IPCS INCHEM Home

Isopropyl alcohol

ISOPROPYL ALCOHOL

International Programme on Chemical Safety
Poisons Information Monograph 290
Chemical

1. NAME

1.1 Substance

Isopropyl alcohol

1.2 Group

Aliphatic alcohol hydrocarbon

1.3 Synonyms

Pseudo propyl alcohol; 2-propanol;
dimethyl carbinol; isopropanol;

sec-propyl alcohol; persprit;
secondary propyl alcohol; IPA;
propan-2-ol; alcohol isopropylicus;
petrohol

1.4 Identification numbers

1.4.1 CAS number

67-63-0

1.4.2 Other numbers

UN/NA (PIN): 1219
EC Number: 603-003-00-0
RTECS: NT 8050000
(Budavari, 1996)

1.5 Main brand names/main trade names

Avantine (UK)
IPS.1 and IPS/C (Shell)
Sterets (Schering-Prebbles UK)
Sterile Pack Fluid (Ethicon)

1.6 Manufacturers, importers

Manufactured by: Shell Chemicals
British Petroleum
Mobil Oil

2. SUMMARY

2.1 Main risks and target organs

Coma, central bnervous system (CNS) depression, liver, kidney, cardiovascular depression and brain damage.

2.2 Summary of clinical effects

Drowsiness, ataxia and stupor.

Coma and respiratory depression.

Irritation of mucous membranes and eyes.

Gastritis, gastric haemorrhage, vomiting and pancreatitis.

Cold and clammy skin, hypothermia, miosis pupils, tachycardia, respiration slow and noisy.

Hypotension

Cardiodepression

Brain, liver and kidney damage at a later stage.

2.3 Diagnosis

Blood and urine for biomedical analysis should be collected.

Fluid balance.

Electrolytes and blood gases.

Blood glucose.

Urinalysis

Hepatic and renal function tests.

Blood pressure, temperature and vital signs.

Blood levels of isopropyl alcohol and acetone are clinically useful.

2.4 First-aid measures and management principles

Seek medical attention immediately and transfer patient to hospital.

Inhalation: Remove source of contamination or move patient into fresh air. Begin artificial respiration immediately if victim is not breathing and administer supplemental oxygen if available. Obtain medical attention.

Eye contact: Flood affected eye(s) with copious amounts of lukewarm, gently running water for at least 15 minutes. During this time the upper and lower eyelids should be held apart.

Skin contact: Rinse the affected area(s) with copious amounts of lukewarm, gently running water for at least 15 minutes. Remove contaminated clothing and shoes. Wash/thoroughly clean all clothing before re-using or discard. If skin irritation exists, seek medical attention.

Ingestion: Induce emesis at an early stage if not contraindicated. Empty stomach by gastric aspiration and lavage, care being taken not to induce pulmonary aspiration of the return flow. Keep the patient warm. If respiration is depressed or absent, endotracheal intubation and assisted respiration may be required. Obtain medical assistance immediately.

In severe poisonings:

maintain respiration;
maintain fluid balance;
initiate haemodialysis or peritoneal dialysis;
treat convulsions with intravenous (IV) benzodiazepines.

First aid summary:

Provide the patient with general supportive measures (warmth, comfort and rest). Obtain medical advice and/or assistance for all cases of ingestion or eye contact, and all but trivial cases of inhalation or skin contact.

3. PHYSICO-CHEMICAL PROPERTIES

3.1 Origin of the substance

Synthetic: Prepared from propylene, which is obtained in the cracking of petroleum or by the reduction of acetone (Budavari, 1996).

3.2 Chemical structure

Chemical name: Isopropyl alcohol

Molecular weight: 60.09

Structural formula: CH_3-CH(OH)-CH_3

3.3 Physical properties

3.3.1 Colour

Colourless

3.3.2 State/form

Liquid-other

3.3.3 Description

Boiling point: 82.5°C

Melting point: 89.5°C

Flash point: 11.7°C (closed cup)
17°C (open cup)

Autoignition temperature: 455.6°C

Relative density: 0.785 (Water = 1)

Vapour pressure: 4399.62 Pa at 20°C
7879.33 Pa at 30°C

Solubility: In water in all proportions at 20°C
In alcohol in all proportions at 20°C
In ether in all proportions at 20°C

Also soluble in acetone, benzene and chloroform, insoluble in salt solutions.

Relative molecular mass: 60.09
(C: 59.96%; H: 13.42%; O: 26.62%)

pH: N/A

Viscosity: 0.00243 Poises (2.43 cP) at 20°C

Odour: Slight odour resembling a mixture of ethanol and acetone.
Taste: Slightly bitter taste

3.4 Hazardous characteristics

Explosive limits:
Lower explosive limit: 2.5% (in air)
Upper explosive limit: 12% (in air)

Dangers associated with the vapour:

Dispersion: inflammable mixture with air.
Possible ignition: flammable liquid; may autoignite. Vapour is heavier than air and will travel along the ground, into work pits, etc.

Possible chemical reaction in air: none.

Possible chemical reactions with other chemicals:

Strong oxidizing agents (nitrates, perchlorates, peroxides).
Phosgene - forms isopropyl chloroformate and hydrogenchloride.
Iron salts - explosive thermal decomposition may occur.
Hydrogen-palladium-mixture can ignite in air.
Potassium t-Butoxide
Trinitromethane (Nitroform) (Cheminfo, 1989).

Electrical and thermal conductivity with possible consequences: no data available.

Products of combustion: carbon dioxide and carbon monoxide.

Environmental risks:

Safe disposal - dispose in a designated landfill site, or burn in an approved solvent burner. If small amounts are disposed of into a sink or sewer, rinse with copious amounts of water to prevent accumulation of flammable vapours. Notify environmental agencies (local health and wildlife officials) in the event of any significant release of this material into the environment.

Isopropanol is dangerous to aquatic life in high concentrations, so prevent entry into water intakes and waterways.

Clean up spills - stop the flow if it can be done safely. Contain the spill. Recover liquid for recycling or disposal if feasible. Otherwise absorb the liquid on clay, sand, sawdust or other absorbent material.

Food chain concentration potential: None.

(EPS Canada, 1984).

4. USES/HIGH RISK CIRCUMSTANCES OF POISONING

4.1 Uses

4.1.1 Uses

Antifreeze;
vehicle;
industrial Solvent;
industrial
Miscellaneous industrial function

4.1.2 Description

In antifreeze products; solvent for gums, shellac, essential oils, in quick drying oils, creosote and resins: extraction of alkaloids; in quick drying inks; in denaturing ethyl alcohol; in body rubs, hand lotions, after-shave lotions, cosmetics and pharmaceuticals; in manufacture of acetone, glycerol, isopropyl acetate; antiseptic; rubefacient pharmaceutic aid (solvent) (in concentrations up to 70%) (Ellenhorn, 1988).

