ATSDR

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Case Studies in Environmental Medicine

15

Cyanide Toxicity

Environmental ALERT . . .

- ☑ ***Cyanide is one of the most rapidly acting poisons.***
- ☑ ***Cyanide poisoning is a hazard in many enclosed-space fires, and its occurrence in smoke-inhalation victims may be underestimated.***
- ☑ ***Acute cyanide exposure results primarily in CNS, cardiovascular, and respiratory effects; thyroid function abnormalities also have been noted in persons chronically exposed.***

This monograph is one in a series of self-instructional publications designed to increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients. *See page 21 for more information about continuing medical education credits and continuing education units.*

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How to use this issue...

This issue begins with a composite case study that describes a realistic encounter with a patient. This description is followed by a pretest. The case study is further developed through Challenge questions at the end of each section. To fully benefit from this monograph, readers are urged to answer each question when it is presented. (Answers to the Pretest and Challenge questions are found on pages 18-19.) The monograph ends with a posttest, which can be submitted to the Agency for Toxic Substances and Disease Registry (ATSDR) for continuing medical education (CME) credit or continuing education units (CEU). See page 21 for further instructions on how to receive these credits.

The objectives of this monograph on cyanide are to help you:

- **Explain why cyanide is an acute and chronic health hazard**
- **Describe the known factors contributing to cyanide toxicity**
- **Identify potential environmental or occupational sources of exposure to cyanide**
- **Identify evaluation and treatment protocols for persons exposed to cyanide**
- **List sources of information on cyanide**

Contents

This issue is prepared with the assistance of those who share a common concern for physician education, public health, and the environment, including the following organizations: American Academy of Family Physicians (AAFP), American Academy of Pediatrics (AAP), American College of Emergency Physicians (ACEP), American College of Occupational and Environmental Medicine (ACOEM), American Medical Association (AMA), Association of State and Territorial Health Officials (ASTHO), and the Society of Teachers of Family Medicine (STFM). Final responsibility for the contents and views expressed in this monograph resides with ATSDR.

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Case Study

Hypotension and coma in a 5-year-old victim of smoke inhalation

You are alerted by paramedics who are en route to the emergency department. They will arrive within 10 minutes with two apparent smoke-inhalation victims: a young woman, approximate age 35, and her son, approximate age 5. Upon arrival at the scene, firefighters found both victims unconscious near the doorway; the entire house was smoke-filled. The fire, which was confined to the child's bedroom and two adjacent rooms, was started by a toy that the child poked into an electric space heater. It is probable that the woman was in another part of the house when the fire began and attempted to rescue the child but was overcome by the thick black smoke. Both victims are unconscious. The paramedics report no evidence of trauma or burns in either victim. The mother has nonpurposeful movements, but the child is flaccid and unresponsive to painful stimuli. Soot is present in the child's nose and throat.

En route to the hospital, IVs were started. The woman's vital signs include BP 70/50 mm Hg, pulse 120/min, respiration rate 30/min. She is being administered 100% oxygen via face mask. The child's vital signs include BP 50/20 mm Hg, pulse 50/min, respiration rate 0/min. He is intubated and mechanically ventilated, and is being administered supplemental oxygen.

Upon arrival at the hospital, the woman is improved but is lethargic and disoriented; the child is still unresponsive even to deep pain. Both victims have adequate pO_2 levels. The mother's initial carboxyhemoglobin level is 25%; the child's carboxyhemoglobin level is 40%. The child remains bradycardic and hypotensive.

The following day, having heard that cyanide may have played a role in the condition of these smoke-inhalation victims, a neighboring couple, who have recently learned that for 2 years they had been drinking well water containing 212 parts per billion (ppb) cyanide, are concerned that they may experience adverse health effects. They ask to be evaluated.

(a) What are the possible causes of coma in the fire victims?

(b) What laboratory tests would help to confirm the diagnosis?

(c) What treatment should be initiated immediately?

(d) What are the possible long-term sequelae for the fire victims and the neighbors who drank cyanide-containing well water?

Answers can be found in Challenge answers (6) through (13) on pages 18-19.

Exposure Pathways

- **Many fruits and vegetables contain cyanide-generating substances.**
- **Cyanide contamination in air and water arises primarily from industrial pollution and vehicle exhaust.**
- **Cigarette smoke contains cyanide.**
- **Cyanide poisoning is an inhalation hazard in many enclosed-space fires.**

Cyanide is one of the most rapidly acting poisons known and accounts for many suicidal and homicidal deaths. Cyanide can exist in many forms. The most common are hydrogen cyanide (HCN) and cyanide salts (potassium cyanide, sodium cyanide, calcium cyanide), which can combine with acid to release HCN. A number of aliphatic nitrile compounds (i.e., acrylonitrile, acetonitrile, proprionitrile) and aliphatic thiocyanates can release cyanide by hepatic metabolism after absorption, resulting in delayed-onset cyanide poisoning. Children have developed symptoms of cyanide poisoning hours after drinking acetonitrile-based artificial nail remover.

Cyanide salts are generally colorless solids, whereas HCN, also known as prussic acid, is a colorless gas at room temperature. Because of its rapid action, HCN is employed in gas chamber executions in the United States and other countries. A cyanide salt was used in the Jonestown massacre, and potassium cyanide was responsible for the deaths of seven persons in the Chicago area who consumed intentionally tainted capsules of an over-the-counter pain reliever.

Cyanide compounds have a faint, bitter almond odor, detectable at a threshold of 0.2 to 5.0 parts per million (ppm). (The current permissible workplace short-term exposure limit [STEL] for hydrogen cyanide is 10 ppm.) The ability to smell cyanide is a genetically determined trait, which is absent in 20% to 40% of the population.

