

Parathion ([C₂H₅O]₂P[S]OC₆H₄NO₂)
CAS 56-38-2; UN 2783

Synonyms include O,O-Diethyl-O-(p-nitrophenyl) phosphorothioate, diethyl parathion, ethyl parathion, parathion ethyl and a variety of trade names such as Alkron, Alleron, Danthion, DNTP, DPP, Etilon, E-605, Stathion, Sulphos, and Thiophos.

- **Persons whose skin or clothing is contaminated with liquid or powdered parathion can cause secondary contamination by direct contact.**
- **Parathion is an organophosphate pesticide. At room temperature, it is a combustible pale yellow to dark brown liquid that may be difficult to ignite. In commercial products, parathion is usually dissolved in hydrocarbon solvents such as toluene or xylene, which are flammable. Parathion has a garlic-like odor, which does not provide adequate warning of hazardous concentrations.**
- **Because parathion has a low vapor pressure, significant inhalation is unlikely at ordinary temperatures. However, the hydrocarbon solvents in commercial preparations can be inhaled. Parathion is rapidly absorbed by ingestion and through intact skin and the eyes, resulting in acute systemic toxicity.**

Description

At room temperature, parathion is a yellow-to-brown liquid with an odor of garlic. It is often dissolved in a hydrocarbon solvent before use. Parathion itself is not volatile. It is almost insoluble in water, slightly soluble in petroleum oils, and miscible with many organic solvents.

Routes of Exposure

Inhalation

Toxic inhalation of parathion vapor is unlikely at ordinary temperatures because of its low volatility, but toxic effects can occur after inhalation of parathion sprays or dusts. The hydrocarbon solvents (most commonly toluene and xylene) used to dissolve parathion are more volatile than parathion itself, and toxicity can result from inhalation of solvent vapor as well. The odor threshold of parathion is five times the OSHA PEL (0.1 mg/m³) and **does not provide adequate warning of hazardous concentrations.**

Children exposed to the same levels of parathion as adults may receive a larger dose because they have greater lung surface area:body weight ratios and increased minute volumes:weight ratios.

Skin/Eye Contact

Parathion is not irritating to the skin or eyes, but is rapidly absorbed through intact skin and eyes, contributing to systemic toxicity.

Children are more vulnerable to toxicants absorbed through the skin because of their relatively larger surface area:body weight ratio.

<i>Ingestion</i>	Acute toxic effects, including rapidly fatal systemic poisoning, can result from ingestion of parathion.
Sources/Uses	Parathion is prepared by the reaction of diethyl phosphorothionchloridate with sodium p-nitrophenate. It is widely used as an agricultural insecticide. It has been used extensively by U.S. farmers on major crops such as wheat, fruit, vegetables, nuts, citrus fruits, alfalfa, corn, soybeans, and other field crops.
Standards and Guidelines	OSHA PEL (permissible exposure limit) = 0.1 mg/m ³ (skin) (averaged over an 8-hour workshift) NIOSH IDLH (immediately dangerous to life or health) = 10 mg/m ³
Physical Properties	<i>Description:</i> Pale-yellow to dark-brown liquid at room temperature <i>Warning properties:</i> Weak odor of garlic at 0.47 mg/m ³ ; inadequate warning for acute and chronic exposures. Does not generally produce skin irritation. <i>Molecular weight:</i> 291.3 daltons <i>Boiling point:</i> (760 mm Hg): 707 °F (375 °C) <i>Freezing point:</i> 43 °F (6.1 °C) <i>Specific gravity:</i> 1.27 (water = 1) <i>Vapor pressure:</i> 4 x 10 ⁻⁵ mm Hg at 68 °F (20 °C) <i>Water solubility:</i> insoluble (0.001% at 68 °F) (20 °C) <i>Flammability:</i> 392 °F (200 °C)
Incompatibilities	Parathion reacts with strong oxidizers and alkaline materials.