4.2 High risk circumstances of poisoning

Accidental ingestion of rubbing alcohols/toiletries by children.

Dermal/inhalation exposure in children during isopropyl alcohol sponging for control of fever.

Intentional ingestion for alcoholic effect, or in suicide attempts.

Occupational or accidental exposure to liquid or its vapour.

4.3 Occupationally exposed populations

Process workers in the pharmaceutical industry.
Process workers in the cosmetic industry.
Process workers in the chemical industry.
Process workers in the petroleum industry.
Analytical and other laboratory workers.
Printers.
Painters.
Carpenters and cabinet makers.

5. ROUTES OF EXPOSURE

5.1 Oral

Oral ingestion of rubbing alcohols and toilet preparations, constitute the most common route of exposure.
Doses of above 20 mL may produce toxic effects.

5.2 Inhalation

Inhalation of vapour from preparations.

Inhalation may occur in children being sponged with isopropyl alcohol to control fever.

Short-term exposure limit is 500 ppm. At levels above this, respirator or self-contained breathing apparatus is necessary. A level of 12,000 ppm is immediately dangerous to health and life.

5.3 Dermal

Dermal exposure to the liquid and vapour.

There is little absorption through intact skin, but significant delayed absorption over 4 hours postulated (Martinez, 1986).

Note risk of inhalation after prolonged skin exposure (sponging, etc.).

5.4 Eye

Eye exposure to liquid and vapour.

Both the liquid and solvent are severely irritant.

5.5 Parenteral

No data available.

5.6 Others

No data available.

6. KINETICS

6.1 Absorption by route of exposure

6.1.1 Oral

80% of an oral dose is absorbed within 30 minutes. Absorption is complete within 2 hours although this may be delayed in a large overdose (Ellenhorn, 1988).

6.1.2 Inhalation

Alveolar concentration is correlated to the environmental concentration at any given time, and the mean alveolar clearance has been calculated at 8 L/min (Ellenhorn, 1988).

6.1.3 Dermal contact

Absorbed through intact skin on prolonged exposure (Martinez, 1986).

6.2 Distribution by route of exposure

Isopropyl alcohol distributes in body water with an apparent volume of distribution of 0.6 to 0.7 L/kg. Two hours are required for complete tissue distribution (Ellenhorn, 1986).

6.3 Biological half-life by route of exposure

Isopropyl alcohol most closely follows first order kinetics, with a half-life of 2.5 to 3.2 hours (Daniel, 1981). The elimination half-life of the active metabolite, acetone, is significantly prolonged to about 5 hours in rats (Teramoto, 1987) and 22.4 hours in man (Natowicz et al., 1985).

6.4 Metabolism

20 to 50% of an absorbed dose is excreted unchanged. Most isopropyl alcohol is oxidized in the liver by alcohol dehydrogenase to acetone, which is probably further metabolized to acetate, formate, and finally carbon dioxide. Acetone may contribute to the CNS depression seen in isopropanol poisoning.

6.5 Elimination and excretion by route of exposure

20 to 50% of an absorbed dose is excreted unchanged (2% of dose excreted in exhaled air).

Acetone is slowly eliminated by the lung (40%) or kidney. Clinically insignificant excretion occurs into the stomach and saliva (Teramoto, 1987).

Related ketoacids are not produced in sufficient quantities to cause a severe metabolic acidosis.

7. TOXICOLOGY

7.1 Mode of action

Isopropyl alcohol is an irritant to mucous membranes and eyes.

Isopropyl alcohol is a potent central nervous system (CNS) depressant, and in large doses causes cardiovascular depression. Acetone, its main metabolite, can potentiate and lengthen the duration of the CNS symptoms. Mild acidosis may develop from the conversion of acetone to acetic acid and formic acid. The alcohol dehydrogenase-induced shift in NAD/NADH may cause decreased gluconeogenesis and hypoglycaemia (Addison, 1962).

Inebriation, peripheral vasodilation and hypothermia may also occur.

In children, hypoglycaemia is particularly severe when poisoning follows fasting, exercise or chronic malnutrition.

Lactic acidosis may occur in patients with severe liver disease, pancreatitis or receiving biguanide therapy, or as a result of the hypovolaemia which frequently accompanies severe intoxication.

In rat hepatocytes (Hormann et al., 1989), the following sequence has been observed:

marked depletion of glutathione
increased malondialdehyde production
decreased protein sulphydryls content
leakage of lactic dehydrogenase with loss of membrane integrity.

7.2 Toxicity

7.2.1 Human data

7.2.1.1 Adults

Adults: 1 mL/kg of a 70% solution, but as little as 0.5 mL/kg may cause symptoms.

The lethal dose may be as low as 240 mL (2 to 4 mL/kg). Death has been reported at a blood level of 150 mg/dL (25 mmol/L); however, survival after dialysis has been reported at levels as high as 560 mg/dL.

Maximal air concentration allowable to prevent irritation of eyes, nose and throat is:

440 ppm (USA)
400 ppm long-term (UK)
500 ppm short-term (UK)
200 ppm (Japan).

7.2.1.2 Children

6 mL/kg (9 mL/kg of 70%) has produced coma with a blood level of 380 mg/dL. The lethal dose is approximately 100 mL

Pediatric patients have survived serum levels from 128 to 520 mg/dL with supportive care.

(Ellenhorn, 1988; Martinez, 1986).

7.2.2 Animal data

LD_{50} (rat, oral): 4.42 to 5.84 g/kg
LD_{50} (mouse, oral): 4.8 g/kg
LD_{50} (rabbit, oral): 7.9 g/kg
(Lehman, 1944: 6.4 mL/kg)
LD_{50} (rabbit, dermal): 13 g/kg

Irritant dose (rabbit, skin): 500 mg/24 h (mild)
Irritant dose (rabbit, eye): 0.1 mL 70% solution (severe eye irritant)

Common signs of acute isopropyl alcohol intoxication of animals are hind leg paralysis, unsteadiness, lack of muscular coordination, respiratory depression and stupor (Cheminfo, 1989).

7.2.3 In-vitro data

No data available.

7.2.4 Workplace standards

Threshold Limit Values (TLVs)/American Conference of Governmental Industrial Hygienists 1987 to 1988 are:

Time weighted average:

TLV-TWA 400 ppm (980 mg/m^3)

Short-term exposure limit:

TLV-STEL 500 ppm (1225 mg/m^3)
IDLH Value 20,000 ppm
(NIOSH, 1985; ACGIH, 1988)

OSHA PEL Final Rule Limits (OSHA, 1989):

PEL-TWA Final Rule Limit:400 ppm (>>980 mg/m^3)
PEL/STEL Final Rule Limit:500 ppm (>>1225 mg/m^3)
no ceiling limit

Odour threshold: 90 mg/m^3 (Chris, 1985).

