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TOXICOLOGY PROFILES FOR CHEMICALS IDENTIFIED IN THE GROUNDWATER FROM
THE OLD CAMDEN COUNTY LANDFILL NSB KINGS BAY GA
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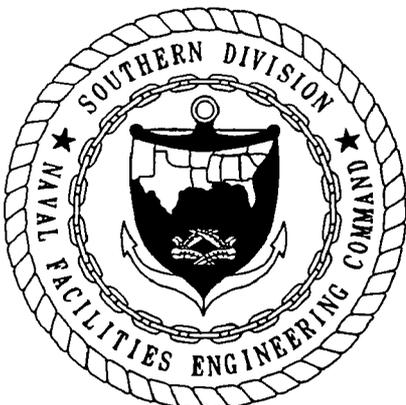


**TOXICOLOGY PROFILES FOR CHEMICALS
IDENTIFIED IN THE GROUNDWATER FROM
THE OLD CAMDEN COUNTY LANDFILL**

**SUBMARINE BASE KINGS BAY
KINGS BAY, GEORGIA**

**NAVY CLEAN - DISTRICT I
CONTRACT NO. N62467-89-D-0317**

SEPTEMBER 1992



**SOUTHERN DIVISION
NAVAL FACILITIES ENGINEERING COMMAND
CHARLESTON, SOUTH CAROLINA
29411-0068**

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INTRODUCTION

The medical, engineering, environmental, and industrial hygiene personnel at the Submarine Base Kings Bay identified a need for current information on the known toxicological effects of the chemicals identified in the groundwater plume emanating from the Old Camden County Landfill. This document was developed in response to this need. It provides the officials a quick reference guide on the toxicology associated with both acute and chronic exposure to four chemicals identified at this site.

This document was derived from the TOMES[®] Medical Management Database[®] 1992 and was edited by a board certified, human health toxicologist. In the interest of brevity and readability, this document was not designed to give the officials all of the information available concerning the toxicology of these chemicals. Rather, it gives them the most relevant information concerning the toxicology of these chemicals as well as the full literature citations supporting the information. Parties interested in more complete information concerning the toxicology of these and other chemicals identified may wish to consult other documents such as the Toxicology Profiles developed by the Agency for Toxic Substances and Disease Registry (ATSDR).

The chemicals covered in this document are listed in alphabetical order. The organization of each chemical summary is the same. The first part of each chemical profile is a summary of the toxicology by organ system, e.g. cardiovascular or genitourinary system. The next part gives information concerning the carcinogenicity, teratogenicity, effects on pregnancy, and genotoxicity or chromosomal aberration potential of the chemical. The third section deals with clinical parameters which may be altered by exposure to the chemical. The fourth section covers decontamination and listed treatment protocols for each chemical by route of exposure, e.g. inhalation or dermal exposure. The fifth section gives additional information concerning the toxicology of the chemical and case reports of persons exposure to the chemical. A full listing of references cited in each profile are given in the final section.

This document is written for health care professionals. It uses abbreviations and terminology which are commonly used in the medical field. For further information you may want to consult a reference such as Dorland's Illustrated Medical Dictionary.

The following chemicals are covered in this document:

- Benzene
- Methylene Chloride
- Trichloroethylene
- Vinyl Chloride

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BENZENE

INTRODUCTION

This document provides information to the health professional concerning the known toxic effects of benzene in humans. In addition it provides information on the symptomology which may be encountered in a person exposed to benzene.

This document also provides the health professional suggestions concerning medical surveillance and laboratory tests which may be of use in the medical management of the exposed patient. This information is derived from the TOMES[®] Medical Management Database, 1987-1992, from Micromedix, Inc. This document is not a comprehensive guide to the medical management of the patients exposed to benzene. Rather, it is designed to give the health care professional up-to-date information on the toxic effects of benzene and provide information on the management of these patients that the health care professional may find useful. The health care professional may wish to consult additional sources of information concerning the management of patients exposed to benzene.

I. SUMMARY OF SYMPTOMOLOGY BY ORGAN SYSTEM

Acute benzene toxicity from inhalation results in CNS effects. Ingestion has been reported, associated with toxicity. Chronic exposure has resulted in aplastic anemia, acute myeloblastileukemia, erythroleukemia, and death. The possibility of chronic poisoning in employees of gasoline filling stations and bulk gasoline loading facilities exists due to the high concentration of benzene in gasoline.

1.0 HEAD, EYES, EARS, NOSE AND THROAT

EYES: Splash contact causes a moderate burning sensation, with slight transient epithelial cell injury, and rapid recovery (Grant, 1986).

1.1 CARDIOVASCULAR

Ventricular fibrillation can occur due to myocardial sensitization (Nahum & Hoff, 1934).

1.2 RESPIRATORY

Bronchial and laryngeal irritation may occur after inhalation. Ingestion may cause substernal pain, cough, and hoarseness. Death can occur due to respiratory failure. Pulmonary edema may occur.

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1.3 NEUROLOGIC

Initially euphoria is seen followed by headache, giddiness, vertigo, and ataxia (Harrington, 1917). High doses can result in confusion, convulsions, and coma. Chronic exposure can produce headache (Harrington, 1917), fatigue, dizziness, and loss of appetite. Transverse myelitis has been reported (Herregods et al. 1984).

1.4 GASTROINTESTINAL

Burning develops in mucous membranes of the mouth, pharynx, and esophagus shortly after ingestion. Stomach pain, nausea, and vomiting occur early in intoxication.

1.5 HEPATIC

Toxicity is more common chronically but may occur after a large exposure.

1.6 GENITOURINARY

No information is available.

1.7 HEMATOLOGIC

Exposures producing acute myelotoxicity can result in delayed hematopoietic changes (Gosselin et al. 1984). Chronic exposure can produce suppression of hematopoiesis resulting in spontaneous bleeding, anemia, and leukopenia. Large single-dose exposure has been postulated to produce acute myelotoxicity or leukemia (Gerarde, 1960).

Aplastic anemia and leukemia can occur (Aksoy, 1985a; Aksoy, 1985b). Work exposure to 100 ppm resulted in 140 excess deaths from leukemia per 1000 exposed; 10 ppm resulted in 14 excess deaths (Landrigan & Rinsky, 1984). Bone marrow toxicity in exposed individuals may be triggered by individual susceptibility - women more than men, pregnancy, infection, and alcoholism. All elements of the marrow are affected.

Blood diseases that have been associated with benzene exposure include pancytopenia, leukopenia, bone marrow hypoplasia or aplasia, thrombocytopenia, granulocytopenia, and lymphocytopenia (NTP, 1986).

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1.8 DERMATOLOGIC

Skin contact may cause erythema, blistering, and dermatitis due to the defatting action of benzene.

