PHOSPHINE	ICSC: 0694

Date of Peer Review: August 1997

Phosphorus trihydride
Hydrogen phosphide

CAS #	**7803-51-2**	**PH_3**
RTECS #	**SY7525000**	**Molecular mass: 34.00**
UN #	**2199**	
EC Annex 1 Index #	**015-181-00-1**	
EC/EINECS #	**232-260-8**	

TYPES OF HAZARD / EXPOSURE	ACUTE HAZARDS / SYMPTOMS	PREVENTION	FIRST AID / FIRE FIGHTING
FIRE	Extremely flammable. Gives off irritating or toxic fumes (or gases) in a fire.	NO open flames, NO sparks, and NO smoking. NO contact with hot surfaces.	Shut off supply; if not possible and no risk to surroundings, let the fire burn itself out; in other cases extinguish with powder, carbon dioxide.
EXPLOSION	Gas/air mixtures are explosive.	Closed system, ventilation, explosion-proof electrical equipment and lighting.	In case of fire: keep cylinder cool by spraying with water. Combat fire from a sheltered position.

EXPOSURE		**STRICT HYGIENE!**	**IN ALL CASES CONSULT A DOCTOR!**
Inhalation	Cough. Nausea. Burning sensation. Diarrhoea. Abdominal pain. Headache. Dizziness. Dullness. Ataxia. Pain and tightness in the chest. Tremors. Shortness of breath. Vomiting. Convulsions.	Breathing protection.	Fresh air, rest. Half-upright position. Artificial respiration may be needed. Refer for medical attention.
Skin	ON CONTACT WITH LIQUID: FROSTBITE.	Cold-insulating gloves. Protective clothing.	ON FROSTBITE: rinse with plenty of water, do NOT remove clothes. Refer for medical attention.
Eyes		Safety goggles or eye protection in combination with breathing protection.	First rinse with plenty of water for several minutes (remove contact lenses if easily possible), then take to a doctor.
Ingestion			

SPILLAGE DISPOSAL	PACKAGING & LABELLING
Evacuate danger area! Consult an expert! Ventilation. Personal protection: chemical protection suit including self-contained breathing apparatus.	
EMERGENCY RESPONSE	**STORAGE**
Transport Emergency Card: TEC (R)-20G2TF NFPA Code: H3; F4; R2.	Fireproof.

IPCS
International Programme on Chemical Safety

Prepared in the context of cooperation between the International Programme on Chemical Safety and the Commission of the European Communities © IPCS, CEC 2005

SEE IMPORTANT INFORMATION ON BACK

PHOSPHINE ICSC: 0694

IMPORTANT DATA

PHYSICAL STATE; APPEARANCE:
COLOURLESS COMPRESSED LIQUEFIED GAS

PHYSICAL DANGERS:
The gas is heavier than air.

CHEMICAL DANGERS:
The substance decomposes on heating or on burning producing toxic fumes including phosphorus oxides. Reacts violently with air, oxygen, oxidants such as chlorine and nitrogen oxides, metal nitrates, halogens and other many substances, causing fire and explosion hazard. Attacks many metals.

OCCUPATIONAL EXPOSURE LIMITS:
TLV: 0.3 ppm as TWA, 1 ppm as STEL; (ACGIH 2005).
EU OEL: 0.1 ppm, 0.14 mg/m³ as TWA; 0.2 ppm, 0.28 mg/m³ as STEL (EU 2006).

ROUTES OF EXPOSURE:
The substance can be absorbed into the body by inhalation.

INHALATION RISK:
A harmful concentration of this gas in the air will be reached very quickly on loss of containment.

EFFECTS OF SHORT-TERM EXPOSURE:
The substance is severely irritating to the respiratory tract. Inhalation of the gas may cause lung oedema (see Notes). Rapid evaporation of the liquid may cause frostbite. The substance may cause effects on the central nervous system, cardiovascular system, heart, gastrointestinal tract, liver and kidneys , resulting in impaired functions. Exposure above the OEL may result in unconsciousness or death. The effects may be delayed. Medical observation is indicated.

EFFECTS OF LONG-TERM OR REPEATED EXPOSURE:
Chronic poisoning may cause toothache, swelling of the jaw, phossy jaw, spontaneous fractures of bones and anaemia. Effects are cumulative.

PHYSICAL PROPERTIES

Boiling point: -87.7℃
Melting point: -133℃
Relative density (water = 1): 0.8
Solubility in water, ml/100 ml at 17℃: 26
Vapour pressure, kPa at 20℃: 4186
Relative vapour density (air = 1): 1.17

Flash point: Flammable Gas
Auto-ignition temperature: 38℃
Explosive limits, vol% in air: 1.8-?

ENVIRONMENTAL DATA

NOTES

The technical product often ignites spontaneously at room temperature because of the presence of other phosphorus hydrides (especially P_2H_4) as impurities. Odourless when pure at concentrations up to 200 ppm, a highly toxic level. Technical product has a garlic-like odour due to impurities. The symptoms of lung oedema often do not become manifest until a few hours have passed and they are aggravated by physical effort. Rest and medical observation is therefore essential. Immediate administration of an appropriate inhalation therapy by a doctor or a person authorized by him/her, should be considered. The odour warning when the exposure limit value is exceeded is insufficient. Turn leaking cylinder with the leak up to prevent escape of gas in liquid state. Card has been partly updated in October 2005 & 2006. See sections: Occupational Exposure Limits, EU classification, Emergency Response. Card has been partially updated in August 2007: see Occupational Exposure Limits.

ADDITIONAL INFORMATION

PHOSPHINE

International Programme on Chemical Safety
Poisons Information Monograph 865
Chemical

1. NAME

1.1 Substance

Phosphine

1.2 Group

Phosphorous hydride

1.3 Synonyms

Hydrogen phosphide; trihydrogen phosphide; phosphorus trihydrides; phosphoretted hydrogen; 2phospane; celphos; delicia; detia; gas-ex-B; celphos

1.4 Identification numbers

1.4.1 CAS number

7803-51-2

1.4.2 Other numbers

DOT UN 2199 (DOT = Dept. of Transport)

1.5 Main brand names/Trade names

Al Pare Alutal; Celphide; Celphine; Celphos; Delicia Gas toxin; Detia Gas Ex-B/P/T; L-Fume; Phosphine; Phostex; Phostoxin; Quickfos; Zedesa

1.6 Main manufacturers/main importers

To be completed by each poison control centre.

2. SUMMARY

2.1 Main risks and target organs

Phosphine is a colourless gas which is odourless when pure, but the technical product has a foul odour, described as "fishy" or "garlicky", because of the presence of substituted phosphine and diphosphine (P_2H_4).

Other impurities may be methane, arsine, hydrogen and nitrogen. For fumigation, it is produced at the site of hydrolysis of a metal phosphide (AlP, Zn_3P_2, Mg_3P_2) and supplied in cylinders either as pure phosphine or diluted with nitrogen.

Phosphine is flammable and explosive in air and can autoignite at ambient temperatures. It is slightly soluble in water and soluble in most organic solvents. Metal

phosphides are usually powders of various colours, which hydrolyse to yield phosphine and metal salts.

Inhalation of phosphine may cause severe pulmonary irritation leading to acute pulmonary oedema, cardiovascular dysfunction, CNS excitation, coma and death. Gastrointestinal disorders, renal damage and leukopenia may also occur.

Exposure to 1400 mg/m^3 (1000 ppm) for 30 minutes may be fatal.

Ingestion of phosphides, particularly aluminum and zinc phosphides, may induce severe gastrointestinal irritation leading to haemorrhage, cardiovascular collapse, acute neuropsychiatric disorders, respiratory and renal failure within a few hours. Hepatic damage may develop later.

2.2 Summary of clinical effects

Initial clinical manifestations of mild phosphine inhalation mimic an upper respiratory tract infection. Other symptoms may include nausea, vomiting, diarrhoea, headache, fatigue and dizziness. In severe exposure, lung irritation with persistent coughing, ataxia, paraesthesia, tremor, diplopia and jaundice may also occur. Very severe cases may progress to acute pulmonary oedema, cardiac dysrhythmias, convulsions, cyanosis and coma. Oliguria, proteinuria and finally anuria may be induced.

Deliberate ingestion of phosphides, especially AID (Phostoxin), causes nausea, vomiting, and sometimes diarrhoea, retrosternal and abdominal pain, tightness in the chest and coughing, headache and dizziness. In severe cases, gastrointestinal haemorrhage, tachycardia, hypotension, shock, cardiac arrhythmias, hypothermia, metabolic acidosis, cyanosis, pulmonary oedema, convulsions, hyperthermia and coma may occur. Clinical features of renal insufficiency and hepatic damage including oliguria, and jaundice may develop later, if the patient does not die.

Death, which may be sudden, usually occurs within four days but may be delayed for one to two weeks. Postmortem examinations have revealed focal myocardial infiltration and necrosis, pulmonary oedema and widespread small vessel injury.

Chronic poisoning from inhalation or ingestion of phosphine/phosphides may cause toothache, swelling of the jaw, necrosis of mandible, weakness, weight loss, anaemia, and spontaneous fractures.

2.3 Diagnosis

Major accidental release of stored phosphine presents serious toxic and explosion/fire hazards for man and even animals. The diagnosis of phosphine poisoning is easy, but the clinical manifestations of phosphine and the phosphides may be similar to those of other toxic chemicals such as arsenic sulphide and calcium oxide. A silver nitrate-

impregnated paper test can be used for the breath and gastric fluid of the patients exposed to phosphine/phosphide: silver nitrate and phosphine/phosphides react to form silver phosphide which confirms the diagnosis. Other laboratory investigations such as cell blood counts, haemoglobin, haematocrit, arterial blood gas analyses, renal and liver function tests and cardiopulmonary monitoring and investigations (ECG and chest X-ray) are essential for the assessment of organ effects and the management of phosphine/phosphide poisoning.

