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# IODIDES

## MICROMEDEX® POISINDEX® MANAGEMENT

POISINDEX Managements provide information on the evaluation, clinical effects, range of toxicity, and treatment protocols for exposures to drugs, chemicals and physical/environmental agents.

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## POISINDEX® Managements

# IODIDES

### 0.0 OVERVIEW

LIFE SUPPORT

CLINICAL EFFECTS

LABORATORY/MONITORING

TREATMENT OVERVIEW

RANGE OF TOXICITY

### 0.1 LIFE SUPPORT

**A)** This overview assumes that basic life support measures have been instituted.

### 0.2 CLINICAL EFFECTS

**0.2.1** SUMMARY OF EXPOSURE

**A)** Acute reactions which may occur following ingestion include hypersensitivity reactions which may present as angioedema, arthralgia, eosinophilia, lymphadenitis, or urticaria.

**B)** Chronic ingestion of small or excessive amounts may result in iodism which is characterized by salivation, coryza, sneezing, conjunctivitis, headache, fever, laryngitis, bronchitis, stomatitis, and various skin rashes.

**C)** Unlike iodine, the iodide salts are not caustic and no human fatalities are reported due to acute overdosage. Up to 10 grams of sodium iodide has been administered IV without signs or symptoms of toxicity. Chronic ingestion of small or excessive amounts may result in iodism.

**0.2.4** HEENT

**A)** A metallic taste, increased salivary and bronchial secretions may be noted.

**0.2.5** CARDIOVASCULAR

**A)** Periarteritis nodosa has been associated with iodide use.

**0.2.8** GASTROINTESTINAL

**A)** Vomiting and abdominal pain may be noted, but do not result in caustic effects on the gastrointestinal tract. Parotitis has also been reported.

**0.2.14** DERMATOLOGIC

**A)** Chronic oral administration can produce various cutaneous manifestations, including erythema nodosum, polymorphic eruptions, urticaria, vasculitis, and petechia.

**0.2.16** ENDOCRINE

**A)** Chronic iodide therapy has produced goiters, hypothyroidism and rarely hyperthyroidism.

**0.2.19** IMMUNOLOGIC

**A)** Acute hypersensitivity reactions including angioedema, Stevens Johnson syndrome, systemic vasculitis, and serum-sickness-like reactions may occur.

**0.2.20** REPRODUCTIVE

**A)** Cretinism and goiter have been reported in children born to mothers chronically taking **iodides** during pregnancy.

### 0.3 LABORATORY/MONITORING

**A)** Plasma iodide levels are not clinically useful.

**B)** Protein bound iodine (PBI) will be elevated for up to 3 weeks after ingestion of pharmacologic doses of inorganic **iodides**.

**C)** Iodide-induced hypothyroidism is accompanied by decreased serum T4 levels and usually increased serum TSH levels.

### 0.4 TREATMENT OVERVIEW

**0.4.2** ORAL/PARENTERAL EXPOSURE

**A)** ACTIVATED CHARCOAL: Administer charcoal as a slurry (240 mL water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents, 25 to 50 g in children (1 to 12 years), and 1 g/kg in infants less than 1 year old.

**B)** ACUTE ALLERGIC REACTION - Administer epinephrine (ADULT and CHILD: 0.01 mL/kg/dose of a 1:1000 solution to a maximum of 0.5 mL SC or IM; repeat every 15 minutes if needed).

**1)** Antihistamines (Diphenhydramine 25 to 50 mg/dose IV or IM or orally) may be administered depending on severity.

**C)** SERUM-SICKNESS-LIKE REACTIONS should be managed with antihistamines (Diphenhydramine 25 to 50 mg/IV or IM or orally may be administered until symptoms remit).

**D)** DIURESIS with IV fluids and mannitol may be useful in the management of chronic iodide poisoning to enhance the renal excretion of iodide or after exposure to methyliodide.

### 0.5 RANGE OF TOXICITY

**A)** Toxicity following acute ingestion is uncommon, however, hypersensitivity reactions may occur and are potentially life-threatening (angioedema and laryngeal edema).

**B)** Up to 10 grams of sodium iodide has been administered IV without toxicity.

**C)** Chronic iodide poisoning (iodism)occurs more commonly.

## 1.0 SUBSTANCES INCLUDED/SYNONYMS

THERAPEUTIC/TOXIC CLASS

SPECIFIC SUBSTANCES

AVAILABLE FORMS/SOURCES

### 1.1 THERAPEUTIC/TOXIC CLASS

**A) Iodides** have been used as expectorants for cystic fibrosis and asthma, as well as, in the management of various thyroid disease (Dolan & Gibson, 1971).

**B)** An iodide is the drug of choice for the lymphocutaneous form of Sporothrix schenckii infections (Anon, 1988).

**C)** Methyl iodide is used as a methylating agent in pharmaceutical and chemical synthesis, in microscopy, as a reagent, as a catalyst in production of organic lead compounds, as an etching agent, as a component in fire extinguishers, and formerly as a soil fumigant (IARC, 1986).

