PESTICIDE PROTECTION

a training manual for health personnel

U.S. DEPARTMENT
of HEALTH, EDUCATION, and WELFARE

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF PESTICIDE PROGRAMS

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a training manual for health personnel

A guide for recognizing, managing, preventing and verifying poisonings caused by organophosphates, carbamates, and other selected pesticides

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INTRODUCTION

This manual is for all health personnel involved in the prevention, recognition, and treatment of pesticide poisoning. However, the information should be of particular interest to nurses[1], especially those working in rural clinics, hospital emergency rooms, and departments of public health. They are the personnel most intimately involved with the care of patients suffering from pesticide poisoning.

Nurses are concerned not only with treatment, but also with prevention. If they understand the nature and use of pesticides, they can work with their public health personnel and community health workers toward the development of safety measures. They can contribute to improved case finding and the followup of illness due to pesticide exposure. They are also ideally suited to educate agricultural workers and their families.

In the primary care setting, nurses in the clinic are in a strategic position to recognize cases early and to rapidly implement necessary treatment measures. In this situation, therefore, they must know the signs and symptoms of pesticide poisoning, and the emergency room measures which save lives and decrease morbidity.

In the intensive care unit, the nurse's ability to recognize the reappearance of cholinesterase signs or to detect evidence of atropine excesses can significantly contribute to the successful management of this medical emergency.

Ambulance attendants and other health personnel also need this information so they can begin the rapid treatment which is necessary for lifesaving before the patient arrives at the hospital.

There is an urgent need for accurate and verified data on acute pesticide poisonings. Present reporting is incomplete, and the potential of the laboratory to verify possible pesticide illness is poorly understood. This book describes the steps necessary to confirm suspected pesticide illnesses.

This book deals mainly with two major types of pesticide illness:

- acute systemic poisoning
  - –severe
  - –mild
- topical (local) effects
  - –eyes
  - –skin.

Actual case examples of pesticide poisoning are used to illustrate:

- the essential diagnostic and management needs of the patient, and
- the potential of the laboratory in verifying the event.

[1]There is no intention by the author to stereotype by sex any health personnel or workers. To avoid a cumbersome text, the words "he" and "she" have been used interchangeably.

CHAPTER I
PESTICIDES

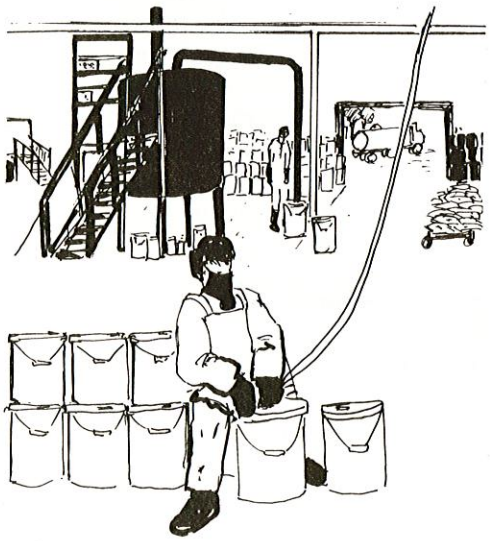
What Are Pesticides?

Pesticides are a diverse group of chemicals which have been developed to kill, prevent, or suppress a wide variety of pests. Six of the most common types are:

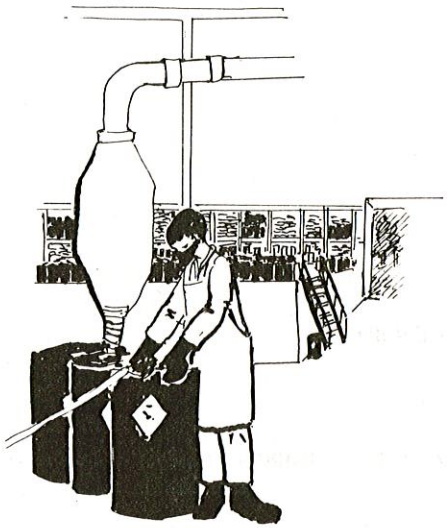
- insecticides (insects)
- herbicides (weeds)
- fungicides (fungi)
- molluscicides (snails and slugs)
- nematicides (nematodes)
- rodenticides (rodents).

Others include miticides (mites), defoliants (remove unwanted plant growth), repellants (keep pests away), attractants (lure pests), and plant growth regulators (stop, speed up, or otherwise change normal plant processes).

The essential component of a pesticide is the active ingredient. This is the material that actually controls the pest. It is produced in a manufacturing plant.



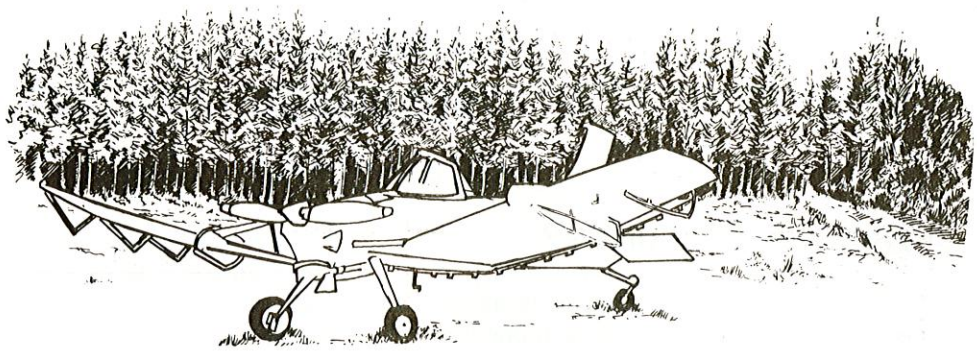
Following manufacture, the active ingredient usually goes to a formulating plant, where it is mixed with other chemicals and a carrier for effective delivery. These are the inert ingredients. They may include such materials as talcs, oils, kerosene, and binding agents (to increase adherence).



These formulated products are sold in many forms, the most common of which are liquids, wettable powders, granules, and dusts. Each form is available in several concentrations.

From the formulating plant, the products move to a wide variety of users, including farmers, commercial pesticide applicators, and the general public.

Application is by various types of ground equipment, hand equipment, or aircraft. It is estimated that aircraft supply about 65 percent of all pesticides used by agriculture.



Registration

More than 1 billion pounds of pesticides are produced each year in the United States. Before any pesticide product can be sold, the manufacturer must register it with the Environmental Protection Agency (EPA). The manufacturer must provide EPA the results of many kinds of tests on the product before it can be registered.