2.4 First aid measures and management principles

Remove the patient from exposure site, and keep at rest. If the patient is unconscious and breathing stops, immediately ventilate artificially and if the heart stops, begin cardiopulmonary resuscitation. In case of ingestion, after consideration of tracheal intubation, perform gastric aspiration and lavage with cold water and preferably sodium bicarbonate solution (2%). Do not give milk, fats or saline emetics. Administration of repeated doses of activated charcoal through the gastric tube may be useful. Monitor and support vital functions, particularly cardiopulmonary, G.I., renal and hepatic functions.

Treat shock conventionally and correct acidosis based on blood gas analyses.

No antidote is available for phosphine/phosphide poisoning. Early recognition and management of the poisoning is essential.

3. PHYSICO-CHEMICAL PROPERTIES

3.1 Origin of substance

Phosphine is extremely rare in nature. It occurs transiently in marsh gas and other sites of anaerobic degradation of phosphorus-containing matter.

Although phosphorus could be expected to occur naturally as a phosphide, the only phosphide in the earth's crust is found in iron meteorites as the mineral schreibersite $(Fe,Ni)_3P$, in which cobalt and copper may also be found (WHO, 1988).

Atmospheric phosphine results from emission and effluents from industrial processes and from the use of phosphides as rodenticides and fumigants.

Unexpected focal release of phosphine may occur due to the action of water on phosphides present as impurities in some industrial materials. Although some phosphine is supplied in cylinders, it is often produced as and when required, by hydrolysis of a metal phosphide. Phosphine is also produced as a by-product or evolved incidentally in various industrial processes (WHO, 1988; Casarett, 1991).

3.2 Chemical structure

Phosphine is trihydrogen phosphide
Molecular formula: PH_3

Molecular weight: 34

Metal phosphides that are commonly used as rodenticide and fumigants are:

zinc phosphide (Zn_3P_2, CAS No. 1314-84-7, molecular weight = 258.1)

aluminum phosphide (AlP, CAS No. 20859-73-8, molecular weight = 57.96)

magnesium phosphide (Mg_3P_2, CAS No. 12057-74-8, molecular weight = 134.87)

(Deichman & Gerarde, 1964; WHO, 1988).

3.3 Physical properties

3.3.1 Colour

Colourless

3.3.2 State/form

Gas

3.3.3 Description

Pure phosphine is a colourless gas at ambient temperature and pressure.
Melting point: -133.5°C
Boiling point -87.4°C
Phosphine is odourless when pure, at least up to a concentration of 282 mg/m^3 (200 ppm), which is highly toxic level. The odour of technical phosphine depends on the presence of odoriferous impurities and their concentrations and odour threshold is usually in the range 0.14 to 7 mg/m^3.

Pure phosphine has an autoignition temperature of 38°C, but because of the presence of other phosphorus hydrides, particularly diphosphine (P^2H^4), as impurities, the technical product often ignites spontaneously at room temperature.

Phosphine has intense ultraviolet absorption in the 185 to 250 nm (1850 to 2000 A) region.

3.4 Hazardous characteristics

Phosphine forms explosive mixtures with air at concentrations greater than 1.8%. The relative molecular mass of phosphine is 34. It dissolves in water to form a neutral solution, but its water solubility is very low (0.25 at room temperature). Phosphine dissolves more easily in organic solvents, particularly in trifluoroacetic acid and carbon disulphide (Beliles, 1981).

In air, the upper and lower explosion limits depend on the temperature, pressure, and proportion of phosphine, oxygen, inert gases and water vapour present, and also on the level

of ultraviolet irradiation. In aqueous solutions, oxidation of phosphine results in the production of hypophosphorous acid.

The technical grade of phosphine contain impurities of higher phosphines (diphosphine) and substituted phosphines, which are responsible for the characteristic foul odour of phosphine which is often described as "fishy" or "garlicky".

Depending on the method of manufacturer, other impurities may include methane, arsine, hydrogen and nitrogen (Polson et al., 1983).

An important reaction of phosphine is with metal, especially with copper and copper-containing alloys, which causes severe corrosion. The reaction is enhanced in the presence of ammonia or moisture and salt. Eighteen carat gold jewellery reacts at one-eighth of the rate of copper (WHO, 1988).

Phosphine and the metal phosphides have only been detected in the general environment in relation to the recent use of metal phosphides in pest control and in relation to a number of industrial activities.

The metal phosphides are solid with grey colour and melting points of more than 750°C. They hydrolyse very quickly and produce phosphine which is more toxic than the metal phosphide.

The volumes released in industrial operations are much smaller and are therefore of less significance in relation to atmospheric pollution.

Residues in fumigated foods are 0.01 mg/m^3 (0.01 ppm) or less and are negligible. Higher residue levels may be found with storage at low temperature. About 10% of the residues are water soluble and appear to be hypophosphite and pyrophosphate. The remainder may have included insoluble aluminium salts (WHO 1988).

Residue levels of phosphine in fumigated foods are generally regulated at 0.1 mg/kg (0.1 ppm) or sometimes 0.01 mg (0.01 ppm). However, among populations whose diet in mainly derived from stored products, the daily intake would be unlikely to exceed 0.1 mg/day, even if the phosphine and phosphides survived cooking.

4. USES/HIGH RISK CIRCUMSTANCES OF POISONING

4.1 Uses

4.1.1 Uses

Fumigants
Pesticide for use on vertebrate animals

4.1.2 Description

Phosphine is mainly used as a fumigant in pest

control. Zinc phosphide is used as a rodenticide because of its reaction with stomach acid in the rodent to release phosphine. For fumigation, the acid has to be supplied. Since they hydrolyse in neutral moist conditions, aluminum and magnesium phosphides

are preferred as fumigants. Aluminum phosphide has also been used as a rodenticide; magnesium phosphide may be used as a pesticide.

Zinc phosphide is available in bulk, typically to a specification of at least 80% Zn_3P_2, and as paste containing 5% or 2.5% for use as a rodenticide by mixing in bait. Aluminum and magnesium phosphides are available in a number of commercial formulations. Aluminum phosphide formulations usually contain approximately 75% active ingredient and magnesium phosphide products contain 43% active ingredient (WHO, 1988).

4.2 High risk circumstances of poisoning.

No subgroups of the general population have been identified to be at special risk from phosphine and the phosphides except children, who might find and eat bait containing phosphides. Zinc phosphide pastes and tablets of zinc, aluminum and magnesium phosphides which are available without restriction in some countries may be used in suicide attempts. Many reports of high mortality (> 50%) due to metal phosphide poisonings in India have recently been published.

4.3 Occupationally exposed populations.

Occupational exposure can be divided into 4 general categories: (a) workers producing phosphine and phosphides; (b) workers in operations that can release phosphine, e.g., welding, metallurgy, semi-conductors (c) fumigators and pest-control operators; and (d) transport workers. e.g. drivers, seamen. Exposure patterns and the potential for control of exposure differ from case to case.

Exposure to phosphine and phosphorus oxides, which occurs during the manufacture of metal phosphides, varies according to the method of manufacture. High levels of exposure may occur in the direct methods involving the reaction of red phosphorus with powdered metal, in which the air phosphine concentrations of 0.4 to 1.6 mg/m^3 (0.3 to 1.13 ppm) may occur. Concentration of > 2 mg/m^3 require the use of personal respiratory protection.

In recorded cases, atmospheric levels to which operatives were exposed while adding zinc/aluminum phosphides to wheat were undetectable. Levels encountered when stores were re-entered for loading or turning were much higher, ranging from 18 to 35 mg/m^3 (13 to 25 ppm).

Exposure to phosphine has also been described in the operation of acetylene generators and in the production of phosphorus. A badly ventilated cargo of ferrosilicon,

particularly in barges, can release phosphine accidentally by the reaction of water with calcium phosphide, one of the impurities present.

Many metals contain phosphorus in small amounts, and phosphine can be generated in a variety of metallurgical processes.

Although phosphine is used extensively in semi-conductor manufacture, there are no published figures for occupational exposure in this industry. There are also no published data relating to exposure to phosphine in the synthesis of organophosphine or phosphonium derivatives. The occupational exposure limit for phosphine in various countries differ from 0.1 mg/m^3 to 0.5 mg/m^3 in long term and up to 1.5 mg/m^3 (1.1 ppm) in short term exposure (WHO, 1988; Deichmann & Gerarde, 1969).

5. ROUTES OF EXPOSURE

5.1 Oral

Deliberate oral ingestion of the metal phosphides, particularly AlP (Phostoxin), is not rare in some parts of the world. Accidental oral ingestion of the metal phosphides, particularly zinc phosphide, have also been reported.

5.2 Inhalation

Inhalation is the commonest route of phosphine poisoning.

5.3 Dermal

The skin is not a common route of absorption of phosphine and phosphides. However, dermal absorption of zinc phosphide in rabbits was reported by US National Pest Control Association (WHO, 1988).

5.4 Eye

No data available.

5.5 Parenteral

Stephenson (1967) mentioned the possibility of zinc phosphide injection.

5.6 Others

No data available.

6. KINETICS

6.1 Absorption by route of exposure

Inhaled phosphine is generally considered to be rapidly absorbed through the lungs. After inhalation, aluminum and magnesium phosphides deposited on the moist surfaces of the respiratory tract would release phosphine, but zinc

phosphide, which hydrolyses significantly only under acid conditions, would be stable for some time. However, the transfer of a proportion of inhaled zinc phosphide to the intestinal tract by particulate clearance mechanisms in the lung would permit hydrolysis to phosphine by gastric acid, as well as absorption of the zinc phosphide. The lung also absorbs particles and it is known that zinc phosphide is absorbed intact from the gut. Inhaled zinc phosphide dust might be absorbed directly via the respiratory tract and then hydrolysed in the tissues.