Cyanogenic glycosides, which occur naturally in a number of plants, release HCN after ingestion. In the United States, cyanide intake through food consumption is normally low since foods high in cyanogenic substances are not a major part of the American diet. However, eating large amounts of seeds, pits, and stone fruits of certain plants (or blending them in "milkshakes") reportedly has caused illness, especially in children, and even death. As many as 1,000 plants contain cyanogenic glycosides. The more common include the following:

- apple (seeds)
- bamboo (sprouts)
- cassava (beans and roots)
- Christmas berry
- crab apple (seeds)
- cycad nut
- elderberry (leaves and shoots)
- hydrangea (leaves and buds)
- lima beans (black bean grown in tropical countries)
- pear (seeds)
- *Prunus* species (leaves, bark, seeds)
  - apricot
  - bitter almond
  - cherry laurel
  - chokecherry
  - mountain mahogany
  - peach
  - pin cherry
  - plum
  - western chokeberry
  - wild black cherry

Adapted from Kingsbury JM. *Poisonous Plants of the United States and Canada.* Englewood Cliffs, NJ: Prentice-Hall, 1964, p 26. Used by permission of Prentice-Hall.

Laetrile, a compound used for cancer treatment by some nontraditional practitioners, contains amygdalin, which releases cyanide when administered orally. Laetrile has been sanctioned in 22 states, despite lack of Federal Drug Administration (FDA) approval. No evidence of laetrile's efficacy exists, and several cases of serious and fatal cyanide poisonings have been reported. Sodium nitroprusside, $NaFe(CN)_5NO$, is an intravenous medication used in acute hypertensive crises. Its cyanide metabolite can reach toxic levels if the recommended dosage is exceeded, if infusion is prolonged or rapid, or if renal failure occurs. Phencyclidine (PCP), an illicit street drug, may contain cyanide if improperly manufactured.

For the general population, the single largest source of airborne cyanide exposure is vehicle exhaust. Vehicle emission rates can be reduced from 14 milligrams (mg) cyanide per mile to about 1 mg per mile by catalytic converters operating optimally. Other atmospheric sources include emissions from chemical processing industries, iron and steel mills, metallurgical industries, metal plating and finishing industries, petroleum refineries, municipal waste incinerators, and cigarette smoke.

Water sources can be contaminated with cyanide by industrial effluents, migration of cyanide from landfills, and to a lesser extent, by runoff from cyanide-containing salts used on roads. The largest sources of cyanide in water are discharges from organic chemical industries, iron and steel plants, and wastewater treatment works. A 1978 U.S. Environmental Protection Agency (EPA) survey of interstate drinking water supplies showed that about 7% of the samples had cyanide concentrations greater than 10 parts per billion (ppb). (The anticipated drinking water standard to be proposed by EPA in 1990 is 200 ppb.) Cyanide has been detected in some surface waters at concentrations above the level safe for aquatic life.

Virtually any substance containing both carbon and nitrogen can release cyanide when burned under certain conditions. HCN is released during pyrolysis of synthetic polymers containing nitrocellulose, acrylonitrile, or urea formaldehyde. Many common textiles, foam, and plastic materials in the home may be sources of HCN in a fire. Some natural products such as silk and wool can also release cyanide when burned.

Challenge

(1) Explain how the smoke-inhalation victims described in the case study could have been poisoned by cyanide.

(2) What additional information would you gather from the neighboring couple regarding their exposure to cyanide?

Who's at Risk

- **Workers have the greatest likelihood of exposure to high concentrations of cyanide.**
- **One lighted cigarette can produce 20 to 450 µg of cyanide, increasing the risk to smokers.**
- **The incidence of cyanide poisoning may be underestimated in smoke-inhalation victims.**

A 1981-1983 National Institute for Occupational Safety and Health (NIOSH) survey estimated that 143,720 workers in a wide variety of occupations were potentially exposed to cyanide compounds. The workers include, but are not limited to, the following:

- blacksmiths
- chemical laboratory workers
- electroplaters
- fumigant applicators
- manufacturers and users of plastics and paint (acrylates, methacrylates, nitriles)
- metallurgists
- metal cleaners
- photoengravers
- reclaimers of silver from photographic materials
- steel manufacturers
- tanners

Firefighters may be exposed to HCN generated during combustion of certain types of plastics, foams, and textiles. Effects due to occupational exposures are usually the result of inhalation.

Each pack of cigarettes smoked releases 250 to 10,000 micrograms (µg) of cyanide, much of which the smoker may inhale. Smokers generally have higher blood cyanide levels than nonsmokers and are at increased risk of cyanide's nervous system effects, particularly tobacco amblyopia and retrobulbar optic neuritis.

Significant blood cyanide levels have been reported in many fire-related smoke-inhalation victims. In one study of 52 fire fatalities, some victims had very high cyanide levels with relatively low carbon monoxide levels. Data suggest that the risk of cyanide intoxication in enclosed-space smoke inhalation injury is probably underestimated. HCN in the air can cause weakness and loss of muscle coordination, making escape from the area of a fire more difficult.

Challenge

(3) Both victims described in the case study were overcome in the same fire. Would you expect them to have similar blood cyanide levels? Explain.

(4) What are other possible causes of an elevated blood cyanide level?

Biologic Fate

Cyanide is absorbed through the lungs, GI tract, and skin. Symptoms can occur within seconds of HCN inhalation, within minutes after ingestion of cyanide salts, and onset may be delayed up to 12 hours after ingestion of cyanogenic glycosides, nitriles, or thiocyanates. Absorption time after ingestion depends on gut pH and solubility of the cyanide-containing compound. Cyanide is readily absorbed via the mucous membranes and eyes. Clinical cases of cyanide poisoning after dermal exposure are rare and most often have involved burns with molten cyanide salts or immersion in cyanide solutions.

Once cyanide is absorbed, it is rapidly distributed by the blood throughout the body. Cyanide exerts toxic effects by combining with the ferric (+3) iron in cytochrome oxidase, which inhibits cellular oxygen utilization. Blockage of the cytochrome oxidase system

- ❑ **Cyanide is absorbed by all routes.**
- ❑ **Cyanide causes cellular asphyxiation by inhibiting the cytochrome oxidase system.**

causes anaerobic metabolism with resultant lactate production and severe metabolic acidosis. Cyanide also inhibits other enzymes and can combine with certain metabolic intermediates.