Labeling

EPA requires certain information to be on every pesticide label. Much of this information can be of help to health personnel in diagnosis and treatment of pesticide poisonings. When you know or suspect that a pesticide is involved in an illness, try to get the product label or a copy of it as soon as possible.

Information which must be on a pesticide label includes:

- brand name
- common name (simplified chemical name)
- active ingredients (chemical or common name, plus percent of the contents they make up)
- inert ingredients (need not be named, but label must tell what percent of contents they make up)
- net contents
- name and address of manufacturer
- registration number (shows that the product has been registered with EPA)
- establishment number (identifies the factory which made the product)
- signal words (one of the following)

Signal Words	Toxicity	Approximate amount needed to kill the average person
DANGER	Highly toxic	a taste to a teaspoonful
WARNING	Moderately toxic	a teaspoonful to a tablespoonful
CAUTION	Low toxicity or Comparatively free from danger	an ounce to more than a pint
All products must bear the statement "Keep out of reach of children."		

- skull and crossbones (must appear, with the word "poison", on all highly toxic materials)
- hazards to humans and animals (includes ways in which the product may be poisonous and protective equipment needed)
- environmental hazards
- physical and chemical hazards (special fire, chemical, or explosion hazards)
- statement of practical treatment (emergency first aid measures and information for physicians on the treatment of poisoning)
- statement of use classification (tells whether the pesticide is restricted to use by certified applicators)
- directions for use (may include reentry times, storage and disposal instructions).

Chemical Groups and Mode of Action

Because most pesticides kill unwanted organisms, they are obviously toxic materials. Their mode of action depends on the chemical group to which they belong. The five major chemical groups are:

- organophosphates and carbamates
- organochlorines
- nitro and chloro phenols
- anticoagulants
- bipyridyls.

The *organophosphate and carbamate* group is the greatest public health problem. It contains many widely used insecticides. In the United States, severe organophosphate poisoning results more often from ethyl parathion and phosdrin. Carbamates commonly used include carbaryl (Sevin), propoxur (Baygon), and methomyl (Lannate or Nudrin).

Organophosphates and carbamates inhibit the enzyme cholinesterase (ChE). This inhibition causes a buildup of acetylcholine in the body. Acetylcholine is the primary chemical transmitter for:

- the preganglionic neurons of the sympathetic and parasympathetic fibers
- the postganglionic parasympathetic fibers
- the central nervous system.

These nerve fibers are called cholinergic fibers. Anticholinesterase poisoning causes parasympathetic effects in the organs they supply. Some of these *muscarinic effects* are shown in the following table.

Organ	Effects	Physical Findings
Eyes - Pupil Ciliary Muscle	Constricted Stimulated	Miosis Blurred Vision
Glands - Lacrimal Salivary Gastric	Stimulated Stimulated Stimulated	Tearing Salivation Increased Secretions
Heart - Muscle	Slow Rate	Bradycardia
Lungs - Bronchi	Constricted	Bronchospasm
Intestines - Lumen Sphincters	Stimulated Relaxation	Increased Peristalsis Evacuation
Bladder - Detrusor muscle Trigone	Stimulated Inhibition	Increased Peristalsis Evacuation

Acetylcholine is also secreted at the skeletal nerve endings, where, in excess, it produces weakness and paralysis. These neuromuscular effects are called *nicotinic effects.* Acetylcholine is also the chemical mediator between the sympathetic nerve fibers and the sweat glands. This is the reason for excessive sweating in poisonings with these cholinergic chemicals.

Atropine is the specific antidote for cholinergic poisoning. It blocks the effects of acetylcholine. Atropine has no effect on the neuromuscular nerve endings, however. This is where the oxime drugs are beneficial. They break up the chemical binding between the pesticide and the cholinesterase enzymes. This frees cholinesterase to stop the acetylcholine action at the neuromuscular junction and thus end the paralytic effects. Oximes are contraindicated, however, in cases of carbamate poisoning.

The effects of this group of chemicals are both systemic and topical. If there is a topical eye exposure, the effects are those of topical effects of acetylcholine on the eye:

- the pupils are constricted
- the ciliary muscles are stimulated, causing blurring of vision and an eyebrow headache.

The *organochlorine* pesticides are powerful nervous system stimulators, but their modes of action are not completely known. These chemicals are soluble in fat, accumulate in the human body, and are very persistent in the environment. Most uses of these pesticides (such as DDT, aldrin, dieldrin) are prohibited in the United States, so acute poisonings are not frequent. Systemic poisonings occur most often with endrin, which is one of the most toxic members of this group.

The *nitro and chloro phenols* are strong metabolic stimulators causing increased metabolism and hyperthermia. These are widely used as fungicides and herbicides.

The *anticoagulants* are rodenticides, such as Warfarin. They produce their effects by inhibiting prothrombin and causing capillary damage.

The *bipyridyls* include paraquat and diquat, which are widely used in agriculture for weed control and defoliation. These chemicals produce proliferative changes in a variety of tissues.

CHAPTER II
PESTICIDE HAZARDS AND HOW EXPOSURE OCCURS

Hazards of Pesticide Use

The hazard of a pesticide–its potential for producing injury–depends on:

- the inherent toxicity of the active ingredient (see Appendix 2)
- the dose and/or concentration of the pesticide
- the physical and chemical properties of the material
- the route of absorption of the chemical
- the duration of exposure.

The *dose* (amount) of pesticide taken into the body is the most important factor in determining the hazard of a chemical. A small amount of some pesticides may cause severe illness; large doses of others may be fairly harmless. Pesticide concentrates are the most hazardous form. Persons working with them are at the greatest risk of getting a harmful dose.

The *physical and chemical properties* of some pesticides make them more hazardous in certain situations. Parathion, for example, changes to a more toxic chemical (paraoxon) at high temperatures.

The three possible *routes of absorption* are:

- ingestion–the result of accidents or suicide or homicide attempts; usually causes the most serious effects.
- inhalation–occurs mainly in confined spaces (warehouses, pesticide tanks); usually causes less serious effects than ingestion.
- dermal absorption–most common method of occupational exposure; causes the least severe effects.

The *duration of exposure* helps determine the dose absorbed. Brief exposure to a concentrate could produce effects similar to longer exposure to the dilute pesticide.

How Exposure Occurs

There is some degree of hazard at each step of pesticide manufacture and use. Because they work with pesticide concentrates, workers in pesticide manufacturing and formulating plants are in positions with a high potential hazard. Most manufacturing plants, however, use a closed system which does not expose the workers to the pesticide. Safe occupational practices and good industrial hygiene help to minimize the danger at both these stages of pesticide production.