**D)** For information regarding iodinated contrast media. Please refer to the IODINATED CONTRAST MEDIA management for further information.

### 1.2 SPECIFIC SUBSTANCES

**A)** POTASSIUM IODIDE

**1)** Iodeto de Potassium
**2)** Kali Iodidum
**3)** Kali Jodidum
**4)** Kali Iodetum
**5)** Kalium Jodatum
**6)** Potassii Iodidum
**7)** Potassium (Iodure de)
**8)** CAS 7681-11-0
**9)** Molecular Formula: K-I
**10)** References: Reynolds, 1989

CALCIUM IODIDE

**1)** CAS 10102-68-8
**2)** Molecular Formula: Ca-I2
**3)** References: Reynolds, 1989

SODIUM IODIDE
**1)** Sodium (Iodure de)
**2)** Sodii Iodidum
**3)** Iodeto de Sodio
**4)** Natrii Iodetum
**5)** Natrii Iodidum
**6)** Natrium Iodatum
**7)** Natrii Jodidum
**8)** CAS 7681-82-5
**9)** Molecular Formula: Na-I
**10)** References: Reynolds, 1989
METHYL IODIDE
**1)** Iodomethane
**2)** CAS 74-88-4

### 1.6 AVAILABLE FORMS/SOURCES

**A)** FORMS
**1)** POTASSIUM IODIDE (Benitz & Tatro, 1995)
**a)** Saturated Solution (SSKI): 1 gram/mL (50 mg/drop)
**b)** Syrup: 325 mg/5 mL
**2)** SODIUM IODIDE
**a)** Solution: </=50% (not more than 500 mg/mL)
**3)** Calcium iodide is used in several multi-ingredient formulations, such as Bepro(R), Calcidrine(R), and Norisodrine with Calcium Iodide(R).
**4)** Methyl iodide is used as a methylating agent in pharmaceutical and chemical synthesis, in microscopy, as a reagent, as a catalyst in production of organic lead compounds, as an etching agent, as a component in fire extinguishers, and formerly as a soil fumigant (IARC, 1986).
**B)** USES
**1) IODIDES** - For the prophylaxis and treatment of iodine deficiency disorders. It may be given as potassium or sodium iodide, iodized oil, or potassium iodate (Sweetman, 2002).
**2) Iodides** have bactericidal activity. They are also active against fungi, viruses, protozoa, cysts, and spores. Potassium iodide has been used in the treatment of fungal infections, such as sporotrichosis.
**3)** POTASSIUM IODIDE - has been used as a thyroid blocking agent following exposure to radioisotopes of iodine such as from a nuclear reactor accident.
**a)** It has also been used as an expectorant, but due to a lack of evidence of clinical efficacy other agents are usually preferred.

## 3.0 CLINICAL EFFECTS

SUMMARY OF EXPOSURE

VITAL SIGNS

HEENT

CARDIOVASCULAR

RESPIRATORY

NEUROLOGIC

GASTROINTESTINAL

ACID-BASE

HEMATOLOGIC

DERMATOLOGIC

ENDOCRINE

IMMUNOLOGIC

REPRODUCTIVE

OTHER

**3.1 SUMMARY OF EXPOSURE**

**A)** Acute reactions which may occur following ingestion include hypersensitivity reactions which may present as angioedema, arthralgia, eosinophilia, lymphadenitis, or urticaria.
**B)** Chronic ingestion of small or excessive amounts may result in iodism which is characterized by salivation, coryza, sneezing, conjunctivitis, headache, fever, laryngitis, bronchitis, stomatitis, and various skin rashes.
**C)** Unlike iodine, the iodide salts are not caustic and no human fatalities are reported due to acute overdosage. Up to 10 grams of sodium iodide has been administered IV without signs or symptoms of toxicity. Chronic ingestion of small or excessive amounts may result in iodism.

**3.3 VITAL SIGNS**

**3.3.3** TEMPERATURE

**A)** FEVER - **Iodides** may cause transient fevers (Horn & Kabins, 1972; Steffen, 1965). Kurtz & Aber (1982) reported one case of iodide-induced fever that is suggested to have lasted 15 years.

**3.4 HEENT**

**3.4.1** SUMMARY

**A)** A metallic taste, increased salivary and bronchial secretions may be noted.

**3.5 CARDIOVASCULAR**

**3.5.1** SUMMARY

**A)** Periarteritis nodosa has been associated with iodide use.

**3.5.2** CLINICAL EFFECTS

**A)** ARTERITIS

**1)** PERIARTERITIS NODOSA has been associated with the use of **iodides** (Wahlbert & Wilstrom, 1963; (Davies, 1969).

**3.6 RESPIRATORY**

**3.6.2** CLINICAL EFFECTS

**A)** SPUTUM ABNORMAL - AMOUNT

**1)** SECRETIONS - Increased bronchial secretions may be noted.

**3.7 NEUROLOGIC**

**3.7.2** CLINICAL EFFECTS

**A)** DROWSY

**1)** Methyl iodide inhalation may result in lethargy (Appel et al, 1975).

**B)** SEIZURE

**1)** Visual disturbances may occur from methyl iodide (organic iodide) inhalation. This symptom may precede seizures, coma, and death (Appel et al, 1975).