Eighty percent of absorbed cyanide is detoxified in the liver by the mitochondrial enzyme rhodanese, which catalyzes the transfer of sulfur from a sulfate donor to cyanide, forming less toxic thiocyanate. Thiocyanate is readily excreted in urine. Other detoxification pathways exist, including reaction with hydroxocobalamin (vitamin B_{12a}) to form cyanocobalamin. A small amount of cyanide is eliminated as CO_2 in expired air, along with small amounts of HCN.

A number of compounds have been found to act synergistically with cyanide, producing toxic effects. Smoke-inhalation victims have experienced additive or synergistic effects from carbon monoxide and cyanide, and only recently has attention been focused on the potential for combined poisoning in victims of enclosed-space fires.

Physiologic Effects

- **The cardiovascular, respiratory, central nervous, and endocrine systems may be adversely affected in cyanide poisoning.**

Cyanide adversely affects the cardiovascular, respiratory, central nervous, and endocrine systems. It is unlikely that cyanides are carcinogenic. Epidemiologic studies indicate that cyanide may be teratogenic in humans, but data are scarce. Reproductive effects of cyanide in humans have not been studied.

There is great variability among "lethal doses" reported in the literature, probably due to differences in supportive care and therapy rendered. The potential lethal oral adult dose of cyanide salts in the absence of medical care is 200 to 300 mg, although persons ingesting 1 to 3 grams (g) of cyanide salts have survived. Inhaling 600 to 700 ppm hydrogen cyanide for 5 minutes or approximately 200 ppm for 30 minutes may be fatal. Survival in any specific case often depends upon the rapidity and scope of treatment.

Acute Exposure

- **The effects of acute cyanide exposure are dominated by CNS disturbances.**
- **Because of its high metabolic demands, the brain is particularly susceptible to cyanide poisoning.**

An acute cyanide exposure affects primarily the central nervous system, initially producing stimulation, which may be followed quickly by depression. Stimulation of peripheral chemoreceptors produces increased respiration, while stimulation of the carotid body receptors slows the heart rate. These early changes are often transient and may be followed by hypoventilation progressing to apnea and myocardial depression. The result is hypotension and shock, which are rapidly fatal if untreated. Because of the brain's susceptibility to cyanide, electrical activity may cease while the heart is still beating.

Predominance of anaerobic metabolism within a cyanide-poisoned cell induces a decrease in the ATP/ADP ratio and thus alters energy-dependent processes such as calcium homeostasis. Disruption in calcium regulation with resultant changes in neurotransmitter releases can alter the electrical activity in the brain and may be an important factor in the manifestation of cyanide-induced neurotoxic effects such as tremors and convulsions. Delayed onset Parkinson-like syndromes have been described after severe cyanide poisoning as well as after carbon monoxide poisoning, implying that the basal ganglia are sensitive to the neurotoxic effects of both agents.

Chronic Exposure

Central Nervous System

An epidemic of spastic paraparesis in Mozambique was attributed to consumption of the cyanogenic vegetable, cassava. Some members of the affected population developed neurologic abnormalities such as demyelination of peripheral nerves with decreased conduction velocity, optic neuropathy, and deafness. A variety of Parkinson-like signs were also present. However, diets deficient in vitamin B_{12} also can result in nerve cell destruction, even when large amounts of cyanogenic foods are not consumed.

- **Chronic degenerative neuropathy has been associated with cassava consumption in Mozambique.**

Inhalation of cyanide from tobacco by heavy smokers with poor diets has been associated with tobacco amblyopia, retrobulbar neuritis, and optic atrophy, characterized by a loss of visual acuity. Leber's hereditary optic atrophy is associated with a defective rhodanese-catalyzed metabolism of cyanide to thiocyanate. Quantitative data associating neurologic effects with long-term occupational cyanide exposures are limited.

Cardiovascular and Respiratory Effects

Although cyanide appears to affect vascular smooth muscle directly, effects on the respiratory and cardiovascular systems may be at least partially secondary to central nervous system effects. In acute poisoning, depression of the CNS respiratory centers causes hypoventilation leading to apnea. Direct myocardial depression, as well as hypoxemia from hypoventilation, leads to decreased cardiac output and hypotension.

- **Respiratory and cardiovascular effects of cyanide poisoning may be secondary to the CNS effects.**

Electrocardiographic abnormalities, palpitations, and chest pain have been noted in chronically exposed workers. Electroplaters who had been exposed to airborne cyanide concentrations of approximately 6 to 10 ppm for 5 to 15 years also complained of dyspnea on exertion.

Endocrine Effects

- **Thyrotoxicity has been linked with inhalation and ingestion of cyanide.**

One study of electroplaters revealed that over 50% had enlarged thyroid glands. Other studies of cyanide-exposed workers have noted mild subclinical abnormalities of thyroid function. Populations in which the cassava is a staple food show a strong correlation between cassava consumption and endemic goiter and cretinism. Thiocyanate, the detoxification product of cyanide, and dietary deficiencies such as low intake of iodine may play a role in producing these thyroid effects.

Developmental Effects

- **Data regarding cyanide's teratogenic effects on the human fetus are lacking.**

Cyanide and various cyanogenic compounds (laetrile, cassava powder, acetonitrile, proprionitrile, acrylonitrile) have produced a variety of teratogenic effects in animals, most of which can be prevented when the dams are administered a cyanide antidote, sodium thiosulfate. In humans, low birth weights have been noted in the children of women chronically exposed to cyanide.

Challenge

(5) What is the prognosis for each of the smoke-inhalation victims described in the case study?

Clinical Evaluation

History and Physical Examination

Pertinent history may include occupation and hobbies, medications, diet, smoking habits, and drinking water source. Physical examination of chronically exposed patients should include particular attention to neuropsychiatric and ophthalmologic examinations, and cardiovascular system and thyroid functioning.

Because cyanide can kill quickly, acute poisoning requires rapid intervention. Unfortunately, positive diagnosis is often difficult, especially when the history cannot be obtained. Knowledge of the patient's occupation, mental status before poisoning, probability of suicidal intent, or location at onset of symptoms can be vital in making a diagnosis.