Health personnel are most likely to encounter pesticide poisonings in three main groups of people:

- applicators
- pickers
- children.

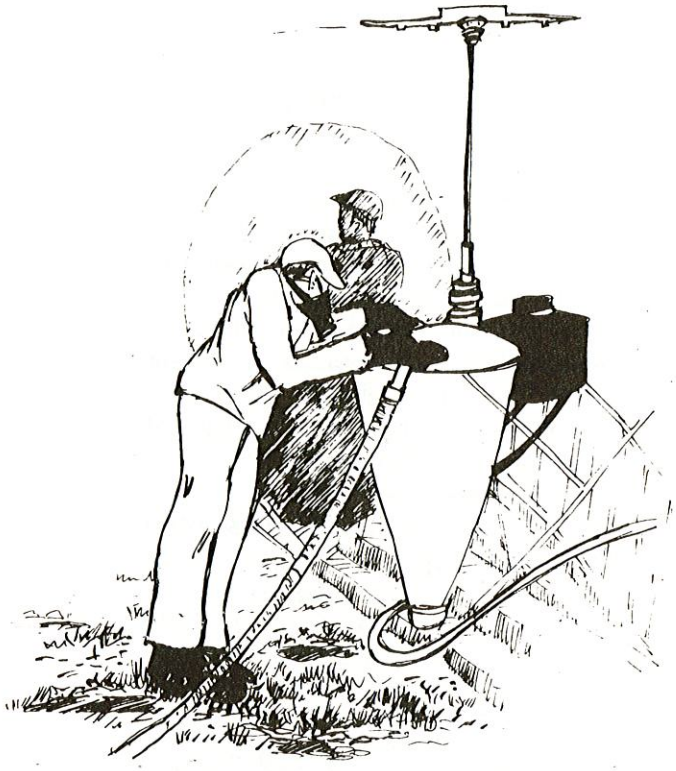
Each is clinically different and each must be recognized at once.

Applicator Poisoning

The hazard to applicators results from the dilution and application of the pesticide concentrate. Hazards exist, therefore, in both mixing and applying the pesticide. The toxicity and concentration of pesticides varies in different applicator situations. Every applicator, however, is at risk of exposure to varying degrees of pesticide concentrate, and therefore is in danger of poisoning. The further down a person is in the chain of pesticide handling and use, the less training he usually has and the greater is his risk of poisoning.

Workers associated with the aerial application of pesticides are especially highly exposed.

It is unwise for a pilot to mix and load chemicals. The normal procedure is to employ aircraft loaders and swampers. They dilute the material and load the aircraft before each run. This is done on the airstrip with fixed wing aircraft. Helicopters are often loaded from an accompanying trailer at the scene of application. Loaders are one of the most highly exposed occupational groups in the entire application process.



An example of applicator poisoning was Tony B., who was employed as a pesticide mixer and loader for a fixed-wing crop dusting firm. He had started loading the aircraft at 6 a.m. with a mixture of parathion 6-3 and toxaphene. Rubber gloves were the only protective clothing worn, and he was a heavy smoker.

He soon began to feel unwell. He was admitted at 11:35 a.m. to the emergency room of a local hospital. He complained of nausea, vomiting, weakness, and blurring of vision. His pupils were constricted and there was profuse perspiration.

A screening cholinesterase test revealed severe inhibition. After being put in a shower and scrubbed all over, he was given a total of 12 mg of atropine intravenously in the emergency room over a brief period of time. The oxime 2-PAM was also administered in a one gram dose in 1,000 cc of D5W. Atropine therapy was continued after his transfer to the medical ward and he proceeded to improve over the next few days.

Subsequent blood and urine metabolite studies by the laboratory confirmed that the poisoning was due to an exposure to ethyl and methyl parathion.

The special lessons of this case are:

- the illness was severe
- no protective clothing was worn
- there was no positive history of an accidental spill
- only one applicator was affected.

Picker Poisoning

Once a pesticide has been diluted to its final concentration and is applied to the crop, the pesticide residue remaining on the fruit and leaves becomes a new source of exposure. The concentrations of these residues are high at first. They decline with time as a result of biodegradation and exposure to light. The rate of dissipation of foliar residues varies considerably with different pesticides and with different concentrations of pesticides. Weather factors also affect the rate of dissipation. Rain removes pesticides more rapidly, and high temperatures favor the changing of some pesticides to more toxic forms.

Too early a reentry to a treated site creates a hazard for the worker. The worker is at risk during the process of thinning and harvesting the crop.

One type of pesticide worker who is at special risk of intoxication is called a "scout." This person goes into the fields regularly to count the number of pests on the plants. Since the job often involves going into the field shortly after application, there is a special risk of residue intoxication.

This type of human illness is sometimes called "picker poisoning." It occurs most frequently with exposures to plants with large leaf surfaces such as citrus, peaches, grapes and tobacco.

Cotton is a crop with a large leaf surface, but it is almost entirely machine-harvested in the United States, so that—except for scouts—residue poisoning is not a serious threat with this crop. This is not the case, however, for Central America, where hand harvesting is still practiced and hundreds of cases of "picker poisoning" have been reported.

Because pesticide poisoning from residues may be milder, it may be overlooked in the health clinic. Health personnel should take special care to be on the lookout for this syndrome with people working in high-exposure crops, especially when the weather is hot.

A nurse may be confronted by a picker who was picking beans or citrus or working in a field with high foliar crops and suddenly became dizzy, developed a headache, and became progressively weak. He would probably complain of sweating profusely. She may soon find that other members of the crew have developed the same symptoms and are waiting outside the clinic or have gone to another doctor.

The clue is that residue poisoning cases:

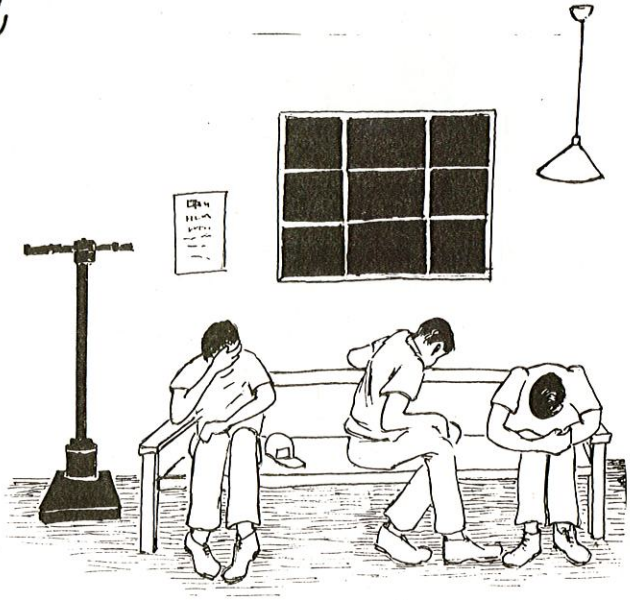
- are usually multiple
- are mild
- require only small doses of antidotes.