**C)** DISTURBANCE IN SPEECH

**1)** Methyl iodide inhalation may result in slurred speech and ataxia (Appel et al, 1975).

**D)** DEMENTIA

**1)** Dementia may be a sequelae of methyl iodide inhalation (Appel et al, 1975).

**E)** EXTRAPYRAMIDAL DISEASE

**1)** Following an acute episode, cerebellar and Parkinsonism symptoms may abate slowly and be followed by psychiatric disturbances (Appel et al, 1975).

## 3.8 GASTROINTESTINAL

**3.8.1** SUMMARY

**A)** Vomiting and abdominal pain may be noted, but do not result in caustic effects on the gastrointestinal tract. Parotitis has also been reported.

**3.8.2** CLINICAL EFFECTS

**A)** VOMITING

**1)** Vomiting and abdominal pain may be noted, but do not result in caustic effects on the gastrointestinal tract.

**B)** TASTE SENSE ALTERED

**1)** Metallic taste may be noted (JEF Reynolds , 2000).

**C)** EXCESSIVE SALIVATION

**1)** Increased salivary and bronchial secretions may be noted (JEF Reynolds , 2000).

**D)** SIALOADENITIS

**1)** PAROTITIS has been caused by **iodides** (Gilman et al, 1985; Katz et al, 1986).

**a)** ONSET usually disappears in 12 hours of exposure with a duration of less than 72 hours. There may be involvement of the submandibular (an adenitis) and laryngeal edema.

## 3.11 ACID-BASE

**3.11.2** CLINICAL EFFECTS

**A)** ANION GAP

**1)** CASE REPORT - Negative ion gap with mild renal insufficiency was reported in a patient who had ingested 180 mEq of iodide. No measure of plasma iodide was done (Fischman et al, 1978).

## 3.13 HEMATOLOGIC

**3.13.2** CLINICAL EFFECTS

**A)** EOSINOPHIL COUNT RAISED

**1)** Ingestion of potassium iodide can result an allergic response which may include eosinophilia (USPDI, 1999).

## 3.14 DERMATOLOGIC

**3.14.1** SUMMARY

**A)** Chronic oral administration can produce various cutaneous manifestations, including erythema nodosum, polymorphic eruptions, urticaria, vasculitis, and petechia.

**3.14.2** CLINICAL EFFECTS

**A)** BULLOUS ERUPTION

**1)** IODODERMA - Topical administration can produce a nonspecific papulovesicular lesion. Iododerma after chronic oral administration can produce carbuncular lesions, which progress to scarring, erythema nodosum, or vasculitis. Other lesions include polymorphic eruptions, urticaria, and petechia (Burnett, 1989).

## 3.16 ENDOCRINE

**3.16.1** SUMMARY

**A)** Chronic iodide therapy has produced goiters, hypothyroidism and rarely hyperthyroidism.

**3.16.2** CLINICAL EFFECTS

**A)** HYPERTHYROIDISM

**1)** **Iodides** can induce thyrotoxicosis in selected cases, but is probably quite rare, although occurring more commonly in Europe. Onset may be up to 4 months after initiation of potassium iodide therapy and may last from 1 to 6 months (Fradkin & Wolff, 1983; (Klein & Levey, 1983) Yoshinari et al, 1988; (JEF Reynolds , 2000).

**B)** GOITER

**1)** CASE SERIES - In one study, 47 of 55 patients on long-term daily iodide therapy for cystic fibrosis of the pancreas developed goiters. Onset was after 2 to 3 years of therapy usually but ranged from 3 months to 12 years (Dolan & Gibson, 1971).

**C)** HYPOTHYROIDISM

**1)** SUMMARY - Hypothyroidism may occur after exposure to **iodides** (JEF Reynolds , 2000).

**2)** CASE SERIES - Hypothyroidism was noted in 14 of 55 patients receiving long-term iodide therapy for cystic fibrosis (Dolan & Gibson, 1971), as well as other patients on therapeutic **iodides** (Gomolin, 1987; Johnson & Rapini, 1988; Bona et al, 1988).

**3)** MECHANISM - Hypothyroidism is caused by inhibition of thyroid hormone synthesis at a critical level of iodide administration, called the "Wolff-Chaikoff effect". Normal subjects will adapt or "escape" from this inhibitory effect with continued administration of iodide (Johnson & Rapini, 1988).

**4)** RISK FACTORS - Patients who are less likely to adapt to the Wolff-Chaikoff effect and are at risk of developing hypothyroidism include patients with Graves' disease, goiter, or Hashimoto's thyroiditis. Susceptibility correlates with higher baseline TSH serum concentrations (Clark, 1990).

## 3.19 IMMUNOLOGIC

**3.19.1** SUMMARY

**A)** Acute hypersensitivity reactions including angioedema, Stevens Johnson syndrome, systemic vasculitis, and serum-sickness-like reactions may occur.

