Phenol

PHENOL

International Programme on Chemical Safety
Poisons Information Monograph 412
Chemical

1. NAME

1.1 Substance

Phenol

1.2 Group

Hydrocarbons, cyclic, alcohol

1.3 Synonyms

acidum carbolicum;
acidum phenolicum;
acidum phenylicum;
baker's P & S liquid ointment;
benzaphenol;
benzene phenol;
benzenol carbolic acid;
carbolic acid;
hydroxybenzene (IUPAC);
monohydroxybenzene;
monophenol;
NCI-C50124;
Oxybenzene;
phenic acid;
phenol alcohol;
phenyl hydrate;
phenyl hydroxide;
phenylic acid;
phenylic alcohol

1.4 Identification numbers

1.4.1 CAS number

108-95-2

1.4.2 Other numbers

NIOSH number: SJ 3325000
Hazchem code: 2X
DOT: 1671/2312/2821

1.5 Main brand names, main trade names

1.6 Main manufacturers, main importers

2. SUMMARY

2.1 Main risks and target organs

Phenol exerts a marked corrosive action on any tissue of contact when ingested, inhaled or after skin exposure. Its cellular uptake is both rapid and passive due to its lipophilic character, and signs of systemic toxicity develop soon after exposure. Phenol's main target organs are the liver and kidney. It may also effect the respiratory and cardiovascular systems.

2.2 Summary of clinical effects

After ingestion phenol produces burning pain and white necrotic lesions in the mouth, oesophagus and stomach, vomiting and bloody diarrhoea. After skin exposure, pain is followed by numbness and the skin becomes blanched. The systemic clinical effects of phenol are independent on the route of exposure, they include: headache, dizziness, hypotension, ventricular arrhythmia, shallow respiration, cyanosis, pallor; excitation and convulsions may occur initially, but it is quickly followed by unconsciousness. A fall in body temperature and pulmonary oedema may occur. Methemoglobinemia and hemolytic anemia have been reported occasionally. The most important effects in short-term animal studies are neurotoxicity, liver and kidney damage and respiratory effects. The available data do not suggest a strong potential for cumulative health effects from chronic exposure.

2.3 Diagnosis

Phenol poisoning can be recognised by the characteristic acrid odour on the breath or it can be detected in the urine, which may be dark coloured. Therefore a urine sample and also a blood sample should be taken. Phenol is corrosive to mucous membranes, eyes and causes burns to the skin.

2.4 First aid measures and management principles

EYES: Immediately flush eye(s) with water (preferably tepid) for at least 15 minutes.

INHALATION: Remove patient from the area of exposure to fresh air. If unconscious, intubate with a cuffed endo-tracheal tube and ventilate mechanically if necessary. If conscious, place in Trendelenburg, left lateral position with succion equipment ready. Treat convulsions, arrhythmias and methaemoglobinemia according to Treatment Guides.

INGESTION: Do not induce vomiting. Do not dilute since it may increase absorption. Gastric aspiration or lavage should be weighed with risk. Give polyethylene glycol solution or activated charcoal with sorbitol.

DERMAL: Wearing protective gloves remove contaminated clothing immediately, flush excess chemical off the skin with water if it is the only available liquid, but preferably wash with polyethyleneglycol molecular weight 300 (Macrogol 300), isopropyl alcohol, industrial methylated spirits or Golytely for at least 30 minutes.

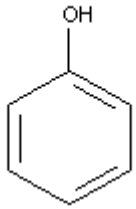
In all cases of exposure the patient should be transferred to a hospital as soon as possible.

3. PHYSICO-CHEMICAL PROPERTIES

3.1 Origin of the substance

Natural, obtained from coal tar, or as a degradation product of benzene. Synthetic, made by fusing sodium benzenesulfonate with NaOH, or by heating monochlorobenzene with aqueous NaOH under high pressure (Windholz, 1983)

3.2 Chemical structure



Chemical formula: C_6H_6O
Molecular weight: 94.11

3.3 Physical properties

3.3.1 Colour

Colourless or white (WHO, 1994).

3.3.2 State/form

Acicular-crystals, or white crystalline mass (WHO, 1994).

3.3.3 Description

The crystals turn pink or red on exposure to air and light, hastened in presence of alkalinity (Windholz, 1983). Phenol has an acrid smell and a sharp burning taste.

In the molten state, it is a clear, colourless liquid with a low viscosity. It is soluble in most organic solvents, and solubility is limited in aliphatic

solvents. Phenol's solubility. In water is limited at room temperature, above 68°C it is entirely water soluble. The vapour is heavier than air (WHO, 1994).