Persistent acidosis or hypotension in a smoke-inhalation victim should raise the suspicion of concomitant cyanide poisoning. In an unresponsive victim, trauma should be ruled out by a thorough examination, which should be repeated frequently. When clinically indicated, confirmatory tests, such as peritoneal lavage for intraperitoneal hemorrhage and a CAT scan for intracranial injury, should be performed.

Signs and Symptoms

Acute Exposure

- **CNS and cardiovascular signs predominate in persons acutely exposed.**
- **Posthypoxic encephalopathy, Parkinson-like syndromes and delayed posthypoxic myocardial lesions are sequelae of severe cyanide poisoning.**

Signs and symptoms of acute cyanide toxicity reflect cellular hypoxia and initially may be nonspecific, generalized, and nondiagnostic. With acute inhalation of HCN gas, death may occur within seconds.

Central nervous system symptoms include faintness, flushing, anxiety, excitement, perspiration, vertigo, headache, drowsiness, prostration, opisthotonos and trismus, hyperthermia (with cyanogenic glycosides), tremors, convulsions, stupor, paralysis, coma, and death. Retinal veins and arteries may appear similarly red in color because cyanide blocks cellular utilization of oxygen, elevating venous Po_2.

Respiratory symptoms initially include tachypnea and dyspnea, progressing rapidly to respiratory depression, with hypoventilation and apnea. Noncardiogenic pulmonary edema may be noted after cyanide inhalation or ingestion. Because of increased venous Po_2 and percent O_2 saturation, cyanosis may be absent despite respiratory depression. Severe metabolic acidosis results from anaerobic metabolism with increased lactic acid production.

Cardiovascular signs include initial transient hypertension with reflex bradycardia and sinus dysrhythmia, followed by tachycardia with hypotension and cardiovascular collapse. Electrocardiogram changes include an elevated or depressed ST segment or a shortened ST segment with fusion of the T wave into the QRS complex. Varying degrees of atrioventricular block, erratic supraventricular rhythms, ventricular fibrillation, and asystole may also be seen.

Sequelae of severe acute exposure may include neuropsychiatric manifestations similar to those seen with posthypoxic or post-carbon monoxide encephalopathy, Parkinson-like syndromes, and cardiovascular signs of delayed posthypoxic myocardial lesions.

In smoke-inhalation victims, the hypoxia resulting from carbon monoxide is initially indistinguishable from that due to cyanide, which makes diagnosis of concomitant cyanide poisoning difficult. Characteristics of patients with significant cyanide poisoning secondary to smoke inhalation may include the following:

Persistent hypotension	Respiratory depression/apnea
Coma	Noncardiogenic pulmonary edema
Seizures	Peak carboxyhemoglobin levels (>30%)
Cardiac dysrhythmias	Persistent metabolic acidosis (pH <7.25)
Cardiac ischemia	Increased venous % O_2 saturation and PO_2; absence of cyanosis

Chronic Exposure

- **Long-term effects of chronic cyanide exposure include cardiovascular, respiratory, and thyroid function abnormalities.**

Symptoms reported after chronic exposure in occupational settings include breathing difficulty, headache, dizziness, nausea or vomiting, a bitter or almond taste, hoarseness, conjunctivitis, palpitations, chest pains, weight loss, weakness, sleep disturbances, and altered mental status. Mild subclinical abnormalities in vitamin B_{12}, folate, thyroid stimulating hormone (TSH) levels, and thyroid function have been found in silver-reclaiming workers 7 months after cyanide exposure had ceased.

Laboratory Tests

Direct Biologic Indicators

- **Whole blood cyanide levels can be used to confirm diagnosis.**

Blood cyanide. Laboratory analysis of whole blood cyanide takes at least 4 to 6 hours, and therapeutic interventions usually must be made before levels are available. However, whole blood cyanide levels are useful in confirming and documenting the diagnosis. One of the most significant problems in measuring cyanide is its instability in collected samples. Consult the laboratory for proper techniques in specimen handling.

Peak whole blood cyanide levels lower than 0.2 micrograms per milliliter (µg/mL) usually do not cause symptoms, although poisoning has occurred at lower levels. Whole blood cyanide levels in smokers may reach 0.4 µg/mL without causing symptoms. At cyanide concentrations between 0.5 and 1.0 µg/mL, untreated patients may be conscious, flushed, and tachycardic. Stupor and agitation can appear with peak blood levels between 1.0 and 2.5 µg/mL. Cyanide

levels over 2.5 μg/mL are associated with coma and are potentially fatal without treatment. Typically, plasma cyanide levels are one-tenth the level of the corresponding whole blood specimen but are seldom measured.

Indirect Biologic Indicators

Plasma or serum thiocyanate. Cyanide in the body is biotransformed to thiocyanate. The relative proportion of thiocyanate to cyanide in body fluids is about 1000 to 1. Thiocyanate can be measured in serum or plasma, but interpretation of levels in a cyanide-poisoned patient is difficult. Little correlation has been found between simultaneously obtained whole blood cyanide and plasma thiocyanate levels. Normal plasma thiocyanate can range up to 10 μg/mL in nonsmokers and smokers alike. Lethal thiocyanate levels may range from 50 to 200 μg/mL. The value of thiocyanate levels in the diagnosis of chronic cyanide poisoning is unknown.

❑ **Plasma or serum thiocyanate levels are of limited value in assessing patients with acute cyanide poisoning.**

Other. Whenever smoke inhalation is the potential source of cyanide exposure, carboxyhemoglobin and methemoglobin levels should also be obtained. Both are measured in most hospital laboratories using a co-oximeter, and both may be elevated in smoke-inhalation victims. Either may be measured in heparinized venous blood, although an arterial blood gas specimen is commonly used and provides data on pulmonary function and acid-base status as well.

Pulse oximetry or ear oximetry are unreliable when carboxyhemoglobin or methemoglobin is present and cannot be used to accurately measure oxygen saturation.

Challenge

(6) In cyanide-poisoned patients, venous Po_2 and percent O_2 saturation may be higher than expected. What is the explanation for this?

(7) What are possible causes of the coma experienced by the victims described in the case study?

(8) What are the key diagnostic signs of cyanide poisoning in a smoke-inhalation victim?

(9) What laboratory tests would help confirm a diagnosis of cyanide toxicity?