**3.19.2** CLINICAL EFFECTS

**A)** ACUTE ALLERGIC REACTION

**1)** Reactions including angioedema, urticaria, serum-sickness-like reaction, lymphadenitis, and systemic vasculitis may occur when applied topically or administered systemically (Eeckhout et al, 1987; USPDI, 1999; JEF Reynolds , 2000).

**B)** STEVENS-JOHNSON SYNDROME

**1)** Stevens Johnson Syndrome is associated with the use of **iodides** (Araugo & Flowers, 1984).

## 3.20 REPRODUCTIVE

**3.20.1** SUMMARY

**A)** Cretinism and goiter have been reported in children born to mothers chronically taking **iodides** pregnancy.

**3.20.3** EFFECTS IN PREGNANCY

**A)** GOITER

**1)** **Iodides** are readily diffused across the placenta. Neonatal deaths from respiratory distress secondary to goiter have been reported (Galina et al, 1962; Visconti, 1981).

**B)** HYPOTHYROIDISM

**1)** CRETINISM - **Iodides** alone should not be used in pregnancy since they are not reliable or effective for long-term management of hyperthyroidism and severe fetal complications such as cretinism and death from obstructive goiter have resulted (Herbst & Selenkow, 1965).

**C)** PREGNANCY CATEGORY

| | |
|---|---|
| POTASSIUM IODIDE | D |
| SODIUM IODIDE | D |
| SODIUM IODIDE I-131 | X |
| SODIUM IODIDE I-125 | X |
| HYDRIODIC ACID | D |
| Reference: Briggs et al, 1998. | |

## 3.23 OTHER

**3.23.2** CLINICAL EFFECTS

**A)** CHRONIC POISONING

**1)** Chronic ingestion can result in iodism associated with a variety of symptoms including unpleasant metallic taste, burning in the mouth and throat, soreness in the teeth and gums, increased salivation, rhinorrhea, sneezing, conjunctival irritation, swelling of the eye lids, productive cough, enlarged and tender salivary glands, acneiform skin lesions, other skin eruptions, gastric irritation, diarrhea, fever and depression.

## 4.0 LABORATORY/MONITORING

### 4.1 MONITORING PARAMETERS/LEVELS

**4.1.1** SUMMARY

**A)** Plasma iodide levels are not clinically useful.

**B)** Protein bound iodine (PBI) will be elevated for up to 3 weeks after ingestion of pharmacologic doses of inorganic **iodides**.

**C)** Iodide-induced hypothyroidism is accompanied by decreased serum T4 levels and usually increased serum TSH levels.

**4.1.2** SERUM/BLOOD

**A)** ENDOCRINE

**1)** Iodide-induced hypothyroidism is accompanied by decreased serum T4 levels and usually increased serum TSH levels.

**2)** Protein bound iodine (PBI) will be elevated for up to three weeks after ingestion of pharmacologic doses of inorganic **iodides** while more specific measures of circulating thyroxine may not be affected.

**B)** LABORATORY INTERFERENCE

**1)** Some laboratory chloride determinations are performed with the Technicon Station System, a potentiometric measurement. This system may give an extremely high serum chloride reading and negative anion gap in the presence of high serum **iodides** (Fischman et al, 1978).

## 6.0 TREATMENT

LIFE SUPPORT

MONITORING

ORAL EXPOSURE

### 6.1 LIFE SUPPORT

**A)** Support respiratory and cardiovascular function.

### 6.4 MONITORING

**A)** Plasma iodide levels are not clinically useful.

**B)** Protein bound iodine (PBI) will be elevated for up to 3 weeks after ingestion of pharmacologic doses of inorganic **iodides**.

**C)** Iodide-induced hypothyroidism is accompanied by decreased serum T4 levels and usually increased serum TSH levels.

### 6.5 ORAL EXPOSURE

**6.5.1** PREVENTION OF ABSORPTION/PREHOSPITAL

**A)** EMESIS -

**1)** INDICATIONS/CAUTIONS

**a)** Ipecac should only be considered for treatment of patients in whom:

**1)** There is no contraindication.

**2)** There is a substantial chance of serious toxicity based on the substance and quantity ingested.
**3)** There is no alternative available to decrease GI absorption.
**4)** There will be a delay of more than 1 hour before a patient can reach an emergency medical facility.
**5)** Ipecac can be administered within 30 to 90 minutes of ingestion (Manoguerra & Cobaugh, 2005).
**6)** It should generally NOT be administered if the patient is already vomiting, or if ipecac induced emesis might interfere with more definitive treatment provided at a hospital (Manoguerra & Cobaugh, 2005). Ipecac administration has never been shown to alter clinical outcome after overdose. Several studies (involving volunteers and patients) have shown that ipecac administration soon after ingestion reduces serum drug levels (Bond et al, 1993; McNamara et al, 1989; Danel et al, 1988; Tenenbein et al, 1987; Neuvonen & Olkkola, 1984; Neuvonen et al, 1983). Ipecac is NOT recommended for use in the emergency department (Krenzelok et al, 1997).
**7)** CONTRAINDICATIONS to ipecac-induced emesis include: ingestion of toxicant that might compromise airway protective reflexes or require advanced life support within 60 minutes; coma; seizures; compromised airway protective reflexes, signs of oral, pharyngeal, or esophageal irritation; central nervous system excitation or depression; ingestion of a corrosive substance; ingestion of a substance with a high aspiration potential (particularly hydrocarbons); debilitated elderly patients or those with medical conditions that might be adversely affected by induced emesis (American Academy of Clinical Toxicology & European Association of Poisons Centers and Clinical Toxicologists, 2004).
**8)** Emesis is most effective if initiated within 30 minutes of ingestion.