The following outbreak is a typical example:

An emergency room nurse had three patients with mild symptoms resembling those seen in organophosphate poisonings.

Three other patients were in the waiting room with similar symptoms, and four more were on their way to the hospital. All were weak, sweating and complaining of abdominal cramps, nausea, and vomiting. Some had difficulty with breathing. Others had diarrhea as well as vomiting. Most had miotic pupils.

By the end of the evening, 10 of a crew of 17 migrant workers who had been detasseling corn that morning had been admitted to the hospital. They received atropine intravenously and were beginning to respond after decontamination and intravenous fluids.



The epidemic was clearly due to an anticholinesterase pesticide, a fact that was later confirmed when 64 mg of a carbamate insecticide called methomyl was identified from a methylene chloride extraction from the shirt of one of the exposed victims.

At 7 a.m. they had entered a cornfield where an airplane had sprayed the field with methomyl. Their clothes and canvas shoes had become moistened from the dew on the ground and from the moisture on the leaves.

Child Poisonings

Children are the group at the greatest risk of accidental poisoning from pesticides which have not been stored or disposed of properly. Prompt action is especially important in these poisonings.

The nurse must always be on the lookout for these dire emergencies. In this type of case, the nurse is usually confronted with a *very sick* child who is semicomatose, vomiting profusely, and has diarrhea and pinpoint pupils. For example:

A 4-1/2-year-old boy who was playing in his grandfather's barn spilled some liquid over his pants at about 1 p.m. When he returned home at 6 p.m. he was not feeling well and he looked pale and listless. He was put to bed. At 9 p.m., his parents noted that he was drowsy and that his respirations were labored. His father rushed him from his home to the emergency room of a nearby hospital. His mother checked his clothes and noticed they had an insecticide smell. The grandfather checked the barn and found a bottle of ethyl parathion.

By the time the boy reached the emergency room, he was moribund, in deep coma with his eyes rolled back, and barely breathing. His respirations soon ceased and he had to be kept alive by artificial respiration and endotracheal intubation. When the mother reported that parathion had been spilled on the child, he was stripped, washed all over, and oxygenated. Atropine was administered intravenously every 10 minutes through the night, and 2-PAM was given. By 5:30 a.m. he was much improved and continued to recover over the next 2 days.

Here, the point is that exposure might not be recognized. Children get extremely ill very rapidly and treatment must be prompt and vigorous.

There is a high incidence of pica among migrant families. This is a situation where the ingestion of a pesticide might not be realized. Geophagia is not uncommon.

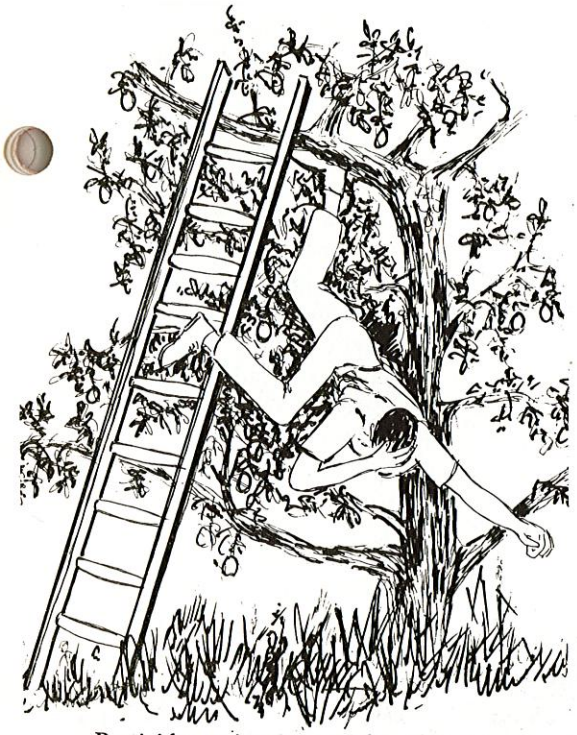
A 3-year-old child was taken to a local migrant clinic with extreme miosis, foaming at the mouth and nose, and diffuse rales throughout both lung fields. An exposure history revealed that the child had been playing in a strawberry field which had been heavily treated within the last three days with phosdrin, Kelthane, and parathion. Pesticide analyses showed total inhibition of cholinesterase. Parathion was identified in the gastric content. It was later determined that the intoxication was the result of ingestion of soil containing parathion.

This case illustrates the point that children eat dirt—which is not commonly recognized. It exemplifies the real possibility of this mechanism of poisoning when a toddler is playing in the fields where the mother is working.

Emergency Personnel

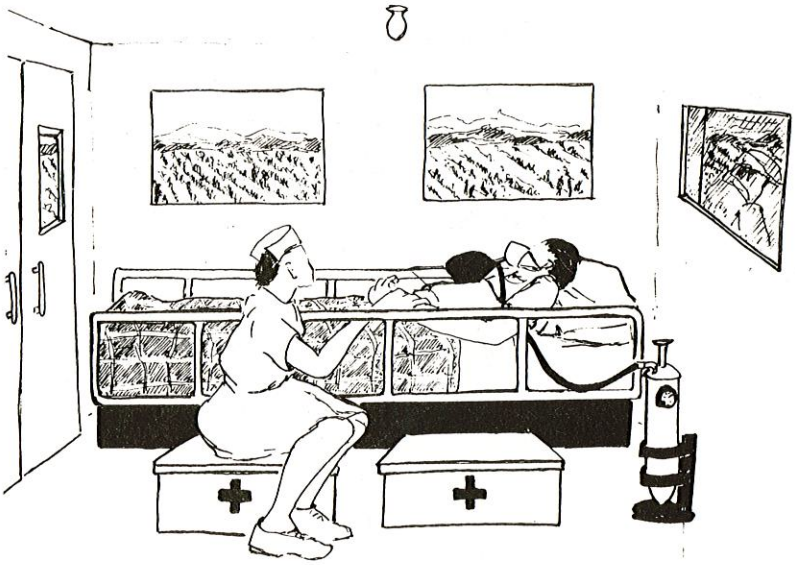
In a rural area, the emergency personnel in the ambulance can expect to encounter anticholinesterase poisonings. Recognition and early treatment are of vital importance.