II. CARCINOGENICITY/TERATOGENICITY

2.0 CARCINOGENICITY

Benzene is classified by the EPA as a group A carcinogen and by IARC as a group 1 carcinogen. Both of these designations mean that benzene is a confirmed human carcinogen.

Although benzene consistently tests negative in most tests for short-term mutagenicity, OSHA assessment has revealed that a 40-year exposure to 1 ppm in the workplace doubles the risk of dying from acute myelogenous leukemia (Goldstein, 1989).

Aplastic anemia and leukemia can occur (Aksoy, 1985a; Aksoy, 1985b). Work exposure to 100 ppm resulted in 140 excess deaths from leukemia per 1000 exposed; 10 ppm resulted in 14 excess deaths (Landrigan & Rinsky, 1984). Risk assessment studies have reported estimated lifetime excess deaths due to leukemia of 14 to 104 per 1000 workers exposed to 10 ppm (Austin et al. 1988).

Bone marrow toxicity in exposed individuals may be triggered by individual susceptibility - women more than men, pregnancy, infection, and alcoholism. All elements of the marrow are affected.

Acute myeloblastic leukemia has been the most common malignancy associated with benzene exposure (Goldstein, 1989).

2.1 PREGNANCY

No information is available.

2.2 TERATOGENICITY

Studies in animals have generally been negative. More animal and epidemiological studies are needed before more definitive conclusions can be made (NTP, 1986).

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2.3 CHROMOSOME ABERRATIONS

There are reports of short-lived chromosome abnormalities, chromatid gaps with occasional breaks of chromatids and chromosomes, and some sister chromatid exchanges. At least 23 studies have shown significantly increased chromosomal aberrations, some persisting for years after exposure has ceased (NTP, 1986).

III. LABORATORY STUDIES

3.0 SERUM/BLOOD

Obtain a baseline CBC (complete blood count) on anyone with acute exposure to benzene. Periodic hematologic examination may be necessary until the clinical signs and symptoms of acute exposure have resolved.

3.1 URINE

Inoue et al (1986) found a good correlation between urine phenol levels and benzene concentrations in the breathing zone. Monitoring urine phenol levels are useful in monitoring respiratory exposure. Some OTC medications containing phenol may give false positives and/or increased urine phenols. Examples are Pepto Bismol(R) and Chloraseptic(R) (Baselt & Cravey, 1989).

Urine phenol levels in non-exposed individuals are less than 10 mg/L. Urine phenol levels after chronic exposure to 0.5 to 4 ppm are less than 30 mg/L. Urine phenol levels after exposure to 25 ppm average 200 mg/L (Baselt & Cravey, 1989).

3.3 OTHER

Monitor EKG for cardiac arrhythmias.

IV. TREATMENT BY EXPOSURE ROUTE

4.0 INHALATION EXPOSURE

4.0.1 DECONTAMINATION

Move patient to fresh air. Monitor patient for respiratory distress. Emergency airway support and 100 percent humidified supplemental oxygen with assisted ventilation

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may be needed. If a cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, pneumonitis.

4.0.2 TREATMENT

Monitor patient for respiratory depression. If a cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, and pneumonia. Toxic effects of benzene are produced chiefly by inhalation of the vapor. Treatment should include recommendations listed in the ORAL EXPOSURE section when appropriate.

4.1 DERMAL EXPOSURE

4.1.1 DECONTAMINATION

Wash exposed area extremely thoroughly with soap and water. A physician may need to examine the area if irritation or pain persists after washing.

4.1.2 TREATMENT

Treatment should include recommendations listed in the ORAL EXPOSURE section when appropriate.

4.2 EYE EXPOSURE

4.2.1 DECONTAMINATION

Exposed eyes should be irrigated with copious amounts of room temperature water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist after 15 minutes of irrigation, an ophthalmologic examination should be performed.

4.2.2 TREATMENT

Treatment is symptomatic since benzene is an eye irritant.

4.3 ORAL EXPOSURE

4.3.1 PREVENTION OF ABSORPTION

EMESIS: Emesis may be indicated in recent substantial ingestion unless the patient is or could rapidly become obtunded, comatose or convulsing. Is most effective if initiated within 30 minutes.

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ACTIVATED CHARCOAL/CATHARTIC: Activated charcoal has been demonstrated to decrease absorption of benzene in animals (Laass, 1980). Administer charcoal as slurry. Charcoal slurry may be aqueous, or mixture of charcoal with saline cathartics or sorbitol.

4.3.2 TREATMENT

Monitor EKG for cardiac arrhythmias. Avoid epinephrine because of the possibility of myocardial sensitization.

V. OTHER INFORMATION

No further information is available.

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METHYLENE CHLORIDE

INTRODUCTION

This document provides information to the health professional concerning the known toxic effects of methylene chloride in humans. In addition it provides information on the symptomology which may be encountered in a person exposed to methylene chloride.

This document also provides the health professional suggestions concerning medical surveillance and laboratory tests which may be of use in the medical management of the exposed patient. This information is derived from the TOMES[®] Medical Management Database, ° 1987-1992, from Micromedix, Inc. This document is not a comprehensive guide to the medical management of the patients exposed to methylene chloride. Rather, it is designed to give the health care professional up-to-date information on the toxic effects of methylene chloride and provide information on the management of these patients that the health care professional may find useful. The health care professional may wish to consult additional sources of information concerning the management of patients exposed to methylene chloride.

I. SUMMARY OF SYMPTOMOLOGY BY ORGAN SYSTEM

Methylene chloride may be absorbed by inhalation, by ingestion, or dermally. It is metabolized in part to carbon monoxide and can produce carboxyhemoglobin levels as high as 9% after an 8 hour exposure to 180 to 200 ppm. Levels up to 40% have been produced after prolonged exposure to higher concentrations, but those above 20% were extremely rare, and are usually in smokers, who have a second source of carbon monoxide from smoking. Toxicity is due to methylene chloride, the carbon monoxide it generates once within the body, and if combustion occurs, additionally to phosgene.

1.0 HEAD, EYES, EARS, NOSE, and THROAT

EYES: Eye irritation has been reported.

1.1 CARDIOVASCULAR

No information is available.

1.2 RESPIRATORY

Inhalation can produce chemical pneumonitis and pulmonary edema (Buie et al. 1986).

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1.3 NEUROLOGIC

This pleasant smelling compound has been used on occasion as an anesthetic. All the phases of anesthesia beginning with excitation may occur up to and including coma and death. Individuals exposed by inhalation may become syncopal, develop anorexia, and headaches.

Ingestion can result in CNS depression preceded by excitement, which can occasionally not completely disappear, leaving chronic CNS effects (Cherry & Waldron, 1962; Barrowcliff & Knell, 1979).

Three year occupational exposure to 300 to 1000 ppm methylene chloride caused memory loss with intellectual impairment and balance disturbances in a 55 to 58 year old man. Bilateral temporal lobe degeneration occurred. Persistent high level of endogenous blood CO was responsible for toxicity (Barrowcliff & Knell, 1979).