NIOSH REL: 400 ppm (TWA)
800 ppm (ceiling for 15 min) (CDC, 1988).

7.2.5 Acceptable daily intake (ADI)

400 ppm (TWA OSHA).

Other guideline levels:

Respiratory Protection Guidelines (NIOSH):

up to 1,000 ppm: Use a powered air purifying respirator with organic vapour cartridge, or full facepiece chemical cartridge.

up to 10,000 ppm: (CCROVF) respirator with organic vapour cartridge; use a supplied air respirator operating in continuous flow mode (GMOVc).

up to 12 000 ppm: use a gas mask with organic vapour canister; or full facepiece self-contained breathing apparatus; or, full facepiece supplied air respirator, (GMOVfb/SAF/SCBAF).

Escape Respirator: GMOV/SCBA.

7.3 Carcinogenicity

Not carcinogenic by animal controls (Cheminfo, 1989).

A waste product, isopropyl oil, has been suspected to cause respiratory tract neoplasms after occupational exposure (Weil et al., 1952).

7.4 Teratogenicity

Decreased fetal weights and increased skeletal malformations were noted when pregnant rats were exposed by inhalation to concentrations that also caused maternal toxicity (Nelson et al., 1988).

7.5 Mutagenicity

Not mutagenic in animal models (RTECS, 1989).

7.6 Interactions

Isopropyl alcohol potentiates the hepatic and renal toxicity produced by halokenes (e.g., carbon tetrachloride) (Ellenhorn, 1981).

8. TOXICOLOGICAL AND OTHER ANALYSES

8.1 Sample

8.1.1 Collection

Whole blood, urine, stomach contents. Collect and store in tightly closed containers and store in a cool place. Samples should be processed quickly to be of use clinically. Sample size at least 5 mL. Glass containers are preferable to plastic.

Urine samples should be collected hourly. If the air is to be sampled for vapour concentrations, at least 50 mL is required, and this needs to be stored in a tightly closed container.

For active material, use a sampling pump with appropriate collecting medium: NIOSH Method S 65

(NIOSH, 1985).

8.1.2 Storage

Store in a cool place, sample could be inflammable.

8.1.3 Transport

Transport to nearest laboratory as quickly as possible in cool containers. Material may be inflammable.

8.2 Toxicological analytical methods

8.2.1 Test for active ingredient

Simple qualitative:

Acetest tablets can be used in the presumptive diagnosis of isopropyl alcohol ingestion. These will detect the presence of acetone in the urine, which appears approximately 3 hours after ingestion. As the level of isopropyl alcohol drops, the level of acetone will initially rise and then decline.

Recommended quantitative:

Gas-liquid chromatography or other specific methods (Parker et al., 1962) similar to those used for ethanol detection constitute the methods of choice.

The GLC should be equipped with flame ionization detection. Ideally the apparatus should have a capsule sampler and recorder and the results should be processed with calculating integrator.

Acetone can be assayed using the same apparatus. The blood sample, 9.5 mL, is placed in an aluminium capsule (3 mm diam. x 6 mm length), and cold welded using a crimping tool. The sealed capsule is inserted into the injection port of the gas chromatograph. The injector is flushed with carrier gas (e.g., helium 30 mL/min), to remove any atmosphere introduced with the probe. The capsule is held in the heated zone for 60 seconds to allow for vapourization of the solvents within the capsule. The capsule is then pierced and carrier gas takes the volatiles into the hollow needle and then on to the chromatographic columns. A suitable column is 6' x 1/8" S.S., with 20% Carbowax 20 M on Chromosrob W, AW-DCMS, 60/80 mesh.

The retention times are: isopropanol 2.17 minutes, and acetone: 1.22 minutes.

Suitable temperatures are: injector: 200°C; column: 70°C; and detector: 200°C.

Typical flow rates are: carrier gas He = 30 mL/min; hydrogen = 35 mL/min; air = 450 mL/min.

Calibration curves may be determined using concentrations of 0.005 to 1.5 mg/mL of isopropanol injected into fresh blood samples.

The concentration of each standard solution can be calculated on a mg/mL basis, using the specific gravity of blood and isopropyl alcohol and acetone. A calibration graph is plotted by plotting peak height (cm) against isopropyl alcohol and acetone concentrations (mg/mL). The limit detection is 0.001 mg/mL for both solvents (Laham et al., 1979, 1980).

8.2.2 Test for biological sample

No data available.

8.3 Other laboratory analyses

8.3.1 Haematological investigations

8.3.1.1 Blood

Full blood count
Blood film.
Haematocrit.

8.3.1.2 Urine

Myoglobinuria (secondary to coma-induced rhabdomyolysis)
Red and white blood cells

8.3.2 Biochemical investigations

8.3.2.1 Blood

Osmolal Gap:

Used to estimate blood isopropanol concentration using the following equation:

Osmolal gap x 6 = predicted isopropyl alcohol level mg/dL

Osmolal gap = measured osmolality - calculated osmolality

Anion gap
Serum electrolytes
Blood urea nitrogen
Creatinine
Glucose
Serum acetone
Hepatic transferases

8.3.2.2 Urine

Glucose
Ketone bodies (acetone)

8.3.2.3 Other

No data available.

8.3.3 Arterial blood gas analysis

Arterial pO_2 and pCO_2 concentrations.

Acid-base balance.

8.3.4 Other relevant biochemical analyses

No data available.

8.4 Other biomedical (diagnostic) investigations and their interpretation

Isopropyl alcohol values do not correlate with severity of poisoning as the metabolite acetone also interferes with symptomatology.

High serum ketones with minimal acidosis are common in isopropyl alcohol ingestion.

Osmolal Gap: A rise in blood isopropyl alcohol of 2 mg/dL produces a change of 0.17 mosm/kg H_2O in the osmolal gap. A 50 mg/dL toxic blood level produces an 8 to 9 mosm increase in the osmolal gap, and seriously toxic levels of 200 mg/dL produce a 34 mosm change in osmolal gap. Acetone levels must also be considered as they produce similar changes.

8.5 Overall interpretation of all toxicological analyses and toxicological investigations

Sample collection

Isopropanol serum levels are clinically useful and significant. Collect in glass where possible, keep cool and tightly closed, until analysed. During treatment, measurement of isoprpyl alcohol in hourly urine samples is clinically useful.

Retention of samples of gastric lavage if performed, and obtaining samples of product ingested, may be useful if diagnostic confirmation is desired.

Biomedical analysis

Routine blood and urine analysis should be performed, with special emphasis on:

Blood glucose (for hypoglycaemia).

Osmolal gap by freezing point depression (for predicted isopropanol levels).

Arterial blood gas analysis (for assessment metabolic acidosis).

Serum acetone (serum ketone bodies).

Urinary acetone (for presumptive diagnosis with Acetest tablets).

Monitoring of fluids and electrolytes. (Acid-base balance for normoglycaemic ketoacidosis).