**2)** DOSE OF IPECAC SYRUP

**a)** ADULT: Dose: 15 to 30 milliliters (American Academy of Clinical Toxicology & European Association of Poisons Centers and Clinical Toxicologists, 2004)
**b)** ADOLESCENT: Dose: 15 to 30 milliliters (American Academy of Clinical Toxicology & European Association of Poisons Centers and Clinical Toxicologists, 2004)
**c)** CHILD 1 TO 12 YEARS: Dose: 15 milliliters (American Academy of Clinical Toxicology & European Association of Poisons Centers and Clinical Toxicologists, 2004)
**d)** CHILD 6 TO 12 MONTHS: Dose: 5 to 10 milliliters (American Academy of Clinical Toxicology & European Association of Poisons Centers and Clinical Toxicologists, 2004). Position child in left lateral decubitus position to reduce risk of aspiration.
**e)** CHILD UNDER 6 MONTHS OF AGE: NOT recommended for prehospital use.
**f)** FLUIDS

**1)** Prior to or after the dose is given, encourage clear fluids, 8 ounces (240 milliliters) in adults and adolescents and 4 to 8 ounces (120 to 240 milliliters) in a child (American Academy of Clinical Toxicology & European Association of Poisons Centers and Clinical Toxicologists, 2004).

**g)** ADVERSE EFFECTS

**1)** Common complications may include diarrhea, lethargy/drowsiness, and prolonged vomiting (American Academy of Clinical Toxicology & European Association of Poisons Centers and Clinical Toxicologists, 2004).
**2)** Refer to the IPECAC TREATMENT management for further information on administration and adverse reactions as indicated.

**B)** ACTIVATED CHARCOAL -

**1)** PREHOSPITAL ACTIVATED CHARCOAL ADMINISTRATION

**a)** Consider prehospital administration of activated charcoal as an aqueous slurry in patients with a potentially toxic ingestion who are awake and able to protect their airway. Activated charcoal is most effective when administered within one hour of ingestion. Administration in the prehospital setting has the potential to significantly decrease the time from toxin ingestion to activated charcoal administration, although it has not been shown to affect outcome (Alaspaa et al, 2005; Thakore & Murphy, 2002; Spiller & Rogers, 2002).
**1)** In patients who are at risk for the abrupt onset of seizures or mental status depression, activated charcoal should not be administered in the prehospital setting, due to the risk of aspiration in the event of spontaneous emesis.
**2)** The addition of flavoring agents (cola drinks, chocolate milk, cherry syrup) to activated charcoal improves the palatability for children and may facilitate successful administration

(Guenther Skokan et al, 2001; Dagnone et al, 2002).

**2)** CHARCOAL DOSE

**a)** Use a minimum of 240 milliliters of water per 30 grams charcoal (FDA, 1985). Optimum dose not established; usual dose is 25 to 100 grams in adults and adolescents; 25 to 50 grams in children aged 1 to 12 years (or 0.5 to 1 gram/kilogram body weight) ; and 0.5 to 1 gram/kilogram in infants up to 1 year old (Chyka et al, 2005).

**1)** Routine use of a cathartic with activated charcoal is NOT recommended as there is no evidence that cathartics reduce drug absorption and cathartics are known to cause adverse effects such as nausea, vomiting, abdominal cramps, electrolyte imbalances and occasionally hypotension (None Listed, 2004).

**b)** ADVERSE EFFECTS/CONTRAINDICATIONS

**1)** Complications: emesis, aspiration (Chyka et al, 2005). Aspiration may be complicated by acute respiratory failure, ARDS, bronchiolitis obliterans or chronic lung disease (Golej et al, 2001; Graff et al, 2002; Pollack et al, 1981; Harris & Filandrinos, 1993; Elliot et al, 1989; Rau et al, 1988; Golej et al, 2001; Graff et al, 2002). Refer to the ACTIVATED CHARCOAL/TREATMENT management for further information.

**2)** Contraindications: unprotected airway (increases risk/severity of aspiration) , nonfunctioning gastrointestinal tract, uncontrolled vomiting, and ingestion of most hydrocarbons (Chyka et al, 2005).

**6.5.2** PREVENTION OF ABSORPTION

**A)** ACTIVATED CHARCOAL

**1)** There is little or no information concerning how well **iodides** are adsorbed by activated charcoal. Iodine is absorbed in vitro (Mitchell et al, 1989; Rausch, 1935).

**2)** CHARCOAL ADMINISTRATION

**a)** Consider administration of activated charcoal after a potentially toxic ingestion (Chyka et al, 2005). Administer charcoal as an aqueous slurry; most effective when administered within one hour of ingestion.