A chemist, after years of exposure, developed toxic encephalosis with acoustical and optical delusion and hallucinations (ACGIH, 1980).

1.4 GASTROINTESTINAL

No information is available.

1.5 HEPATIC

Brief dermal exposure to methylene chloride in a splash accident resulted in elevated LFTs one week later which normalized within 2 weeks (Puurunen & Sotaniemi, 1985). In another case dermal exposure for approximately 4 hours resulted in mildly elevated LFT, nausea, fever, chills, and headache (Cordes et al. 1988).

1.6 GENITOURINARY

Kidney toxicity is not likely except in exposure to high doses or chronic exposure.

Acute tubular necrosis was described following inhalation of a tile remover containing methylene chloride, mineral spirits, and methanol by a 19 year old man. Liver function tests were also abnormal and myoglobinuria was present (Miller et al. 1985).

A 16 year old girl exposed to methylene chloride and trichloroethane developed Goodpasture's syndrome after 10 months of workplace exposure with no exhaust ventilation (Keogh et al. 1984).

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1.7 HEMATOLOGIC

Toxicity to the bone marrow is not considered likely except in extreme toxic symptoms or chronic high dose effect.

In a study of volunteers exposed to 200 ppm for 7.5 hour work days, the maximum COHb level was 6.8% (Divincenzo & Kaplan, 1981). Levels ranged from 7 to 15% in subjects exposed to 986 ppm for 2 hours (Stewart et al. 1972a). COHb levels of 26 and 40% were reported following the use of methylene chloride-containing paint removers (Langehennig et al. 1976).

1.8 DERMATOLOGIC

Skin irritation and burns may occur due to defatting action of the solvent. Methylene chloride is mildly irritating to skin on repeated contact. The problem may be accentuated by chemical being sealed to skin by shoes or tight clothing. The situation is most severe with paint remover formulations that form "a skin" or film.

II. CARCINOGENICITY/TERATOGENICITY

2.0 CARCINOGENICITY

Methylene chloride has been shown to be carcinogenic in animals but there is inadequate evidence of carcinogenicity in humans. It is considered a B2 carcinogen by the EPA and a class 3 carcinogen by IARC. These designation indicate that methylene is a probable human carcinogen. This means that there is adequate evidence of carcinogenicity in animals but that evidence of carcinogenicity in humans is either lacking or inadequate.

2.1 PREGNANCY

Methylene chloride crosses the placenta and has been found in breast milk of occupationally exposed women. It is unlikely to have adverse reproductive effects at the TLV of 100 ppm (AMA, 1985).

2.3 TERATOGENICITY

There is limited evidence that methylene chloride is not teratogenic at exposures to sub-toxic doses in animals. It is mutagenic by the Ames test.

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III. LABORATORY STUDIES

3.0 SERUM/BLOOD

Carboxyhemoglobin levels should be followed. Liver function tests should also be followed.

3.1 URINE

No information is available.

3.2 RADIOLOGICAL

Methylene chloride was radio-opaque in vitro (Dally et al. 1987).

IV. TREATMENT BY EXPOSURE ROUTE

4.0 INHALATION EXPOSURE

4.0.1 DECONTAMINATION

Move patient to fresh air. Monitor patient for respiratory distress. Emergency airway support and 100 percent humidified supplemental oxygen with assisted ventilation may be needed. If a cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, pneumonitis.

4.1 DERMAL EXPOSURE

4.1.1 DECONTAMINATION

Wash exposed area extremely thoroughly with soap and water. A physician may need to examine the area if irritation or pain persists after washing.

4.1.2 TREATMENT

Treatment should include recommendations listed in the ORAL EXPOSURE section when appropriate.

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4.2 EYE EXPOSURE

4.2.1 DECONTAMINATION

Exposed eyes should be irrigated with copious amounts of room temperature water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist after 15 minutes of irrigation, an ophthalmologic examination should be performed.

4.3 ORAL EXPOSURE

4.3.1 PREVENTION OF ABSORPTION

EMESIS: Emesis may be indicated in substantial recent ingestions unless the patient is obtunded, comatose or convulsing or is at risk of doing so based on ingestant. Emesis is most effective if initiated within 30 minutes of ingestion.

ACTIVATED CHARCOAL/CATHARTIC: Administer charcoal as slurry. Charcoal slurry may be aqueous, or mixture of charcoal with saline cathartics or sorbitol. Administer one dose of a saline cathartic or sorbitol, mixed with charcoal or administered separately.

4.3.2 TREATMENT

CARBOXYHEMOGLOBIN

Determine a carboxyhemoglobin level. Methylene chloride is converted in part to carbon monoxide. Administer oxygen, 100% humidification, for short periods, then concentrations comfortable to the patient.

The comparative efficacy between 100% normobaric oxygen and hyperbaric oxygen has not been studied in animals or humans.

V. OTHER INFORMATION

5.0 CASE REPORTS

Pike & Shaney (1987) reported a group of 82 out of 200 postal workers who were occupationally exposed to a cresylic acid, phenol, and methylene chloride spill. Clinical effects were elevated liver function tests (21%), headache (66%), nausea (39%), diarrhea (32%), dizziness (28%), weakness (23%), shortness of breath (19%), blurred vision (11%),

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abdominal pain (8%), vomiting (5%), and rash (5%). Some workers complained of symptoms lasting more than 6 days.

Kaufman et al (1989) reported the case of 5 men exposed to methylene chloride; two were working with paint remover in an enclosed space and three were rescue workers. Both workers were wearing paper masks while using the paint remover. The rescuers displayed either mild symptoms of nausea and dizziness or were asymptomatic. Both workers presented in a state of cardiac arrest. One was unable to be resuscitated and the other was resuscitated but died on the 4th day. The carboxyhemoglobin value of the resuscitated worker rose from 0% to 8% over nine hours despite being on 40 to 50% oxygen. The cause of death in these workers was thought to be due to solvent induced narcosis.

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TRICHLOROETHYLENE

INTRODUCTION

This document provides information to the health professional concerning the known toxic effects of trichloroethylene in humans. In addition it provides information on the symptomology which may be encountered in a person exposed to trichloroethylene.

This document also provides the health professional suggestions concerning medical surveillance and laboratory tests which may be of use in the medical management of the exposed patient. This information is derived from the TOMES[®] Medical Management Database, ° 1987-1992, from Micromedix, Inc. This document is not a comprehensive guide to the medical management of the patients exposed to trichloroethylene. Rather, it is designed to give the health care professional up-to-date information on the toxic effects of trichloroethylene and provide information on the management of these patients that the health care professional may find useful. The health care professional may wish to consult additional sources of information concerning the management of patients exposed to trichloroethylene.