A migrant worker might fall off a ladder while picking citrus which had been treated with parathion. His pesticide exposure might not be recognized while the patient is being transported to the hospital, particularly if he appears dazed. The diagnosis may be clouded by other evidence of trauma which he may have sustained during his fall. The questions which should be asked are, "Why did the worker fall?" and, "Was the fall due to pesticides?"



Pesticide poisoning might also go unrecognized in a pilot who has had an aircraft accident. The possibility of injury is likely to be of greater concern than the possibility of chemical intoxication which may have been the result of a spill before, during, or after the accident. It is essential to evaluate whether the pilot has sustained a serious pesticide exposure. If the exposure is allowed to pass unrecognized, it could cost him his life.

Emergency personnel who are transporting suspected cases to the hospital should always be in radio contact with the hospital. The patient's condition might suddenly deteriorate in transit, requiring immediate therapy. If the physician is alerted ahead of time, he can communicate with the emergency personnel to prescribe whatever is necessary.



CHAPTER III

SYSTEMIC ORGANOPHOSPHATE AND CARBAMATE POISONING

Symptoms and Signs

Symptoms and signs of systemic organophosphate and carbamate poisonings are almost entirely due to cholinergic manifestations. They include both muscarinic and nicotinic effects and are the result of acetylcholine accumulation.

Early symptoms depend on the route of absorption and the severity of the intoxication.

- Gastric symptoms appear earlier if the material has been ingested.
- Shortness of breath, salivation, and excessive bronchial secretions occur if the material has been inhaled.
- With dermal exposure, gastrointestinal and respiratory symptoms appear at the same time.
- In children, a convulsion may be the first symptom.
- In serious intoxication, both muscarinic and nicotinic symptoms and signs begin shortly after exposure.

Muscarinic effects, which usually precede nicotinic effects, include:

- anorexia
- nausea
- vomiting
- abdominal cramps
- diarrhea
- involuntary defecation and urination
- sweating
- salivation
- lacrimation
- pain in the chest
- excessive bronchial secretions
- blurring of vision due to miosis.

Nicotinic effects include:

- muscle twitching
- fasciculations
- weakness
- flaccid paralysis.

With involvement of the muscles of respiration, further respiratory failure occurs from bronchial constriction, blockage by secretions, and depression of the respiratory center.

Central nervous signs and symptoms include anxiety, restlessness, giddiness, headache, drowsiness, convulsions, and coma.

In the advanced state, the patient is pale, sweating, and frothing at the mouth. The pupils usually are miotic and non-responsive to light. Pupils will sometimes be dilated if the patient is in extremis. They will then become miotic with initial treatment.

The most important neurological findings are:

- Fasciculations—localized and generalized involuntary twitching may be elicited by tapping the muscles over the cheekbone, over the thorax, or on the arms.
- Sometimes generalized clonic seizures may be observed. The plantar reflex is extensor and electroencephalographic changes may be noted.
- Miosis—the pupil is small, usually less than 5 mm. The diameter of the pupil in millimeters should be recorded.

Metabolic signs and symptoms include the following:

- Blood sugar may be elevated at first, and glycosuria may be observed. The level of hyperglycemia is much less than levels observed with diabetic coma. Ketoacidosis is not seen.
- Serum electrolytes are usually normal, though hypokalemia may occur and be aggravated by diuretic therapy. Serum K levels should be checked early.
- Fever is not a constant finding. The patient's temperature usually is normal or subnormal, though severe dehydration may occasionally cause fever.
- Polymorphonuclear leukocytosis is common.

Clinical Diagnosis

In severe poisoning, the initial diagnosis and institution of appropriate treatment must be made on clinical grounds alone, since there is not enough time to wait for confirmatory laboratory results.

The most important factors in the clinical diagnosis of organophosphate poisoning are:

- a pesticide exposure history
- symptoms and signs typical of an anticholinesterase illness
- the presence of atropine refractoriness.

Pesticide Exposure History

The first prerequisite to diagnosis is to determine whether the patient has been exposed to any of the anticholinesterase pesticides. Symptoms begin shortly after exposure. They are seen especially early after ingestion or inhalation.

With few exceptions the patient will begin to feel ill within 15 minutes to an hour after ingestion.

If a patient has ingested a pesticide, this is usually known or admitted, except in the case of a toddler. Ingestion of a pesticide as a result of pica might not be realized.

Dermal exposure may be obvious and the patient may recall spilling the pesticide on his skin and clothing. On the other hand, he may not be aware that pesticides can be absorbed in this way. He may not mention that his shirt, pants, or shoes were wet from the pesticide or from moist residue on leaves. In arid areas, dermal exposure occurs from dry, dusty residues on the leaves of the plant and there is no sensation of wetness. In all suspected cases, however, diagnosis is materially helped if the attending health personnel are made aware that the patient has sustained a pesticide exposure. Use the exposure history form in Appendix 8.

Symptoms and Signs

Symptoms and signs compatible with cholinergic excess are the second most important variables contributing to the clinical diagnosis of anticholinesterase poisoning.

Although symptoms of cholinergic poisoning may be easily confused with those of other conditions, a pesticide cause should always be considered. Physical signs are less subject to misinterpretation. Miosis is a rare condition in the clinic setting. It should always lead to first consideration of exposure to anticholinesterase pesticides. Miosis is doubly significant if it is accompanied by nicotinic and muscarinic symptoms and muscle fasciculations. These signs, together with the general appearance of the patient, should prompt the nurse and the physician to a diagnosis of organophosphate poisoning.

Atropine Refractoriness

This is the third important clinical observation which helps substantiate the diagnosis of an anticholinesterase illness. When a physician prescribes a larger than normal dose of atropine in a person *not* exposed to anticholinesterase pesticides, the early signs of atropine toxicity soon become apparent. These signs include:

- dry mouth
- flushed skin
- increased heart rate
- dilated pupils.

If the patient has anticholinesterase poisoning, large doses of atropine are required to produce these normal reactions.

Differential Diagnosis

Mild anticholinesterase poisoning causes such symptoms as:

- headache
- fatigue
- dizziness
- blurred vision
- excessive sweating
- nausea and vomiting
- stomach cramps
- diarrhea
- salivation.

These symptoms are shared by many illnesses not related to pesticides, such as influenza, heat stroke or heat exhaustion, and gastroenteritis.