In the rat, ingestion of zinc phosphide results in detectable amounts of acid-hydrolysable phosphide in the liver. Human ingestion of tablets containing aluminum phosphide yielded evidence of acid-hydrolysable phosphide in blood and liver. These results indicate that metal phosphides can be absorbed directly. In the rat, recovery of phosphide from the following administration of zinc phosphide in corn oil was 4 times higher than when administered in water, suggesting that absorption of unhydrolysed material is greater.

In general, dermal absorption of phosphine and phosphides is insignificant.

6.2 Distribution by route of exposure

Inhaled phosphine produces neurological and hepatic symptoms suggesting that it reaches the nervous system and liver. Ingested phosphides have been shown to reach the blood and liver in rats and human beings. On the other hand, muscle tissue of animals poisoned with supralethal doses of zinc phosphide does not contain detectable levels of phosphine or phosphide and does not produce toxic effects when fed to test animals. The presence of acid-hydrolyzable phosphide in the kidney and liver of a fatal case of zinc phosphide has been reported (WHO, 1988).

6.3 Biological half-life by route of exposure

The biological half-life of phosphine and phosphides in man has not been reported and may be difficult to estimate. Experimentally, the amount of acid-hydrolyzable phosphide found in the liver of a rat fed phosphide for 15 days is nearly twice that of a rat fed for 7 days. However, this limited study cannot be considered to provide evidence of a long biological half-life and/or the accumulation of metal phosphides (WHO, 1988).

6.4 Metabolism

Metal phosphides are hydrolysed to phosphine. In the rat, phosphine that is not excreted in the expired air is oxidized and appears in the urine, chiefly as hypophosphite and phosphite. The fact that (a) phosphine is incompletely oxidized; and (b) the proportion of an administered dose that is eliminated as expired phosphine increases with the dose, suggests that the oxidative pathway is slow (WHO, 1988). Oxyhaemoglobin is denatured and a variety of enzymes are inhibited by reaction with phosphine (WHO, 1988).

6.5 Elimination and excretion

Zinc phosphide suspended in corn oil was given to rats by gavage and phosphine concentrations were measured in a metabolic chamber over the following 12 hours. After doses of 0.5, 1, 2, 3 and 4 mg, the proportions of the administered doses as phosphine were 1.5%, 1.7%, 3.2%, 15.6% and 23.5%, respectively, but some or much of this could have been derived from faeces or intestinal gas rather than by desorption and exhalation. Hypophosphite is the principal urinary excretion product (WHO, 1988).

7. TOXICOLOGY

7.1 Mode of action

Phosphine reduces the respiration of wheat partly by damaging the microflora present. The activity of glutamate decarboxylase is reduced when the moisture content is 18% or more. Alcohol dehydrogenase activity is reduced to zero within 7 days as a result of phosphine treatment of the grain at a moisture content of more than 24%. Catalase activity in wheat is reduced by about 20% after 2 weeks exposure to phosphine fumigations. Phosphine markedly inhibits respiration and the growth of microorganisms in wheat with a moisture content up to 29%. The amount of adenosine triphosphate (ATP) is reduced by phosphine fumigation, but adenosine diphosphate (ADP) is not, indicating that the respiratory activity in treated grain is markedly reduced (WHO, 1988).

Studies on isolated rat liver showed that mitochondrial oxygen uptake is inhibited by phosphine due to its reaction with cytochrome C and cytochrome C oxidase. Phosphine inhibits insect catalase, though this appears to be an indirect effect and might be a consequence not a cause of toxicity.

There have not been any systemic studies on the mechanism of phosphine toxicity in man. Various effects on intermediary metabolism have been described. Dose-related increases in blood and urinary porphyrin concentrations due to zinc phosphide have been reported. In a study on rabbits, changes in serum glutamic-pyruvic and glutamic oxalacetic transaminase, leucine aminopeptidase, aldolase, alkaline phosphatase and albumin in the first 24 hours of zinc phosphide poisoning have been observed. Dysfunction of hepatic fat metabolism was also reported. Loss of cell viability and cell membrane integrity accounts for the raised hepatic enzymes and the bronchiolitic effect. There is no adequate explanation for the fact that phosphine does not cause the haemolysis that is characteristic of arsine.

Although the exact mechanism of action of phosphine in man is not known, non-competitive inhibition of mitochondrial cytochrome oxidase in mouse liver, housefly and granary weevil is mentioned by some authors (Singh et al., 1985; Chopra et al., 1986; Khosta et al., 1988).

7.2 Toxicity

7.2.1 Human data

7.2.1.1 Adults

Phosphine and the metal phosphides are highly toxic to human beings and animals.

The odour of phosphine depends on the impurities it contains. When pure it has no odour, even at a concentration of 28 mg/m^3. Phosphine prepared conventionally without purification, has a fishy or garlic-like odour due to its impurities. These may be absorbed by stored products during fumigation with a resultant loss of odour, even though phosphine remains at toxic concentrations. Phosphine is in class D of the safety classification, because 20 to 50% of attentive persons can detect the threshold

limit value (TLV) of 0.42mg/m^3 by smell. However, the smell of phosphine cannot be relied on as a warning of toxic concentrations.

Zinc phosphide baits and formulated aluminium phosphide pellets are widely used. Occasional accidental or more usually suicidal exposure to the metal phosphides may be encountered. Ingestion, the only highly toxic route, has almost always been with suicidal intent and the symptoms are always acute.

There is negligible exposure of the general population to phosphine. Many cases of acute phosphine poisoning due to occupational exposure have been reported in the literature (WHO, 1988).

In one incident, 12 inhabitants of an apartment house developed nausea and one died when phosphine was emitted from an adjacent warehouse containing bags of aluminum phosphide which became damp. Some passengers on ships and barges carrying cargoes of ferrosilicon of grain under fumigation have also been poisoned by phosphine, with symptoms similar to those of acute occupational poisoning. In a further incident, 2 adults and one child died when a granary sharing a party wall with their house was fumigated. It was estimated that phosphine concentration in the bedroom reached 1.2 mg/m^3. Symptoms were initially non-specific and insidious and illustrate the risk of sustained exposures to relatively low concentrations. At autopsy, there was congestion of all organs; pulmonary oedema and focal emphysema were found in the lungs

and there was vacuolation in the liver (WHO, 1988).

Many cases of acute deliberate zinc phosphide poisoning by ingestion have been reported in the literature. Stephenson (1967) reviewed 20 patients with zinc phosphide poisoning by ingestion in which the approximate doses were recorded. Of these, 10 patients died after ingestion of 4.5 to 180 g; 6 cases had ingested 20 g or more. In the 10 non-fatal cases, the doses ranged from 0.5 to 50 g and 7 ingested less than 20 g. The main clinical

manifestations were metabolic acidosis, methaemoglobinaemia, hypocalcaemic tetani, reduced blood coagulation, pulmonary oedema; and gastrointestinal, neuropsychiatric and cardiovascular disorders. Postmortem findings included blood in all the serous cavities, pulmonary congestion and oedema, haemorrhagic changes in the intestinal epithelium, centrilobular congestion and necrosis and yellow discolouration of the liver, and patchy necrosis of the proximal convoluted - tubules of the kidneys.

An unsuccessful suicidal attempt by a 25 year-old man who ingested 6 tablets of AID (Phostoxin) in water was reported. Immediate symptoms were severe retrosternal pain, a generalized burning sensation and vomiting. There was circulatory collapse necessitating resuscitation and subsequently cerebral, renal and hepatic dysfunction appeared.

Harger and Spobyar (1958) reviewed 54 cases of acute phosphine poisoning with 26 deaths since 1900. In 6 of 11 reports, cargoes of ferrosilicon were cited as the source of phosphine and in these cases, the victims were passengers or crew members of the ships or barges concerned. Other cases involved the exposure of welders to calcium carbide and raw acetylene and of submariners to sodium phosphide. The most common autopsy finding was congestion of the lungs with marked oedema.

Metal workers at a large shipyard in Norway, drilling deep holes in spheroidal graphite iron, became ill during work. The symptoms were mostly nausea, dizziness, chest tightness, dyspepsia and disturbances of smell and taste. Measurement of phosphine concentration in the worker's breathing zone (with Drager tubes) showed a phosphine concentration of about 1.4 mg/m^3 (1 ppm). After installing local exhaust ventilation on the drilling machines, there were no longer any measurable amounts of phosphine, and

there were no complaints from the workers. When the local exhaust ventilation was removed for technical reasons 5 years later illness among the workers recurred. Measurement of phosphine levels just above the machines, showed concentration up to 56

to 70 mg/m^3 (40 to 50 ppm). When the local exhaust ventilation was re-installed, the phosphine concentrations dropped to unmeasurable amounts, and no further cases were reported. (WHO 1988).

7.2.1.2 Children

Two children and 29 of 31 crew members aboard a grain freighter became acutely ill after inhaling the toxic fumigant phosphine; one child died. Predominant symptoms were headache, fatigue, nausea, vomiting, cough and shortness of breath. Abnormal physical findings included jaundice, paraesthesia, ataxia, intention tremor and diplopia. Focal myocardial infiltration with necrosis, pulmonary oedema and widespread small vessel injury were found postmortem. The surviving child showed ECG and echocardiographic evidence of myocardial injury and transient elevation of MB fraction of serum creatinine phosphokinase. Phosphine gas was found to have escaped from the holds through a cable housing located near the midship ventilation intake and around hatch covers on the forward deck (Wilson et al., 1980).

Occasional reports on accidental phosphine poisoning in children have been published.

Reports of deaths of children and adults in chemical accidents involving phosphine have been published (Wilson et al., 1980).

Acute phosphine poisoning following ingestion of aluminum phosphide has been reported in young children and adults. Eight patients aged 14 to 25 years with acute aluminum phosphide poisoning reported by Misra et al. (1988). The clinical picture consisted of acute gastritis, altered sensorium and peripheral vascular failure, cardiac arrhythmias, jaundice and renal failure. Six patients died, the mean hospital stay was 19 (range 4 to 72) hours. These patients had taken 2 or more AlP tablets, whereas the two patients survived had taken one tablet or less.

