Camphor and Mothballs
• Camphor has been used medicinally for centuries, most commonly as a rubefacient.

• Accidental ingestion of 20 %camphorated oil, mistaken for castor oil, has caused numerous cases of toxicity.

• Although camphorated oil is no longer available and the efficacy of camphor itself is not widely accepted, camphor remains a common ingredient in many over-the-counter preparations.
• **20 grams** of Vicks VapoRub, **10 mL** of Campho-Phenique, and **16 mL** of Vicks VapoSteam each contain about **1 g** of camphor
• Clinically significant camphor toxicity has not been reported with ingestions of less than 30 mg/kg.

• Uncommon when less than 50 mg/kg of camphor is ingested.

• The human lethal dose of camphor is reported to range from 50 to 500 mg/kg.

• In adults, 10 mL of 20% camphorated oil (2 g) has been reported to cause symptoms,

• In 19-month-old child 5 mL (1 g) resulted in the death
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Ayurvedic Health Benefits of Camphor Oil

- Relieves severe pain, spasms and inflammation.
- Combats microbes and skin infections.
- Alleviates cold and other respiratory problems.
- Supports digestive functions.
- Pacifies the nervous system.
Toxicokinetics/Pathophysiology

• Camphor is a cyclic terpene with a characteristic, penetrating odor and a pungent, aromatic taste.

\[(1S,4S)-1,7,7\text{-trimethyl}bicyclo[2.2.1]heptan-2\text{-one}\]
\[\text{Camphor}\]

• Camphor is rapidly absorbed from the mucous membranes and the gastrointestinal tract.

• Its lipophilic nature is responsible for central nervous system (CNS) toxicity, which is believed to involve excitatory mechanisms.
• It is also absorbed through inhalation, through dermal application, and by nasal instillation.

• It has been abused as an abortifacient orally and by intrauterine injection and readily crosses the placenta.

• Pulmonary elimination of camphor causes a distinctive odor on the breath that is helpful in diagnosis.
Clinical Presentation

- Symptoms of camphor toxicity usually begin 5 to 90 minutes after ingestion and are often abrupt in onset.

- Spontaneous emesis, with the odor of camphor readily apparent, typically occurs first.

- **CNS stimulation** ensues with restlessness, confusion, delirium, and increased muscular activity.

- **Severe toxicity** may include seizures, apnea, and coma.

- Death results from respiratory depression or status epilepticus
Clinical Presentation

- Chronic ingestion of camphor can cause a variety of symptoms clinically similar to Reye’s syndrome.

- **Gastrointestinal symptoms** may include nausea, vomiting, epigastric pain, and hepatic enzymes elevation.

- Pathologic hepatic changes often include such findings as granulomatous hepatitis and fatty metamorphosis.

- With chronic dermal exposure, systemic effects and contact dermatitis can occur as well as significant allergic responses.

- **Ocular exposure** results primarily in irritation only, although oral intake has been associated with visual problems.
Treatment

• **Activated charcoal** should be administered for gastrointestinal decontamination.

• Due to prominent CNS effects, the induction of emesis is contraindicated.

• If liquid camphor is ingested, a nasogastric tube can be used to aspirate gastric contents before instillation of activated charcoal.

• **Alcohols and oil solutions should be avoided** because they have been reported to enhance absorption of camphor.
Treatment

• **Benzodiazepines such as lorazepam or diazepam** are indicated for symptoms of CNS hyperactivity, such as agitation, tremors, and seizures.

• **Phenobarbital** can be used for recurrent or prolonged seizures.

• **Thorough flushing** of exposed eyes and skin is usually sufficient treatment to relieve transient irritation produced by camphor.
MOTHBALLS

- Naphthalene and paradichlorobenzene are sole ingredients in moth repellents in the form of balls, flakes, or crystals.
- Naphthalene can produce serious hematologic and CNS effects, necessitating treatment,
MOTHBALLS

• Differentiating between these two white, crystalline solid compounds is important.

• Differentiation is possible through a variety of methods.
  1. Naphthalene feels dry to the touch; paradichlorobenzene feels moist and oily.
  2. A naphthalene will float, whereas the paradichlorobenzene-containing mothball will sink in a saturated salt solution (3 heaping tablespoonfuls of salt and 4 ounces of tepid water stirred vigorously until the salt will not dissolve further.)
MOTHBALLS

3-On an abdominal radiograph, paradichlorobenzene is densely radiopaque whereas naphthalene-containing mothballs are radiolucent or faintly radiopaque.

4-Odors are characteristic and can be compared with known samples.
Naphthalene

- Naphthalene is well absorbed after oral, dermal, and inhalational exposure. It is soluble in fats and oils, and the administration of milk or food after ingestion may aid absorption.