Hepatic function tests (for hepatic aminotransferases).

Toxicological analysis

Plasma levels of isopropanol are indicative of the level of poisoning. The time of ingestion is critical in determining

treatment options.

Other investigations
Respiratory function tests to gauge the degree of respiratory depression may be useful in selected, severe poisonings.

Blood pressure (for hypotension).

Temperature (for hypothermia).

8.6 References

9. CLINICAL EFFECTS

9.1 Acute poisoning

9.1.1 Ingestion

This is the common route of poisoning. Absorption (80%) occurs within 30 minutes, and is complete within 2 hours. Symptoms are drowsiness, gastrointestinal pain, cramps, nausea, vomiting and diarrhoea, with unconsciousness and death following massive exposure. In children, more than three swallows of 70% isopropyl alcohol results in symptoms requiring medical observation.

Isopropyl alcohol intoxication has a rapid onset of action (30 to 60 minutess) with peak effects occurring within several hours. Severe poisoning presents early with stupor leading to deep coma, respiratory depression and hypotension. Other symptoms are dizziness, poor coordination, headache, confusion, gastric irritation, abdominal pain, vomiting, haematemesis, hypotension, tachycardia, (and cardiodepression) and loss of deep tendon reflexes.

9.1.2 Inhalation

Common route of poisoning. Mild irritation of the respiratory tract occurs at 400 ppm. High concentrations can cause nausea, headache, lightheadedness, drowsiness, ataxia and deep narcosis.

9.1.3 Skin exposure

Brief exposures are not irritating, but prolonged contact (4 hours) has led to toxicity with central nervous system (CNS) effects (Martinez, 1986). Sponging children with isopropyl alcohol for fever control may result in significant absorption.

9.1.4 Eye contact

Vapour is mildly irritating at 400 ppm. Direct eye contact with the liquid can cause severe irritation (Cheminfo, 1989) and even corneal abrasion (Osborn and Rosales, 1981).

9.1.5 Parenteral exposure

No data available.

9.1.6 Other

No data available.

9.2 Chronic poisoning

9.2.1 Ingestion

No significant changes were found in the chemical or cellular composition of the blood or urine after humans ingestion of 6.4 mg/kg of isopropyl alcohol on a daily basis for 6 weeks (Cheminfo, 1989).

No long-term health effects have been reported in humans. Rapid metabolism in the body precludes accumulation.

9.2.2 Inhalation

An excess of sinus cancers and laryngeal cancers has been found among workers producing isopropyl alcohol. This could be due to the by product, isopropyl oil.

9.2.3 Skin exposure

Chronic toxic exposure may lead to coma and death (Broughton, 1944).

Drying, cracking and eczema may result from repeated or prolonged skin contact.

9.2.4 Eye contact

Oxygen uptake of corneal epithelium is reduced in the rabbit (Roseman, 1987).

9.2.5 Parenteral exposure

No data available.

9.2.6 Other

No data available.

9.3 Course, prognosis, cause of death

Central nervous system depression often lasts for 24 hours. The development of hypotension is a poor prognostic feature. Haemodialysis should be used in severely poisoned patients to remove the isopropyl alcohol, and to shorten the duration of coma, together with other supportive measures.

Death results from central nervous system (CNS) and respiratory depression during coma.

9.4 Description of clinical effects by system

9.4.1 Cardiovascular

Hypotension from peripheral dilatation
Tachycardia
Serious arrhythmias have not been reported.

(Ellenhorn, 1988).

9.4.2 Respiratory

Respiratory depression and death.
Acetone can be detected on the breath.
(Teramoto, 1987; Buckley, 1986).

9.4.3 Neurologic

9.4.3.1 Central Nervous system (CNS)

Dizziness, poor coordination, headache, confusion, progressing to stupor, coma and loss of deep tendon reflexes. Serious nervous system depression often lasts for 24 hours. Elation does not occur.

9.4.3.2 Peripheral nervous system

No data available.

9.4.3.3 Autonomic nervous system

Pupils are often miotic, and nystagmus is normally present.

9.4.3.4 Skeletal and smooth muscle

Deep tendon reflexes are not present during coma.

9.4.4 Gastrointestinal

Gastric irritation appears early, abdominal pain and vomiting are prominent, and haematemesis may occur (Buckley, 1986).

9.4.5 Hepatic

Hepatic dysfunction has been reported (Kulig, 1984).

9.4.6 Urinary

9.4.6.1 Renal

Acute tubular necrosis and myoglobinuria have been reported (Buckley, 1986).

9.4.6.2 Others

No data available.

9.4.7 Endocrine and reproductive systems

No data available.

9.4.8 Dermatologic

Dryness, irritation, allergic eczema, with repeated or chronic direct contact.

9.4.9 Eye, ear, nose, throat: local effects

Eye: vapour causes irritation, liquid may cause intense irritation including corneal abrasion.

9.4.10 Haematological

Myoglobinuria and haemolytic anaemia have been reported.

9.4.11 Immunologic

No data available.

9.4.12 Metabolic

9.4.12.1 Acid-base disturbances

Mild metabolic acidosis may develop from the conversion of acetone to acetic and formic acid.

9.4.12.2 Fluid and electrolyte disturbances

Abnormalities of serum in electrolytes, blood urea nitrogen, creatinine, may be noted.

9.4.12.3 Others

Hypoglycaemia, and hypothermia from peripheral vasodilation.

Osmolal gap may be clinically significant in suspecting the diagnosis.

Elevations of hepatic aminotransferases may occur.

9.4.13 Allergic reactions

Allergic eczema has been reported.

9.4.14 Other clinical effects

No data available.

9.4.15 Special risks: pregnancy, breast feeding, enzyme deficiencies.

No data available.

9.5 Others

No data available.

9.6 Summary

10. MANAGEMENT

10.1 General principles

If a small quantity of isopropyl alcohol has been recently ingested, i.e., within 30 minutes, then emesis with syrup of ipecac may be used. It should not be used with

ingestion of large quantities, or in the presence of central nervous system (CNS) depression.

Gastric lavage is the treatment of choice for the removal of large volumes of isopropyl alcohol from the stomach. In the case of skin contamination, wash the skin thoroughly with soap and water. If irritation and pain persist seek medical advice.

Exposed eyes should be irrigated with copious amounts of room temperature water for at least 15 minutes. If irritation, pain, swelling, lachrymation persist after 15 minutes of irrigation, seek ophthalmological advice.

10.2 Life supportive procedures and symptomatic treatment

Support respiratory function.

Support cardiovascular function.

Treat hypotension with intravenous (IV) fluids and place in the Trendelburg position. If the patient is unresponsive, correct dehydration and acidosis with fluids and electrolytes and administer dopamine.