Postmortem examination revealed pulmonary

oedema, gastrointestinal mucosal congestion, and petechial haemorrhages on the surface of liver and brain.

7.2.2 Relevant animal data

Animal experiments have revealed that rabbits exposed to 70 mg phosphine/m^3 (50 ppm) for 10 minutes do not develop any symptoms but exposure to 140 mg/m^3 (500 ppm) is fatal in 2.5 to 3 hour, and 700 mg/m^3 (500 ppm) is fatal 25 to 30 minutes. Rats survive exposure to 80 and 800 mg/m^3 for 4 and 1 hour, respectively. All animals exhibited signs of respiratory irritation and died of pulmonary oedema. Pathological examination of the lungs revealed bronchiolitis and atelectasis; there was no evidence of haemolysis but all organs were hyperaemic. The liver showed fatty infiltration and there was cloudy swelling of kidney tubular cells. Neurohistological studies in rats revealed widening of the perivascular spaces, vacuolization of the nuclei of ganglion cells, a reduction in the Purkinje cells and a glial reaction. In one study a 4-hour LC_{50} for phosphine inhalation in male rats was estimated as 15 mg/m^3 (11 ppm), but in another study on female rats it was reported as 55 mg/m^3. The LC_{95} was 420 (260 to 670) mg/h/m^3. The US National Pest Control Association submitted a value of 19.6 mg/L for an inhalation LC_{50} of 10% zinc phosphide powder in rats.

In an oral study on 35 rats of both sexes administered doses of 20, 40, 50 and 80 mg/kg LD_{50} for zinc phosphide was 40.5 to 2.9 mg/kg body weight. The LD_{50} for kit fox was reported as 93 mg zinc phosphide/kg body weight. A dose of 100 mg/kg bodyweight of zinc phosphide was fatal for dogs after starving but not after feeding.

An acute dermal LD_{50} of 2000 to 5000 mg/kg body weight for zinc phosphide (94% Zn_3P_2) in rabbits is reported by the US National Pest Control Association (WHO, 1988).

Inhalation exposure to phosphine at 28 mg/m^3 (20 ppm) for 4 hours a day was fatal for rabbits and guinea pigs. Pretreatment with sub-lethal concentrations of phosphine reduced resistance to near-lethal concentrations. At low concentrations (up to 14 mg/m^3), animals displayed no signs until about 0.5 hour before death when they exhibited diminished

reactivity, became stuporous with shallow respiration and died in coma and occasionally with signs of pulmonary oedema.

Zinc phosphide was mixed with the diet of rats at 0 (control), 50, 100, 200 and 500 mg/kg. Deaths occurred at the two higher dosage regimens in 1/12 and 10/12 animals, respectively. There was a dose-dependent reduction in haemoglobin, red cells and haematocrit

(WHO, 1988).

No long-term studies on phosphine and metal phosphide exposure have yet been reported.

7.2.3 Relevant in vitro data

No data available.

7.2.4 Workplace standards

Occupational exposure limits for phosphine in various countries are shown in Table 1. The recommended exposure limits for phosphine in many countries are higher than the registered regulatory requirement. However, the exposure limits for phosphine varies from 0. mg/m^3 to 1.5 mg/m^3 in short-term exposure.

Table 1 - Occupational exposure limits for phosphine in various countries

Country	Legal	mg/m^3	Comment
Australia	Rec	0.4	TLV TWA
Belgium	Rec	0.4	TLV
Bulgaria	Rec	0.1	MPC
Czechoslovakia	Rec	0.1	MAC TWA
		0.2	MAC Ceiling value
Finland	Reg	0.1	MPC TWA
Germany	Rec	0.15	8.h TWA
		0.3	5 min STEL
I.R.Iran	Rec	0.3	TLV
Italy	Rec	0.4	8.h TWA
Hungary		0.1	
Netherlands	Rec	0.4	TWA
		1.5	STEL
Poland	Reg	0.1	Ceiling value
Romania	Reg	0.2	TWA
		0.5	Ceiling value
Sweden	Reg	0.4	1-day TWA
Switzerland	Reg	0.15	TWA
United Kingdom	Rec	0.4	8-h TWA
		1.0	10 min TWA
USA	Rec	0.4	TWA
		1.0	STEL
USSR	Reg	0.1	Ceiling value
Yugoslavia	Reg	0.1	MAC TWA

Rec.=Recommendation
Reg.=Registered regulatory requirement
TLV=Threshold limit value
TWA=Time - weighted average
MPC=Maximum permitted concentration
MAC=Maximum allowable concentration
STEL=Short - term exposure limit

7.2.5 Acceptable daily intake

Residues of phosphine or metal phosphides in fumigated foods are considered negligible at 0.01 mg/kg or less. Reported various national and international standards for phosphine residues in food are 0.01 mg/kg (1 ppm) except for whole food grains in India, for which the standard is 0.05 mg/kg. However, the acceptable limit of phosphine residue in milled food grain in this country was reported as 0.01 mg/kg. Therefore the acceptable daily intake of phosphine/phosphide residues could be extrapolated as 0.01 mg or less (WHO, 1988).

7.3 Carcinogenicity

No data available.

7.4 Teratogenicity

No data available.

7.5 Mutagenicity

No data available.

7.6 Interactions

No data available.

8. TOXICOLOGICAL/TOXINOLOGICAL ANALYSES AND BIOMEDICAL INVESTIGATIONS

8.1 Material sampling plan

8.1.1 Sampling and specimen collection

8.1.1.1 Toxicological analyses

Different sampling methods for phosphine and the metal phosphides are available.

I.Gaseous phosphine: Workplace air monitoring and fumigation control demand a measurement range from approximately 0.04 mg/m^3 to greater than the lower explosion limit of 25000 mg/m^3. Thus, methods covering concentrations differing by six orders of magnitude are required. Techniques are available that (a) directly indicate the concentration in a grab sample or a time-weighted average sample, (b) absorb or adsorb phosphine from a known volume of air for subsequent analysis directly or by desorption and gas analysis and (c) give a continuous record of time-dependent concentrations. Some methods are given in table 2.

Table 2 - Methods of sampling and analysis

Method	Range		Efficiency	Interference
	ppm	mg/m^3		
Sampling				
Silver nitrate (0.1 N) impregnated paper	0.05-8.0	9.07-11.3	90%	
Ethanolic mercuric chloride	0.05-3.0	0.07-4.2		NH_3
Acidic potassium permanganate (0.1N) impinger	0.01-0.05		100%	H_3S
Silver diethyl-dithiocarbomate (0.5%) bubbler	0.6-18	0.85-25	54-86.2%	H_2S, AsH_3, SbH_3
Mercuric chloride (0.5%) aqueous bubbler	10-28	14-28		AsH_3
Toluene impinger			41.5%	
Mercuric chloride (0.1%) conductance cell	0.05-2.5	0.07-3.5	88.0%	SO_2, H_2S, AsH_3, SbH_3
Silver nitrate impregnated silica gel	0.05-4.1	0.07-5.8	95%	H_2A, AsH_3
Auric chloride impregnated silica gel	0.01-1000	0.014-1 400	100%	AsH_3, SbH_3
Ethanolic mercuric chloride (0.1%)	0.0006		88-100%	AsH_3, SO_2, HCN, H_2S
Mercuric cyanide impregnated silica gel	0.014-1.18	0.02-1.7	80%	

Phosphine can be detected by filter paper impregnated with a mixture of silver nitrate and mercuric chloride. Direct-indicating detector tubes are commercially available for spot sampling.

There are directly-indicating continuous samplers in which phosphine-containing gas is passed through a paper tape impregnated with a mixture containing silver nitrate which develops a colour corresponding to the phosphine concentrations.

II. Residues: Fumigated foodstuffs may contain gaseous phosphine (adsorbed or in trapped air) and residual aluminium or magnesium phosphide. Interstitial and adsorbed phosphine can be purged by nitrogen and trapped in reagents for classical analyses.

Total phosphine and phosphide is measured by extraction of the fumigated stored product with silver nitrate or with sulphuric acid. The sulphuric acid method is preferred because it also measures the capacity of the product to release phosphine and this is of more biological significance than the measurement of free phosphine only.

III. Metal phosphide: Hydrolysis of metal phosphides with acid yields phosphine, which can be measured by any of the methods already described.

IV. Inhaled expiration: Silver nitrate impregnated paper test can be used for the breath of patients exposed to phosphine. Silver nitrate and phosphine react to form silver phosphide which confirms the diagnosis.

V. Gastric fluid: Gastric fluid (vomited or through gastric tube) must be collected in a clean glass tube or beaker for toxicological analysis.

VI. Blood: Phosphine that is not excreted in the expired air is oxidized and has no significant effect on diagnosis. Thus blood sampling for toxicological analysis may not be required except for research purposes.

V. Urine: Oxidative metabolites mainly as phosphite and hypophosphate may be present in the urine . Urine sampling for the estimation of the metabolites may be required.

8.1.1.2 Biomedical analyses

Blood samples

In case of cardiac dysfunction, estimation of cardiac enzymes including LDH and CPK may be indicated. Biochemical analyses particularly require liver and kidney function tests. Urine samples including a 24-hour collection are necessary to perform routine urine analysis and to estimate creatinine clearance and other investigations such as beta-macroglobulin and N-acetyl beta-glucose aminidase (NAG) as required.

8.1.1.3 Arterial blood gas analysis

Arterial blood samples must be taken for urgent estimation of PH, PaO_2, $PaCO_2$, bicarbonate and the other parameters as usual in severely poisoned patients in order to assess and correct acidosis and pulmonary dysfunction. Repeated sampling for arterial blood gases may be required for the management of patients with severe phosphine/phosphide poisoning.

8.1.1.4 Haematological analyses

Blood samples must be taken in the usual haematology tubes for cell blood count, haemoglobin, haematocrit. Estimation of prothrombin time rate may be indicated clinically.