**3)** CHARCOAL DOSE

**a)** Use a minimum of 240 milliliters of water per 30 grams charcoal (FDA, 1985). Optimum dose not established; usual dose is 25 to 100 grams in adults and adolescents; 25 to 50 grams in children aged 1 to 12 years (or 0.5 to 1 gram/kilogram body weight) ; and 0.5 to 1 gram/kilogram in infants up to 1 year old (Chyka et al, 2005).

**1)** Routine use of a cathartic with activated charcoal is NOT recommended as there is no evidence that cathartics reduce drug absorption and cathartics are known to cause adverse effects such as nausea, vomiting, abdominal cramps, electrolyte imbalances and occasionally hypotension (None Listed, 2004).

**b)** ADVERSE EFFECTS/CONTRAINDICATIONS

**1)** Complications: emesis, aspiration (Chyka et al, 2005). Aspiration may be complicated by acute respiratory failure, ARDS, bronchiolitis obliterans or chronic lung disease (Golej et al, 2001; Graff et al, 2002; Pollack et al, 1981; Harris & Filandrinos, 1993; Elliot et al, 1989; Rau et al, 1988; Golej et al, 2001; Graff et al, 2002). Refer to the ACTIVATED CHARCOAL/TREATMENT management for further information.

**2)** Contraindications: unprotected airway (increases risk/severity of aspiration) , nonfunctioning gastrointestinal tract, uncontrolled vomiting, and ingestion of most hydrocarbons (Chyka et al, 2005).

**6.5.3** TREATMENT

**A)** ANAPHYLAXIS

**1)** SUMMARY

**a)** Mild to moderate allergic reactions may be treated with antihistamines with or without inhaled beta adrenergic agonists, corticosteroids or epinephrine. Treatment of severe anaphylaxis also includes oxygen supplementation, aggressive airway management, epinephrine, ECG monitoring, and IV fluids.

**2)** BRONCHOSPASM

**a)** ALBUTEROL

**1)** ADULTS: 2.5 to 5 milligrams in 2 to 4.5 milliliters of normal saline delivered per nebulizer every 20 minutes up to 3 doses. If incomplete response administer 2.5 to 10 mg every 1 to 4 hours as needed, or 10 to 15 mg/hr by continuous nebulization as needed (National Heart,Lung,and Blood Institute, 2007). CHILDREN: 0.15 milligram/kilogram (minimum 2.5

milligrams) per nebulizer every 20 minutes up to 3 doses. If incomplete response administer 0.15 to 0.3 mg/kg (up to 10 mg) every 1 to 4 hours as needed, or 0.5 mg/kg/hr by continuous nebulization (National Heart,Lung,and Blood Institute, 2007).

**3)** CORTICOSTEROIDS

**a)** METHYLPREDNISOLONE - Adults: 1 to 2 milligrams/kilogram intravenously every 6 to 8 hours. Children: 1 to 2 milligrams/kilogram intravenously (maximum 125 milligrams) every 6 hours.

**b)** PREDNISONE - Adults: 40 to 60 milligrams/day. Children: 1 to 2 milligrams/kilogram/day divided twice daily. Prolonged therapy generally not needed.

**4)** MILD CASES

**a)** DIPHENHYDRAMINE

**1)** ADULTS: 50 milligrams orally, intravenously, or intramuscularly initially, then 25 to 50 milligrams orally every 4 to 6 hours for 24 to 72 hours.

**2)** CHILDREN: 1.25 milligrams/kilogram orally, intravenously, or intramuscularly initially, then 5 milligrams/kilogram/day orally in four divided doses for 24 to 72 hours.

**5)** MODERATE CASES

**a)** EPINEPHRINE: 0.3 to 0.5 milliliter of a 1:1000 solution subcutaneously or intramuscularly (children: 0.01 milliliter/kilogram, 0.5 milliliter maximum); may repeat in 20 to 30 minutes (American Heart Association, 2005).

**6)** SEVERE CASES

**a)** EPINEPHRINE

**1)** INTRAVENOUS BOLUS: 1:10,000 solution, 5 to 10 milliliters diluted in 10 milliliters 0.9% saline slow intravenous push over 5 to 10 minutes (American Heart Association, 2005) (children: 0.1 milliliter/kilogram); give if systolic blood pressure less than 70 mmHg (adults); it is safest to titrate to effect in small increments, 1 to 2 milliliters at a time.

**2)** INTRAVENOUS INFUSION: An alternative method of intravenous epinephrine by constant infusion has been advocated as safer: 1 milligram of a 1:1000 dilution of epinephrine added to 250 milliliters dextrose 5 percent in water. Start infusion at 1 microgram/minute and titrate to systolic blood pressure of 100 mmHg (or mean arterial pressure of 80 mmHg).