Treatment and Management

- **Administration of 100% oxygen, followed by antidotes, is the best therapy for cyanide poisoning.**
- **Nitrite/thiosulfate is the only approved cyanide antidote in the United States.**

Acute Exposure

Treatment of cyanide poisoning consists of removal from exposure, administration of 100% oxygen, aggressive cardiorespiratory support, and administration of antidote. Inhalation of amyl nitrite has been recommended as a first-aid measure. Rescuers should not enter areas with potentially high airborne cyanide levels without self-contained breathing apparatus and adequate protective clothing, lest they become secondary victims. If contaminated by liquid or powder, the victim's clothes should be removed and the skin thoroughly cleansed with soap and copious amounts of water. If breathing is absent or labored, ventilatory assistance should be provided. Because of the rapid onset of cyanide toxicity, gut decontamination should follow antidote therapy unless both can be performed simultaneously. Ipecac-induced emesis is contraindicated; activated charcoal should be administered as soon as possible. Gastric lavage is of questionable value unless performed immediately after ingestion. Seizures and hypotension should be treated with anticonvulsants and vasopressors.

Several cyanide-poisoned patients have survived with only aggressive supportive care. Routine use of 100% oxygen is recommended even in the presence of normal Po_2 since oxygen acts synergistically with other antidotes. Metabolic acidosis (pH below 7.35) should be corrected with sodium bicarbonate solution. Close cardiac monitoring is essential; cardiac dysrhythmias often resolve without administration of other pharmacologic agents when the cyanide antidote kit is used. Monitoring for possible development of noncardiogenic pulmonary edema should be instituted.

Hyperbaric oxygen (HBO) is of theoretical benefit because it greatly increases the partial pressure of oxygen in cells. Anecdotal case reports and data from some animal studies support its use in cyanide poisoning. Whether HBO offers any added clinical advantage over 100% normobaric oxygen and the cyanide antidote kit is unknown. Smoke inhalation is frequently associated with chemical pneumonitis, skin burns, carbon monoxide poisoning, and cyanide poisoning; HBO has theoretical or documented benefit in the treatment of all four conditions.

The decision to administer antidotes in acute cyanide poisoning must often be made by clinical suspicion of exposure since pathognomonic signs may be lacking. The odor of cyanide (bitter almonds, "musty") on the victim's breath or in gastric contents is helpful, but its absence does not rule out cyanide poisoning. A hypotensive, bradycardic, acidotic, and acyanotic patient may have cyanide poisoning, especially if CNS, respiratory, and cardiovascular de-

pression subsequently develop. Rapid onset of coma and severe metabolic acidosis together are also indicative of acute cyanide exposure. Cyanide can be measured in body fluids to confirm a diagnosis, but not rapidly enough to guide emergency treatment.

For severely poisoned victims, antidote should be administered as quickly as possible. Only the 3-component Lilly Cyanide Kit is approved in the United States. (Detailed instructions are in the kit.) The kit includes amyl nitrite perles for inhalation, which can be placed under the victim's nose as a first-aid measure. This step can be bypassed if the IV is already established. The second step is slow IV administration of sodium nitrite with careful blood pressure monitoring, followed within minutes by the third step, IV administration of sodium thiosulfate. Nitrites are believed to induce methemoglobinemia, which detoxifies cyanide by forming cyanomethemoglobin. Thiosulfate serves as a sulfur donor in the rhodanese-catalyzed conversion of cyanide to less toxic thiocyanate.

Methemoglobin levels should be followed serially after the cyanide antidote kit is used. There is no established therapeutic methemoglobin level in the treatment of cyanide poisoning. The determinant of whether further doses of sodium nitrite may be required is the patient's clinical status. Administering further sodium nitrite to a patient who has recovered in order to maintain an arbitrary therapeutic methemoglobin level is both unnecessary and dangerous. One hour after administration of nitrite/thiosulfate, methemoglobin should not exceed 20% (peak level). At methemoglobin levels of 40%, symptoms of cyanosis are evident, and levels of 70% or greater can be fatal, especially in children. If excessive methemoglobinemia occurs, methylene blue treatment can be used. Since this treatment can cause deterioration by re-establishing high cytochrome ferro cyanide levels, it should be administered only by highly trained professionals experienced in managing cyanide poisoning. HBO and exchange transfusion are alternative therapies in severe cases.

Smoke-inhalation victims present a dilemma because administration of nitrite antidote can worsen the effects of carbon monoxide poisoning by forming methemoglobin, which further restricts the oxygen-carrying capacity of the blood. When carboxyhemoglobin levels are greater than 30%, administration of nitrites becomes dangerous. One option is administering the thiosulfate component of the antidote kit first and beginning HBO before giving the nitrite component.

The most promising treatment for smoke-inhalation victims is 100% oxygen and a combination of hydroxocobalamin (vitamin B_{12a}) and sodium thiosulfate, which has been used since the late 1960s in France to treat cyanide-poisoned patients. Hydroxocobalamin does not form methemoglobin nor does it cause hypotension. It is currently an investigational orphan drug undergoing clinical trials in the United States. Rare cases of urticaria reported with use of hydroxocobalamin may have been related to the vehicle in which the material was formerly prepared or to other drugs given concomitantly.

A variety of other agents are effective antidotes for cyanide poisoning, including 4-dimethylaminophenol (DMAP), used clinically in Germany, and dicobalt ethylenediaminetetraacetic acid (cobalt EDTA), used in Britain, France, and Australia. The results of animal studies have shown alpha-ketoglutaric acid, calcium channel blockers, stroma-free methemoglobin solution, and rhodanese to be efficacious. Although several of these cyanide antagonists show clinical promise, none has received FDA approval for routine use.

Chronic Exposure

- **The therapy for chronic cyanide poisoning is removal of the patient from the exposure source and treatment of end-organ damage.**

In cases of chronic poisoning, the patient must be separated from the source of cyanide and treated symptomatically. Nutritional deficiencies may exacerbate the chronic effects of cyanide and should be corrected.

(10) What treatment should be initiated immediately for smoke-inhalation victims?