8.1.1.5 Other (unspecified) analyses

8.1.2 Storage of laboratory samples and specimens

8.1.2.1 Toxicological analyses

Blood and urine samples should be stored at -20°C for further analyses. However, no data are available on the stability of phosphine/phosphides in biological fluids.

8.1.2.2 Biomedical analyses

8.1.2.3 Arterial blood gas analysis

8.1.2.4 Haematological analyses

8.1.2.5 Other (unspecified) analyses

8.1.3 Transport of laboratory samples and specimens

8.1.3.1 Toxicological analyses

Transportation of samples must follow the required safety regulations.

8.1.3.2 Biomedical analyses

8.1.3.3 Arterial blood gas analysis

8.1.3.4 Haematological analyses

8.1.3.5 Other (unspecified) analyses

8.2 Toxicological Analyses and Their Interpretation

8.2.1 Tests on toxic ingredient(s) of material

8.2.1.1 Simple Qualitative Test(s)

Phosphine can be detected by filter papers impregnated with a mixture of silver nitrate and mercury (II) chloride. Aluminum and magnesium phosphide can be hydrolysed conventionally but zinc phosphide requires acid hydrolysis to produce phosphine for detection.

8.2.1.2 Advanced Qualitative Confirmation Test(s)

Direct-indicating detector tubes are commercially available for qualitative confirmation of phosphine. Other direct-indicating tubes of lower sensitivity are available for the estimation of the higher phosphine concentrations used in fumigation.

8.2.1.3 Simple Quantitative Method(s)

Colorimetric method is a simple quantitative technique for phosphine. Filter papers impregnated with a mixture of silver nitrate and mercury (II) chloride which detect phosphine can be made

semi-quantitatively by appropriate configuration and measurement for stain length of colour comparison. Calzodari (1986) also reported a colorimetric method involving oxidation of PH3 with bromine water and reduction of phosphomolibate.

The quantity of phosphine bubbled through a solution of mercury (II) chloride and undergoing the reaction:

$$PH_3.3HgCl_2 \text{-----}> P(HgCl)_3 + 3\ HCl$$

can be measured by the change in electrical conductivity using a conductance cell or by potentiometric titration of HCl against NaOH.

8.2.1.4 Advanced Quantitative Method(s)

Chan et al. (1983) reported a headspace gas chromatographic technique using a nitrogen phosphorus detector to estimate phosphine applied to postmortem specimens following ingestion of aluminum phosphide.

Gas chromatography is the most sensitive method for the determination of the phosphine content of air samples.

Usually, samples are adsorbed from a solid absorbent coated with mercury (II) cyanide, although samples taken in syringes, gasbags or tonometers can be used. Microcolorimetric and thermionic detectors have detection limits of 5000 and 20 pg, respectively. The limit for flame photometric and argon and helium beta-ionization detectors is 5 pg and that for mass spectrometry is 1 ng. Photoionisation detection is also commonly used. Flame photometry combines both sensitivity and stability.

8.2.2 Tests for biological specimens

8.2.2.1 Simple Qualitative Test(s)

Silver nitrate impregnated paper test can be used for the breath and gastric fluid of the patients exposed to phosphine/phosphides. Silver nitrate and

phosphide/phosphides react to form silver phosphide which is dark grey (Chugh et al., 1989). Blood and urine samples cannot be used for phosphine detection, because absorbed phosphine is rapidly oxidized and excreted mainly as phosphite and hypophosphite in the urine.

8.2.2.2 Advanced Qualitative Confirmation Test(s)

8.2.2.3 Simple Quantitative Method(s)

8.2.2.4 Advanced Quantitative Method(s)

Khan et al. (1983) estimated phosphine levels in post mortem specimens liberated after acidification and found a small amount in blood (0.5 ng/mL) and liver (3 ng/g) but a large quantity (3000 ng/g) in the stomach and contents. See also 8.2.1.4.

8.2.2.5 Other Dedicated Method(s)

8.2.3 Interpretation of toxicological analyses

Diagnosis of phosphine/phosphide poisoning is normally based on the history of exposure and clinical

manifestation. However, qualitative toxicological analyses confirm the diagnosis and the quantitative tests may be used for the evaluation of the severity and prognosis.

8.3 Biomedical investigations and their interpretation

8.3.1 Biochemical analysis

8.3.1.1 Blood, plasma or serum

Kidney and liver function tests and cardiac enzymes, particularly blood urea, electrolyte, creatinine, bilirubin, alkaline phosphatase, transaminases, lactic dehydrogenases and creatine phosphokinase, should be estimated in all patients hospitalized after phosphine/phosphide poisoning. Further investigation, such as plasma cortisol level (Chugh et al, 1989) and plasma renin activity (Chugh et al, 1990) should only be done as clinically indicated.

In case of zinc phosphide poisoning, serum zinc concentration may be elevated (Stephenson, 1967) - in this case, by 590 to 605 g/100 mL (Normal 120 to 200 g/mL). Serum magnesium and aluminium concentrations may also increase in Mg_3P_2 and AlP poisoning, respectively.

8.3.1.2 Urine

Routine urinalysis and further investigations such as estimation of beta-microglobulin, N-acetyl-glucose aminidase (NAG) and 24 hour urine creatinine are required to evaluate renal function.

8.3.1.3 Other fluids

8.3.2 Arterial blood gas analyses

Serial arterial blood gas analyses may be required in order to assess respiratory and acid-base abnormalities and to correct them.

8.3.3 Haematological analyses

Routine haematological tests such as cell blood count, haemoglobin, haematocrit are required for all patients with phosphide/phosphine poisoning. Further investigations such as prothrombin time ratio should be done as clinically indicated.

8.3.4 Interpretation of biomedical investigations

Biochemical and haematological tests are required to assess the effects of phosphine/phosphide

poisoning.The results should be considered in conjunction with the clinical picture and other paramedical investigation such as electrocardiogram and chest X-ray. Re-evaluation of the patient's condition and repetition of biomedical and haematological tests may be necessary.

8.4 Other biomedical (diagnostic) investigations and their interpretation

Electrocardiographic changes in phosphine poisoning were reported by Roman and Dubey (1985), who found cardiac arrhythmias, usually heart block and myocardial ischaemia. Wilson et al. (1980) also found similar ECG and echocardiographic changes in child after phosphine poisoning; they reported a transient elevation of the MB fraction of

serum creatinine phosphokinase and focal myocardial infiltration with necrosis and widespread small vessel injury at postmortem.

Misra et al. (1988), in a study of 8 cases of attempted suicide by ingestion of aluminum phosphide tablets, found circulatory failure in all cases and cardiac arrhythmias in three patients. ECG changes included sinus arrhythmia with ST segment depression in leads II and III; AVF and T- wave inversion in V5-V6; and premature complexes which were followed by ventricular tachycardia.

8.5 Overall interpretation of all toxicological analyses and toxicological investigations

Cardiac monitoring with serial ECG recording, as well as the other investigations are required for poisoning by phosphine/phosphide.

Sample collection

Blood samples (10mL) for biochemical investigations are usually collected in dry glass tubes without any preservative. Blood samples for haematology should be collected in anticoagulant tubes as instructed by the laboratory. A 24-hour urine collection may be needed for the estimation of creatinine and phosphine metabolite concentrations.

Biochemistry

Routine urinalysis, blood urea, electrolytes, creatinine, bilirubin, alkaline phosphates, transaminases (ALT, AST), lactic dehydrogenase (LDH), creatinine phosphokinase (CPK) should be measured in all patients with phosphine/phosphide poisoning. If the results are abnormal (high LDH and CPK or renal dysfunction), further biochemical investigations (e.g. LDH and CPK, urine creatine, beta-microglobulin, N-acetyl-glucose aminidase) should be determined.

Haematology

Cell blood counts (CBC), haemoglobin (Hb) and haematocrit (HCT) should be investigated in all patients with

phosphine/phosphide poisoning. If there are any abnormalities or signs of gastrointestinal haemorrhage, or hepatic failure, CBC, Hb and HCT must be repeated and further tests including platelet counts, and prothrombin time ratio should also be performed.

Arterial blood gas analyses

Arterial pH and blood gases should be investigated in all patients with respiratory dysfunction. Repeated arterial blood gas analyses may be required in order to correct pH and blood gas abnormalities.

Toxicological analysis

Exhaled air can be tested for phosphine by an impregnated silver nitrate paper. The paper test can also be used to identify phosphine in the gastric contents. Blood samples are of no practical use for the estimation of phosphine/phosphide, since absorbed phosphine is rapidly oxidised in the blood. However, Chan et al. (1983) reported postmortem blood concentrations of phosphine of 5 ng/mL. Urine can be tested for the oxidative metabolites of phosphine (phosphite and hypophosphite).

Other investigations

Since phosphine initially affects the respiratory and cardiovascular system, respiratory function tests (spirometry), chest X-ray and ECG are required. Cardiorespiratory monitoring in an ICU with serial ECG recording is necessary in severe cases. Further investigation such as electroencephalography (EEG) and electromyography (EMG) should be performed as clinically indicated.

8.6 References

9. CLINICAL EFFECTS

9.1 Acute Poisoning

9.1.1 Ingestion

Deliberate ingestion of the metal phosphides particularly AlP (Phostoxin) and Mg_3P_2 tablets or pellets for suicidal purpose is common in the countries in which these fumigants are sold without restriction. Oral use of zinc phosphide paste (Zelio) for suicidal attempts is also common in some countries, including Islamic Republic of Iran, and the author has seen and treated many of these patients (see 9.3).

Acute poisoning by phosphine and the metal phosphides is common in some countries, particularly in India and Iran. Phosphine poisoning is either occupational or accidental, but the acute metal phosphides poisonings are mainly suicidal (Vale & Meredith, 1983).

9.1.2 Inhalation

Phosphine inhalation is the commonest route of intoxication and may occur accidentally or occupationally. The metal phosphides, particularly AlP and Mg_3P_2 may be easily hydrolysed in moisture and produce phosphine. Following oral ingestion of the metal phosphides, phosphine produced in the stomach may also be inhaled (see 9.3).