**7)** AIRWAY MANAGEMENT

**a)** OXYGEN: 5 to 10 liters/minute via high flow mask.

**b)** INTUBATION: Perform early if any stridor or signs of airway obstruction.

**c)** CRICOTHYROTOMY: Use if unable to intubate with complete airway obstruction.

**d)** BRONCHODILATORS are recommended for mild to severe bronchospasm.

**e)** ALBUTEROL: ADULTS: 5 to 10 milligrams in 2 to 4.5 milliliters of normal saline delivered per nebulizer every 20 minutes up to 3 doses. If incomplete response repeat every hour. CHILDREN: 0.15 milligram/kilogram (minimum 2.5 milligrams) per nebulizer every 20 minutes up to 3 doses. If incomplete response repeat every hour.

**8)** MONITORING

**a)** CARDIAC MONITOR: All complicated cases.

**b)** IV ACCESS: Routine in all complicated cases.

**9)** HYPOTENSION

**a)** IF hypotensive give 500 to 2000 milliliters crystalloid initially (20 milliliters/kilogram in children) and titrate to desired effect (stabilization of vital signs, mentation, urine output); adults may require up to 6 to 10 liters/24 hours. Central venous or pulmonary artery pressure monitoring is recommended in patients with persistent hypotension.

**1)** VASOPRESSORS: Should be used in refractory cases unresponsive to repeated doses of epinephrine and after vigorous intravenous crystalloid rehydration.

**2)** DOPAMINE: Mix 400 to 800 milligrams in 250 milliliters of dextrose 5 percent in water (1600 or 3200 micrograms/milliliter). Initial dose is 2 to 5 micrograms/kilogram/minute intravenously; titrate to desired hemodynamic response.

**10)** DIPHENHYDRAMINE

**a)** ADULTS: 50 milligrams intravenously initially, then 25 to 50 milligrams intravenously or orally every 4 to 6 hours for 24 to 72 hours.

**b)** CHILDREN: 2 milligrams/kilogram intravenously initially, then 5 milligrams/kilogram/day intravenously or orally in four divided doses for 24 to 72 hours.

**11)** METHYLPREDNISOLONE

**a)** Adults: 1 to 2 milligrams/kilogram intravenously every 6 to 8 hours. Children: 1 to 2 milligrams/kilogram intravenously (maximum 125 milligrams) every 6 hours.

**12)** DYSRHYTHMIAS

**a)** Dysrhythmias may occur primarily or iatrogenically as a result of pharmacologic treatment (epinephrine). Monitor and correct serum electrolytes, oxygenation and tissue perfusion. Treat with antiarrhythmic agents as indicated.

**B)** TRANSFUSION REACTION DUE TO SERUM PROTEIN REACTION

**1)** DIPHENHYDRAMINE/ADULT DOSE

**a)** Usual dose is 25 to 50 milligrams intravenously over 2 minutes

**b)** Maximum dose: 100 milligrams/dose; 400 milligrams/day

**2)** DIPHENHYDRAMINE/PEDIATRIC DOSE

**a)** 1.25 milligrams/kilogram/dose (37.5 milligrams/M(2)) intravenously over 2 minutes

**b)** Maximum dose: 300 milligrams/day (Prod Info Benadryl(R), diphenhydramine, 1998)

**3)** Corticosteroids should be administered if antihistamines are not effective or if severe symptoms persist.

**C)** DIURESIS

**1)** A diuretic such as mannitol should be administered in treatment of chronic iodide poisoning or methyl iodide exposure to increase renal excretion of iodide. Fluids and sodium chloride intake will also hasten iodide excretion.

**D)** THYROTOXICOSIS

**1)** Discontinuing the inciting agent is usually the only therapy necessary in mild cases. Beta blockers may be used in symptomatic patients. RARELY propylthiouracil, I131, or surgery are necessary.

**E)** HYPOTHYROIDISM

**1)** Discontinuation of the iodide source will usually result in restoration of normal thyroid function within several weeks (Gomolin, 1987). Short-term administration of thyroid hormone supplements may hasten recovery (Johnson & Rapini, 1988).

## 7.0 RANGE OF TOXICITY

SUMMARY

THERAPEUTIC DOSE

MINIMUM LETHAL EXPOSURE

MAXIMUM TOLERATED EXPOSURE

TOXICITY INFORMATION

OTHER

### 7.1 SUMMARY

**A)** Toxicity following acute ingestion is uncommon, however, hypersensitivity reactions may occur and are potentially life-threatening (angioedema and laryngeal edema).

**B)** Up to 10 grams of sodium iodide has been administered IV without toxicity.

**C)** Chronic iodide poisoning (iodism)occurs more commonly.

### 7.2 THERAPEUTIC DOSE

**7.2.1** ADULT

**A)** SPECIFIC SUBSTANCE

**1)** POTASSIUM IODIDE (Benitz & Tatro, 1995) -

**a)** Solution - 1000 mg/mL (available as 30 and 240 mL; SSKI)

**b)** Expectorant - 300 to 1000 milligram dose given after meals; 2 to 3 times daily. May be increased to 1000 to 1500 milligrams/dose three times as tolerated.

**c)** Sporotrichosis - 500 milligrams three times a day. May be increased by 50 milligrams/dose daily until a maximum of 1 to 2 grams/dose.