(11) After administration of 100% oxygen to the patients described in the case study, the mother responds fully, but the child remains comatose and unresponsive to pain after 40 minutes. What additional action should you take?

(12) What are possible side effects of the cyanide antidote kit?

(13) What are the long-term sequelae in persons acutely or chronically poisoned by cyanide?

Standards and Regulations

A summary of standards and regulations is listed in Table 1.

Table 1. Standards and regulations for cyanide

Agency *	Focus	Level	Comments
ACGIH	Air -Workplace	4.7 ppm	Advisory; TWA† for cyanides, based on skin absorption
NIOSH	Air -Workplace	5 mg/m^3	Advisory; 10-minute ceiling limit
OSHA	Air -Workplace	10 ppm	Regulation; PEL§ for HCN
		5 mg/m^3	Regulation; PEL§ for cyanide salts
EPA	Air-Environment	N/A	Regulation: to be covered under the Clean Air Act
	Water-Environment	N/A	Regulation; drinking water standard to be proposed in 1990
		3.5 ppb	Advisory; 24-hour coverage to protect aquatic life
	Food	25 ppm	Regulation; residual HCN, when used as a postharvest fumigant in dried beans, peas, and nuts; 250 ppm in spices

* ACGIH = American Conference of Governmental Industrial Hygienists; EPA = Environmental Protection Agency; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration

† TWA (Time-Weighted Average) = time-weighted average concentration for a normal workday and 40-hour workweek to which nearly all workers may be repeatedly exposed.

§ PEL (Permissible Exposure Limit) = highest level in air, averaged over an 8-hour workday, to which a worker may be exposed.

Workplace

Air

- **The current OSHA permissible exposure limit for HCN is 10 ppm; for cyanide salts, it is 5 mg/m^3.**

The Occupational Safety and Health Administration (OSHA) current permissible exposure limit (PEL) for HCN is 10 ppm and for cyanide salts, 5 milligrams per cubic meter (mg/m^3). NIOSH recommends a 10-minute ceiling level of 5 mg/m^3 (4.7 ppm HCN). Air levels of 50 ppm HCN are considered immediately dangerous to life or health.

Environment

Air

- **EPA currently has no standard for cyanide emissions to ambient air.**

EPA anticipates cyanide in air will be regulated under the Clean Air Act proposed for 1990.

Drinking Water

- **EPA anticipates proposing a drinking water standard for cyanide in June 1990.**

EPA will propose a maximum contaminant level (MCL) for cyanide in drinking water in June 1990. This limit is anticipated to be 200 ppb.

Food

- **Foods are regulated when they have been treated with HCN, a postharvest fumigant.**

EPA tolerances for HCN in foods when used as a postharvest fumigant range from 25 ppm in dried beans, peas, and nuts, to 250 ppm in spices. Reference doses were extrapolated from studies of rats fed cyanide salts for 2 years at levels resulting in no observable effect.

Suggested Reading List

Reviews

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Clark CJ, Campbell D, Reid WH. Blood carboxyhaemoglobin and cyanide levels in fire survivors. Lancet 1981;1:1332-5.

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Hall AH, Kulig KW, Rumack BH. Suspected cyanide poisoning in smoke inhalation; complications of sodium nitrite therapy. J Toxicol Clin Exp 1989;9:3-9.

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Symington IS, Anderson RA, Thomson I, Oliver JS, Harland WA, Kerr JW. Cyanide exposure in fires. Lancet 1978;2:91-2.

Related Government Documents

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Environmental Protection Agency. Drinking water criteria document for cyanide. Washington DC: US Environmental Detection Agency, Criteria and Standards Health Effects Division, 1987. NTIS report no. PB/86/117793/LL.

Fiskel J, Cooper C, Eschenroeder A, Goyer M, Perwak J. Exposure and risk assessment for cyanide, 1981. Report no. EPA/440/4-85/008; NTIS report no. PB85-220572.

Answers to Pretest and Challenge Questions

Pretest questions are on page 1. Challenge questions begin on page 4.

(1) Many smoke-inhalation victims are poisoned by both carbon monoxide (elevated carboxyhemoglobin levels) and HCN. HCN is formed during pyrolysis of synthetic and natural materials containing nitrogen. The child placed a toy through the heater grill, it ignited, and the fire spread to carpets, drapes, furniture, wire insulation, etc. Under the right conditions, these items could produce HCN, as well as other noxious gases.

(2) The known source of cyanide for the neighbors is contaminated drinking water. Other possible sources include cyanogenic foods, vehicle exhaust, active or passive cigarette smoke, and the fire described in the case study. Information to gather would include other residences, diet, smoking history, occupation, hobbies, and other illnesses.

(3) The two victims would not necessarily have similar blood cyanide levels, even though they were victims of the same fire. The blood cyanide level is a function of the amount and composition of the smoke inhaled. If the mother was in another part of the house and remained unaware of the fire until it had spread, she probably would not have inhaled as much HCN as the child, who was near the fire source. The mother likely received exposure to a lower concentration of smoke and for a shorter duration than the child did.

(4) Causes of an elevated blood cyanide level might include occupational exposure, heavy cigarette smoking, ingestion of large quantities of cyanogenic plants, nitroprusside therapy, smoke inhalation, or a possible laboratory error. The physician should also consider a suicide or homicide attempt using cyanide salts.

(5) Assuming vital signs have been adequately maintained and the patients were well supported, the prognosis is generally good unless significant hypoxia, hypotension, or acidosis occurred before treatment and caused end-organ damage. Survival after 4 hours usually indicates recovery. A delayed Parkinsonian syndrome can occur after cyanide poisoning, as well as after carbon monoxide poisoning.

(6) The blockage of cellular respiration by cyanide prevents extraction and utilization of the oxygen carried in the arteries. As a result, the presence of unused oxygen will raise the oxygen partial pressure and percent O_2 saturation of the venous blood.

(7) Causes of coma may be grouped as toxicologic or nontoxicologic. Toxicologic causes include carbon monoxide poisoning, cyanide poisoning, or drug overdose. Drug overdose could have resulted in unconsciousness before the fire started. Hypoxemia resulting from any of the above causes may also contribute to continuing coma. Nontoxicologic causes include trauma, particularly to the head, occurring before the fire or to a victim trying to escape the fire.