- Hepatic metabolism generates α-naphthol, β-naphthol, α-naphtholquinone, and β-naphtholquinone metabolites.

- The α-naphthol oxidative metabolite is responsible for the severe hemolytic activity, acute tubular necrosis, and methemoglobinemia.

- Less than one mothball containing 200 to 500 mg of naphthalene can cause hemolysis in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency.
- Infants due to inadequate conjugation of toxic metabolites.
Clinical Presentation

- Nausea, vomiting, abdominal pain, diarrhea, and fever may develop as early as 1 day after exposure.

- Development of clinically recognized hemolysis may be delayed 1 to 5 days and is typically preceded by gastrointestinal symptoms.

- Signs of hemolysis, including pallor, tachypnea, weakness, jaundice, and dark urine, may also be present or evolve over the next several days.
Clinical Presentation

- **Cyanosis** may indicate methemoglobinemia.

- **Lethargy and seizures** are other possible effects.

- Hemoglobin frequently is decreased with an increase in red cell fragmentation.

- **Anisocytosis and poikilocytosis** with Heinz body formation may occur in severe cases.

- **Chronic exposure** has resulted in nausea, vomiting, fever, jaundice, lethargy, aplastic anemia, and hepatic centrilobular necrosis.
Treatment

• Treatment of naphthalene toxicity is primarily supportive.

• Treatment should be initiated for ingestions of one half or more of a naphthalene-containing mothball.

• In early therapy, ipecac syrup-induced emesis, in the first 2 hours after ingestion, is preferred to lavage, owing to the size of the mothballs.

• activated charcoal should also be administered.

• Milk or fatty meals that increase absorption should be avoided for several hours.

• Transfusion may be indicated in cases of severe hemolysis.

• Intravenous fluids to maintain urinary flow may prevent renal deposition of cell breakdown products.

• oxygen and methylene blue to patients symptomatic from methemoglobinemia
Treatment

• Exposed skin should be thoroughly washed with soap and water.

• Naphthalene is not easily removed from clothing, and contaminated clothing should be discarded to prevent further exposure.
Paradichlorobenzene

- The toxicity of mothballs containing paradichlorobenzene is low, with ingestion causing possible nausea and vomiting only.

- After chronic exposure, hepatotoxicity, anemia, and pulmonary granulomatosis have been reported.

- **Emesis is indicated when large amounts have been ingested.**
  - **Activated charcoal** can also be administered.

- **Milk and fatty foods** should be withheld for several hours after ingestion to minimize absorption.

- **If the composition of a mothball is unknown**, gastric decontamination and treatment should proceed as outlined for naphthalene.
PARADICHLOROBENZENE
Antiseptics, Disinfectants, and Sterilizing Agents
Iodophors, Iodine, and Iodide

1. iodide (I⁻),
2. free iodine (I₂), and
3. iodine bound to povidone, a carrier molecule that limits the availability of free iodine.
The iodophors are used as
✓ antiseptics and irrigants.
✓ Iodine-containing solutions are also used as expectorants
✓ as vaginal irrigants
✓ for contrast studies in radiology
✓ as therapy for acute hyperthyroidism. Lugol’s iodine solution, commonly used in the acute treatment of hyperthyroidism, contains 5 % I₂ and 10 % potassium iodide (I⁻).

• The antibacterial property of iodide is facilitated by a covalent linkage to bacterial cell surface glycoproteins, which enhances the bactericidal properties of polymorphonucleocytes
• The mechanism of iodine cellular toxicity has been attributed to interference with cellular oxidative metabolism and ATP production.

• Chronic exposure results in disturbances of thyroid structure and function and may be manifest as hyperthyroidism, hypothyroidism, goiter, or thyroiditis.

• These paradoxical responses are due to the stimulation of increased thyroid hormone production in some individuals (Jod basedow reaction) and the blockade of thyroid hormone production in others (Wolff-Chaikoff effect).

• Iodine toxicity occurs through ingestion, through absorption during wound irrigation with iodophor-containing solutions, through mucosal exposure, and during topical application to dermal burns.
Clinical Presentation

- Acute ingestion of iodine (I$_2$)-containing solutions results in gastrointestinal irritation, gastrointestinal ulceration, and chemical pneumonitis.

- These effects are concentration dependent, so tincture of iodine or Lugol’s iodine solution results in far more tissue injury than does ingestion of povidone-iodine.

- Any acute exposure may lead to acute tubular necrosis, hemolytic anemia, and hyperthyroidism.

- Early toxic symptoms are related to stimulation of increased secretions, leading to rhinorrhea, conjunctivitis, and cough productive of serous fluid.
Clinical Presentation

- Acute respiratory distress
- severe lactic acidosis
- renal dysfunction
- cardiogenic shock, and death occur within hours.

- Anaphylactic and anaphylactoid reactions also occur

- Chronic ingestion of iodine leads to thyroid disorders in some individuals.