Melting point is 43°C, the commercial product has an impurity that increases the melting point (Windholz, 1983).

Boiling point: 181.75°C
Flash point: 80°C (closed cup); 79°C to 85°C (open cup)
Relative vapour density: 3.24 (WHO, 1994).
Explosive limits are 1.7% to 8.6% (Allen, 1991).
Vapour pressure at 20°C is 47 Pascal
pKa 10,0 at 25°C
pH of aqueous solutions is approximately 6.0
It is liquefied by mixing with about 8% water (Windholz, 1983).

3.4 Hazardous characteristics

Autoignition temperature is 715°C. Phenol is a volatile, combustible solid that when heated gives off flammable vapours and carbon dioxide. Explosive or violent reactions occur with acetylaldehyde; aluminium chloride plus nitrobenzene; aluminium chloride-nitromethane; butadine; calcium hypochlorite; peroxomonosulphuric acid; peroxodisulphuric acid; sodium nitrate; and sodium nitrate-trifluoroacetic acid (Allen, 1991).

Phenol is sensitive to oxidising agents. Splitting of the hydrogen atom from the phenolic hydroxyl group is followed by resonance stabilisation of the resulting phenyloxy radical. The radical that is formed can be further oxidised. This makes phenol suitable as an antioxidant, functioning as a radical trapping agent (WHO, 1994). The taste threshold is 0.3 mg/L (0.00003%) in water (WHO, 1994).

4. USES

4.1 Uses

4.1.1 Uses

4.1.2 Description

The main use of phenol is as a feedstock for phenolic resins, bisphenol A and caprolactam (an intermediate in the production of nylon-6). It is used in the manufacture of many products including insulation materials, adhesives, lacquers, paint, rubber, ink, dyes, illuminating gases, perfumes, soaps and toys (IARC, 1989; WHO, 1994). Also used in embalming and research laboratories. It is a product of the decomposition of organic materials, liquid manure, and the atmospheric degradation of benzene.

It is found in some commercial disinfectants, antiseptics, lotions and ointments. Phenol is active against a wide range of microorganisms, and there are some medical and pharmaceutical applications including topical anaesthetic and ear drops, sclerosing agent.

It is also used in the treatment of ingrown nails in the "nail matrix phenolization method" (Kimata et al., 1995). Another medical application of phenol is its use as a neurolytic agent, applied in order to relieve spasms and chronic pain (Wood, 1978; Geller, 1997).

It is used in dermatology for chemical face peeling.

4.2 High risk circumstance of poisoning

Deliberate, accidental or occupational exposure.

4.3 Occupationally exposed populations

Workers involved in the production of phenol by the cumene process, or the production of phenol from chlorobenzene are at risk. Phenol may be emitted into the air during the processing of phenolic resins, production of phenol and phenol derivatives, production of caprolactam, production of cokes and insulation materials. Wood workers, and workers at plywood plants are at risk of exposure. Phenol is used in iron and steel foundries - in the manufacture of moulds or kernels, or during the operation of an electric furnace in a steel factory and also in coal gasification and liquefication plants, bakelite factories, and synthetic fibre

and fibrous glass wool factories. In creosote impregnation plants, with highest exposure levels during the cleaning of creosote warming chambers. In embalming situations, where the embalming solution contains high phenol concentrations (Allen, 1991; WHO, 1994).

In chemical accidents, responders or health staff may be exposed by direct contact, by vomitus, or by off-gassing of contaminated clothing.

5. ROUTES OF EXPOSURE

5.1 Oral

Phenol is readily absorbed from the gastrointestinal tract (WHO, 1994).

5.2 Inhalation

Phenol is readily absorbed from the lungs (Allen, 1991).

5.3 Dermal

When spilt on the skin, intact or abraded, it is rapidly absorbed and may lead to systemic poisoning (Brooks & Riviere, 1996).

5.4 Eye

Phenol is absorbed through the mucous membranes of the eye (WHO, 1994).

5.5 Parenteral

Therapeutic use: phenol can be administered by intrathecal injection to relieve pain and spasticity (Geller,

1997), and has been used as a sclerosing agent.

5.6 Other

No data available.

6. KINETICS

6.1 Absorption by route of exposure

Phenol is rapidly absorbed through skin, lungs, and the gastrointestinal tract.

Eight humans exposed to 6 to 20 mg/m^3 by inhalation for 8 hours absorbed 70 to 80% of the phenol dose.

