, et al. Toxicity of an intravenous infusion of isopropyl alcohol. *J Toxicol Clin Toxicol* 1997;35:503.
5. Norback D. Skin and respiratory symptoms from exposure to alkaline glutaraldehyde in medical services. *Scand J Work Environ Health* 1988;14:366-71.
6. Corrado OJ, Osman J, and Davies RJ. Asthma and rhinitis after exposure to glutaraldehyde in endoscopy units. *Hum Toxicol* 1986;5:325-8.
7. Chan-Yeung M, McMurren T, Catonio-Begley F, et al. Occupational asthma in a technologist exposed to glutaraldehyde. *J Allergy Clin Immunol* 1993; 91(5):974-8.
8. Stenton SC, Beach JR, Dennis JH, et al. Glutaraldehyde, asthma, and work – a cautionary tale. *Occup Med* 1994;44(2):95-8.
9. Mucklow ES. Accidental feeding of a dilute antiseptic solution (chlorhexidine 0.05% with cetrimide 1%) to five babies. *Hum Toxicol* 1988;7:567-9.
10. Wilson JT and Burr IM. Benzaldehyde poisoning in infant twins. *AJDC* 1975;129:1208-9.
11. Chan TY. Poisoning due to savlon (cetrimide) liquid. *Hum Exp Toxicol* 1994;13:681-2.
12. Zargar SA, Kochhar R, Mehta S, et al. The role of fiberoptic endoscopy in the management of corrosive ingestion and modified endoscopic classification of burns. *Gastrointest Endosc* 1991;37:165-9.
13. Consensus: POISINDEX[®] Editorial Board consensus opinion poll, Irritants/Caustics Specialty Board. Englewood, CO: Micromedex, 1988.
14. Wong WK, Goh CL, and Chan KW. Contact urticaria from chlorhexidine. *Contact Dermatitis* 1990;22:52.
15. Okano M, Nomura M, Hata S, et al. Anaphylactic symptoms due to chlorhexidine gluconate. *Arch Dermatol* 1989;125:50-2.
16. Tabor E, Bostwick DC, and Evans CC. Corneal damage due to eye contact with chlorhexidine gluconate. *JAMA* 1989;261:557-8.
17. Massano G, Ciocatto E, Rosabianca C, et al. Striking aminotransferase rise after chlorhexidine self-poisoning. *Lancet* 1982;1:289.
18. Hardin RD and Tedesco FJ. Colitis after hibiclens enema. *J Clin Gastroenterol* 1986;8:572-5.
19. Ingram TA. Response of the human eye to accidental exposure to sodium hypochlorite. *J Endod* 1990;16:235-8.

20. French RJ, Tabb HJ, and Rutledge LJ. Esophageal stenosis produced by ingestion of bleach. *South Med J* 1970;63:1140-4.
21. Landau GD and Saunders WH. The effect of chlorine bleach on the esophagus. *Arch Otolaryngol* 1964;80:174-6.
22. Mrvos R, Dean BS, Krenzelok EP, et al. Home exposure to chlorine/chloramine gas: review of 216 cases. *South Med J* 1993;86:654-7.
23. Reisz GR and Gammon RS. Toxic pneumonitis from mixing household cleaners. *Chest* 1986;89:49-52.
24. Kurt TL, Hnilica V, Bost R, et al. Fatal iatrogenic iodine toxicity in a 9-week old infant. *Vet Hum Toxicol* 1992;34:333.
25. Means LJ, Rescorla FJ, and Grosfield JL. Iodine toxicity: An unusual cause of cardiovascular collapse during anesthesia in an infant with Hirschsprung's Disease. *J Pediatr Surg* 1990;25:1278-9.
26. Ponn RB. Continuous povidone-iodine irrigation (letter). *Ann Thorac Surg* 1987;43:239.
27. Pietsch J and Meakins JL. Complications of povidone-iodine absorption in topically treated burn patients. *Lancet* 1976;7:280-2.
28. Campistol JM, Cipiano A, Nogué S, and Bertrán A. Acute renal failure in a patient treated by continuous povidone-iodine mediastinum irrigation. *J Pediatr Surg* 1988;29:410-2.
29. Pegg SP and Campbell DC. Children's burning due to cresol. *Burns* 1985;11:294-6.
30. Arthus GJ, Wise CC, and Coles GA. Poisoning by cresol. *Anaesthesia* 1977;32:642-3.
31. Chan TK, Mak LW, and Ng RP. Methemoglobinemia, heme bodies, and acute massive intravascular hemolysis in lysol poisoning. *Blood* 1971;38:739-44.
32. Mullick FG. Hexachlorophene toxicity – Human experience at the Armed Forces Institute of Pathology. *Pediatrics* 1973;51(2)II:395-9.
33. Anderson JM, Cockburn F, Forfar J, et al. Neonatal spongiiform myelinopathy after restricted application of hexachlorophane skin disinfectant. *J Clin Pathol* 1981;34:25-9.
34. Martin-Bouyer G, Lebreton R, Toga M, et al. Outbreak of accidental hexachlorophene poisoning in France. *Lancet* 1982;1:91-5.
35. Slamovitz TL, Burde RM, and Klingele TG. Bilateral optic atrophy caused by chronic oral ingestion and topical application of hexachlorophene. *Am J Ophthalmol* 1980;89:676-9.
36. Prasad GV, Rajendra W, and Indira K. Brain ammonia metabolism in hexachlorophene-induced encephalopathy. *Bull Environ Contam Toxicol* 1987;38:561-4.
37. Boehm RM and Czajka PA. Hexachlorophene poisoning and the ineffectiveness of peritoneal dialysis. *J Toxicol Clin Toxicol* 1979;14(3):257-62.
38. Nagy L and Orosz M. Occupational asthma due to hexachlorophene. *Thorax* 1984;39:630-1.
39. Koppel C, Tenczer J, Tennesmann U, et al. Acute poisoning with pine oil - Metabolism of monoterpenes. *Arch Toxicol* 1981;49:73-8.
40. Litovitz TL, Schmidz BF, Matyunas N, et al. 1987 Annual Report of the American Association of Poison Control Centers National Data Collection System. *Am J Emerg Med* 1988;6:479-515.