(8) One of the initial diagnostic signs of cyanide poisoning in a smoke-inhalation victim is the absence of cyanosis despite respiratory depression. The persistence of acidosis and continued bradycardia and hypotension also suggest cyanide poisoning, and antidote should be administered.

(9) A whole blood cyanide level of greater than 1.0 μg/mL is diagnostic of significant cyanide exposure. The laboratory requires 4 to 6 hours to perform this test, however, so it is useful only for confirmation or documentation of the diagnosis and cannot be used to guide emergency management.

An elevated carboxyhemoglobin level (greater than 30%) in a smoke-inhalation victim should provoke suspicion of a concomitant cyanide poisoning. Persistent metabolic acidosis after hypotension has been alleviated, which is consistent with cyanide intoxication, can be determined from the pH or CO_2 content. An

elevated venous Po_2, resulting from the inability of cells to properly extract oxygen from the blood, is also a valuable clue. However, because respiratory depression or pulmonary damage are likely to occur in such patients, and because inspired oxygen concentrations may vary, a normal venous or mixed venous Po_2 level in a given setting may be unknown.

(10) For smoke-inhalation victims, 100% oxygen should be administered immediately and IV access established.

(11) The biologic half-life of carbon monoxide using 100% oxygen is 60 to 90 minutes. If hypotension, bradycardia, or acidosis persist, cyanide poisoning is likely, and antidote should be administered. Methemoglobin levels should be monitored carefully throughout nitrite therapy. Blood pressure also should be monitored and the nitrite infusion rate slowed if hypotension occurs.

(12) The two major side effects of sodium nitrite from the antidote kit are excessive methemoglobinemia and hypotension. Methemoglobinemia is due to the oxidation of ferrous (+2) iron in hemoglobin to ferric (+3) by nitrite; therefore, care must be taken to avoid excessive nitrite doses. Hypotension results from the vasodilating action of nitrite.

(13) Short-term sequelae of patients acutely poisoned by cyanide may include tremors and convulsions. Longer-term effects may include posthypoxic brain damage and myocardial lesions.

The long-term sequelae in persons chronically exposed to cyanide depend on exposure level and duration. In occupational settings, mild abnormalities in vitamin B_{12}, folate, TSH levels, and thyroid function have been reported. Enlarged thyroid, dyspnea on exertion, psychosis, encephalopathy, and myocardial lesions have also been noted but may be due to multiple episodes of subacute poisoning rather than true chronic exposure. In populations consuming large quantities of cassava, endemic goiter, neuropathies, and cretinism have been noted, although dietary deficiencies may have contributed to these effects. Retrobulbar optic neuritis in heavy smokers has also been linked to chronic, low-level cyanide exposure from cigarette smoke.

Whether the neighbors described in the case study who have had chronic cyanide exposure through drinking water will experience long-term effects is difficult to predict. The level of cyanide in their drinking water (212 ppb) is close to what the EPA will propose in 1990 (200 ppb). The amount of cyanide ingested through cassava consumption in the Mozambique population is unknown, and comparisons are not warranted. Persons chronically exposed to low levels of cyanide have not been adequately studied. However, if neurologic, ophthalmologic, and thyroid examinations are normal, reassurance should be given that no ill effects from consumption of the water are evident. The local or state health department could be consulted to obtain assistance with possible control measures to remove cyanide from the well water.

Sources of Information

More information on the adverse effects of cyanide and the treatment and management of cyanide-exposed persons can be obtained from ATSDR, your state and local health departments, and university medical centers. *Case Studies in Environmental Medicine: Cyanide Toxicity* is one of a series. For other publications in this series, please use the order form on the back cover. For clinical inquiries, contact ATSDR, Division of Health Education, Office of the Director, at (404) 639-6204.

Notes

Posttest and Credits

Continuing education credit is available to health professionals who use this monograph and complete the posttest. The criterion for awarding continuing medical education (CME) credits and continuing education units (CEU) is a posttest score of 70% or better.

The Centers for Disease Control and Prevention (CDC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education for physicians, and by the International Association for Continuing Education and Training (IACET) to sponsor continuing education units for other health professionals.

The Agency for Toxic Substances and Disease Registry, in joint sponsorship with CDC, is offering 1 hour of CME credit in Category 1 of the Physician's Recognition Award of the American Medical Association and 0.1 hour of CEU for other health professionals upon completion of this monograph.

In addition, the series *Case Studies in Environmental Medicine* has been reviewed and is acceptable for credit by the following organizations:

The **American Academy of Family Physicians (AAFP)**. This program has been reviewed and is acceptable for 1 prescribed hour by the American Academy of Family Physicians. (Term of Approval: beginning January 1992.) For specific information, please consult the AAFP Office of Continuing Medical Education.

The **American College of Emergency Physicians (ACEP)**. Approved by the American College of Emergency Physicians for one hour per issue of ACEP Category I credit.

The **American Osteopathic Association (AOA)**. AOA has approved this issue for 1 credit hour of Category 2-B credit.

The **American Association of Occupational Health Nurses (AAOHN)**. AAOHN has approved this program for 1.2 contact hours. Applicant will receive the assigned code number in the award letter.

The **American Board of Industrial Hygiene (ABIH)**. ABIH has approved this program for 0.5 certification maintenance (CM) point per 3 Case Studies. The CM approval number is 2817.

To receive continuing education credit (CME or CEUs), complete the Posttest on page 22 in the manner shown in the sample question below. **Circle all correct answers.**

Which of the following is known to precipitate migraine headaches?

(a) fatigue
(b) alcohol
c. grapefruit
(d) sunlight
e. sleep

After you have finished the Posttest, please transfer your answers to the answer sheet on the inside back cover and complete the evaluation on the lower half of that page. Fold, staple, and mail the back cover to Continuing Education Coordinator, Agency for Toxic Substances and Disease Registry, Division of Health Education, E33, 1600 Clifton Road, Atlanta, GA 30333. Your confidential test score will be returned with an indication of where the correct answers can be found in the text. Validation of earned CME credit and CEU will also be forwarded to participants, and their names, if requested, will be placed on the mailing list to receive other issues in the *Case Studies in Environmental Medicine* series.