9.1.3 Skin Exposure

Skin exposure is not a common route of intoxication by phosphine and the metal phosphide, because skin absorption is not significant.

9.1.4 Eye contact

It seems that phosphine does not affect the eyes significantly. There are no data available on the effects of phosphine/phosphides on the eyes either in animals or man.

9.1.5 Parenteral Exposure

No data available.

9.1.6 Other

No data available.

9.2 Chronic poisoning

9.2.1 Ingestion

No data available.

9.2.2 Inhalation

No long-term studies of chronic exposure to phosphine and the metal phosphides have been reported. Chronic poisoning is generally occupational, but no reports with evidence of chronic poisoning by phosphine and the metal phosphine have been published. Chronic effects include anaemia, bronchitis, gastrointestinal disorders, speech and motor disturbances, toothache, swelling of the jaw, mandibular necrosis, weakness, weight loss and spontaneous fracture have been reported but these are by no means general (WHO, 1988). Complications of acute poisoning may occur but are distinct from the effects of chronic poisoning.

9.2.3 Skin contact

No data available.

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

The initial clinical manifestations of mild phosphide inhalation may mimic upper respiratory tract infection including cough, feelings of cold, sore throat, tachypnea, respiratory irritation and tightness of breath. Other symptoms may include nausea, vomiting, diarrhoea, headache, fatigue and dizziness. In severe exposure, lung irritation with persistent coughing, ataxia, paraesthesia, tremor, diplopia, hypotension, weak pulse and jaundice may also occur. Very severe cases may progress to acute pulmonary oedema, cardiac dysrhythmia, convulsion, cyanosis, hypothermia followed by hyperthermia and coma. Severe metabolic acidosis, cardiovascular collapse, oliguria, proteinuria and finally anuria may occur which may require haemodialysis.

Most severely poisoned patients may die within a few hours due to cardiovascular collapse, myocardial injury or pulmonary oedema.

In a study of acute phosphine poisoning aboard a grain freighter, the predominant symptoms in 29 crew members and two children were headache, fatigue, nausea, vomiting, cough and shortness of breath (Wilson et al., 1980). Abnormal physical findings included jaundice, paraesthesia, ataxia, intention tremor and diplopia. Focal myocardial infiltration with necrosis, pulmonary oedema and widespread small vessel injury were found at postmortem examination of a dead child. The surviving child showed ECG and echocardiographic evidence of myocardial injury and transient elevation of the MB fraction of creatinine phosphokinase.

Deliberate ingestion of the metal phosphides especially AlP (Phostoxin) causes nausea, vomiting, retrosternal and abdominal pain, tightness in the chest and coughing, headache, dizziness and sometimes diarrhoea. In severe

cases, gastrointestinal haemorrhage, tachycardia, hypotension, shock, cardiac arrhythmias, cyanosis, pulmonary oedema, metabolic acidosis, convulsions and coma may occur.

Clinical features of renal failure and hepatic damage including oliguria, proteinuria, anuria and jaundice may develop later if the patient survives. In 8 cases of attempted suicides by ingestion of aluminum phosphide tablets, the clinical picture consisted of acute gastritis, peripheral vascular failure, cardiac arrhythmia, jaundice and renal failure (Misra et al, 1988). Six patients died and postmortem examination in two of them revealed pulmonary oedema, gastrointestinal mucosal congestion, and petechial haemorrhages on the surface of the liver and brain.

In 15 cases of aluminum phosphide poisoning reported by Khosla et al. (1988), all had severe symptoms such as shock, cardiac arrhythmias, pulmonary oedema, and renal failure, of which, only 7 patients survived.

In a prospective study of 16 cases of aluminum phosphide poisoning by Chopra et al. (1986), profuse vomiting, pain in the upper abdomen and shock were the most common presenting features. Only 6 patients succumbed to their illness. Analysis of various prognostic factors revealed that ingestion of aluminum phosphide tablets taken from a freshly opened bottle was associated with a greater risk of fatal outcome.

The mortality of attempted suicide by acute phosphine/phosphide poisoning is 37 to 80% (Singh et al., 1985; Chopra et al.,1988; Khosla et al., 1988.) in suicidal patients. However, in occupational or accidental exposure to phosphine, the mortality is much lower and depends on the severity of exposure, age and other predisposing factors of the patients.

Death, which may be sudden, usually occurs within four days but may be delayed for one to two weeks. Acute metal phosphide poisoning, particularly deliberate aluminum phosphide (Phostoxin) poisoning, may cause death within a few hours (Singh et al., 1985; Chopra et al., 1986; Khosla et al., 1988; Misra et al., 1988).

Severity of phosphine/phosphide poisoning:

Deliberate ingestion of the metal phosphides, particularly aluminum phosphide (Phostoxin), is usually more severe than occupational phosphine intoxication. However, the clinical severity of phosphine/phosphide poisoning could be classified as follows.

(a) Mild exposure may present as slight respiratory, gastrointestinal and neuropsychiatric disorders such as cough, shortness of breath, nausea, vomiting, headache, fatigue and dizziness.

(b) Moderate exposure may cause cardiovascular, renal and hepatic dysfunction, as well as more severe respiratory, gastrointestinal and neuropsychiatric involvement, e.g. tachycardia, hypotension, persistent coughing, paraesthesia, tremor, diplopia, ataxia, intention tremor, retrosternal and abdominal pain, shortness of breath, oliguria, jaundice and diarrhoea.

(c) Severe exposure may progress to shock, gastrointestinal haemorrhage, pulmonary oedema, cardiac arrhythmias, metabolic acidosis, cyanosis, convulsions and coma. Renal failure and liver damage may also occur.

Common causes of death following phosphine/phosphide poisoning are pulmonary oedema, cardiac arrhythmias and myocardial injury. A secondary cause of death may be renal failure.

Stephenson (1967) classified patients seriously poisoned by phosphine into 3 groups: (a) those who die within a few hours with pulmonary oedema (b) the majority of fatal cases who die after about 30 hours, and (c) those who survive the first 3 days who may not be in danger, despite extensive liver damage and renal dysfunction.

9.4 Systematic description of clinical effects

9.4.1 Cardiovascular

Cardiovascular effects of aluminum phosphide poisoning were studied by Khosla, Nand and Kumar (1988). Twenty-five cases of aluminum phosphide poisoning were observed by the authors over a period of 2 years; 16 cases (64%) had evidence of cardiac dysfunction. Despite adequate treatment, 40% of the patients died. Shock and cardiac dysrhythmia were the main effects. In another study by Singh & Rastogi (1989), out of 32 cases of aluminum phosphide poisoning, cardiac arrhythmia (28), dyspnoea (25), palpitation (25), cyanosis (12), hypotension (12) and shock (15) were the main clinical manifestations. Hypermagnesaemia due to myocardial and liver damage occurred in 13 patients.

Roman & Dubey (1985) and Khosla et al. (1988) have reported circulatory failure, cardiac dysrhythmias, myocarditis and cardiac failure; the dysrhythmias included complete heart block, atrial fibrillation, chaotic atrial and ventricular tachycardia.

9.4.2 Respiratory

The respiratory tract is a major target for phosphine poisoning. The initial symptoms include cough, sore throat, tightness in the chest, retrosternal pain, dyspnoea, followed by persistent coughing, pulmonary oedema and respiratory distress syndrome which may induce mortality. In a study of 59 cases of phosphine poisoning by Harger & Spolyar (1958), 26 patients died mainly due to respiratory disorders. The commonest finding at autopsy was congestion of the lungs with marked oedema.

Wilson et al. (1980), in a study of 2 children and 29 crew members aboard a grain freighter with phosphine poisoning, reported cough, shortness of breath and pulmonary oedema. On postmortem examination they found pulmonary oedema and pleural effusion. Misra et al. (1988) on postmortem examination in two patients, found pulmonary oedema and desquamation of the lining epithelium of the bronchioles. In a study by Khosla et al. (1988) on 15 cases of aluminum phosphide poisoning, pulmonary oedema was the main cause of mortality in 7 patients.

Chugh et al. (1989) reported 4 cases of adult respiratory distress syndrome (ARDS) following aluminum phosphide poisoning. All their patients had

shock on admission and developed ARDS within 6 hours. Exhalation of phosphine was detected by positive silver nitrate test. In a study by Khosla and Nand (1988) on 15 cases of aluminum phosphide poisoning, pulmonary oedema was one of the main findings which contributed to the cause of death in 8 patients. Chemical pneumonia may also be associated with pulmonary toxic effects.

9.4.3 Neurologic

9.4.3.1 Central nervous system (CNS)

The CNS is a major target in phosphine poisoning. Neurologic symptoms included headache, vertigo, tremors, and unsteady gait, progressing to convulsion, coma and death. Wilson et al. (1980) described CNS symptoms of acute phosphine

poisoning in 2 children and 29 crew members aboard a grain freighter as headache, fatigue, drowsiness, dizziness and paraesthesia weakness, followed by tremor on physical examination, intention tremor in 9 patients ataxia in 2 patients, convulsion and coma in a child who died. Disturbances of smell and taste, dizziness and other clinical manifestations of phosphine poisoning were observed in the workers at a large shipyard in Norway (WHO, 1988).

Miara et al. (1988) described CNS effects in 8 cases of acute phosphine poisoning as drowsiness (3), stupor (2) and delirium (1). On postmortem examination, the brain was markedly congested with areas of exudation, and small haemorrhages were observed.

9.4.3.2 Peripheral nervous system

Some patients with phosphine/phosphide poisoning develop paraesthesia, fatigue and weakness (Wilson et al., 1980; Misra et al., 1988). Peripheral neuropathy (neuritis) may occur, but no studies of the effects of phosphine/phosphide on the peripheral nervous system have been reported.