Skin absorption of phenol vapour (5 to 25 mg/m^3) occurs rapidly. The absorption in 8 humans exposed to phenol vapour at concentrations of 6 to 20 mg/m^3 , by skin only, for 6 hours was also 70 to 80%. Concentrations between 5 and 10% phenol denature epidermal protein and this can therefore partly prevent absorption. The phenol-protein complex is not stable and by dissociation of phenol the substance may exert its action over a period of time. In an *in vitro* study with human abdominal skin, 10.9% of the dose was absorbed. The period of exposure and the concentration of phenol are both factors that determine the extent of absorption, but the area of skin exposed affects the extent of absorption more than the concentration. A single dose of 25 mg/kg body weight dermally administered to rats, pigs and sheep was more than 95% absorbed (WHO, 1994).

6.2 Distribution by route of exposure

Rapidly distributed to all tissues in exposed animals. After a single oral administration of 207 mg/kg phenol to rats, the highest concentration ratios between tissue and plasma were found in the liver (42%), followed by spleen, kidney, adrenal, thyroid and lungs, with a peak tissue level occurring after 0.5 hours.

In rabbits 15 minutes after an oral dose of 0.5 g/kg, the highest concentrations of phenol were in the liver, followed by the CNS, lungs and blood. After 82 minutes the phenol was relatively uniformly distributed in all tissues (WHO, 1994).

6.3 Biological half-life by route of exposure

The half-life of conjugated phenol in humans is 1 hour (Leikin & Paloucek, 1996-7), but there have also been reported half-lives of 4 to 5 hours in humans (WHO, 1994).

6.4 Metabolism

After oral uptake of phenol, there is a large first-pass metabolism. It is unclear whether phenol also undergoes first pass pulmonary metabolism, there have been conflicting results (Dickenson & Taylor, 1996). The liver, lungs and the gastrointestinal mucosa are the most important sites of

phenol metabolism (WHO, 1994). Conjugation with glucuronic

acid to phenyl glucuronide and sulphation to phenyl sulphate, have been shown to be major metabolic pathways in several species. A shift from sulphation to glucuronidation was observed in rats after increasing the phenol doses, which is thought to be due to a saturation of the overall sulphation process, by the limited availability of 3-phosphoadenosine-5-phosphosulfate. The formation of sulphate and glucuronic metabolites occurs in the hepatocytes, and then transported to the bile or back into the blood (Ballinger et al, 1995). *In vitro* studies have shown the formation of the reactive metabolites 4,4'-biphenol and diphenoquinone by neutrophils and activated leukocytes. Both in vivo and in vitro tests have shown covalent binding of phenol to tissue and plasma proteins, some phenol metabolites also bind to proteins (WHO, 1994).

6.5 Elimination and excretion

Urinary (renal) excretion is the major route of phenol elimination in animals and humans. The rate of excretion varies with different species, dose and route of administration. Three men after an oral administration of 0.01 mg/kg phenol, excreted 90% of the dose in the urine within 24 hours, mainly as phenyl sulfate and phenyl glucuronide. A minor part is eliminated in the faeces and expired air. Urinary excretion of humans exposed to phenol vapour via inhalation or skin, occurred with an excretion rate constant of k 0.2/hour. On oxidation to quinones the metabolites may tint the urine green.

The half life is estimated to be between 1 and 4.5 hours with 52% eliminated unchanged in the urine (Leikin & Paloucek, 1996-7). The natural presence of phenols in food and drug metabolites, makes biological monitoring impossible. A minor part is eliminated in expired air and faeces. (Reynolds, 1993; Ellenhorn & Barceloux, 1988)

7. TOXICOLOGY

7.1 Mode of action

Cellular uptake of phenol is due to its lipophilic character. It denatures proteins (WHO, 1994). Phenol is known to disrupt disulphide bridges in keratin in the skin (Brooks & Riviere, 1996).

In vitro studies have shown the formation of the reactive metabolites 4,4'-biphenol and diphenoquinone by neutrophils

and activated leukocytes. Both *in vivo* and *in vitro* tests have shown covalent binding of phenol to tissue and plasma protein, some phenol metabolites also bind to proteins (WHO, 1994). It produces coagulation necrosis.

The acute lethality of phenol, associated with exposure to high dose concentrations, is customarily attributed to a depressant effect on the CNS.

7.2 Toxicity

7.2.1 Human data

7.2.1.1 Adults

The lethal dose ranges from 1 g to 15 g (Reynolds, 1993). Ingestion of 4.8 g resulted in death after 10 minutes in one person (Anderson, 1869).