Health Effects

Systemic toxicity due to parathion can result from all routes of exposure. Symptoms include abdominal cramps, vomiting, diarrhea, pinpoint pupils and blurred vision, excessive sweating, salivation and lacrimation, wheezing, excessive tracheobronchial secretions, agitation, seizures, bradycardia or tachycardia, muscle twitching and weakness, and urinary bladder and fecal incontinence. Seizures are much more common in children than in adults.

Death results from loss of consciousness, coma, excessive bronchial secretions, respiratory depression and cardiac irregularity.

Commercial parathion products often contain hydrocarbon solvents, such as xylene or toluene, which themselves can cause toxicity.

Toxicity of parathion depends on metabolic activation; thus, symptoms may be delayed for 6 to 24 hours after exposure.

Acute Exposure

Parathion, like all organophosphate pesticides, inhibits acetylcholinesterase and alters cholinergic synaptic transmission at neuroeffector junctions (muscarinic effects), at skeletal myoneural junctions and autonomic ganglia (nicotinic effects), and in the CNS. Inhibition occurs when a metabolite of parathion binds to acetylcholinesterase; thus, symptoms may be delayed after exposure. Signs and symptoms of poisoning vary according to age, dose, and concentration.

Muscarinic effects include: pinpoint pupils; blurred vision; hypersecretion by salivary, lacrimal, sweat, and bronchial glands; narrowing of the bronchi; nausea, vomiting, diarrhea, and crampy abdominal pains; urinary and fecal incontinence; and slow heart rate.

Nicotinic effects include muscle twitching, cramping, and weakness. Nicotinic stimulation can obscure certain muscarinic effects and produce rapid heart rate and high blood pressure.

CNS

CNS effects are often the earliest manifestations of poisoning in adults and constitute the major signs and symptoms in children. CNS effects include irritability, nervousness, giddiness, fatigue, lethargy, impairment of memory, confusion, slurred speech, visual disturbance, depression, impaired gait, convulsions, loss of consciousness, coma, and respiratory depression.

<i>Peripheral Neurologic</i>	Peripheral neurologic effects include muscle twitching and weakness due to inhibition of acetylcholinesterase at neuromuscular junctions.
<i>Respiratory</i>	<p>Respiratory failure is the most common cause of death due to parathion poisoning. Narrowing of the bronchi and markedly increased bronchial secretions can occur. Respiratory failure results from respiratory depression coupled with paralysis of the respiratory muscles and progressive airway obstruction from bronchorrhea. In addition, pulmonary aspiration of the hydrocarbon solvents found in many commercial preparations can cause inflammation of the lungs.</p> <p>Children may be more vulnerable because of relatively increased minute ventilation per kg and failure to evacuate as area promptly when exposed.</p>
<i>Cardiovascular</i>	Most exposure victims experience bradycardia, but pulse rate may be increased initially and tachycardia is more common in very severe poisoning. Irregular heartbeat may occur.
<i>Gastrointestinal</i>	Nausea, vomiting, abdominal cramps, diarrhea, and fecal incontinence are common manifestations, regardless of the exposure route. These are generally the earliest symptoms to occur.
<i>Metabolic</i>	Profuse sweating is likely to occur and may lead to profound dehydration. This is somewhat less common in children.
<i>Dermal</i>	<p>Parathion is not generally irritating, but is readily absorbed through the skin. Skin contact can result in systemic poisoning.</p> <p>Because of their relatively larger surface area:body weight ratio, children are more vulnerable to toxicants absorbed through the skin.</p>
<i>Ocular</i>	Systemic poisoning typically causes pinpoint pupils and spasm of the muscle of visual accommodation (i.e., ciliary muscle) leading to blurred vision and aching pain in the eye. However, organophosphate poisoning may still be present without pinpoint pupils, and dilation of the pupils may even be noted occasionally. Eye irritation, if it occurs, is most likely caused by the hydrocarbon solvents used in commercial pesticide preparations.
<i>Potential Sequelae</i>	Complete recovery generally occurs within 10 days unless severe lack of oxygen has caused residual brain damage. CNS effects such as confusion, fatigue, irritability, nervousness, and

impairment of memory can occasionally last for several weeks. Six to twenty-one days after acute exposure to some organophosphate compounds, onset of nerve disorders of mixed sensory-motor type may occur; peripheral nerve recovery may never be complete. It is uncertain if parathion produces this delayed polyneuropathy.