9.4.3.3 Autonomic nervous system

There is no evidence of direct toxic effects of phosphine/phosphide on the autonomic nervous system, but indirect effects through the adrenal gland and the central nervous system may induce tachycardia, hypotension, shock, and gastrointestinal disorders.

9.4.3.4 Skeletal and smooth muscle

Transient elevation of MB fraction of creatinine phosphokinase in a surviving child with phosphine poisoning reported by Wilson et al. (1980) revealed cardiac and skeletal muscle involvement. Gastrointestinal and vascular disorders, such as abdominal pain and vascular collapse, may be associated with smooth muscle constriction.

9.4.4 Gastrointestinal

Initial symptoms following ingestion of the metal phosphides, particularly aluminum phosphide are nausea, vomiting and abdominal pain (Chopra et al., 1986). As Misra et al. (1988) reported, within 5 minutes of ingestion of aluminium phosphide tablets, patients develop epigastric pain and vomiting; dryness of the mouth, abdominal cramp and diarrhoea may also occur. In severe cases, haematemesis and melaena may develop but gastrointestinal haemorrhage has not been recorded as a cause of death (in phosphine/phosphide poisoning). At autopsy, gastrointestinal mucosal congestion and haemorrhage have been found.

9.4.5 Hepatic

The liver may be affected by phosphine/ phosphide poisoning, but the effects are delayed and rarely cause death. Jaundice may occur 24 hours or more after exposure. In 31 cases of phosphine poisoning studied by Wilson et al. (1980), jaundice occurred in 52% of the patients. Liver function tests were abnormal in a further 10 patients. Abnormalities included elevations of transaminases (mainly SGPT) and lactic dehydrogenase (5 patients).

Misra et al. (1988) found one patient with jaundice among 8 patients with phosphine poisoning they studied. The patient died because of renal and hepatic failure and ventricular tachycardia. On autopsy, petechial haemorrhages were seen on the surface of the liver and histopathological examination showed vascular degeneration of hepatocytes.

9.4.6 Urinary

9.4.6.1 Renal

Toxic effects of phosphine and the metal phosphides on the kidneys are rare and may be delayed. In 31 cases of phosphine poisoning studied by Wilson et al. (1980), renal symptoms were not prominent. Urinalyses of 30 patients revealed abnormalities in 8, usually as microscopic haematuria and bile in urine. None of these abnormalities persisted and all patients improved within a week except one child who died due to cardiovascular and pulmonary toxic effects.

Misra et al. (1988) found one patient with acute renal failure of 8 cases of phosphine poisoning reported. This patient developed anuria with blood urea of 80 mg/dL and serum creatinine of 3.5 mg/dL on the second day of admission. Because of persistent anuria and uraemia, the patient underwent peritoneal dialysis but died 72 hours after admission because of hepato-renal failure and ventricular tachycardia. On postmortem examination, the kidneys were congested with focal areas of exudation and small haemorrhages.

Chopra et al. (1986) reported one patient with significant proteinuria (4.8 g/day) which gradually disappeared over 10 days, and another patient who developed renal failure.

Plasma renin activity (PRA) is increased in shock due to aluminum phosphide poisoning (Chugh et al., 1990). An initially high PRA continued to rise, probably due to slow release of toxic phosphine gas, which was detected by a positive silver nitrate paper test. The rise in PRA was directly proportional to the dose of aluminum phosphide consumed and there was a direct relationship between mortality and an increased PRA. The authors concluded that angiotensin converting enzyme inhibitors may have a role in combating shock in AlP poisoning.

9.4.6.2 Others

No data available.

9.4.7 Endocrine and reproductive systems

There is little information on the effects of phosphine/phosphide on the endocrine and reproductive systems. Adrenocortical involvement in aluminum phosphide poisoning was studied by Chugh et al. (1986) in 50 cases. A significant rise in plasma cortisol (> 1048 nmol/L) was observed in 20 patients. Postmortem examination in 10 patients revealed mild to moderate adrenal cortex changes including congestion, oedema, and cellular infiltration. There was no evidence that adrenal insufficiency or haemorrhage was the cause of shock in these patients.

9.4.8 Dermatologic

There have been no reports of dermal symptoms in phosphine/phosphide poisoning.

9.4.9 Eye, ear, nose, throat

Local effects

There has been no reports on the local effects of phosphine/phosphide on the eyes and ears. The irritant effects of phosphine on the nose and throat are probably trivial in comparison with those on the lung. Initial clinical manifestations of mild phosphine inhalation may mimic upper respiratory tract infection, but these are overshadowed by the other effects of phosphine poisoning including pulmonary, gastrointestinal and cardiovascular disorders.

9.4.10 Haematologic

The haematologic system is not a major target in phosphine/phosphide poisoning. However, marked congestion of the spleen with focal areas of exudation and small haemorrhages were found during the post mortem examination of a patient who died due to phosphine poisoning, though this was not the cause of death (Misra et al., 1988). A reduction in red blood cells, haemoglobin and haematocrit due to phosphide poisoning has been reported in animal experiments (WHO, 1988) but the only report in humans was of a patient who developed purpura, with transient reduction of red blood cells, platelets and haemoglobin which was ascribed to phosphine/phosphide poisoning.

9.4.11 Immunologic

No data available.

9.4.12 Metabolic

9.4.12.1 Acid-base disturbances

Metabolic acidosis is a common problem in severe phosphine poisoning, although it has not been reported in detail.

9.4.12.2 Fluid and electrolyte disturbances

Fluid and electrolyte disturbances may occur in severe phosphine/phosphide poisoning, particularly hypokalaemia associated with metabolic acidosis, renal dysfunction, and hypermagnesaemia.

9.4.12.3 Others

No data available.

9.4.13 Allergic reactions

There has been a single case report of purpura ascribed to phosphine poisoning. The platelet count was reduced to 60,000/mm^3 and red blood cell to $3.1 \times 10^6/mm^3$. On recovery, both the platelet and

red cell counts increased to 210,000/mm^3 and 4.8×10^6 /mm^3, respectively.

9.4.14 Other clinical effects

No data available.

9.4.15 Special risks: Pregnancy, breastfeeding, enzyme deficiencies

No data available.

9.5. Others

No data available.

9.6 Summary

10. MANAGEMENT

10.1 General principles

Management depends on the route of exposure and proper first aid treatment must be performed.

(a) First aid

In case of phosphine inhalation, the patient must be removed from the exposure site and rested. Rescuers should follow fully safety procedures. If a patient is unconscious, place in the semi-prone recovery position or otherwise maintain the airway and give oxygen if required. If breathing stops, immediately ventilate the patient artificially (mouth-to-mouth/nose or mechanically with oxygen if available). If the

heart stops, begin cardiopulmonary resuscitation (CPR). The patient must then be referred to the nearest medical centre for further treatment (Vale and Meredith, 1983).

In case of ingestion of a metal phosphide, do not give milk, fats or saline emetics by mouth. If the patient is conscious, induce vomiting. After vomiting, administer activated charcoal (50 g in water by mouth) if available. Early clearance of zinc phosphide from the gut was recommended by Stephenson (1967) although he found Zn_3P_2 in gastric contents at autopsy when gastric lavage had been performed.

(b) Medical treatment

1. Gastric lavage, with tracheal intubation if appropriate, using 2% sodium bicarbonate solution (to limit hydrolysis of zinc phosphide). Stephenson (1967) used copper sulphate as the precipitation solution for gastric lavage for Zn3P2. Indian authors (Chopra et al, 1986; Khosla et al., 1988; Misra et al, 1988) applied potassium permanganate for gastric lavage.

2. Activated charcoal or medicinal liquid paraffin may limit absorption of phosphine and zinc phosphide respectively and may be administered by mouth or stomach tube (although it did

not work in the patient reported by Stephenson,1967). Repeated doses of activated charcoal together with sorbitol (to avoid constipation) may be useful and has been used by the author but has not been yet reported for phosphine/ phosphide poisoning.

3. Monitor and support vital functions, particularly cardiovascular, respiratory, hepatic and renal functions. Treat shock conventionally (Chopra et al, 1986; Khosla et al, 1988). Dopamine and hydrocortisone succinate have been used to overcome the shock.

4. Perform arterial blood gas analysis and correct respiratory dysfunction by clearing the airways, giving oxygen and perform artificial (mechanical) respiration if required. Metabolic acidosis must also be treated by giving sodium bicarbonate according to the results of arterial pH and blood gas analyses.

5. Hepatic and renal failure should be treated as required, with consultation with an experienced hepatologist and nephrologist.

10.2 Life supportive procedures and symptomatic treatment

Dehydration and shock was treated by infusion of dextrose-saline, dopamine hydrochloride and hydrocortisone hemisuccinate by Khosla et al (1988). Severe metabolic acidosis must also be promptly treated by giving intravenous sodium bicarbonate. Calcium gluconate has been used as a membrane stabilizing agent. It was effective in controlling excitement and convulsions in some patients. However, if convulsions do not respond to calcium, an anticonvulsant drug such as diazepam should be administered intravenously.

Severe cases of phosphine/phosphide poisoning must be treated in an intensice care unit (ICU) in which vital facilities, particularly cardiopulmonary monitoring and resuscitation, would be available. Mechanical respiration may be required in severely poisoned patients. Unfortunately there is no specific treatment for phosphine/phosphide poisoning. Therefore, life supportive procedures and symptomatic treatment should be applied whenever clinically indicated.

10.3 Decontamination

Depending on the route of entry different procedures for decontamination must be performed. In case of inhalation, the patient must be removed from the contaminated area. With the patient at rest, clear the airway and give oxygen and artificial respiration as required. In the case of metal phosphide ingestion, vomiting should be induced while preparation is made for gastric aspiration and lavage. Syrup of ipecac can be used as an emetic. Alternatively, copper sulphate 0.5 g as 1% aqueous solution can be given and has the additional theoretical benefit of forming insoluble copper phosphide (Stephenson 1967). Indian physicians (Chopra et al., 1986; Khosla et al., 1988, Misra et al., 1988) have used potassium permanganate solution (1:1000) as an oxidative agent for gastric lavage, although experimental and clinical evidence is lacking.