However, an adult survived a 26.7 g dose ingestion after a 15-day stormy hospital course without permanent sequellae (Haddad et al., 1979).

Survival has been reported with up to 350 mg/kg orally (Christiansen & Klaman, 1996).

7.2.1.2 Children

Children have died after the application of 5 % phenol compresses (Ellenhorn, 1996).

7.2.2 Relevant animal data

LD_{50} (oral) rodent values ranged from 300 to 600 mg/kg body weight.

LD_{50} (dermal) values for rats and rabbits range from 670 to1400 mg/kg body weight respectively.

LC_{50} (8 hour) for rats by inhalation was more than 900 mg/m^3 (WHO, 1994).

In two multiple dose rat studies, NOAEL values obtained were 40 mg/kg/day and 60 mg/kg /day and the

LOAEL values were 53 mg/kg/day and 120 mg/kg/day. In a mouse study the NOAEL was 140 mg/kg/day and the LOAEL was 280 mg/kg/day (WHO, 1994).

Chronic vapour exposures in rats (0.02 to 1ppm for 2 months) produced changes in the blood enzyme activity and time for excitation of extensor muscles. At higher exposures, phenol may lead to decrease in body weight. In various animal species the inhalation of phenol affected the lungs by causing hyperaemia, infarcts, pneumonia, purulent bronchitis and hyperplasia of the peribronchial tissues (WHO, 1994). There does not appear to be a strong potential for cumulative health effects from chronic exposure (WHO, 1994)

7.2.3 Relevant in vitro data

No data available.

7.2.4 Workplace standards

OSHA PEL: TWA 5 ppm (skin)
ACGIH TLV: TWA 5 ppm (skin)
IDLN: 100 ppm
DFG MAK: 5 ppm (19 mg/m^3)
NIOSH REL: TWA 20 mg/m^3; CL 60 mg/m^3/15 minutes
(Sax & Lewis, 1989)

In Britain the occupational exposure standard is 19

mg/m^3 (long term) and 38 mg/m^3 (short term). In the United States the permissible level is 19 mg/m^3 and the recommended is 20 mg/m^3 (long-term) and the maximum short term is 60 mg/m^3 (Reynolds, 1993).

7.2.5 Acceptable daily intake (ADI)

A Task Group derived a tolerable daily intake (TDI) using the lowest NOAEL's for kidney and developmental effects in rats which is in the range of 12 to 40 mg/kg body weight /day. With an uncertainty factor of 200, the range of 60 to 200 µg/kg/day was recommended as the upper limit of the TDI (WHO, 1994)

The estimated maximal total daily intake of phenol for a 70 kg individual is calculated to be 0.1 mg/kg body weight per day (WHO, 1994).

However, according to IRIS (Integrated Risk Information Sytem, 1996), the reference dose (Rfd) is 0.6 mg/kg/day, using the NOAEL (reduced fetal body weight in rats) of 60 mg/kg/day, with an uncertainty factor (Uf) of 100.

7.3 Carcinogenicity

An IARC review in 1989 found that the carcinogenicity evidence for phenol was inadequate (group 3) (WHO, 1994). US EPA classifies phenol in group D.

Two-stage carcinogenicity studies have shown that phenol, applied repeatedly to mouse skin, has promoting activity (WHO, 1994).

7.4 Teratogenicity

Phenol has been identified as a developmental toxicant in studies with rats and mice (WHO, 1994).

7.5 Mutagenicity

The majority of bacterial mutagenicity tests have demonstrated negative results. In mammalian cells, mutations, chromosomal damage and DNA effects have been observed. Phenol has shown no effect on intercellular communication in cultured mammalian cells. The induction of micronuclei in bone marrow cells of mice has been observed in some studies at high doses. No micronuclei were observed in mice studies at lower dose (IARC, 1989; WHO, 1994).

7.6 Interactions

No data available.

8. TOXICOLOGICAL AND BIOMEDICAL INVESTIGATIONS

8.1 Material sampling plan

8.1.1 Sampling and specimen collection

8.1.1.1 Toxicological analyses

After swallowing a significant concentrated dose, an intense burning of the mouth and throat is felt (necrosis of the skin and mucous membranes of the throat), and pain in the abdominal area, with gastrointestinal irritation including nausea, vomiting, sweating and diarrhoea. The face is usually pale and sweaty, the pupils may be contracted or dilated; cyanosis is usually marked; the pulse is usually weak and slow, occasionally it may be racing; respiration may initially be increased in rate, but later decreased in rate and magnitude; body temperature may fluctuate. Excitation may occur initially, but it is quickly followed by unconsciousness. Occasionally isolated twitching of muscles or convulsions may be observed. Acute renal failure can develop resulting from systemic absorption. Ingestion is usually fatal (Foxall et al., 1989; Allen, 1991; Reynolds, 1993; WHO, 1994).