Chronic Exposure

Persistent weakness and impaired memory have been reported to occur from low-level exposures to organophosphates in the absence of acute cholinergic effects.

Carcinogenicity

The International Agency for Research on Cancer has determined that parathion is not classifiable as to its carcinogenicity to humans. However, EPA lists parathion as a possible human carcinogen.

Reproductive and Developmental Effects

Studies have been reported in which parathion was embryo-toxic and fetotoxic in rodents. There are no studies addressing reproductive or developmental effects in humans exposed to parathion. Parathion is not included in *Reproductive and Developmental Toxicants*, a 1991 report published by the U.S. General Accounting Office (GAO) that lists 30 other chemicals of concern because of widely acknowledged reproductive and developmental consequences.

Prehospital Management

Parathion is highly contaminating. Victims whose skin or clothing is contaminated with liquid or powdered parathion can secondarily contaminate response personnel by direct contact or evaporation of solvent vapor. Clothing and leather goods (e.g., belts or shoes) cannot be reliably decontaminated; they should be incinerated.

Systemic effects of parathion poisoning can occur from all routes of exposure. Symptoms of parathion poisoning can include headache, nausea, vomiting, abdominal cramps, diarrhea, generalized muscle weakness and twitching, slurred speech, pinpoint pupils, excessive secretions and shortness of breath.

Severely poisoned patients may develop seizures, skeletal-muscle paralysis, cardiac arrhythmias and respiratory failure, or may become comatose.

Commercial parathion products often contain hydrocarbon solvents, such as xylene or toluene, which themselves can cause toxicity. Treatment for breathing the solvent is fresh air.

Treatment for parathion poisoning consists of thorough decontamination, cardiorespiratory support, and administration of the antidotes atropine and pralidoxime. Antidotes should be administered as prevention even if the diagnosis is in doubt.

Hot Zone

Rescuers should be trained and appropriately attired before entering the Hot Zone. If the proper equipment is not available, or if rescuers have not been trained in its use, assistance should be obtained from a local or regional HAZMAT team or other properly equipped response organization.

Rescuer Protection

Parathion is a highly toxic systemic poison that is absorbed well by all routes of exposure.

Respiratory Protection: Positive-pressure, self-contained breathing apparatus (SCBA) is recommended in response situations that involve exposure to potentially unsafe levels of parathion.

Skin Protection: Chemical-protective clothing is recommended because parathion is rapidly absorbed through the skin and may cause systemic poisoning.

ABC Reminders

Quickly access for a patent airway, ensure adequate respiration and pulse. If trauma is suspected, maintain cervical immobilization manually and apply a cervical collar and a backboard when feasible.

Victim Removal

If victims can walk, lead them out of the Hot Zone to the Decontamination Zone. Victims who are unable to walk may be removed on backboards or gurneys; if these are not available, carefully carry or drag victims to safety.

Consider appropriate management of chemically-contaminated children, such as measures to reduce separation anxiety if a child is separated from a parent or other adult.

Decontamination Zone

All victims suspected of parathion ingestion, or substantial exposure to aerosolized parathion, or who have skin or eye exposure to liquid or powdered parathion require thorough decontamination as described below.

Rescuer Protection

If exposure levels are determined to be safe, decontamination may be conducted by personnel wearing a lower level of protection than that worn in the Hot Zone (described above).

ABC Reminders

Quickly access for a patent airway, ensure adequate respiration and pulse. Stabilize the cervical spine with a collar and a backboard if trauma is suspected. Administer supplemental oxygen as required. Assist ventilation with a bag-valve-mask device if necessary.

Basic Decontamination

Rapid and thorough decontamination is critical, but must proceed concurrently with supportive and antidotal measures.