**7.2.2** PEDIATRIC

**A)** SPECIFIC SUBSTANCE

**1)** POTASSIUM IODIDE (Benitz & Tatro, 1995) -

**a)** CHILDREN - Expectorant - 150 to 500 milligrams/dose after meals 2 to 3 times daily. May increase to 500 to 750 milligrams/dose, three times daily, as needed and tolerated.

**b)** PRE-SCHOOLER - Sporotrichosis - Initially, 50 milligrams/dose three times a day which may be increased by 50 milligrams/dose increments at daily intervals.

**1)** MAXIMUM - 500 milligrams/dose three times a day.

**c)** OLDER CHILD - Sporotrichosis - Initially, 250 milligrams/dose three times a day which may be increased by 50 milligrams/dose at daily intervals.

**1)** MAXIMUM - 1 to 2 grams/dose three times a day.

## 7.3 MINIMUM LETHAL EXPOSURE

**A)** CASE REPORTS

**1)** At least one fatal case of periarteritis nodosa has been described.

## 7.4 MAXIMUM TOLERATED EXPOSURE

**A)** CHRONIC

**1)** Chronic iodide poisoning (iodism) is more common. Sufficiently high dose levels will cause toxicity in all individuals. On occasion goiter and, even less commonly, hypothyroidism may be induced by prolonged administration of iodide.

**2)** Chronic iodide administration during pregnancy has been associated with a few case reports of congenital goiter and even hypothyroidism.

## 7.7 TOXICITY INFORMATION

### 7.7.1 TOXICITY VALUES

**A)** METHYL IODIDE -

**1)** LD50- (INTRAPERITONEAL)MOUSE:

**a)** 172 mg/kg (RTECS, 2000)

**2)** LD50- (SUBCUTANEOUS)MOUSE:

**a)** 110 mg/kg (RTECS, 2000)

**3)** LD50- (INTRAPERITONEAL)RAT:

**a)** 101 mg/kg (RTECS, 2000)

**4)** LD50- (ORAL)RAT:

**a)** 76 mg/kg (RTECS, 2000)

**B)** POTASSIUM IODIDE -

**C)** SODIUM IODIDE -

**1)** LD50- (INTRAPERITONEAL)MOUSE:

**a)** 430 mg/kg (RTECS, 2000)

**2)** LD50- (ORAL)MOUSE:

**a)** 1 g/kg (RTECS, 2000)

**3)** LD50- (ORAL)RAT:

**a)** 4340 mg/kg (RTECS, 2000)

## 7.9 OTHER

**A)** OTHER

**1)** GENERAL

**a)** Acute poisoning is uncommon but the hypersensitivity reactions may be dangerous, especially if angioedema is accompanied by laryngeal edema. Serum-sickness-like reactions may include fever, lymphadenitis, arthralgia, and arthritis.

# 8.0 KINETICS

ABSORPTION

DISTRIBUTION

EXCRETION

**8.1 ABSORPTION**
**A)** SUMMARY
**1)** Rapidly and readily absorbed.

**8.2 DISTRIBUTION**
**8.2.1** DISTRIBUTION SITES
**A)** TISSUE/FLUID SITES
**1)** Appears in the saliva, sweat and milk. Diffuses across the placenta.

**8.4 EXCRETION**
**8.4.1** KIDNEY
**A)** Excreted mainly in the urine.

## 9.0 PHARMACOLOGY/TOXICOLOGY

**9.1 PHARMACOLOGIC MECHANISM**
**A) Iodides** do not normally have much pharmacological activity when acutely administered. They are employed primarily as expectorants because of their stimulatory effects on bronchial secretions, but attempts to demonstrate decreased sputum viscosity have not been successful.
**B)** Potassium iodide is the drug of choice for treatment of lymphocutaneous sporotrichosis. It is also used to treat erythematous dermatoses, including erythema nodosum, erythema multiforme, and nodular vasculitis.
**C) Iodides** in small quantities (about 150 mg/day) are ingested normally and plasma inorganic iodide concentration is normally less than 10 mg/L (about 10 to 20% of total plasma iodine concentration). Distribution is throughout extracellular body water with concentration in the thyroid gland and salivary secretions.
**D) Iodides** are also used to increase the solubility of iodines such as in iodine tinctures.

## 10.0 PHYSICOCHEMICAL

PHYSICAL CHARACTERISTICS

MOLECULAR WEIGHT

**10.1 PHYSICAL CHARACTERISTICS**
**A)** POTASSIUM IODIDE
**1)** Colorless, opaque, or white crystals or crystalline powder with no odor
**2)** Slightly hydroscopic
**3)** There are 6 mmol of potassium and of iodide per gram.
**4)** Should be protected from light.
**B)** SODIUM IODIDE
**1)** A colorless, odorless, crystal or whitish powder
**2)** Deliquescent in moist air
**3)** Develops a brownish color upon decomposition.
**4)** There are 6.7 mmol of sodium and of iodide per gram.

**10.3 MOLECULAR WEIGHT**
**A)** 166.0 (Potassium Iodide)

**B)** 149.9 (Sodium Iodide)

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