I. SYMPTOMOLOGY BY ORGAN SYSTEM

Trichloroethylene is toxic by ingestion, inhalation or dermal exposure. Vasodilation and malaise ("Degreasers Flush") occur in workers who drink ethanol after exposure to trichloroethylene. Inhalation abuse of typewriter correction fluid has been reported.

Optic neuritis and blindness has been reported following ingestion. Eye exposure causes pain and irritation but permanent injury is unlikely. Cardiac arrhythmias (ventricular fibrillation), hypotension, conduction defects, and myocardial injury have been noted.

Respiratory depression and cyanosis; pulmonary hemorrhages and edema have been reported following ingestion and inhalation, respectively. CNS depression, light headedness, dizziness, and euphoria, most notably after inhalation, have been noted. Trigeminal nerve impairment has been noted in individuals chronically poisoned by trichloroethylene.

1.0 HEAD, EYES, EARS, NOSE AND THROAT

HEAD: Weakness and numbness of the face, as well as trigeminal neuralgias may be seen (NIOSH, 1973).

EYES: Optic neuritis and blindness have been reported following inhalation as have double vision and blurred vision (Mitchell & Parsons-Smith, 1969; Baxton & Hayward, 1967;

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NIOSH, 1973; Grant, 1986). The majority of these reports appear to be associated with trichloroethylene and its decomposition products from exposure to heat and alkali.

Contact with the eye produces irritation but, permanent injury is unlikely. However, first and second degree burns have been attributed to the vapors (NIOSH, 1973; Browning, 1965; Grant, 1986).

EARS: Bilateral, symmetrical, eighth cranial nerve deafness, slight for low frequencies but complete for tones over 1000 cycles/sec has been reported with trichloroethylene (NIOSH, 1973).

THROAT: Difficulty in swallowing and salivation occurred in 3 coal miners accidentally poisoned by trichloroethylene subjected to decomposition in closed circuit oxygen respirators (Grant, 1986).

TASTE: Loss of taste has been noted (Mitchell & Parsons-Smith, 1969).

Dysphagia has been reported in workers exposed to trichloroethylene (Lawrence & Partyka, 1981; Grant, 1986).

1.1 CARDIOVASCULAR

Ventricular fibrillation may occur as a result of cardiac sensitization to endogenous catecholamines and is thought to be the cause of death in fatal exposure (Anon, 1974).

Hypotension and first degree heart block have been reported following ingestion of trichloroethylene (Wells, 1982).

Bradycardia, occasional premature contractions, and in a few cases, a rapid irregular pulse have been reported during trichloroethylene anesthesia (Barnes & Ives, 1944; Gutch et al. 1965).

Prolongation of PR interval (0.26 seconds) occurred following an ingestion of nearly 5 ounces of trichloroethylene in a 22 year old (Stentiford & Logan, 1956).

1.2 RESPIRATORY

Respiratory depression resulting in cyanosis is common (Derobert, 1952).

Pulmonary hemorrhages and edema have been reported (Koch, 1931; Patel et al. 1973).

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Bronchial irritation and shortness of breath from trichloroethylene inhalation exposure have been noted (Anderssen, 1957; McCarthy & Jones, 1983).

1.3 NEUROLOGIC

Ingestion and inhalation will produce central nervous system depression, coma, visual disturbances, mental confusion, ataxia, dizziness, loss of coordination, and fatigue (McCunney, 1988; Stentiford & Logan, 1956; McCarthy & Jones, 1983). Residual encephalopathy was present 16 years after a single acute exposure to trichloroethylene (Feldman et al. 1985).

Multiple nerve palsies and peripheral neuropathy have been observed following trichloroethylene intoxication (NIOSH, 1973; Joron et al. 1955).

Inhalation may result in euphoria and addiction (Reynolds, 1988).

Clinical hypesthesia of the trigeminal nerve has been noted in some individuals suffering from chronic poisoning (Mitchell & Parsons-Smith, 1969; Baxton & Hayward, 1967); tests of somatosensory evoked potentials seem to be useful in assessing chronic intoxication caused by trichloroethylene (Barret et al. 1982; Arezzo et al. 1985). There is some evidence that suggests that trichloroethylene decomposition products or impurities may be responsible for cranial nerve injuries (Grant, 1986).

Headache has been reported (Derobert, 1952).

Resting tremor may occur (McCunney, 1988).

Anorexia has been reported (McCunney, 1988).

A 46-year-old worker developed carpal spasm of six weeks duration following cleaning of a vapor degreaser. He wore an air purifying respirator during the cleaning procedure that took about one hour to complete (McCunney, 1988).

Organic dementia has been described in workers exposed to trichloroethylene (Mitchell & Parsons-Smith, 1969; Bardodej & Vyskocil, 1956; Grandjean et al. 1955; Trense, 1965).

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1.4 GASTROINTESTINAL

There are reports associating primary Pneumatosis Cystoides Intestinalis with exposure to trichloroethylene prior to onset of the disease. No cause and effect relationship could be established (Yamaguchi et al. 1985; Sato et al. 1987).

Gastric pain and vomiting were reported following inhalation exposure to trichloroethylene (Derobert, 1952; McCarthy & Jones, 1983).

1.5 HEPATIC

Acute hepatic damage has been reported infrequently and usually only after massive exposure (Joron et al. 1955; James, 1963; AMA, 1985).

A 28-year-old man developed hepatitis that was associated with the use of trichloroethylene in a small, unventilated, basement room. Other causes of hepatitis were ruled out. His 24 hour urinary trichloroacetic acid level, 6 months after the hepatitis, was 9 mg/L (McCunney, 1988).

Hepatitis has also been reported following trichloroethylene anesthesia (Herdman, 1945).

1.6 GENITOURINARY

Acute tubular necrosis and renal failure may follow oral or respiratory exposure (Gutch et al. 1965; Reynolds, 1988).

A 42-year-old man developed gynecomastia and impotence following prolonged exposure to trichloroethylene (Barlow & Sullivan, 1982).

1.7 HEMATOLOGIC

No information is available.

1.8 DERMATOLOGIC

Trichloroethylene is mildly irritating to the skin and prolonged contact may give rise to eczema and dermatitis (Anon, 1974).

Chronic exposure may produce rough, chapped skin (Clayton & Clayton, 1982).

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Exfoliative dermatitis occurred on two separate occasions following inhalational exposure to trichloroethylene in a 29-year old male (Goh & Ng, 1988). A 21-year-old male developed exfoliative dermatitis with mucous membrane involvement, fever, and liver dysfunction associated with occupational exposure to trichloroethylene. A positive patch-test reaction was observed to trichloroethylene and trichloroethanol (Nakayama et al. 1988).

Progressive systemic sclerosis was associated with dermal exposure to trichloroethylene of 2.5 hours duration in a 47 year old previously healthy woman (Lockey et al. 1987).

1.9 OTHER

Menstrual irregularities occurred in a 20-year-old woman following an exposure in the workplace to a high concentration of trichloroethylene vapor. Basal body temperature indicated lack of ovulation (Barlow & Sullivan, 1982).