Victims who are able may assist with their own decontamination. Quickly remove and double-bag contaminated clothing and personal belongings. Clothing (especially leather items) is extremely difficult to decontaminate; in most cases, contaminated clothing should be incinerated as directed by hazardous materials experts.

Victims should be washed repeatedly with copious amounts of soap and water. Rescuers should wear rubber gloves as vinyl gloves provide no protection against skin absorption. It is important to observe the patient closely for sudden appearance of symptoms. It is important to thoroughly clean hair, fingernails, and skin folds. Use caution to avoid hypothermia when decontaminating children or the elderly. Use blankets or warmers when appropriate.

Irrigate exposed or irritated eyes with plain water or saline for 15 minutes. Remove contact lenses if easily removable without additional trauma to the eye. If a corrosive material is suspected or if pain or injury is evident, continue irrigation while transferring the victim to the Support Zone.

In cases of ingestion, **do not induce emesis**. If the victim is alert and asymptomatic, administer a slurry of activated charcoal (at 1 gm/kg, usual adult dose 60–90 g, child dose 25–50 g). A soda can and straw may be of assistance when offering charcoal to a child.

Consider appropriate management of chemically contaminated children at the exposure site. Provide reassurance to the child during decontamination, especially if separation from a parent occurs.

Transfer to Support Zone

As soon as basic decontamination is complete, move the victim to the Support Zone.

Support Zone

Be certain that victims have been decontaminated properly (see *Decontamination Zone* above). Victims who have undergone decontamination or have been exposed only to vapor generally pose no serious risks of secondary contamination to rescuers. However, the Support Zone team should wear disposable aprons or gowns and rubber gloves for protection.

ABC Reminders

Quickly access for a patent airway. If trauma is suspected, maintain cervical immobilization manually and apply a cervical collar and a backboard when feasible. Ensure adequate respiration and pulse. Administer supplemental oxygen as required and establish intravenous access if necessary. Place on a cardiac monitor. Airway suctioning may be required for excessive bronchial secretions.

Additional Decontamination

Continue irrigating exposed skin and eyes, as appropriate.

In cases of ingestion, **do not induce emesis**. If the victim is alert and asymptomatic, administer a slurry of activated charcoal if it has not already been given (at 1 gm/kg, usual adult dose 60–90 g, child dose 25–50 g). A soda can and straw may be of assistance when offering charcoal to a child.

Advanced Treatment

In cases of respiratory compromise, coma, or to facilitate removal of excessive pulmonary secretions secure airway and respiration via endotracheal intubation. If not possible, perform cricothyroidotomy if equipped and trained to do so.

When possible, atropine (see *Antidotes*, below) should be given under medical supervision to all symptomatic patients who have known or strongly suspected parathion poisoning.

Patients who are comatose, hypotensive, or having seizures or cardiac arrhythmias should be treated according to advanced life support (ALS) protocols.

Antidotes

Two antidotes are administered to treat organophosphate poisoning. Atropine is a competitive antagonist of acetylcholine at muscarinic receptors and is used to control the excessive bronchial secretions which are often responsible for death. Pralidoxime relieves both the nicotinic and muscarine effects of organophosphate poisoning by regenerating acetylcholinesterase and can reduce both the bronchial secretions and the muscle weakness associated with poisoning.

The initial intravenous dose of atropine in adults should be determined by the severity of symptoms: An initial adult dose of 1.0 to 2.0 mg or pediatric dose of 0.01 mg/kg (minimum 0.01 mg) should be administered intravenously. If intravenous access cannot be established, atropine may also be given subcutaneously or via endotracheal tube. Doses should be repeated every 15 minutes until excessive secretions and sweating have been controlled. Once bronchial secretion has been controlled, atropine administration should be repeated whenever the secretions begin to recur. In seriously poisoned patients, very large doses may be required. Alterations of pulse rate and pupillary size should not be used as indicators of treatment adequacy.