It is obviously important to clear the metal phosphide (AlP. Zn_3P_2, Mg_3P_2) from the entire gastrointestinal tract. A large dose (100 g) of mineral oil is recommended, but it is not always effective. In such circumstances the dose should be repeated and, if necessary, followed by a magnesium sulphate purge, bearing in mind that this may lead to further water and electrolyte losses. Activated charcoal with sorbitol (Medicoal) may be effective and 5 to 10 g should be given every 2 to 3 hour by mouth or through a nasogastric tube. Milk, fats and saline emetics must not be given as they may induce more toxicity with phosphine/phosphide.

10.4 Elimination

Since observed phosphine/phosphide is rapidly oxidised in the blood it seems that elimination techniques such as forced diuresis, alkinisation, haemoperfusion and dialysis will be ineffective. Stephenson (1967) reported that forced diuresis was not effective in one patient. However, correction of dehydration and metabolic acidosis by intravenous administration of isotonic solution and sodium bicarbonate is required. Repeated doses of activated charcoal with sorbitol by mouth or through a nasogastric tube (gastrointestinal dialysis) may be effective. Haemodialysis is required for the treatment of acute renal failure which may complicate phosphine poisoning.

10.5 Antidote

10.5.1 Adults

No antidote is available for phosphine/ phosphide poisoning.

10.5.2 Children

No antidote is available for phosphine/ phosphide poisoning.

10.6 Management discussion

Since the exact mechanism of toxicity of phosphine/phosphide poisoning is not clear in human beings, no specific treatment is available. A review of the European cases by Stephenson (1967) suggests that early vomiting improves the prognosis. Two young women swallowed similar quantities of zinc phosphide in a suicide pact. One woman was induced to vomit by mechanical means shortly after poisoning; she had only transient symptoms and recovered completely. Her friend would not vomit and despite gastric lavage one hour after poisoning, she died within 24 hours.

Early recognition and treatment of phosphine/phosphide poisoning is therefore of great importance. Treatment of shock and metabolic acidosis together with the intensive care therapy of the cardiopulmonary effects are essential.

11. ILLUSTRATIVE CASES

11.1 Case reports from literature

Cases of acute phosphine poisoning reported in the literature were reviewed by Harger & Spolyar (1958). Since 1900, a total of 59 cases with 26 deaths have been recorded.

In 6 of 11 reports, cargoes of ferrosilicon were cited as the source of phosphine and in these cases the victims were passengers or crew members on the ships or barges concerned. Other cases involved the exposure of welders to calcium carbide and/or raw acetylene and of submariners to sodium phosphide.

Stephenson (1967) reported a fatal case of zinc phosphide poisoning and reviewed the European literature. A 37-year old woman drank a mixture of 180 g zinc phosphide and water with suicidal intent. The zinc phosphide was 85% technical grade powder used by a game-keeper to prepare rodent baits. Vomiting began one hour after ingestion and was frequent and violent. She was discovered in a state of shock after about 5 hours. Her skin was cold and blue; blood pressure was unrecordable and heart sounds were inaudible. Occasional ronchi were heard over the right lung. The breath smelt of phosphine; one pint of pungent black fluid, smelling of phosphine was aspirated from her stomach. After this, her rectal temperature was 92°F (33°C). Arterial blood gas analysis is revealed severe metabolic acidosis corrected by 1200 mEq sodium bicarbonate over 8 hours. White blood cells were 15000/mm^3 with 94% neutrophils. Serum zinc concentration was 590 to 605 ng/100 mL (normal 120 to 200 ng/100 mL). The ECG showed sinus tachycardia and slight S-T depression in the left ventricular leads. Severe abdominal pain, hepatic factor and refractory tetany persisted for several hours. Urine output diminished; fever and tachypnea preceded a rapidly developing confusional state and unexpected cardiac arrest occurred 41 hours after ingestion. Postmortem examination revealed congestion in all organs. The lungs were oedematous, the gastric mucosa were deeply haemorrhagic and some centrilobular necrosis of the liver and patchy necrosis of the convoluted tubules of the kidneys were observed.

Wilson et al. (1980) reported 31 cases of acute phosphine poisoning aboard a grain freighter. These included 2 children, one of whom died. The predominant symptoms were headache, fatigue, nausea, vomiting, cough and shortness of breath. The abnormal physical findings included jaundice, paraesthesia, ataxia, intention tremor, and diplopia. Focal myocardial infiltration with necrosis, pulmonary oedema and widespread small vessel injury were found at postmortem examination.

Singh et al (1985) reported 15 patients who had ingested 1.5 to 9 g (mean 4.7 g) of phostoxin pellets or tablets (containing 58% aluminum phosphide); 13 cases were attempted suicides. Repeated vomiting and hypotension occurred in all patients, and 13 were in shock on admission. Other common features included impaired sensorium, restlessness, tachycardia, tachypnea, pulmonary crepitations, oliguria,

anuria and jaundice. Half of the patients had raised blood

urea, creatinine, bilirubin and transaminases. Electrocardiographic abnormalities were observed in 6 patients. Metabolic acidosis with blood pH values of 6.97 to 7.31 and bicarbonate of 4.6 to 14.5 m mol/L were present in all 6 patients tested. Eleven patients died and postmortem examination revealed upper gastrointestinal congestion and in 2 cases haemorrhagic fluid was present in the stomach. Lungs were congested and heavy and showed fibrinous pulmonary oedema. Examination of the liver revealed mild fatty infiltration and areas of centrizonal in two cases with haemorrhages in another.

Chopra et al. (1980) described 16 patients suffering from aluminum phosphide poisoning which accounted for half the total number of cases of acute poisoning in their medical centre. Profuse vomiting, upper abdominal pain and shock were the commonest presenting features. Six patients who had taken unexposed tablets of AlP died because of cardiovascular collapse, pulmonary oedema and acute renal failure.

Khosla et al. (1988) presented 25 cases of aluminum phosphide poisoning in which 16 (64%) cases had evidence of cardiac dysfunction; the mortality was 40%. Peripheral circulatory failure, cardiac dysrhythmias, myocarditis and cardiac failure were the main cardiovascular findings.

Misra et al. (1988) reported 8 cases of phosphine poisoning following ingestion of aluminum phosphide tablets for suicidal attempt. The clinical picture consisted of gastritis, altered sensorium and peripheral vascular failure in all cases, cardiac arrhythmia (3), jaundice and renal failure (1 each). Six patients died with a mean hospital stay of 19 hours. Post mortem examinations revealed pulmonary oedema, vascular degeneration of hepatocytes, dilatation of hepatic central veins and areas of nuclear fragmentation.

12. ADDITIONAL INFORMATION

12.1 Specific preventive measures

The most important factor in the safe handling of phosphine and metal phosphides and in their formulation, is proper work practices. Management should identify these, provide training for the operatives, and ensure that these practices are carried out. Personal protective measures recommended to reduce the likelihood of absorption of phosphide preparations include the wearing of:

(a) Synthetic rubber gloves
(b) Rubber boots
(c) Lightweight impervious overalls, and
(d) Suitable eye protection

Adequate washing facilities should be available at all times during handling. Eating, drinking and smoking should be prohibited during handling. The means to measure the concentration of phosphine in the air should be available and used as required. When necessary, respiratory protective equipment should be worn. In fumigation, each operator or

other person liable to be exposed to the gas must be provided with an efficient means of respiratory protection. Persons exposed to magnesium or aluminium phosphide (or any other readily hydrolysed phosphide), which may give rise to an airborne dust, should be protected by respiratory protective equipment. This should be protective against gaseous phosphine, since hydrolysis of dust in the filter of a dust mask or respirator may give rise to high phosphine exposure (WHO, 88).

No occupational accidents have been reported since 1957. It seems that the established safety precautions are satisfactory. Stephenson (1967) recommended the prohibition of sale and distribution of zinc phosphide to all but experts. This should also apply to aluminum and magnesium phosphides.

Wilson et al. (1980) pointed out that ship crew members who work with toxic substances must be adequately educated in preventive measures. Multilingual signs should be placed aboard ships as a reminder of toxic hazards. Most important, ship owners and masters ought, whenever possible, to consider substitution of less toxic fumigation for such highly poisonous agents as phosphine.

Singh et al (1985), suggested that since the mortality of aluminum phosphide poisoning is so high and there is no specific antidote, a less toxic but equally effective agent should be sought to replace this lethal substance.

Chopra et al (1988) indicated that the United Nations Organisation and its agencies WHO and FAO and others in consultation with state governments should quickly take appropriate steps to prevent further loss of lives as a result of self-poisoning with aluminum phosphide.

Misra et al (1988) pointed out that the high mortality and lack of specific antidotes should caution the authorities dealing with the distribution and use of this pesticide.

12.2 Other

Leaks, spillages, and residues

Small leaks and residues of compressed gas can be discharged slowly to the atmosphere in the open air. Larger quantities should be burned using an appropriate burner.

Spillages and residues of metal phosphides in containers will evolve phosphine for several days by reaction with atmospheric moisture. Respiratory protective equipment will be required by those dealing with them.

Residues at the site of spillage should be washed away using a large quantity of water and the area kept secure and well ventilated until the gas is no longer measurable.

Combustible packages can be incinerated at high temperature (>1000°C) using proper facilities. Containers should not cleaned for re-use, but should be disposed of by deep burial, at an approved site, well away from habitation and where

there is no danger of contamination of water sources (WHO, 1988). Sowunmi (1985) measured the phosphine residues on cowpeas fumigated with phostoxin tablets and showed them to be well below the 0.1 ppm tolerance limits for grain at all treatment levels.

Calzolari (1990) compared the residue formation of phostoxin (AlP) with magnesium phosphide (Mg_3P_2) and reported that the latter left a much lower residue concentration, with no detectable PH3 after 120 hours.

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