A condition called "Degreaser's Flush" has been reported in workers who drink ethanol after exposure to trichloroethylene. It consists of vasodilation and feeling of malaise. The cutaneous flushing fades with the reduction of blood alcohol level (Waters et al. 1977).

II. CARCINOGENICITY/TERATOGENICITY

2.0 CARCINOGENICITY

Trichloroethylene is designated a B2 carcinogen by the EPA and but a class 3 carcinogen by IARC. This indicates that the EPA considers trichloroethylene to be a probable human carcinogen while IARC does not consider it to be carcinogenic.

Trichloroethylene is not considered to be carcinogenic in man (AMA, 1985; Kimbrough et al. 1985). Hepatocellular carcinoma was reported in B6C3F1 mice only after chronic administration of high, cytotoxic dose levels (AMA, 1985).

2.1 PREGNANCY

Trichloroethylene has been used as an anesthetic in obstetrics. It may aggravate the normal acidosis and hypoxia of second stage of labor (Phillips & Macdonald, 1971).

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2.3 TERATOGENICITY

Animal studies have not found trichloroethylene to be particularly teratogenic or fetotoxic (Schwetz et al. 1975; Dorfmueller et al. 1979; Land et al. 1981).

III. LABORATORY STUDIES

3.0 SERUM/BLOOD

Blood concentration of trichloroethylene in anesthesia are 5 to 10 mg/dL (Polson & Tattersall, 1969).

3.1 URINE

No information is available.

3.2 RADIOLOGICAL

Trichloroethylene was radiopaque in vitro (Dally et al.1987).

3.3 OTHER

Breath analysis for trichloroethylene analyzed by gas chromatography appeared to be an accurate indicator of the time-weighted vapor exposure to a worker (Stewart et al. 1974).

IV. TREATMENT BY EXPOSURE ROUTE

4.0 INHALATION EXPOSURE

4.0.1 DECONTAMINATION

Move patient to fresh air. Monitor patient for respiratory distress. Emergency airway support and 100 percent humidified supplemental oxygen with assisted ventilation may be needed. If a cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, pneumonitis.

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4.0.2 TREATMENT

Treatment should include recommendations listed in the ORAL EXPOSURE section when appropriate.

4.1 DERMAL EXPOSURE

4.1.1 DECONTAMINATION

Wash exposed area extremely thoroughly with soap and water. A physician may need to examine the area if irritation or pain persists after washing.

4.1.2 TREATMENT

Treatment should include recommendations listed in the ORAL EXPOSURE section when appropriate.

4.2 EYE EXPOSURE

4.2.1 DECONTAMINATION

Exposed eyes should be irrigated with copious amounts of room temperature water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist after 15 minutes of irrigation, an ophthalmologic examination should be performed.

4.3 ORAL EXPOSURE

4.3.1 PREVENTION OF ABSORPTION

EMESIS: Emesis may be indicated in substantial recent ingestion unless the patient is obtunded, comatose or convulsing or is at risk of doing so based on ingested amount. Emesis is most effective if initiated within 30 minutes of ingestion.

GASTRIC LAVAGE: Gastric lavage with a large-bore orogastric tube may be indicated if performed soon after ingestion, or in patients who are comatose or at risk of convulsing.

ACTIVATED CHARCOAL/CATHARTIC: Administer charcoal as slurry. Charcoal slurry may be aqueous, or mixture of charcoal with saline cathartics or sorbitol.

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4.3.2 TREATMENT

Monitor cardiac function closely. Epinephrine or other stimulants should not be used because of the danger of cardiac sensitization, especially by beta-adrenergic agents.

Pulmonary edema, renal failure and liver damage should be treated symptomatically.

Propranolol 40 to 80 milligrams orally in adults MAY be useful to reverse "degreaser's flush."

4.3.3 ENHANCED ELIMINATION

Koppel et al (1988) estimated 70% of the absorbed dose was eliminated in expired air when hyperventilation therapy was used in a 32-year-old male following a suicidal ingestion of 150 grams of trichloroethylene and ethanol. Previous reports indicated 75% of the absorbed dose is metabolized in the liver when hyperventilation treatment was not utilized (Baselt,1982). No liver or renal dysfunction were noted and level of consciousness improved with hyperventilation therapy.

V. OTHER INFORMATION

5.0 CASE REPORTS

Troutman (1988) reported 3 deaths from the intentional inhalation abuse of a typewriter correction fluid containing both 1,1,1-trichloroethane and trichloroethylene. One patient was found collapsed in a field with a bottle of this material in his hand, one was found locked in a bathroom with a bag containing the material over his head, and the third collapsed during an altercation after abusing the material throughout the day. None of the three patients could be resuscitated. Although mustard oil has been added to this product to discourage abuse, it did not deter these three victims.

Bernad et al (1987) reported a cohort of 22 persons exposed to trichloroethylene at low levels (8 to 14 parts per million) in well water for a 5 to 20 year period. Hyperesthesia was present on current perception threshold measurement in 21 of 22 patients. Ten of 10 adults complained of fatigue, somnolence, lack of energy, and numbness and tingling, while 8 of 10 had headache and dizziness, and 4 of 10 had tremor. Three of the 10 had also been evaluated for cardiac arrhythmias. Of the 12 children, 9 had behavioral difficulties (poor learning, aggressive behavior, poor attention span), and one had multiple congenital anomalies.

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Ventricular fibrillation and cardiac arrest were reported in a 15-year-old male who inhaled typewriter correction fluid (Wodka & Jeong, 1991). Serial electrocardiograms and subsequent stress echocardiogram findings were consistent with recent antero-apical subendocardial infarction. The patient was discharged 4 days after admission.

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VINYL CHLORIDE

INTRODUCTION

This document provides information to the health professional concerning the known toxic effects of vinyl chloride in humans. In addition it provides information on the symptomology which may be encountered in a person exposed to vinyl chloride.

This document also provides the health professional suggestions concerning medical surveillance and laboratory tests which may be of use in the medical management of the exposed patient. This information is derived from the TOMES[®] Medical Management Database, 1987-1992, from Micromedix, Inc. This document is not a comprehensive guide to the medical management of the patients exposed to vinyl chloride. Rather, it is designed to give the health care professional up-to-date information on the toxic effects of vinyl chloride and provide information on the management of these patients that the health care professional may find useful. The health care professional may wish to consult additional sources of information concerning the management of patients exposed to vinyl chloride.

I. SUMMARY OF SYMPTOMOLOGY BY ORGAN SYSTEM

Acute exposures and deaths are most often due to CNS depression and subsequent respiratory depression. The primary toxic hazard is to vinyl chloride monomer gas rather than any polyvinyl chloride product, and the primary individuals exposed are workers present during the polymerization process and those in maintenance and descaling. Unpolymerized vinyl chloride monomer may be found in polyvinyl chloride particles and inhalation of polyvinyl chloride particles or dust may result in vinyl chloride monomer toxicity. There may be a long latent between initial exposure and onset of symptoms (Walker, 1981).