Pralidoxime should be administered as early in poisoning as possible as its efficacy may diminish when given more than 24 to 36 hours after exposure. Doses are as follows: adult 1.0 g; pediatric 25 to 50 mg/kg. The drug should be administered intravenously over 30 to 60 minutes, but in a life-threatening situation, one-half of the total dose can be given per minute for a total administration time of 2 minutes. Treatment should begin to take effect within 40 minutes with a reduction in symptoms and the amount of atropine necessary to control bronchial secretion. The initial dose can be repeated in 1 hour and then every 8 to 12 hours until the patient is clinically well and no longer requires atropine.

Transport to Medical Facility

Only decontaminated patients or patients not requiring decontamination should be transported to a medical facility. "Body bags" are not recommended.

Report to the base station and the receiving medical facility the condition of the patient, treatment given, and estimated time of arrival at the medical facility. Severely ill patients should be taken to a medical facility immediately.

If parathion has been ingested, prepare the ambulance in case the victim vomits toxic material. Have ready several towels and open plastic bags to quickly clean up and isolate vomitus.

Multi-Casualty Triage

Consult with the base station physician or the regional poison control center for advice regarding triage of multiple victims.

Patients with evidence of significant exposure, such as nausea or excessive sweating, and all persons who have ingested parathion should be transported to a medical facility for evaluation. Others may be discharged at the scene after their names, addresses, and telephone numbers are recorded. It is very important to ensure that individuals have been completely decontaminated before discharge; otherwise life-threatening symptoms may occur in the absence of assistance. Those discharged should be advised to seek medical care promptly if symptoms develop (see *Patient Information Sheet* below).

Emergency Department Management

Parathion is highly contaminating. Patients whose skin or clothing is contaminated with liquid or powdered parathion can secondarily contaminate hospital personnel by direct contact or off-gassing of solvent vapor from clothing, skin, or vomitus. Clothing and leather goods (e.g., belts or shoes) cannot be reliably decontaminated; they should be incinerated.

Systemic effects of parathion poisoning can occur from all routes of exposure. Symptoms of parathion poisoning can include headache, nausea, vomiting, abdominal cramps, diarrhea, generalized muscle weakness and twitching, slurred speech, pinpoint pupils, excessive secretions and shortness of breath.

Severely poisoned patients may develop seizures, skeletal-muscle paralysis, cardiac arrhythmias and respiratory failure, or may become comatose.

Commercial parathion products often contain hydrocarbon solvents such as xylene or toluene, which themselves can cause toxicity.

Treatment consists of thorough decontamination, cardiorespiratory support, and administration of the antidotes atropine and pralidoxime. Antidotes should be

Decontamination Area

Unless decontaminated previously, all patients suspected of skin or eye contact with liquid or powdered parathion require decontamination immediately. If a solvent such as xylene or toluene is involved, decontamination should take place outdoors or with proper ventilation.

Health care personnel should don butyl rubber aprons and butyl rubber gloves. If butyl rubber items are not available, limited protection will be provided by two layers of latex gloves and a waterproof apron or chemical-resistant jumpsuit. Wash hands frequently during decontamination and promptly dispose of latex gloves. Personnel who come in contact with parathion secondarily should undergo decontamination.

Be aware that use of protective equipment by the provider may cause fear in children, resulting in decreased compliance with further management efforts.

Because of their relatively larger surface area:body weight ratio, children are more vulnerable to toxicants absorbed through the skin. Also emergency room personnel should examine children's mouths because of the frequency of hand-to-mouth activity among children.

ABC Reminders

Evaluate and support airway, breathing, and circulation. In cases of respiratory compromise, coma, or to facilitate tracheal suctioning, secure airway and respiration via endotracheal intubation. If not possible, surgically create an airway. Suctioning may be required for excessive bronchial secretions.

Patients who are comatose, hypotensive, or have seizures or ventricular arrhythmias should be treated in the conventional manner.