9.1.2 Inhalation

Phenol vapours are irritating to the upper respiratory tract. Ocular and nasal irritation, tremors and incoordination were reported in rats exposed to phenol via inhalation to 906 mg/m^3 for 8 hours (WHO, 1994). Wheezing may occur. Other symptoms associated with inhalation include anorexia, weight loss, headache, salivation, vertigo (WHO, 1994) and dark urine (dark/brown/green) (Leikin & Paloucek, 1996-7).

9.1.3 Skin exposure

Phenol is a local anaesthetic, so upon initial contact, no pain is felt. By the time pain is felt, serious burns and absorption through the skin may have occurred (Allen, 1991). Local damage to the skin includes erythema, inflammation, and necrosis. The effects are worse when the application sites are bandaged (WHO, 1994). A white, brown or red discolouration of the skin may occur (Leikin &

Paloucek 1996-7). Systemic intoxication can occur from absorption (WHO, 1994); roughly 50 % of all reported cases have a fatal outcome (Horch et al., 1994).

In the Kligman maximization test phenol did not cause sensitisation in 24 human volunteers (Kligman, 1966).

9.1.4 Eye contact

Phenol is an eye irritant (WHO, 1994). Solutions can be corrosive to the eyes, and can cause severe ocular damage including corneal opacification.

9.1.5 Parenteral exposure

After intraperitoneal and subcutaneous doses of phenol, tremors, convulsions, coma and death have been reported (WHO, 1994). Injection of 30 mL of 89% phenol instead of 10% for celiac plexus nerve block resulted in coma, hypotension, respiratory insufficiency and ventricular tachycardia (Christiansen & Klaman, 1996).

9.1.6 Other

No data available.

9.2 Chronic poisoning

9.2.1 Ingestion

Severe gastrointestinal irritation, cardiovascular, CNS and respiratory effects, hypothermia and decreased body weight. Brown or discoloured urine has also been observed in chronic poisoning (Goldfrank & Bresnitz, 1990).

Repeated oral exposure for several weeks (estimated intake 10 to 240 mg/day) resulted in mouth sores, diarrhea and dark urine. Examination 6 months after the exposure revealed no residual effects (Baker et al., 1978).

9.2.2 Inhalation

There does not appear to be a strong potential for cumulative health effects from chronic exposure (WHO, 1994)

9.2.3 Skin exposure

Chronic doses may result in onychronosis (yellowing of the skin) and skin eruption (WHO, 1994). Death has been observed from repeated application of small doses (Olson, 1994).

In former times phenol 5 to 10% was used as a skin disinfectant giving rise to the "carbolic marasmus" characterized by anorexia, headache, vertigo, salivation, dark urine and increased skin and scleral pigmentation (Merliss, 1972).

9.2.4 Eye contact

No data available

9.2.5 Parenteral exposure

No data available.

9.2.6 Other

No data available.

9.3 Course, prognosis, cause of death

Hypotension, renal failure, apnea, laryngeal oedema and ARDS can develop soon after exposure leading to death (WHO, 1994). Coma and seizures usually occur within minutes to a few hours after exposure. Toxic effects may be delayed up to 18 hours.

9.4 Systematic description of clinical effects

9.4.1 Cardiovascular

Heart rate at first increases and then becomes slow and irregular. Blood pressure at first increases slightly, then falls markedly (hypotension) (WHO, 1994). Cardiovascular collapse, atrial and ventricular arrythmias have been reported (Leikin & Paloucek, 1996-7). Deep venous thrombosis has been reported following injection of phenol (WHO, 1994).

Cardiac dysrhythmias have been observed in skin peeling and nerve blockade (Forrest & Ramage, 1987; Lober, 1987; Sorkin, 1988; Gaudy et al., 1993; Lalanne et al., 1994; Zamponi & French, 1994).

9.4.2 Respiratory

Respiration may initially be increased in rate, but later decreased in rate. Pulmonary oedema, wheezing, coughing, dyspnea, pneumonia are common signs (Leikin & Paloucek, 1996-7). The cause of death from phenol exposure is often respiratory failure (WHO, 1994).