Moderately severe poisoning causes all of the symptoms found in mild poisoning, but in addition, the patient:

- is unable to walk
- often complains of chest discomfort and tightness
- exhibits marked miosis
- exhibits muscle twitching.

These symptoms might be reasonably mistaken for such conditions as pneumonia, myocardial infarction, and encephalitis.

Severe poisoning results in:

- unconsciousness
- local or generalized seizures
- the manifestation of a florid cholinergic crisis.

In these cases, several alternative causes of coma enter into the differential diagnosis.

If there is glycosuria, diabetic coma might be considered. The miosis might lead to consideration of a cerebrovascular accident, particularly a pontine hemorrhage.

Screening Tests

At all three levels of clinical severity, a screening cholinesterase test can help confirm a cholinergic poisoning diagnosis. These are colorimetric procedures using filter paper impregnated with a color reagent which is sensitive to pH change. The time taken for the color to change is a crude index of the degree of cholinesterase inhibition. The severity of inhibition is categorized as none, suspicious, or severe.

Although these tests have several limitations, they offer the most immediate confirmation which is within the laboratory expertise in any hospital or clinic. The Acholest test is the most reliable of the several available screening methods for plasma cholinesterase determinations. It can detect inhibition as low as 20 percent of plasma cholinesterase. The results correlate well with more quantitative procedures.

The test should be administered:

- to any patient who claims to have been exposed to a pesticide
- to any worker having regular and heavy pesticide exposure, such as spraying chemicals or loading aircraft (see Chapter VII–PREVENTION)
- to any patient complaining of three or more of the previously cited symptoms
- to any patient with any one of the following physical signs:
  –miosis (less than 5 mm in size)
  –muscle fasciculations
  –bronchial exudation
  –bradycardia (pulse rate of 50 or less per minute).

To prepare a blood sample for the Acholest or other qualitative screening test, collect 1 cc of blood in a *green stoppered* vacutainer tube (heparin-lined) and separate the plasma by centrifugation.

(See Appendix 3 for complete description of the test.)

No further laboratory confirmation is needed in the clinical setting.

Definitive verification and validation of the poisoning episode, however, calls for:

- quantitative measurements of the plasma and red cell cholinesterase
- specific analysis of the intact pesticide and/or urinary pesticide metabolites to help identify the specific offending pesticide.

Laboratory Diagnosis

Three types of laboratory investigations can help confirm a clinical diagnosis of cholinergic poisoning:

- cholinesterase determination
- urinary metabolite studies
- intact pesticide studies.

Cholinesterase Determination

The levels of cholinesterase in the red blood cells and in the plasma are used to confirm human poisoning. They correlate well with nervous system cholinesterase inhibition. It is believed that ChE values of 0.5 or less (Michel method) for either red blood cell or plasma represent abnormal depressions for most individuals.

The four laboratory techniques most commonly used for quantitative expressions of these enzyme activities are the electrometric (Michel), the titrimetric (pH stat), the colorimetric (Ellman) and gas chromatographic (Cranmer) methods (see Appendix 4).

In two instances, laboratory tests may not show low levels of cholinesterase enzymes, even though cholinergic poisoning is present. These are:

- overexposure to carbamate pesticides–With this group of chemicals, cholinesterase reactivation is rapid. In vitro reactivation often occurs before the blood reaches the laboratory. Normal red cell and plasma levels may be reported even in the presence of obvious cholinergic illness.
- red cell cholinesterase determinations made after the administration of 2-PAM–if this oxime is given early in the case, red cell cholinesterase reactivates rapidly, even in the presence of continued cholinergic symptoms.

Low plasma cholinesterase levels may sometimes be due to other causes including:

- liver diseases, malnutrition, hyperpyrexia, myocardial infarction, dermatomyositis
- after certain drugs
- as a result of genetically determined low plasma cholinesterase. This condition may lead to respiratory arrest after being anesthetized with succinylcholine. The defect occurs in about 3 percent of the population. Recognition of this defect is primarily of medico-legal importance. Special laboratory procedures permit differentiation of pesticide exposure from this phenotypic mechanism.

Low red blood cell cholinesterase is found:

- in paroxysmal hemoglobinuria
- in the newborn after complicated delivery
- with disseminated sclerosis

Urinary Metabolite Studies

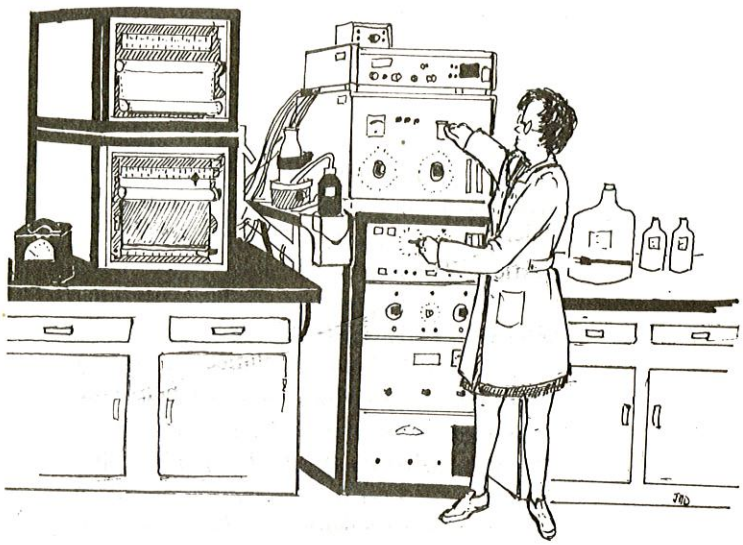
These breakdown products are excellent measures of exposure. Qualitatively they often provide valuable information on the exact type of pesticide which has caused the illness. Quantitatively, their concentrations in urine can be used as a measure of:

- the severity of the poisoning
- its probable duration.

The analysis of these breakdown products, therefore, is the second laboratory technique which will assist in the confirmation of the illness. (See Appendix 4)

Intact Pesticide Studies

The identification of the intact pesticide is the third way in which the laboratory can confirm an anticholinesterase pesticide poisoning. In the case of ingestion of the material, the intact pesticide can be identified by gas chromatographic studies of the gastric contents. In severe exposures, the intact pesticides may even be identified in the blood and other tissues. (See Appendix 4)



Collecting Specimens

Confirmatory tests must be done by a special pesticide analytical laboratory. When confirmatory tests are required, specimens should be handled in specific ways:

Blood–If the Acholest test is positive, collect an additional 8 cc of blood for definitive red blood cell and plasma cholinesterase determination. A 10 ml *green stoppered* vacutainer tube (heparin-lined) is the best method of collection. Invert the tube gently once or twice to insure proper mixing. Take care to avoid hemolysis. Label the tube to show:

- the name of the patient
- the time and date of collection
- whether a sample was obtained before or after 2-PAM administration.