Basic Decontamination

Patients who are able may assist with their own decontamination. Remove and double-bag contaminated clothing and personal belongings. Dispose of contaminated clothing and leather goods (e.g., belts, wallets, and shoes); they should be incinerated as directed by hazardous materials experts.

Victims should be washed repeatedly with copious amounts of soap and water. Rescuers should wear rubber gloves as vinyl gloves provide no protection against skin absorption. It is important to observe the patient closely for sudden appearance of symptoms. It is important to thoroughly clean hair, fingernails, and skin folds. Use caution to avoid hypothermia when decontaminating children or the elderly. Use blankets or warmers when appropriate.

Flush exposed eyes with plain water or saline for 15 minutes. Remove contact lenses if easily removable without additional trauma to the eye. Continue irrigation during transport to the Critical Care Area.

In cases of ingestion, **do not induce emesis**. If the victim is alert and asymptomatic, administer a slurry of activated charcoal if it has not already been given (administer at 1 gm/kg, usual adult dose 60–90 g, child dose 25–50 g). A soda can and straw may be of assistance when offering charcoal to a child.

Critical Care Area

Be certain that appropriate decontamination has been carried out (see *Decontamination Area* above).

ABC Reminders

Evaluate and support airway, breathing, and circulation as in ABC Reminders above. Establish intravenous access in seriously ill patients. Continuously monitor cardiac rhythm.

Patients who are comatose, hypotensive, or have seizures or cardiac arrhythmias should be treated in the conventional manner.

Inhalation Exposure

Administer supplemental oxygen by mask to patients who have respiratory symptoms. Refer to *Antidotes and Other Treatments* below for appropriate clinical treatment.

Skin Exposure

Parathion does not generally burn or irritate the skin, but dermal exposure produces systemic toxicity. Thorough washing of the skin, as described in *Basic Decontamination* above, should be performed and may need to be repeated. Unexplained, persistent symptoms may indicate inadequate decontamination.

Because of their relatively larger surface area:body weight ratio, children are more vulnerable to toxicants affecting the skin.

Eye Exposure

Continue irrigation for at least 15 minutes. Test visual acuity. Examine the eyes for corneal damage and treat appropriately. Immediately consult an ophthalmologist for patients who have severe corneal injuries.

Ingestion

Do not induce emesis. If the victim is alert and asymptomatic, administer a slurry of activated charcoal if it has not already been given (administer at 1 gm/kg, usual adult dose 60–90 g, child dose 25–50 g). A soda can and straw may be of assistance when offering charcoal to a child.

Gastric lavage is useful in certain circumstances to remove toxic material. Consider gastric lavage with a small nasogastric tube if: (1) a large dose has been ingested; (2) the patient's condition is evaluated within 30 minutes; (3) the patient has oral lesions or persistent esophageal discomfort; and (4) the lavage can be administered within one hour of ingestion. Care must be taken when placing the gastric tube because blind gastric-tube placement may further injure the chemically damaged esophagus or stomach.

Because children do not ingest large amounts of toxic materials, and because of the risk of perforation from NG intubation, lavage is discouraged in children unless performed under endoscopic guidance.

Toxic vomitus or gastric washings should be isolated, e.g., by attaching the lavage tube to isolated wall suction or another closed container.

Antidotes and Other Treatments

All patients who have signs or symptoms of systemic toxicity require antidotal treatment. In the United States, primary antidotal treatment for parathion poisoning involves administration of

atropine. In addition, pralidoxime (2-PAM) is indicated for seriously poisoned patients.

Atropine is a competitive antagonist of acetylcholine at muscarinic receptors and is used to control the excessive bronchial secretions which are often responsible for death. Pralidoxime relieves both the nicotinic and muscarine effects of organophosphate poisoning by regenerating acetylcholinesterase and can reduce both the bronchial secretions and the muscle weakness associated with poisoning.