- Large or chronic exposure to iodides results in sensitivity reactions manifest as painful salivary gland enlargement and a variety of dermal eruptions, so-called ioderma.
Treatment

- Good **supportive care** is critical in the management of all cases and is all that is required in most cases of povidone-iodine ingestion.

- After ingestion of significant amounts, **gastric decontamination** may be done by aspiration with a small nasogastric tube.

- Iodine may be converted to less toxic iodide by the administration of starch or milk.

- Sodium thiosulfate will also convert iodine to iodide.

- **Activated charcoal** is indicated unless significant symptoms suggest the need for endoscopic evaluation of caustic injury.

- Iodine is **removed by hemodialysis**.
Boric Acid

• Boric acid had significant medical uses in the past as a **topical antiseptic and irrigant**.

• At one time it was **mixed with honey and glycerine** and sold to treat topical mucosal sores in children.

• Because of its weak antiseptic properties and significant toxicity it is **no longer used** in medicinal preparations.

• A common cause of exposure today is related to **accidental ingestion** of powdered boric acid used to kill cockroaches.

• Suicidal ingestions of large amounts (280 g in one case) may be fatal.
Pathophysiology

• Boric acid is easily absorbed across
  1. mucosal surfaces
  2. abraded skin, and I
  3. gastrointestinal tract.

• It is eliminated unchanged in the kidneys with a half-life of 21 hours in patients with normal renal function.

• Excretion is markedly prolonged in seriously poisoned patients.
Clinical Presentation

• The characteristic presentation of boric acid poisoning is the development of blue-green vomiting and diarrhea

• followed by intense erythroderma that has a boiled lobster appearance.

• This progresses to desquamation and erosion of mucosal surfaces.

• Renal, bone marrow, and hepatic injuries occur.

• CNS symptoms include seizures, delirium, and coma.
Treatment

- Treatment in most cases is supportive.
- Activated charcoal does not absorb boric acid effectively.
- Ipecac-induced emesis for 200 to 400 mg/kg,
- Lavage and emergency department evaluation for more than 400 mg/kg. Lavage was recommended for a 12-g ingestion in larger patients.
- Hemodialysis may be indicated for severely poisoned patients.
- Exchange transfusions have been used in severely poisoned neonates.
Pathophysiology

- Formaldehyde is a protoplasmic poison, precipitating proteins and causing coagulation necrosis.

- It promotes allergic reactions by binding to and altering tissue proteins.

- Repeated exposure is believed to result in allergic contact dermatitis (type IV hypersensitivity reaction).

- Type I hypersensitivity reactions are thought to precipitate the bronchospastic disease associated with formaldehyde.

- Formaldehyde is readily absorbed from the gut and lungs and rapidly metabolized through formate to carbon dioxide and water by the liver and erythrocytes.

- Formaldehyde is a naturally occurring product of tetrafolate reactions with endogenous tissue levels of 3 to 12 ng/g.
Clinical Presentation

• **Inhalational Exposure**

  • Formaldehyde has an easily detectable odor at 0.5 ppm frankly irritating to upper airways and mucous membranes at 1 ppm.

  • These unpleasant attributes serve as an early warning and tend to limit inhalational exposure.

  • Severe respiratory irritation develops at higher concentrations, producing dyspnea at 10 ppm. Inhalation of 100 ppm may be life threatening.

  • Formaldehyde is believed to be a potent sensitizer in immune-mediated bronchospasm.

  • Chronic low-level exposure to up to 1.6 ppm produced fatigue, headache, and mucous membrane irritation but did not produce significant changes in pulmonary function tests.

  • The incidence of dermal sensitization may be as high as 5 per cent in the general population.
• Chronic off-gassing of formaldehyde from urea-formaldehyde foam insulation (UFFI) has been suggested as the cause of a myriad of complaints such as headache, nausea, upper airway irritation, reactive airway disease, dermatitis, and insomnia.
Oral Exposure

- Ingestion of formaldehyde results in gastrointestinal injury with nausea, vomiting, diarrhea, abdominal pain, and lethargy.
- Serious ingestions may be complicated by hemorrhage, shock, coagulation necrosis of stomach and distal esophagus, severe metabolic acidosis, and death.
- Multiple factors contribute to the acidosis, including the metabolism of formaldehyde to formate, increased tissue lactate, and concomitant methanol poisoning.
- Methanol levels were elevated for over 13 hours in a 58-year-old man who ingested 4 ounces of formalin. This prolonged course was attributed to delayed absorption due to “fixation” of the gastric mucosa.
- Perforation and strictures of the gastrointestinal tract may limit recovery.
- Inadvertent parenteral exposure to 2 per cent formalin from incompletely purged hemodialysis machines has resulted in intravascular hemolysis.
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