Cover the label with transparent tape.

Refrigerate (do not freeze) the tube before transferring it to the laboratory. If the specimen must be sent to another laboratory, it should be airshipped. Pack it with crushed ice in a styrofoam container.

Urine and other tissues–In cases of suspected organophosphate and carbamate poisonings, collect 20 ml of urine as soon as possible. Place the urine in a hexane-washed glass bottle with an aluminum foil lined metal screw top. The label should contain:

- patient identification
- the time and date of voiding.

Cover the label with transparent tape and close the lid securely. Freeze the bottle and its contents and airship it with the blood as soon as possible.

In special circumstances, other tissues may be collected for laboratory analysis. Handle them the same way as urine.

Gastric contents–Gastric washings should be labeled and frozen before sending to the laboratory. Collect the first washing for toxicological studies.

Treatment

The five basic steps in emergency treatment are:

- airway clearance
- oxygenation
- antidotal therapy
- decontamination, including gastric lavage
- collection of appropriate biological materials.

Time is of the utmost importance. The prompt action required in a serious intoxication, particularly with a child, is similar to that required by a patient with ventricular fibrillation with cardiac arrest.

What a nurse may or may not do in such circumstances depends on:

- her previous training and experience in the necessary resuscitative procedures
- whether there is a written protocol.

In both the rural health clinic and the emergency room, standing orders should be drawn up ahead of time to cover pesticide emergency situations. The nurse and physician should agree on procedures involved in all five of the steps of emergency treatment.

Airway Clearance and Oxygenation

Remove dentures and use a finger or, preferably, suction to clean mucus and debris from the mouth and pharynx. Introduce an oropharyngeal or nasopharyngeal airway, and administer 50 percent oxygen by mask or nasal catheter. Draw serial blood gases to monitor respiratory and metabolic dynamics.

Antidotal Therapy

Atropine—Atropine sulphate is lifesaving and should be given as soon as possible. It should not be withheld while efforts are being made to overcome any respiratory embarrassment.

The nurse should be instructed by protocol to administer atropine when the patient is first seen. Under no circumstances should such a patient be transferred to the emergency room without having had atropine therapy.

For an adult, the physician will order 2 to 4 mg of atropine sulphate intramuscularly or intravenously every 10 minutes during the early phase of treatment. Doses for children should be proportionate to weight—0.05 mg per kg of body weight.

The pulse rate, pupil size, and amount of bronchial exudate are important variables which influence the frequency of atropine administration. Large doses may be required. In a severe poisoning case, a man was unconscious for 14 days and required atropine continuously for 18 days. The therapeutic goals are to reach and maintain atropinization during the period of poisoning. Dilation of the pupils and a pulse rate of 140 per minute are the indications that atropinization has been reached.

With certain types of organophosphate pesticides, the intoxication period may be prolonged. The signs of cholinesterase inhibition reappear as the effects of atropine wear off. These periods may be followed by periods of atropine excesses with the pulse rate exceeding 140/min and the pupils becoming fully dilated. It is thus very important for the nurse to continuously monitor:

- the pulse rate
- the degree of bronchial secretions
- rate of respiration
- changes in pupil size.

This information helps the physician decide when it is necessary to readminister atropine or when there are signs of atropine toxicity. As recovery occurs, the intervals between atropine administration get longer until there is no further need to continue this treatment. When no further treatment is necessary, the patient should be observed in the hospital for another 24 hours.

Even if the poisoning appears mild and atropinization is reached after only a single dose, observe the patient for 24 hours. The atropine may have produced only temporary relief of symptoms in what may prove to be a serious case of poisoning.

Oxime—The only oxime available in the United States is N-Methyl 2 formyl-pyridinium oxime, used as the chloride (2 PAM-Cl) or Protopam Chloride. This should be available in any health clinic or emergency room likely to have to treat cholinergic poisonings. Give 2-PAM as early as possible and always in conjunction with atropine. The two drugs are complementary in their action.

The oximes are not recommended for use in cases of carbamate poisoning—in fact, they are contraindicated—but more research is needed in this area. Therefore, the physician faces a dilemma when it is not known whether the poisoning has been caused by an organophosphate or a carbamate. Make every possible effort immediately to find out the specific pesticide involved.

The oximes are not active against all of the organophosphates. Depending upon the particular organophosphate, "aging" of the phosphorylated enzyme occurs, at which time the inhibited enzyme can no longer be reactivated by 2-PAM.

With parathion, aging does not occur for 2 days after exposure, so 2-PAM may be used up to that time. With malathion, aging is early. Effects of oxime therapy in this intoxication are generally disappointing.

The usual adult dose is 1 gm intravenously, preferably as an infusion in 250 ml of saline given over 30 minutes. If this is not practicable, give it in not less than 2 minutes. A second dose of 1 gm can be given in 1 hour. In children, 20 to 50 mg per kg is given intravenously in 250 ml of saline over 30 minutes.

If convulsions are troublesome, trimethadione or thiopental may be used. Respiratory embarrassment is usually due to excessive bronchial secretions rather than pulmonary edema. For this reason, opiates, aminophylline, reserpine, phenothiazine, tranquilizers, succinylcholine, and furosemide are contraindicated.

Decontamination

The first step should be to remove the patient from further exposure. Bring him out of the exposure area and try to limit further absorption of the pesticide.

The attendant should strip the patient and place all clothing in a plastic bag. The patient must be thoroughly washed. If he is conscious, place him in a shower and wash him all over with large amounts of soap and water. Be sure to rinse the hair thoroughly and remove any residue from under the nails.

Decontamination also includes the removal of the ingested pesticide. Vomiting should not be induced:

- in stuporous or unconscious patients, or
- if petroleum distillates are part of the pesticide formulation.

In these instances, or if there is doubt about what pesticide is involved, gastric lavage is preferable to the use of an emetic. Place the patient head down and on his side to avoid aspiration of vomitus. Wash out the stomach with large amounts of water.

The use of an esophageal obturator is the best way to avoid aspiration of stomach contents, if the nurse has had previous training in inserting the obturator.

Collection of Biological Materials

(See instructions in previous section, "Laboratory Diagnosis.")