Add 200 to 400 mg dopamine to 250 mL sodium chloride 0.9% (normal saline, NS) or glucose 5% injection, (Dextrose, D5W), to produce 800 or 1600 mg/mL. Alternatively, add 400 mg to 500 mL of NS or D5W to produce 800 mg/mL. Begin infusion at

2 to 5 mg/kg/min, progressing in 5 to 10 mg/kg/min increments as needed. If ventricular arrhythmias occur decrease the rate of administration.

Support neurological function.

10.3 Decontamination

Measures to eliminate isopropanol from the gastrointestinal tract must usually be initiated within 2 hours of ingestion and, preferably within 30 min, due to its rapid absorption.

Due to the onset of CNS depression (which usually occurs within 30 minutes of ingestion), ipecac-induced emesis is not recommended.

Gastric lavage may be indicated if performed soon after ingestion. If coma or convulsions occur, protect the airway by cuffed endotracheal intubation. Use a large bore orogastric tube:

Adult: 36 to 42 French
Child: 24 to 28 French.

Repeat as necessary. Lavage should return approximately the same volume of fluid as has been given.

Lukewarm tap water or normal saline is recommended, at a rate of 150 to 200 mL per wash (in children over 5 and adults) and 50 to 100 mL per wash in young children.

Continue until the lavage return is clear.

Activated charcoal

Administer charcoal as a slurry in water at a dilution of 30 g charcoal to 240 mL aqueous diluent. The diluent may also be a saline cathartic or sorbitol. Usual doses are 30 to 100 g of charcoal in adults (15 to 30 g in children, and 1 to 2 g/kg in infants).

Note: Activated charcoal has limited absorptive properties for isopropanol (1 g absorbs approximately 500 mg of isopropanol), and the optimum dose of charcoal has not been established.

Cathartics

Administer one dose of a saline cathartic or sorbitol mixed with the charcoal or preferably administered separately.

Note: The safety of more than one dose of a cathartic has not been established.

Hypermagnesaemia has been reported after repeated administration of magnesium-containing cathartics in patients with normal renal function. Repeated cathartic dosing should be done with extreme caution if at all; admiistration of cathartics should be stopped when a charcoal-coloured stool appears.

Cathartics should not be used in patients with ileus and saline cathartics should not be used in patients with impaired renal function. Magnesium sulphate or sodium sulphate are suitable cathartics and the usual dose is 20 to 30 g in adults and 250 mg/kg in children over 2 years. Magnesium citrate oral solution *USP* may also be used in a dose for both adults and children of 4 mL/kg/dose up to 200 to 300 mL/dose.

Catharsis may be effective 12 to 24 h after ingestion.

Sorbitol:

Adult: The usual dose for sorbitol will vary but is in the order of 1 to 2 g/kg/dose to a maximum of 150 g.

Children: Over 1 year old, 1 to 1.5 g/kg/dose to a maximum of 150 g/dose. It is administered under direct medical supervision monitoring fluid and electrolyte status. It should be given as a 35% aqueous solution.

Skin decontamination: Wash the skin thoroughly with soap and water. Seek medical advice if pain or irritation persist.

Eyes: Irrigate with copious amounts of tepid water (preferably isotone saline solution) for at least 15 minutes. Seek specialist medical advice if pain, irritation, swelling, lachrymation or photophobia persist.

10.4 Enhanced Elimination

Forced diuresis is not effective and should not be used.

Haemodialysis:

Haemodialysis is useful in patients with severe life-threatening symptoms unresponsive to standard treatment therapy.

Note: Using haemodialysis, the metabolism of isopropanol to acetone is rapid and should be monitored carefully.

Peritoneal dialysis:

This appears to be only minimally effective, and should only be used in selected severe cases of poisoning where haemodialysis is not possible.

10.5 Antidote treatment

10.5.1 Adults

There is no specific antidote.

10.5.2 Children

There is no specific antidote.

10.6 Management discussion

Haemodialysis is used on the basis that it removes isopropanol and shortens the duration of coma. Removal of isopropanol alcohol is 52 times, and of acetone 40 times, more efficient through haemodialysis than through urinary excretion (Rosansky, 1982).

Peritoneal dialysis is considerably less effective. No advantage is to be gained by infusing ethanol to block alcohol dehydrogenase, as the toxicity of isopropanol is principally due to the parent compound.

Isopropanol is oxidized in the liver to acetone and treatment options are limited to the symptomatic and supportive measures.

11. ILLUSTRATIVE CASES

11.1 Case reports from the literature

A 2-year-old boy weighing 21 kg reportedly ingested a small amount of 70% isopropyl alcohol rubbing alcohol. There were no symptoms and emesis with dilute dishwashing liquid was tried. The child refused this and was referred to the nearest hospital. On arrival, 30 minutes after ingestion, the child was in an unresponsive condition with laboured respiration. No emesis had occurred. The child was started on intravenous (IV) fluids, intubated, lavaged, and then transferred to a children's hospital. It was found that the child had been sponged with isopropanol for several hours in an attempt to reduce fever. Three-and-a-half hours after ingestion, the child was still unconscious and was transferred to the intensive care unit (ICU) with artificial ventilation and haemodialysis. Approximately 16 hours after

ingestion and following treatment the child was alert and responsive. Intravenous (IV) fluids were discontinued and the child made an uneventful recovery (Martinez, 1986).

A 38-year-old woman with Type I diabetes mellitus complained

of discomfort after applanation tonometry of the right eye. The tonometer tip had been swabbed with a 70% isopropyl alcohol wipe immediately before use. On examination of the right eye, a circular area of corneal epithelial opacification corresponding to the size of the tonometer tip was noted. This area stained faintly with fluorescein but stained strongly with Rose Bengal. A patch was placed on the eye for 2 h and then removed, the patient seemed comfortable. But 2 to 3 h later she noted significant discomfort that persisted for 16 hours. The discomfort resolved by the next day. A second patient showed lesser symptoms that resolved after 24 hours (Soukiasian, 1988).

An 18-month-old child was wrapped in towels soaked with rubbing alcohol, by her mother, in an attempt to control a 40°C fever due to otitis media. The towels were wrapped about the child's waist for periods up to 4 hours. The child became progressively lethargic and unresponsive to verbal and tactile stimulation. She was admitted to the ICU unit unconscious and unresponsive to pain, with midline fixed miotic pupils, a temperature of 35.8°C, a pulse of 153 beats/min, and blood pressure of 100/50 mm Hg. Nasogastric aspiration yielded strongly haem-positive "coffee-ground" material. Laboratory studies were performed, including arterial blood gases, electrolytes, CSF and urinalysis. Her osmolal gap was calculated at 70 mOsm/kg (70 mmol/kg). She was diagnosed as having mild metabolic acidosis and this coupled with an anion gap of 20 mmol/L (20 mEq/L) and a significant elevation in the osmolality gap, suggested the possibility of acute intoxication with an osmotically active substance of low molecular weight.