The initial intravenous dose of atropine in adults should be determined by the severity of symptoms: An initial adult dose of 1.0 to 2.0 mg or pediatric dose of 0.01 mg/kg (minimum 0.01 mg) should be administered intravenously. If intravenous access cannot be established, atropine may also be given subcutaneously or via endotracheal tube. Doses should be repeated every 15 minutes until excessive secretions and sweating have been controlled. Once bronchial secretion has been controlled, atropine administration should be repeated whenever the secretions begin to recur. In seriously poisoned patients, very large doses may be required. Alterations of pulse rate and pupillary size should not be used as indicators of treatment adequacy.

Pralidoxime should be administered as early in poisoning as possible as its efficacy may diminish when given more than 24 to 36 hours after exposure. Doses are as follows: adult 1.0 g; pediatric 25 to 50 mg/kg. The drug should be administered intravenously over 30 to 60 minutes, but in a life-threatening situation, one-half of the total dose can be given per minute for a total administration time of 2 minutes. Treatment should begin to take effect within 40 minutes with a reduction in symptoms and the amount of atropine necessary to control bronchial secretion. The initial dose can be repeated in 1 hour and then every 8 to 12 hours until the patient is clinically well and no longer requires atropine.

Patients who have seizures most often respond to atropine and pralidoxime; however, if they do not respond, administer diazepam or lorazepam.

Avoid other acetylcholinesterase inhibitors (e.g., physostigmine and edrophonium chloride), and do not use succinylcholine for rapid sequence intubation. The paralyzing effects of succinylcholine are likely to be prolonged because it will not be metabolized normally.

Non-depolarizing neuromuscular blockers (e.g., pancuronium and vecuronium) may not be effective because of unsuccessful competition with acetylcholinesterase for the motor end-plate receptors.

Laboratory Tests

The diagnosis of acute parathion toxicity is primarily clinical and is based on the combination of nausea, excessive sweating and salivation, miosis, and muscle weakness. Routine laboratory studies for all exposed patients include CBC, glucose, and electrolyte determinations. A chest radiograph is useful to examine for hydrocarbon aspiration and non-cardiogenic pulmonary edema. Symptomatic and asymptomatic patients suspected of significant exposure should have determinations of plasma and red blood cell (RBC) cholinesterase activity. Symptoms of acute poisoning are usually present when more than 50% of RBC cholinesterase activity is inhibited. However, these tests are not always readily available and are more useful in diagnosis and follow-up. Blood and urine analyses for the presence of parathion or its metabolite may show whether damage has been done to the brain, heart, lungs, and nerves.

Disposition and Follow-up

Patients with life threatening illness must be hospitalized, also consider hospitalizing patients who have a suspected serious exposure and are symptomatic.

Delayed Effects

Skin absorption can cause delayed or recurrent symptoms. Contaminated clothing and leather items (e.g., shoes, wallets, and belts) should not be reused, even if they have been washed.

Aspiration of commercial parathion preparations that contain hydrocarbon solvents can result in chemical pneumonitis.

Chronic neurologic symptoms have been reported for some patients exposed to organophosphates. Symptoms develop after acute toxicity has resolved (24 to 96 hours after exposure) and can include cognitive impairment, depression, Guillian-Barré syndrome, and peripheral neuropathy.

Patient Release

Patients who remain asymptomatic for 4 to 6 hours after exposure may be discharged with instructions to seek medical care promptly if symptoms develop (see *Parathion—Patient Information Sheet* below).

Follow-up

Obtain the name of the patient's primary care physician so that the hospital can send a copy of the ED visit to the patient's doctor.

Patients who have severe exposure should be evaluated for persistent CNS sequelae and delayed peripheral neuropathy.

Patients who have been acutely poisoned should be advised to avoid further organophosphate exposure until sequential RBC

cholinesterase levels have stabilized in the normal range, a process that may take 3 to 4 months after severe poisoning.

Patients who have corneal injuries or severe skin burns should be reexamined within 24 hours.

Reporting

If a work-related incident has occurred, you may be legally required to file a report; contact your state or local health department.