9.4.3 Neurological

9.4.3.1 Central nervous system (CNS)

Initial signs and symptoms include headache, dizziness and tinnitus. Seizures, coma, respiratory depression and death may ensue quickly. Coma and seizures usually occur within minutes to a few hours after exposure or a delay of up to 18 hours. Phenol may also cause demyelination and axonal damage of peripheral nerves (WHO, 1994).

9.4.3.2 Peripheral nervous system

No data available.

9.4.3.3 Autonomic nervous system

A decrease in body temperature has been reported (WHO, 1994).

9.4.3.4 Skeletal and smooth muscle

Locomotor activity reduced at 244 mg phenol/kg body weight in female Fischer-344 rats (WHO, 1994). Chronic exposure in rats lead to changes in the time for excitation of extensor muscles.

9.4.4 Gastrointestinal

Symptoms include diarrhoea, salivation, vomiting, ulceration and haemorrhage (Leikin & Paloucek, 1996-7). Corrosive damage may involve the entire gastrointestinal tract.

9.4.5 Hepatic

Hepatic necrosis was observed in two(out of six) female Fischer-344 rats when given 244 mg phenol/kg body weight (WHO, 1994).

9.4.6 Urinary

9.4.6.1 Renal

Renal failure has been reported in acute poisoning. Urinalysis may reveal a green to brown discolouration of the urine with albuminuria. Nephritis is reported (Leikin & Paloucek, 1996-7).

9.4.6.2 Other

No data available.

9.4.7 Endocrine and reproductive systems

Increased incidence of preimplantation loss and early postnatal death in the offspring of rats (WHO, 1994).

9.4.8 Dermatological

Chronic doses may result in ochronosis (yellowing of the skin) and skin eruption (WHO, 1994).

9.4.9 Eye, ear, nose, throat: local effects

The fumes are irritating to the eyes and affects the pupil's response to light (miosis) (WHO, 1994). Solutions can be corrosive to the eyes, and can cause severe ocular damage including corneal opacification. Lymph production in the conjunctiva may be increased and will leave the cornea white and hypesthetic (Jaeger, 1987).

Necrosis of the mucous membranes of the throat.

Phenol applied to the inner ear round window of Sprague-Dawley rats caused morphological damage to the organ of Corti in the basal coil. The outer hair cells appeared to be more sensitive to phenol and as a result of the damage, impairment of inner ear function was noted which was permanent for higher frequencies. One experiment in female mice lead to an increase in ear thickness (WHO, 1994)

9.4.10 Haematological

Heinz body haemolytic anaemia and hyperbilirubinemia have been reported occasionally (WHO, 1994).

9.4.11 Immunological

There are no studies in humans. For four weeks groups of five male CD-1 mice were given drinking water containing 0, 4.7, 19.5 or 95.2 mg phenol/L. Total and differential leukocyte counts were unaffected. The highest dose suppressed the stimulation of cultured splenic lymphocytes by the B-cell mitogen lipopolysaccharide, the T-cell mitogen phytohaemagglutinin, and the T and B-cell mitogen pokeweed, but not by concanavatin. Suppression of the animal's antibody production in response to a T-cell-dependent antigen, occurred at the mid and high doses (WHO, 1994).

9.4.12 Metabolic

9.4.12.1 Acid-base disturbances

Metabolic acidosis (WHO, 1994)

9.4.12.2 Fluid and electrolyte disturbances

Fluid loss secondary to burns or shock.

9.4.12.3 Others

No data available.

9.4.13 Allergic reaction

No data available.

9.4.14 Other clinical effects

No data available

9.4.15 Special risks

Phenol has been shown to be a developmental toxicant in rats and mice (WHO, 1994).

9.5 Other

No data available.

9.6 Summary

10. MANAGEMENT

10.1 General principles

When spilt on the skin or in the eyes, there should be an immediate washing with water (preferably tepid) for at least 10 minutes. If available wash with polyethyleneglycol molecular weight 300 (Macrogol 300), isopropyl alcohol, industrial methylated spirits or Goletely (PEG 3550) for at least 30 minutes (Horch et al., 1994).

Do not induce vomiting. Dilution may increase absorption. Gastric lavage should be carefully weighed against the risk of complications. Recommended gastric lavage fluids are polyethylene glycol, water, following administration of activated charcoal, or vegetable oils, such as olive oil, castor oil or cottonseed oil (WHO, 1994).

Treatment is mainly supportive. If there is a systemic intoxication, monitor the respiration and the level of oxygenation, the blood pressure and ECG, the level of methaemoglobinemia, the hepatic and renal functions. Control convulsions and cardiac arrhythmias according to the Treatment Guides.