Toxicological studies performed 8 to 10 hours after ingestion showed a serum level of isopropanol of 162 mg/dl (27 mmol/L), (measured by gas chromatography). The serum acetone level was 180 mg/dL (31 mmol/L). The child received supportive care, including airway management and intravenous hydration. She improved over the next 12 to 24 h and was extubated and discharged to a paediatric ward 36 hours after admission to the ICU. She was discharged home on the 7th day with no evidence of neurologic or other sequelae. The child continued to do well 3 months after the event (Arditi, 1987).

A similar case was reported by Webster et al. (1985) of a 9-month-old girl. Osmolality and anion gap was seen as being diagnostic of isopropanol poisoning. Treatment consisted of

mechanical ventilation and parenteral fluids. She was discharged from hospital 3 days after admission, with no evidence of neurologic or other sequelae.

A 55-year-old woman was found unconscious and unresponsive in her home. There was no palpable pulse, HR 140 beats/min, RR 6 breaths/min. She was intubated, ventilated, but no venous access could be obtained. The patient had been bathed with approximately 1 litre of 70% isopropanol by her daughter. Four hours after admission, an isopropanol level of 98 mg/dL (165 mmol/L) and an acetone level of 43 mg/dL (7 mmol/L) were obtained. The patient had massive skin lesions and it was believed that absorption through the skin had occurred (McGrath, 1989).

A 61-year-old patient was found unconscious and unresponsive after ingesting 1480 mL of 70% isopropyl alcohol. He was

admitted to ICU 4 hours later and was immediately intubated and started on fluid replacement with a dopamine drip for hypotension. On examination, the patient was completely unresponsive; pulse was 60; BP was 80/60, and pinpoint pupils, acetone odour on breath, bradycardia and no response to painful stimuli were noted. The following laboratory tests were performed: blood gases; electrolytes; BUN; serum creatinine; ketone bodies and glucose. Haemodialysis with minimal ultrafiltration was instituted 9 hours after ingestion, and within 1 hour the patient had spontaneous movements and started assisting ventilation. By 2 hours, the patient responded to verbal commands. The paper concludes that haemodialysis is a life-saving procedure in this case, removing approximately 27 g/hour of alcohol (50 times greater than the amount eliminated by urinary excretion) (Rosansky, 1982).

A 4-year-old mentally retarded girl was found unconscious in her bed by her mother, 27 hours after she had been intentionally scalded. The mother subsequently applied gauze soaked with isopropanol to the scald burns. The child was then left alone in her room without food or water until she was discovered dead. The paper suggests that a contributory cause of death was isopropanol intoxication, resulting from application of this chemical to burns (Russo, 1986).

A review of acute isopropyl alcohol poisoning (Lacouture et al., 1983), provides an overview of acute poisoning with special emphasis on clinical management. Two case reports were presented.

A 42-year-old woman was admitted to hospital in an unresponsive and unconscious condition. Initial treatment consisted of endotracheal intubation, respiratory support, administration of intravenous (IV) fluids, and gastric lavage. There was no response to naloxone or glucose. She

was given 4 litres of lactated Ringers solution prior to her transfer to ICU. Her depth of coma decreased and 12 hours after admission she was alert and awake and able to breathe on her own.

A 32-year-old man was admitted to hospital unconscious (Grade III to IV coma). He was areflexic and unresponsive to deep pain. There was no response to naloxone, thiamine or glucose. Laboratory tests included isopropyl alcohol levels, serum electrolytes, BUN, and blood gases. During intubation, the patient suffered a cardiac arrest. He was resuscitated. However, an arryhthmia refractory to standard therapy necessitated insertion of a transvenous pacemaker. He was also started on a dopamine drip and was prepared for haemodialysis. Twenty-four hours after admission he awoke and was orientated and co-operative.

The authors suggest that haemodialysis should be considered in patients with hypotension and those with serum isopropyl alcohol concentrations exceeding 400 to 500 mg/dL (66 to 83 mmol/L). They also conclude that the management of acute isopropanol intoxication may involve supportive care only, or may include haemodialysis.

A 23-month-old boy was admitted to hospital comatose and unresponsive to deep stimuli. He was intubated. His pulse became unperceptible, and cardiac compression was initiated, together with controlled ventilation with 100% oxygen and 20

mmol of sodium bicarbonate by slow intravenous (IV) infusion through a scalp vein. Nonadrenaline was required to correct and maintain the BP within normal limits. It was subsequently discovered that he had ingested approximately 200 mL of isopropanol. He was comatose for 2 days and then slowly recovered (Light, 1969).

A further case reported by Visudhiphan (1971), concerned a 2-year-old boy suffering from isopropanol intoxication., His blood pressure was maintained with metaraminol tartrate and levaterenol bitartrate. His respiration was assisted with a positive pressure respirator. He gradually improved over 2 days and made an uneventful recovery.

A comatose 46-year-old woman was admitted to hospital with isopropanol and acetone levels of 2,000 mg/L and 120 mg/L respectively. Pharmacokinetic analysis showed that both isopropanol and acetone obeyed apparent first order kinetics with half-lives of 6.4 and 22.4 hours respectively, with similar values in cerebrospinal fluid. Two days after admission the patient was alert and responsive. She was subsequently discharged.

A 59-year-old man was admitted in a coma to hospital after ingesting 1 litre of isopropyl alcohol. Laboratory data were normal except for proteinuria and ketonuria. His condition deteriorated rapidly, with development of hypotension and respiratory depression. Pupils were miotic and all deep reflexes were absent. Extra corporeal haemodialysis was instituted; within 1 hour he was responding weakly to painful stimuli. After 3 hour of dialysis the patient was very active, and so the procedure was terminated (Freireich, 1967).

An 850 g girl of 25 weeks gestation was intubated at birth and placed in a ventilator. During surgical preparation for umbilical artery catheterization, isopropyl alcohol was poured over the umbilical stump. After the procedure was completed it was noted that there were two areas of erythema bilaterally on the flank, each approximately 2 x 5 cm. These progressed to areas of second- and third-degree burns. The burns were treated with an antibiotic ointment and a steroid cream, and continued to heal over a period of 3 months (Schick, 1981; Martindale, 1989).

Similar cases were reported by Weintraub (1982), giving details of skin burns in premature infants. The isopropyl alcohol had been used either for conduction on ECG or for arterial catheterization. The 4 premature infants reported were all less than 26 weeks gestational age and their birth weight was less than 750 g; they had suffered severe perinatal asphyxia, hypothermia and acidosis, and they all developed severe respiratory distress insufficiency and died within 48 hours despite mechanical ventilation and other supportive measures. It is recommended that the skin be dried after application of isopropyl alcohol.