Other persons may still be at risk in the setting where this incident occurred. If the incident occurred in the workplace, discussing it with company personnel may prevent future incidents. If a public health risk exists, notify your state or local health department or other responsible public agency. When appropriate, inform patients that they may request an evaluation of their workplace from OSHA or NIOSH. See Appendices III and IV for a list of agencies that may be of assistance.

Parathion Patient Information Sheet

This handout provides information and follow-up instructions for persons who have been exposed to parathion.

What is parathion?

Parathion is a potent organophosphate pesticide. It is a pale yellow-to-brown liquid with an odor like garlic. It is used by farmers as a pesticide on fruits, vegetables, nuts, and grains. Commercial pesticides often contain a hydrocarbon solvent, which itself can cause illness.

What immediate health effects can be caused by exposure to parathion?

Parathion can cause nausea, vomiting, stomach cramps, and diarrhea, as well as confusion, blurred vision, sweating, muscle twitching, irregular heartbeat, convulsions, and death. Symptoms occur when parathion is inhaled, swallowed or absorbed through the skin. Breathing the solvent used to dissolve the pesticide may cause dizziness, headache, and nausea. Generally, the more serious the exposure, the more severe the symptoms.

Can parathion poisoning be treated?

For minor exposures (for example, breathing the pesticide solvent), the only treatment needed is fresh air. For serious parathion poisoning, thorough washing of all exposed skin, removal and burning of exposed clothing, and hospitalization and administration of an antidote may be needed.

Are any future health effects likely to occur?

A single small exposure from which a person recovers quickly is not likely to cause delayed or long-term effects. After a serious exposure, a patient may feel ill for several weeks.

What tests can be done if a person has been exposed to parathion?

Specific tests for the presence of parathion or its breakdown product in blood and urine generally are not useful to the doctor. If a severe exposure has occurred, blood and urine analyses and other tests may show whether damage has been done to the brain, heart, lungs, and nerves. Testing is not needed in every case.

Where can more information about parathion be found?

More information about parathion can be obtained from your regional poison control center; your state, county, or local health department; the Agency for Toxic Substances and Disease Registry (ATSDR); your doctor; or a clinic in your area that specializes in occupational and environmental health. If the exposure happened at work, you may wish to discuss it with your employer, the Occupational Safety and Health Administration (OSHA), or the National Institute for Occupational Safety and Health (NIOSH). Ask the person who gave you this form for help in locating these telephone numbers.

Follow-up Instructions

Keep this page and take it with you to your next appointment. Follow *only* the instructions checked below.

- Call your doctor or the Emergency Department if you develop any unusual signs or symptoms within the next 24 hours, especially:
- irritability, confusion, or fatigue
 - coughing, wheezing, or shortness of breath
 - nausea, vomiting, cramps, or diarrhea
 - muscle weakness or twitching
 - blurred vision
- No follow-up appointment is necessary unless you develop any of the symptoms listed above.
- Call for an appointment with Dr. _____ in the practice of _____.
When you call for your appointment, please say that you were treated in the Emergency Department at _____ Hospital by _____ and were advised to be seen again in _____ days.
- Return to the Emergency Department/ _____ Clinic on (date) _____ at _____ AM/PM for a follow-up examination.
- Do not perform vigorous physical activities for 1 to 2 days.
- You may resume everyday activities including driving and operating machinery.
- Do not return to work for _____ days.
- You may return to work on a limited basis. See instructions below.
- Avoid exposure to cigarette smoke for 72 hours; smoke may worsen the condition of your lungs.
- Avoid drinking alcoholic beverages for at least 24 hours; alcohol may worsen injury to your stomach or have other effects.
- Avoid taking the following medications: _____
- You may continue taking the following medication(s) that your doctor(s) prescribed for you: _____

- Other instructions: _____

- Provide the Emergency Department with the name and the number of your primary care physician so that the ED can send him or her a record of your emergency department visit.
- You or your physician can get more information on the chemical by contacting: _____ or _____, or by checking out the following Internet Web sites: _____; _____.

Signature of patient _____ Date _____

Signature of physician _____ Date _____