10.2 Life supportive procedures and symptomatic/specific treatment

Make a proper assessment of airway, breathing, circulation and neurological status.
Maintain a clear airway.

If unconscious give artificial respiration.
If the patient has breathing difficulties, put them in a sitting position.
Monitor vital signs.
Monitor blood pressure and ECG.
Monitor fluid and electrolyte balance.
Monitor acid-base balance.
Control cardiac dysrhythmias with appropriate drug regimen.
Control convulsions with appropriate drug regimen.

10.3 Decontamination

Remove and discard contaminated clothing.
Irrigate exposed eyes with copious amounts of water.
Wash skin with copious amounts of water or preferably if available wash with polyethyleneglycol molecular weight 300 (Macrogol 300), isopropyl alcohol, industrial methylated spirits or Goletely (PEG 3550) for at least 30 minutes.
Do not induce vomiting, empty stomach by aspiration followed by polyethylene glycol or activated charcoal with cathartic.
Endoscopy.

10.4 Enhanced elimination

If acute renal failure occurs in phenol poisoning, dialysis should probably not be used alone, but in conjunction with charcoal hemoperfusion. Without renal failure the use of charcoal hemoperfusion may also be useful, when the patient has been exposed to 15 to 20 g of phenol (Christiansen & Klaman, 1996)

10.5 Antidote treatment

10.5.1 Adults

No antidote available.

10.5.2 Children

No antidote available.

10.6 Management discussion

No data available.

11. ILLUSTRATIVE CASES

11.1 Case reports from the literature

Occupational - Male

A 27-year-old male spilt 80% phenol on both knees and arrived at the emergency department 30 to 60 minutes after the spillage. Both legs had been washed with copious amounts of water and he had also undergone further irrigation and application of glycerin cream, but continued to be in pain. Further irrigation with 6 litres of saline followed and then it was suggested to irrigate with polyethylene glycol. By mistake Golytely (PEG 3550) was used. The patient reported an immediate soothing of the pain and his long term recovery was favourable (Wahl et al., 1995).

Accidental Injection

A 50-year-old woman inadvertently received a 30 ml dose of 89% phenol (26.9 g, 0.44 mg/kg body weight). At 23 minutes post- injection she was unresponsive, at 27 minutes she had respiratory distress and was intubated. At 62 minutes she developed shock and dopamine was started followed by epinephrine and neosynephrine. At the same time she developed ventricular tachycardia and was treated with lidocaine. 4 hours after the injection she was comatose with a blood pressure of 70/58, while receiving large doses of vasopressors. At 4.5 hours charcoal hemoperfusion was started, and given for 6 hours and 20 minutes. Her clinical status improved with the perfusion and she made a complete recovery. The perfusion enhanced the elimination of free and total phenol (Christiansen & Klaman, 1996).

Five Year Acute Exposure Study

A five year evaluation of acute exposure to phenol disinfectant (26%) studied 80 cases with an age range of 1 to 78 years, 75% under 5 years old. There were 60 oral only exposures, 7 dermal only, 12 oral/dermal and 1 inhalation. 65% were assessed at the emergency department and 33% were admitted. 14% of oral exposures developed rapid CNS depression, without seizures, and 2 patients developed coma after ingestion. Burns occurred in 17 oral exposures and 5 dermal. 17 patients underwent endoscopy. Urine colour change was noted in 5 patients following ingestion. There were no cardiovascular complications, or oliguria and anuria. In all cases there were complete recoveries (Spiller et al., 1993).

Accidental Ingestion - Male Alcoholic, Addict

A report from the Invercargill hospital in New Zealand early in 1997, reports a 29 year old male, alcoholic, addict, who drank 30 mL of 88% phenol from a bottle he found (26.4 g/75 kg = 350 mg/kg). He had a cardiac arrest requiring DC cardioversion, and developed renal failure secondary to rhabdomolysis. He developed a denuded oesophagus, large, superficial, antral ulceration and received continuous haemofiltration and endoscopy. His urine output only recommenced 14 days after the ingestion and his renal function is expected to return to normal (NZNPIC, 1997).

Ingestion - Female

Fatal case reports include a 21 year old female who drank 10 to 20 g of phenol. She went into a deep coma with partial areflexia, a heart rate of 140 and dilated pupils, and had a cardiac arrest 60 minutes post ingestion. She received repeated gastric lavage with water, glycerin and animal charcoal, but died from pulmonary oedema and shock (Stajduhar-Carie, 1968).