"Vinyl chloride disease" is characterized by a scleroderma-like condition of the connective tissue of the fingers, Reynaud's phenomenon followed by acro-osteolysis, liver damage, and sometimes hematologic changes and pulmonary effects. It develops after exposures from 1 month to 3 years and is reversible after cessation of exposure (ATSDR, 1989).

Angiosarcoma, a rare form of liver cancer, has occurred in persons with chronic exposure to at least 50 ppm (ATSDR, 1989).

1.0 HEAD, EYES, EARS, NOSE AND THROAT

EYES: Contact with escaping, compressed gas may cause mechanical injury and frostbite (ACGIH, 1986). There has been a single instance of human corneal injury reported, which healed completely in 48 hours (Grant, 1974). Vapors of vinyl chloride monomer are moderately irritating to the eyes (CCOHS, 1988).

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1.1 CARDIOVASCULAR

Vinyl chloride monomer sensitized the dog heart to epinephrine-induced arrhythmias (Jucker, 1974). Portal hypertension can occur as a result of liver injury (ATSDR, 1989). Ventricular fibrillation may be a cause of sudden death following acute exposure (HSDB, 1989).

1.2 RESPIRATORY

Various pulmonary abnormalities have occurred, including breathlessness, possibly resulting from impaired diffusion capacity. Occupational exposure to high concentrations of vinyl chloride monomer has been loosely implicated to be associated with impaired pulmonary function as measured by single breath transfer for carbon monoxide (TLCO) (Lloyd et al, 1984). Respiratory difficulties may arise in supermarket workers who cut polyvinyl chloride wrap used in wrapping meat. This liberates vinyl chloride monomer and HCL (Anderson Jr, 1974; Stevens, 1974). Pneumoconiosis has been reported in workers exposed to polyvinyl chloride powder or dust (Wagoner, 1983). Respiratory cancer has been reported in vinyl chloride workers, but no dose-response figures were given (ACGIH, 1986).

1.3 NEUROLOGIC

Vinyl chloride monomer has narcotic properties and may cause unconsciousness in air concentrations of 10,000 to 20,000 ppm (Walker, 1981; Proctor & Hughes, 1978). Other signs of CNS depression include headache, dizziness, ataxia and inebriation, euphoria, visual disturbances, numbness and tingling of the extremities, and drowsiness (ATSDR, 1989; HSDB, 1989).

Fatigue has been a symptom reported after chronic exposure, as has paraesthesia (Barlow & Sullivan, 1982). Seizures may occur in deep anesthesia induced with vinyl chloride monomer (Danziger, 1960).

Neurological effects are the main manifestation of acute exposure to vinyl chloride monomer (ATSDR, 1989). Workers exposed to air concentrations less than 50 ppm have shown axonal neuropathy (ATSDR, 1989). EEG changes were seen in vinyl chloride monomer workers (ATSDR, 1989; HSDB, 1989).

1.4 GASTROINTESTINAL

Nausea, vomiting, diarrhea and abdominal pain, sometimes intense enough to mimic an acute surgical abdomen, may follow ingestion of the liquid (HSDB, 1989). Hematemesis may occur following ingestion (HSDB, 1989).

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1.5 HEPATIC

Angiosarcoma of the liver has been reported in individuals exposed when cleaning polyvinyl chloride reaction chambers (Walker, 1981). Angiosarcoma may be preceded by hyperplasia and hypertrophy of hepatocytes and mesenchymal sinusoidal cells, followed by periportal fibrosis and nodular capsular fibrosis leading to prehepatic sclerosis; these changes and subsequent angiosarcoma occur after chronic exposure to air concentrations of 50 to 100 ppm or higher (ATSDR, 1989).

Through 1986 a total of 120 cases of angiosarcoma in vinyl chloride workers had been reported to a worldwide registry (ATSDR, 1989). The latent period between first exposure and diagnosis was from 15 to 29 years, with an average length of exposure of 18.3 years (ATSDR, 1989). The workers with highest risk for developing angiosarcoma have been reactor cleaners, who scraped the solid, caked polymer from the inside of reaction vessels (ATSDR, 1989). This process of hand cleaning is now obsolete. No cases of angiosarcoma have been reported in individuals who have been exposed only to low levels of vinyl chloride monomer (ATSDR, 1989).

Hepatomegaly as well as splenomegaly has been seen in reaction chamber cleaners (Walker, 1981). Hepatic damage seen in man exhibits a sequence of events from proliferation of hepatocytes and sinusoidal cells (may be associated with sinusoidal dilatation) to angiosarcoma. Cirrhosis is generally not observed (Popper et al, 1981). Hepatotoxicity is generally seen after several months or years of exposure to higher concentrations of vinyl chloride monomer, in the range of 500 to 1000 ppm in the air (ATSDR, 1989).

Some preliminary animal studies would indicate that chronic ethanol consumption, concurrent with chronic vinyl chloride monomer exposure, increased risk of hepatic cancers over vinyl chloride monomer alone (Tamburro, 1978).

1.6 GENITOURINARY

Loss of libido was reported in a series of 37 workers (Barlow & Sullivan, 1982). Men exposed to levels of gaseous vinyl chloride monomer as low as 30 mg/m³ 5 years previously showed effects on spermatogenesis (RTECS, 1989).

Male rats breathing 1000 ppm of vinyl chloride in air for 6 hours a day over 55 days prior to mating had lowered fertility (RTECS, 1989).

1.7 HEMATOLOGIC

Thrombocytopenia may be seen in workers exposed to vinyl chloride monomer gas (Walker, 1981). Wide-field capillary microscopy of the hands detected abnormalities in 48 of 152

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(31.6%) of vinyl chloride monomer workers hands and only 3 of 50 (6%) of the hands of non-exposed manual workers (Maricq et al, 1976).

Pathologic porphyrinuria, especially secondary to co-proporphyrinuria, is a consistent pathological finding and may be useful in detecting the incipient toxic phase of vinyl chloride monomer hepatic disease (Doss et al, 1984).

1.8 DERMATOLOGIC

Vinyl chloride monomer-induced sclerodermic skin changes with new collagen formation and capillary abnormalities of the nail folds has been seen as part of acro-osteolysis (Walker, 1981). Vinyl chloride monomer is stored under pressure, and exposure to the escaping gas may cause frostbite (ACGIH, 1986).

1.9 OTHER

MUSCULOSKELETAL: Acro-osteolysis is a condition combining lytic lesions of the bones, usually the terminal phalanges, Raynaud's syndrome and sclerodermatous skin changes. There is a 3 to 5% incidence of this condition in men who clean polyvinyl chloride reaction chambers (Walker, 1981). Cold hands and feet were reported in a group of 37 workers studied (Barlow & Sullivan, 1982). Arthralgia as well as myalgia were reported in this same group of 37 workers (Barlow & Sullivan, 1982).