A 45-year-old white man was comatose on admission to hospital after apparently falling from a stepladder. He was intubated and placed on a respirator. He was found to have an osmolal gap of 304 mOsm/kg. Serum ethanol concentration was determined by enzymatic alcohol dehydrogenase giving a concentration of 1.86 g/L. The osmolal gap indicated that there was another alcohol present, and that the enzymatic

alcohol determination was in error because of its lack of specificity. A gas chromatographic analysis showed the presence of ethanol, acetone and isopropanol. The authors warn of the danger of using an enzymatic procedure for ethanol, because the method is not specific for ethanol. The patient recovered and was discharged 2 days later.

A 28-year-old man ingested 1 litre of rubbing alcohol over a 10 minute period, and was admitted to hospital 45 minutes later. He was comatose and unresponsive. A tracheostomy was performed and therapy with intravenous fluids was initiated. He made little progress and so, 16 hours after admission,

haemodialysis was started. After 2 hours he was agitated, alert and responsible, and haemodialysis was discontinued (King, 1970).

Junco (1969), reported a case of a 35-year-old man who had ingested 1 pint of rubbing alcohol. The patient was inebriated confused and tremulous. His right arm was swollen, erythematous, tender and painful on motion. This swelling progressed, and by the 3rd day the entire limb was swollen. A phlebogram demonstrated blockage of the axillary vein. He received therapy over the next 25 days, being treated for renal failure and thrombosis. The patient was given phenytoin, supportive fluids, electrolytes, vitamins and whole blood. The outcome is unknown.

12.ADDITIONAL INFORMATION

12.1 Specific preventive measures

Respiratory protection:

Use self-contained breathing apparatus (in fire).
Respirator selection: see 7.2.5.1
Gloves: rubber or plastic.
Protective clothing: suitable to prevent repeated or prolonged skin contact, e.g., coveralls, etc.
Eyes: wear suitable eye protection.
Boots: rubber.

Note: remove clothing immediately if wet or contaminated to avoid flammability hazard.

12.2 Other

No data available.

13. REFERENCES

ACGIH (1988). Threshold limit values and biological exposure indices for 1988-89. American Government Conference of Industrial Hygiene, Cincinnati, Ohio

Adelson L (1962). Fatal intoxication with isopropyl alcohol (rubbing alcohol). Am J Clin Pathol 38: 144-151

Alexander CB, McBay AJ, Hudson RP (1982). Isopropanol and isopropanol deaths - 10 years experience. J Forensic Sci 27: 541-548

Antonova VI, Salmina ZA (1978). The maximum permissable concentration of isopropyl alcohol in water bodied with due regard for its action on the gonads and the progeny. Gibiena i

Sanitariya (USSR) 1: 8-11

Arditi M et al (1987). Coma following the use of rubbing alcohol for fever control. AJDC The Pediatric Forum 141 (March): 237-238

Buckley BM, Vale JA. Poisoning by alcohols and ethylene glycol. Prescribers Journal 26(4): 110-115

Budavari S ed. (1996) The Merck Index: An Encyclopedia of Chemicals, Drugs, and Biologicals. 12th ed. Merck & Co., Inc, Whitehouse Station, NJ.

CDC (1988). Recommendations for occupational safety and health standards. MMWR 37 (Suppl S-7): 1-29

Cahaplin MA (1949). Isopropyl alcohol poisoning with acute renal insufficiency. J Maine Med Assoc 40: 288-290

CHEMINFO (1989). Canadian Centre for Occupational Health, 1989 - 05 - 5.

Chris (1985). Hazardous chemical data US Department of Transportation, US Coast Guard, Washington

Coppock RW, Mostrom MS, Lillie LE (1988). The toxicology of detergents, bleaches, antiseptics and disinfectants in small animals. Vet Hum Toxicol 30: 463-473

Corbett J, Meier G (1968). Suicide attempted by rectal administration of drug. J Am Med Assoc 206: 2320-2321

Daniel DR, Mcanalley BH, Garriott JC (1981). Isopropyl alcohol metabolism after acute intoxication in humans. J Anal Toxicol 5: 110-112

Dua SL (1974). Peritoneal dialysis for isopropyl alcohol poisoning. J Am Med Assoc 1974: 230-235

Ellenhorn MJ, Barceloux DG (1988). Medical toxicology - diagnosis and treatment of human poisoning. London, Elsevier Science Publishing

EPS (March 1984). Manual for spills of hazardous materials Canada, Technical Services Branch, Environmental Protection Service

Freirich AW, Cinque TW, Xanthaky G et al (1967). Haemodialysis for isopropanol poisoning. New Engl J Med 277: 699-700

Gadsen RH, Mellette RR, Miller JC (1958). Scrap iron intoxication. J Am Med Assoc 1958: 1168-1220

Garrison RF (1953). Acute poisoning from the use of isopropyl alcohol in tepid sponging. J Am Med Assoc 1953: 317-318

Grant WM (1986). Toxicology of the eye. Springfield, Illinois, Charles C Thomas

Hurst MR (1987). Airborne levels of isopropyl alcohol disinfectant in a laminar-airflow hood. Am J Hosp Pharm 44: 2293-2295

IARC (1979). IARC monographs on the evaluation of the

carcinogenic risk of chemicals to humans. Lyon, France, Supplement I, IARC, 36

ILO (1983). Encyclopaedia of Occupational Health and Safety, p 109

Jatlow PI, Bailey DN (1980). Analytical toxicology In: Sonnewirt AC, Jarett L Ed Gradwohls' Clinical Laboratory Methods and Diagnosis, St Louis, The CV Mosby Company, pp 407-410

Juncos L, Taguchi JT (1968). Isopropyl alcohol poisoning. J Am Med Assoc 204: 732-734

Kelner M, Bailey DN (1983). Isopropanol ingestion: interpretation of blood concentrations and clinical findings. J Toxicol Clin Toxicol 20(5): 497-507

King LH et al (1970). Haemodialysis for isopropyl alcohol poisoning. J Am Med Assoc 211: 1855

Lacouture PG, Heldreth DD et al (1989). The generation of acetonaemia/acetonuria following ingestion of a sub-toxic dose of isopropyl alcohol. Am J Emerg Med 7: 38-40

Lacouture PG, Wason S, Abrams L et al (1983). Acute isopropyl alcohol intoxication. Am J Med 75: 680-686

Laham S, Protvin M et al (1981). Studies on inhalation toxicity of 2-propanol. Drug and Chemical Toxicology 3(4): 343-360

Lehman AJ, Chase HF (1944). The acute toxicity of isopropyl alcohol. J Lab Clin Med 29: 561-567

Light FB, Marx GF (1969). The value of gastric aspiration in a comatose child. Anesthesiology 31: 478-480

Mackison FW, Stricoff RS, Partridge LJ, eds (1981). Occupational health guidelines for isopropyl alcohol. In: Occupational Health Guidelines for Chemical Hazards (DHHS (NIOSH) Publication No 81-123) Washingtron, DC, NIOSH/OSHA (1981)