Occupational - Fatal Dermal Exposure

A 17-year-old male had 30% phenol (as industrial waste) splashed on his face, neck and right trunk. He was washed with water, but 30 minutes later he had a seizure and died. His blood phenol level was 2.7 mg/dL. An autopsy showed red areas to 15% of his skin area, and pulmonary oedema. (WHO, 1994)

Accidental Immersion

A man was partially submerged in a solution of 20% phenol in dichloromethane for a few seconds. He immediately showered , but collapsed, his extremities were cold and he had 50% burns to his body. He developed acute renal failure. Anuria followed, with a rise in plasma creatinine, but treatment with intravenous furosemide and haemodyalisis (daily for seven days, then with decreasing intervals for a further 18 days), allowed adequate urinary volumes to be produced. He also had respiratory distress, treated in intensive care. A spill of 80-100% phenol on the hip, thigh and scrotum with a contact duration of 20 minutes lead to death, while a reported spill of 43.5% to the lower half of the body resulted in shock. A spill of 4 to 5 litres on the upper half of the body of 78%, with a contact duration of 2 to 5 minutes resulted in a coma (WHO, 1994).

Contaminated Drinking Water

A chemical spill in Wisconsin in 1974 contaminated groundwater which was being used as drinking water. One month later there were complaints of health effects, and six months later medical histories were taken from 100 people. The estimated daily exposure was 10 to 240 mg phenol/person. A significantly significant increase in diarrhoea, mouth sores, dark urine and burning of the mouth was found. Gastrointestinal illnesses were also reported in North Wales, when a river used for the preparation of drinking water was contaminated with phenol, and when chlorinated, various chlorophenols formed (WHO, 1994)

Accidental Ingestion

An outpatient was mistakenly given in a measured container one ounce of 89 % phenol. The patient immediatly clutched her throat and collapsed. Within 30 minutes she had an unrecordable blood pressure and sustained respiratory arrest. During endotracheal intubation in the ED the mouth and hypopharynx were noted to be white. A "lamp oil" odour was noted while ventilating the patient with a bag mask. The patient experienced ventricular tachycardia one hour after ingestion and resuscitation was effected by cardioversion. Over the first 24 hours she exhibited ventricular arrhythmia, seizures and metabolic acidosis. (Haddad et al., 1979)

Child Exposure

A 10-year-old boy had a solution of 40% phenol and 0.8% croton oil in hexachlorophene soap and water applied to a large nevus covering his 1.9% of his body surface whilst under anaesthesia. After 55 minutes of treatment, multifocal and coupled premature ventricular complexes were detected by ECG. An intravenous infusion of 250 mg bretylium sulfate suppressed the dysrhythmia (WHO, 1994).

12. ADDITIONAL INFORMATION

12.1 Specific preventative measures

Phenol should be kept in a tightly closed container, in a cool, dry place, away from heat, flame and oxidising agents. It is light sensitive and should be kept in the dark (WHO, 1994).

Protective clothing should will be appropriate to the amount and form of the phenol being handled. It should be handled wearing an approved respirator; viton, butyl rubber or neoprene gloves (not nitrile or PVA gloves), safety goggles and other protective clothing. Safety showers and

polyethylene glycol 300 should be near where phenol is being handled.(Allen, 1991)

12.2 Other

Phenol is not likely to persist in air, soil or sewage, sea or surface water. It readily reacts photochemically, is rapidly biodegraded aerobically to mainly carbon dioxide, and anaerobic biodegradation occurs also at a slower rate. Low removal rates of phenol in ground water and soil may occur e.g. following spills, with subsequent inhibition of the microbial populations. Phenol is toxic to aquatic organisms: the lowest EC_{50} for water organisms is estimated to be 3.1 mg/L. The lowest chronic NOEC is estimated to be 0.2 (g/L).

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Date: August 1997

Peer review: Rio de Janeiro, Brazil, September 1997
(Group members: R. McKeown, W.A. Temple, W.A. Watson)

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Netherlands

Date: March 1998

Accepted: London, United Kingdom, March 1998
(Group members: R.E. Ferner, A. van Heijst, M. Mathieu-Nolf, A.J. Nantel, M.O. Rambourg Schepens (coordinator))

Editor: Mrs J. Duménil
International Programme on Chemical Safety

Date: October 1999

See Also:
- Toxicological Abbreviations
- Phenol (EHC 161, 1994)
- Phenol (HSG 88, 1994)
- Phenol (ICSC)
- PHENOL (JECFA Evaluation)
- Phenol (IARC Summary & Evaluation, Volume 71, 1999)