POSTTEST: CYANIDE

Circle **all** correct answers and transfer your answers to page 23.

1. Potential sources of cyanide exposure include
   a. metallurgical industries
   b. fires
   c. auto exhaust
   d. cigarette smoke
   e. spot removers
2. Dietary deficiencies that may predispose a person to the effects of cyanide include
   a. iodine
   b. vitamin B_{12}
   c. vitamin D
   d. protein
   e. selenium
3. Which of the following statements about thiocyanate are true?
   a. it is less toxic than cyanide
   b. its level can be measured in the serum or plasma, but is of little clinical value
   c. it is readily expired in air
   d. it is a better indication of cyanide poisoning than whole blood cyanide levels
   e. it is excreted via the kidneys
4. Effects of chronic exposure to cyanide may include
   a. hepatomegaly
   b. hypothyroidism
   c. amblyopia
   d. renal failure
   e. palpitations and chest pain
5. Which of the following is (are) likely affected by cyanide?
   a. adrenal cortical hormone balance
   b. thyroid function
   c. central nervous system centers
   d. hormonal influence on the skeleton
   e. bilirubin transport
6. Neurotoxic effects associated with long-term exposure to HCN could be related to
   a. volatile halogenated solvents
   b. certain foods ingested on a regular basis
   c. pica in children from homes with flaking paint
   d. smoking habits
   e. vitamin B_{12} balance
7. Chronic sequela(e) of acute cyanide poisoning may include
   a. Gilles de la Tourette syndrome
   b. Parkinson-like syndrome
   c. amyotrophic lateral sclerosis
   d. sensory peripheral neuropathy
   e. encephalopathy similar to that caused by carbon monoxide poisoning
8. Treatments that might be prescribed for smoke-inhalation victims of enclosed-space fires with a suspected cyanide component include
   a. 100% normobaric oxygen
   b. sodium nitrite/sodium thiosulfate
   c. correction of metabolic alkalosis with ammonium chloride
   d. hyperbaric oxygen therapy
   e. dimercaprol (BAL)

CASE STUDIES IN ENVIRONMENTAL MEDICINE: CYANIDE TOXICITY

If you wish CME credits or CEUs, please indicate your answers to the Posttest questions on page 22 by circling the letters below for the correct answers. Complete the evaluation questionnaire and fill in the information requested on the reverse side. Tear off this last page, and fold, staple, and mail to Continuing Education Coordinator, Agency for Toxic Substances and Disease Registry, Division of Health Education, E33, 1600 Clifton Road, Atlanta, GA 30333.

1. a b c d e
2. a b c d e
3. a b c d e
4. a b c d e
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6. a b c d e
7. a b c d e
8. a b c d e

Evaluation Questionnaire

Please complete the following evaluation by circling the appropriate number.

	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
1. As a result of completing this monograph, I will be able to:					
Explain why cyanide may be an acute and chronic health hazard.	1	2	3	4	5
Describe the known factors contributing to cyanide toxicity.	1	2	3	4	5
Identify potential environmental or occupational sources of exposure to cyanide.	1	2	3	4	5
Identify evaluation and treatment protocols for persons exposed to cyanide.	1	2	3	4	5
List sources of information on cyanide.	1	2	3	4	5
2. The monograph addressed the objectives printed on the inside front cover.	1	2	3	4	5
3. I am more likely to ask patients questions regarding possible environmental exposures as a result of reading this issue.	1	2	3	4	5
4. Independent study was an effective teaching method for the content.	1	2	3	4	5
5. How much time (in minutes) was required to read this monograph and complete the posttest?	40	60	80	100	120

Comments:_____

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Please send me the following *Case Studies in Environmental Medicine.*

- ❑ Arsenic
- ❑ Asbestos
- ❑ Benzene
- ❑ Beryllium
- ❑ Cadmium
- ❑ Carbon Tetrachloride
- ❑ Chlordane
- ❑ Cholinesterase Inhibitors
- ❑ Chromium
- ❑ Cyanide
- ❑ Dioxins
- ❑ Ethylene/Propylene Glycols
- ❑ Exposure History
- ❑ Gasoline
- ❑ Jet Fuel
- ❑ Lead
- ❑ Mercury
- ❑ Methanol
- ❑ Methylene Chloride
- ❑ Nitrates/Nitrites
- ❑ Pentachlorophenol
- ❑ Polyaromatic Hydrocarbons (PAHs)
- ❑ Polychlorinated Biphenyls (PCBs)
- ❑ Radiation
- ❑ Radon
- ❑ Reproductive and Developmental Hazards
- ❑ Skin Lesions
- ❑ Stoddard Solvent
- ❑ Tetrachloroethylene
- ❑ 1,1,1-Trichloroethane
- ❑ Trichloroethylene
- ❑ Toluene
- ❑ Vinyl Chloride

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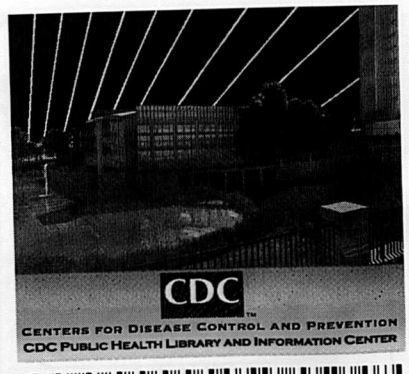

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The state of knowledge regarding the treatment of patients potentially exposed to hazardous substances in the environment is constantly evolving and is often uncertain. In this monograph, the Agency for Toxic Substances and Disease Registry (ATSDR) has made diligent effort to ensure the accuracy and currency of the information presented but makes no claim that the document comprehensively addresses all possible situations related to this substance. This monograph is intended as an additional resource for physicians and other health professionals in assessing the condition and managing the treatment of patients potentially exposed to hazardous substances. It is not, however, a substitute for the professional judgment of a health care provider and must be interpreted in light of specific information regarding the patient available to such a professional and in conjunction with other sources of authority.

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