Martinez TT et al (1986). A comparison of the absorption and metabolism of isopropyl alcohol by oral, dermal and inhalation routes. Vet Hum Toxicol 28(3): 233-236

Mccord WM, Switzer PK, Brill HH (1948). Isopropyl alcohol intoxication. South Med J 41: 639-642

McFadden SW, Haddow JE (1969). Coma produced by tropical application of isopropanol. Pediatrics 43: 622-623

McGrath RB (1989). Absorption of topical isopropyl alcohol in an adult. Critical Care Medicine 1(11): 1233

Mecilaski MB, Depner TA (1982). Peritoneal dialysis for isopropanol poisoning. West J Med 137: 322-325

Mendelson J, Wexler D et al (1957). A study of addiction to nonethyl alcohols and other poisonous compounds. Q J Stud Alcohol 18: 561-580

Natowicz M, Donahue J et al (1985). Pharmacokinetic analysis of a case of isopropanol intoxication. Clin Chem 31: 326-328

Nelson BK, Brightwell DS (1988). Teratogenicity of n-propanol and isopropanol; administered at high concentration to rats. Fd Chem Toxic, 26: 247-254

NIOSH (1985). Pocket guide to chemical hazards Cincinnati, Ohio, National Institute for Occupational Health and Safety

NIOSH (1976). Criteria for a recommended standard: Occupational exposure to isopropyl alcohol, Cincinnati, Ohio, National Institute for Occupational Health and Safety, Document number: 76-142

Osborn LM, Rosales TO (1981). Corneal abrasion during alcohol sponging. Clin Pediatr 20: 782

OSHA (1989). 29 CFR Part 1910: Air contaminants final rule Occupational Safety and Health Administration Federal Register 1989; 54(12): 2332-2893

Parker KD, Fontan CR, Yee JL et al (1962). Gas chromatographic determination of ethyl alcohol in blood for mediolegal purposes. Anal Chem, 34: 1234

Parmeggiani L Ed (1983). International Labour Organisation: Encyclopaedia of occupational health and safety, 34th ed, Vol I. Geneva, IL Office, p 109

Poisindex Information System. Rumach BH, ed. Micromedex, Denver, 1990

Reynolds JEF, ed (1989). Martindale, The Extra Pharmacopoeia: 29th edition London, The Pharmaceutical Press

Rosansky SJ (1982). Isopropyl alcohol poisoning treated with haemodialysis: Kinetics of isopropyl alcohol and acetone removal. J Toxicol Clin Toxicol 19(3): 265-271

Roseman MJ, Hill RM (1987). Aerobic responses of the cornea to isopropyl alcohol, measured in vivo. Acta Opthalmologica 65: 306-312

Rowe VKI, McCollister CB (1982). Alcohols. In: Clayton GD, Clayton FE, Eds. Patty's industrial hygiene and toxicology, 3rd revised edition Vol 2C. Toxicology New York: Toronto, Ontario: Wiley-Interscience, pp 4527-4708

RTECS (1989). 50359 Isopropyl alcohol, RTECS #NT805000 Registry of toxic effects of chemical substances Washington, US Department of Health and Human Sciences

Russo S et al (1986). Scald burns complicated by isopropyl alcohol intoxication: A case of fatal child abuse. Am J For Med Pathology, 7(1): 81-83

Sax NI (1982), ed. Dangerous properties of industrial materials report, 2. New York, Van Nostrand Reinhold Co No 2, 50-52

Schick JB, Milstein JM (1981). Burn hazard of isopropyl alcohol in the neonate. Pediatrics 68: 587-588

Scrimgeour EM (1980). Outbreak of methanol and isopropanol poisoning in New Britain, Papua New Guinea. Med J Austr 7 July: 36-38

Senz EH, Goldfarb DL (1958). Coma in a child following use of isopropyl alcohol in sponging. J Pediatr 53: 322-323

Shepard TH (2986). Catalog of teratogenic agents, 5th ed. Baltimore and London, The John Hopkins University Press, p 329

Sittig, Marshall (1985). Handbook of toxic and hazardous chemicals and carcinogens, 2nd edn Park Ridge, NJ, USA, Noyes Publications, pp 532-534

Soukiasian SH et al (1988). A complication from alcohol-swabbed tonometer tips. Am J Ophthalmology 105(4): 424-425

Teramoto K, Horiguchi S et al (1987). 2-propanol and acetone elimination via exhaled air after 2-propanol administration to rats. Osaka City Med J 33(2): 153-160

US Environmental Protection Agency (1979). Chemical Hazard Information Profile: Isopropyl alcohol. Washington, DC (December 29, 1977), also revised edition (1979)

Vasiliades J et al (1978). Pitfalls of alcohol dehydrogenase procedure for the emergency assay of alcohol. A case study of isopropyl overdose. Clin Chem 24: 383-385

Visudhiphan P, Kaufman H (1971). Increased cerebrospinal fluid protein following isopropyl alcohol intoxications. NY State J Med 71: 887-888

Wallgren H (1960). Relative intoxicating effects in rats of ethyl, propyl, and butyl alcohols. Acta Pharmacol Toxicol 16: 217-222

Weil CS, Smyth HF, Nale TW (1952). Quest for industrial carcinogen. Arch Ind Hyg Occup Med 5: 535-547

Webster HC et al (1985). Diagnostic clinical osmometry in the unconscious infant. Critical Care Medicine 13(12): 1076-1077

Weintraub Z, Iancu TC (1982). Isopropyl alcohol burns (letter). Pediatrics 69(4): 506

Wimer WW, Russel JA, Kaplan HL (1983). Alcohols toxicology, Park Ridge, NJ: Noyes Data Corporation, pp 46-55

Wise IR (1969) Alcohol sponge baths (letter) New Engl J Med 280: 840.

14. AUTHOR(S), REVIEWER(S), DATE(S), COMPLETE ADDRESS(ES)

Authors: Dr Peter Hayes
Lecturer in Pharmaceutical
Technology/Manager Production Services
Department of Pharmacy
Faculty of Medicine
University of Otago
PO Box 913
Dunedin
New Zealand

Tel: 64-3-797272/740999 Ext 8347
Fax: 64-3-797618

Trudi P Martin, MPS

Senior Pharmacist
Pharmacy Production Unit
Dunedin Hospital
Private Bag
201 Gt King Street
Dunedin
New Zealand

Tel: 64-3-740999 Ext 8347
Fax: 64-3-797618

Date: February 1990

Reviewer: Dr Per Kulling
Swedish Poison Information Centre
Karolinska Hospital
Box 60500
10401 Stockholm
Sweden

Tel: 46-8-338765
Fax: 46-8-327584

Date: March 1990

Peer Review: Strasbourg, France, April 1990

Editor: Dr M. Ruse (October, 1997)

See Also:
- Toxicological Abbreviations
- Isopropyl alcohol (ICSC)
- ISOPROPYL ALCOHOL (JECFA Evaluation)