II. CARCINOGENICITY/TERATOGENICITY

2.0 CARCINOGENICITY

Vinyl chloride is classified by the EPA as a group A carcinogen and by International Agency for Research on Cancer (IARC) as a Group 1 carcinogen. Both of these designations mean that vinyl chloride is a confirmed human carcinogen.

HUMAN STUDIES

Angiosarcoma of the liver has been seen in men who clean polyvinyl chloride reactors (Walker, 1981) as has hemangiosarcoma and hepatocellular carcinoma. Vinyl chloride is considered capable of causing cancer directly, that is without a co-carcinogen, and has a latency period of 5 to 20 years (Dietz et al, 1985). Symptoms at clinical onset of angiosarcoma are nonspecific and include fatigue, anorexia and weight loss, nausea, abdominal pain, vomiting, indigestion, diarrhea and chest pain. There is no specific

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diagnostic test other than liver biopsy, which may be dangerous in advanced stages of the disease (CCOHS, 1988; ATSDR, 1989).

The total number of expected occupationally-related cases of angiosarcoma range from approximately 250 to 1500, depending on which predictive model is used (Forman et al, 1985). An update on a mortality study of persons exposed to vinyl chloride monomer at Dow Chemical Company has shown fewer than expected deaths from total cancers (Dahar et al, 1988). This result is consistent with a model predicting a decline in cancer deaths from vinyl chloride monomer as a function of time.

A mortality study on British vinyl chloride monomer workers has found excess deaths only from liver cancer (Jones et al, 1988).

A study of Russian workers exposed to vinyl chloride monomer found excess deaths from leukemias and lymphomas, especially in women, but not angiosarcomas. The reason for this difference in distribution of cancers in contrast with western studies is not readily apparent. It may reflect a sex difference, because the cohort for this study were approximately one-third women (Smulevich et al, 1988).

A study of 5,958 Chinese vinyl chloride monomer workers, however, has found no excess deaths from all malignancies and no cases of angiosarcoma. Hepatomegaly and splenomegaly were increased, suggestive that the latency was too short to allow for full expression of any angiosarcomas (Wu, 1988).

In addition to a higher than normal risk of cancer in polyvinyl chloride plant workers in some studies, there are some preliminary data that suggest that living near (less than 2 miles) a polyvinyl chloride plant may increase the risk of neoplasms. More study is needed in this area before definite conclusions may be drawn (Wagoner, 1983).

A recent review has concluded that the current overall risk to the general public from environmental contamination by vinyl chloride monomer is negligible (Doll, 1988).

A case of hemangiosarcoma of the liver and spleen with metastases in the periportal and epipancreatic lymph node in a vinyl chloride worker has been described (Michot et al, 1987). This tumor was first diagnosed more than 30 years after discontinuance of chronic occupational vinyl chloride exposure. The presenting complaints were weight loss, nausea, anorexia, aching in the shoulders, and spontaneous bleeding. The patient was found to have microangiopathic hemolysis, anemia, thrombocytopenia, hypoprothrombinemia, hypoproteinemia, and enlargement of the spleen and liver. The patient died two days after the onset of the microangiopathic hemolysis.

One report associated exposure to vinyl chloride monomer with an unspecified cancer of the blood (RTECS, 1989), but this has not been confirmed in other studies.

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Some reports have linked brain cancer with occupational exposure to vinyl chloride monomer. In one study, 9 out of 10 cases of brain cancer had a histologic diagnosis of glioblastoma multiform, a rather unusual cell type (HSDB, 1989). Another study of 10,173 men, malignancies of the brain and other parts of the nervous system were the only types of cancer in significant excess (HSDB, 1989).

Nine retrospective mortality studies consistently found elevated risk for brain cancer as well as liver cancer (HSDB, 1989).

An updated mortality study of workers exposed to vinyl chloride monomer and/or polyvinyl chloride found excesses of liver, lung and brain cancer; however, only the liver cancer was related to cumulative dose for vinyl chloride monomer (Wu et al, 1989).

An ongoing study of all the vinyl chloride monomer workers in Italy has reported excess deaths from cancers of the esophagus, liver, and respiratory system for 3 of the 9 plants (Belli et al, 1987).

A prospective cohort study in France of 1,100 vinyl chloride monomer workers and matched controls has found no excess deaths from cancer in the exposed group, but there was increased incidence of angiosarcoma and lung cancers (Laplanche et al, 1987).

Excess deaths from digestive cancers were found in one study of 451 occupationally exposed persons (HSDB, 1989).

An ongoing study of Norwegian workers exposed to vinyl chloride monomer and polyvinyl chloride has found a significant increase in incidence of malignant melanoma (6 cases vs. 1.1 expected) (Heldaas et al, 1987).

ANIMAL STUDIES

Increased liver and kidney tumors were seen in rats exposed to 3,463 mg/kg vinyl chloride monomer by the oral route for 52 weeks. Tumors of the skin and appendages were seen in rats inhaling 1 to 5 ppm, 4 hours daily for 52 weeks. Transplacental endocrine tumors were found when pregnant rats were exposed to 10,000 ppm for 4 hours.

Vinyl chloride was an equivocal tumor agent in rats for skin/appendage and gastrointestinal tumors when given i.p. at 21 mg/kg for 65 weeks or s.c. at 21 mg/kg for 67 weeks. It was carcinogenic in mice at 50 ppm for 30 or 43 weeks for vascular and/or skin/appendage tumors.

Vinyl chloride was carcinogenic in hamsters exposed to air levels of 50 ppm, 4 hours daily for 30 weeks, resulting in lymphomas and skin/appendage tumors. It was also carcinogenic in rats. Air exposure to 50 ppm for 7 hours per day over 26 weeks produced vascular and

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skin/appendage tumors with continued exposure at this dose producing tumors of the respiratory system at 47 weeks.

Vinyl chloride was carcinogenic in rats by the oral route. Doses of 34 mg/kg over 3 years caused an increase in the incidence of angiosarcoma and kidney tumors. In rats, inhalation of 50 ppm, 6 hours per day for 4 weeks produced tumors of the respiratory system and skin/appendages. Exposure to 50 ppm, 6 hours per day over 30 weeks resulted in an increase in the incidence of liver and skin/appendages.

2.1 PREGNANCY

Increased risk for fetal loss was reported among wives of vinyl chloride monomer workers (Infante et al, 1976), but this study has been criticized because its methods of data collection and reporting were indirect (Paddle, 1976).

An excess of birth defects including nervous system defects, deformities of the genital and upper alimentary tracts, and clubfoot were reported in a group of still- and live-born children in three Ohio towns in proximity to vinyl chloride plants (HSDB, 1989).

A recent review of the literature on effects of paternal exposure to vinyl chloride monomer on the unborn has concluded that the data are inconclusive (Uzych, 1988).

Decreased female fertility was seen in rats exposed to 250 ppm 55 days prior to mating (RTECS, 1989).

2.2 TERATOGENICITY

Although there has been some concern that vinyl chloride monomer may cause birth defects, several studies could not substantiate such an association (within their sample sizes) (Therianlt et al, 1983; Barlow & Sullivan, 1982).

Rats exposed to various levels during pregnancy demonstrated via measurements of fetal blood levels and amniotic fluid considerable transfer of vinyl chloride monomer to the fetus. Studies concerning actual teratogenicity have been contradictory (Barlow & Sullivan, 1982). Animal studies also suggest decreased male fertility.

Vinyl chloride monomer was fetotoxic in rats exposed to 500 ppm in the air and caused increased post-implantation mortality in fetal rats at 1500 ppm. Musculoskeletal abnormalities were seen at 500 ppm in mice and rats, and urogenital abnormalities at 2500 ppm (RTECS, 1989).

Increased pre-implantation mortality was seen in embryonic mice where the males had been exposed to 30,000 ppm in the air 5 days prior to mating (RTECS, 1989).

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2.3 CHROMOSOMAL ABERRATIONS

Frequencies of chromosome aberrations associated with vinyl chloride monomer exposure have tended to return to normal within a few months after exposure ceases (Hansteen et al, 1978; Anderson et al, 1980). Positive sister chromatid exchanges in peripheral lymphocytes of persons occupationally exposed have been observed (HSDB, 1989; DPIMR, 1989). Increased frequencies of chromosome aberrations have not been seen when persons were exposed to air levels 5 to 12 ppm in the workplace (Picciano et al, 1977; Rossner et al, 1980).

Vinyl chloride caused sex chromosome loss and nondisjunction in *Drosophila melanogaster* (RTECS, 1989), gene conversion and mitotic recombination in *Saccharomyces cerevisiae* (RTECS, 1989), and chromosomal aberrations in human HeLa cells. It was positive in the micronucleus test in mice after in vivo exposure (RTECS, 1989) and it caused sister chromatid exchanges in hamsters (RTECS, 1989). Vinyl chloride was positive for oncogenic transformation in rat cells after in vivo exposure at 2000 ppm in the air for 14 weeks (RTECS, 1989).

2.4 GENOTOXICITY

Vinyl chloride was positive for DNA damage in *Escherichia coli* (RTECS, 1989), for unscheduled DNA synthesis in rat liver cells and for inhibition of DNA synthesis in rats exposed to the gas in vivo (RTECS, 1989).

2.5 MUTAGENICITY

Vinyl chloride was positive in mutagenicity tests in *Salmonella typhimurium*, *Escherichia coli*, *Saccharomyces pombe*, and rat liver cells (RTECS, 1989). It was positive in the host mediated assay using *Saccharomyces cerevisiae* in rats, *Saccharomyces pombe* in mice, *Saccharomyces cerevisiae* in mice, and in the following mammalian systems: hamster lung cells, hamster ovary cells (RTECS, 1989).

Positive for sex-linked recessive lethals in *Drosophila melanogaster* (DPIMR, 1989). Negative in mammalian spot test using mice exposed to 4,600 ppm in the air (DPIMR, 1989). (Note: this test is considered relatively insensitive.)

III. LABORATORY STUDIES

3.0 SERUM/BLOOD

Standard liver enzymes and liver function tests, CBC with peripheral smear, and serum creatinine are indicated to confirm liver involvement but are of little value in detecting early

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stages of liver injury or associating exposure to low levels of vinyl chloride monomer (ATSDR, 1989; Sugita et al, 1986). Fasting serum bile acids and indocyanine green clearance rate may be sensitive monitors for latent chemical liver injury (ATSDR, 1989).

3.1 URINE

Thiodiglycolic acid in the urine is indicative of recent exposure (ATSDR, 1989). Values found for persons occupationally exposed to 0.14 to 7 ppm ranged from 0.3 to 4.0 mg/L (HSDB, 1989). Clinical tests on urine which may be indicated include coproporphyrin and total urinary porphyrin, bilirubin, BUN and creatinine, and urinalysis (ATSDR, 1989).

Porphyrinuria, especially secondary co-proporphyrinuria with subclinical chronic hepatic porphyria, has been consistently found in the presence of hepatic lesions induced by vinyl chloride monomer (HSDB, 1989).

3.2 OTHER

Periodic medical surveillance should include liver function tests (ATSDR, 1989). Exclude ethanol, carbon tetrachloride, iron overload, vitamin A overdose, and viral infection as possible causes of liver abnormalities. Consider biliary obstruction and metastatic colorectal cancer in differential diagnosis of liver injury (ATSDR, 1989). Liver biopsy may be indicated for confirming specific histopathological changes if liver function tests are abnormal, but may lead to serious bleeding complications in advanced disease (ATSDR, 1989). Wide-field capillary microscopy of the hands may be a useful method to detect early stages of vinyl chloride disease (HSDB, 1989).

IV. TREATMENT BY EXPOSURE ROUTE

4.0 INHALATION EXPOSURE

4.0.1 DECONTAMINATION

Move patient to fresh air. Monitor patient for respiratory distress. Emergency airway support and 100 percent humidified supplemental oxygen with assisted ventilation may be needed. If a cough or difficulty in breathing develops, evaluate for respiratory tract irritation, bronchitis, pneumonitis.

4.0.2 TREATMENT

Vinyl chloride monomer and polyvinyl chloride dust may cause various respiratory abnormalities, and possibly respiratory cancers. Workers exposed to dusts should have

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periodic chest x-rays. Exposures to vinyl chloride monomer vapors may cause hepatic cancers, including angiosarcoma. There is no specific test to detect vinyl chloride monomer toxicity, and authors disagree concerning the usefulness of normal liver enzyme profile testing.

4.1 DERMAL EXPOSURE

4.1.1 DECONTAMINATION

Wash exposed area extremely thoroughly with soap and water. A physician may need to examine the area if irritation or pain persists after washing.

4.1.2 TREATMENT

Scleroderma has been seen after chronic exposure to these agents. Treatment should be symptomatic.

4.2 EYE EXPOSURE

4.2.1 DECONTAMINATION

Exposed eyes should be irrigated with copious amounts of room temperature water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist after 15 minutes of irrigation, an ophthalmologic examination should be performed.

4.2.2 TREATMENT

There have been very few cases of eye toxicity from vinyl chloride. Corneal damage, healing within 48 hours occurred in only one case report.

5.0 CASE REPORTS

Two fatal cases of acute exposure to vinyl chloride monomer occurred in Ontario in 1958 and 1959. Exposures were in excess of 100,000 ppm, and deaths were from respiratory failure (CCOHS, 1988).

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