Carbamate Poisoning

Carbamate pesticides, like the organophosphates, are powerful cholinesterase inhibitors. Special considerations in the diagnosis and treatment of carbamate poisonings include the following:

- Cholinesterase reactivates rapidly after carbamate poisoning. Laboratory cholinesterase determination tests may be misleading.
- Since blood tests may not be reliable, identification of the nonhalogenated phenols—the urinary metabolites of carbamates—becomes more significant.
- The oxime 2-PAM should not be used to treat carbamate intoxications.

Interprofessional Communication

A systemic pesticide poisoning involves a wide variety of health professionals. Good communication among these professionals is essential.

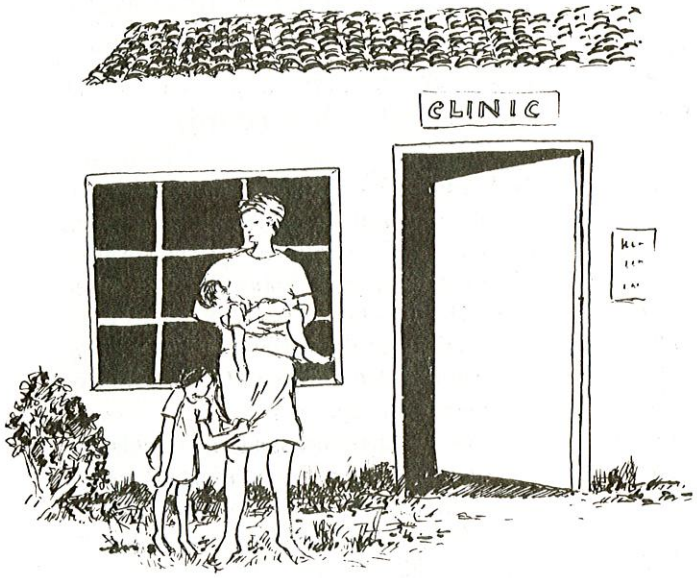
If the clinic nurse is the first point of patient contact, she should contact the attending physician as soon as possible, as well as the patient's next of kin and his employer.

If the physician is not immediately available, the nurse will have to communicate with an ambulance service and the nearest hospital emergency room to arrange for the transfer of the patient. Meanwhile, she must begin emergency treatment to stabilize the patient's condition.

If there is an epidemic of poisonings, the

health department must be informed. These are usually "picker poisoning" incidents. If there is a massive chemical spill with a potential community hazard, the nurse should communicate with CHEMTREC (See Appendix 5).

If a sick child has been brought in from the home and the source of the pesticide has not been identified or removed, other children in the home may be at immediate risk of poisoning. The police should be contacted. The nurse should also contact the regional pesticide analytical laboratory for poisoning verification.



If the patient comes straight from the field or the home to the hospital, the emergency room nurse is primarily concerned with reporting significant changes in her patient's condition to the attending physician. She should contact the immediate family, employer, police, or health department if this has not already been done.

EMT personnel should be in constant radio contact with the emergency room while transporting the poisoned patient to the hospital. If the patient suddenly collapses, the attendant can apply the appropriate treatment while in transit.

The public health nurse or the community health worker will encounter special risk factors when investigating the scene of the accident. These must be reported to the employer or landowner and local health and environmental resources so that further poisonings can be prevented.

The implementation of these communication needs are best insured by working through a checklist of persons who should be contacted. This is shown in Appendix 6.

CHAPTER IV
MISCELLANEOUS POISONINGS

Organochlorine Insecticides

These pesticides:

- are soluble in fat
- build up in the human body
- are persistent (break down slowly and remain unchanged in the environment for a long time)
- are powerful nervous system stimulators. Organochlorine poisoning causes excitation. Convulsions are the most important symptom.

Applicator poisonings and residue intoxication are not common with these pesticides, since most of their uses are prohibited. Most systemic poisonings are caused by accidental oral ingestion of endrin.

Acute poisonings may be caused by the organochlorine pesticides which are no longer sold but still remain in some homes.

A 2-year-old boy was brought to the emergency room of a local hospital. On arrival, he had a convulsive seizure. The parents reported that the child had ingested an unknown amount of an insecticide. The child was hyperactive, ataxic, and unsteady. He fell down when he tried to walk. Pupils were 2 to 3 mm in size. There was no vomiting, sweating, or increased bronchial secretions. The chest was clear. He was given diphenylhydantoin and a large intravenous dose of phenobarbital. He was admitted to the Pediatric Intensive Care Unit for observation. Both heparinized and whole blood were collected. By the Michel method the RBC ChE was 0.69 Δph/hr, and the plasma ChE was 0.90 Δph/hr which are normal enzyme levels with this method. Blood tests for intact pesticide, however, identified dieldrin in a concentration of 407 ppb. This confirmed an organochlorine pesticide poisoning due to dieldrin.

Nursing care for organochlorine poisoning is the same as that for other convulsive disorders. There are no specific antidotes. Sodium phenobarbital and diphenylhydantoin are the drugs most often used to control the convulsive seizures. Trimethadione or thiopental may also be used.

Diagnosis can be confirmed by identifying the intact pesticide in the serum. In a case of suspected organochlorine pesticide poisoning, draw 10 cc into a *red stoppered* vacutainer tube. Label the sample and allow it to clot. Separate the serum and freeze it. If the specimen must be sent to another laboratory, it should be airshipped. Pack it with crushed ice in a styrofoam container.

Dieldrin seldom causes convulsions when blood levels are less than 200 ppb. With blood levels greater than 200 ppb, convulsive seizures may be seen. The worker should be taken off his work with levels of this magnitude.

Bipyridyls

Paraquat is more toxic than diquat and produces proliferative changes in the lung, cornea, lens, nasal mucosa, skin, and fingernails. Diquat affects the lens and gastrointestinal mucosa. It does not produce the lung changes characteristic of paraquat.

Except for eye lesion, illness due to occupational exposure is usually mild and is the result of topical exposure. Epistaxis occurs in workers following droplet inhalation. Conjunctival changes occur with accidental spills.

The clinical picture following accidental or suicidal ingestion is very different. Paraquat ingestions are frequently fatal. Their management is unsatisfactory and largely symptomatic. Three clinical stages follow ingestion of as little as an ounce of paraquat:

- The first is a gastrointestinal phase with burning in the mouth and throat, nausea, vomiting, and abdominal pain with diarrhea.
- Several days after exposure, signs of hepatic and renal toxicity appear. These are due to central zone necrosis in the liver and acute tubular necrosis of the kidney.
- Ten to 20 days after ingestion, progressive proliferative changes develop in the lungs. Hyperplastic changes in the terminal bronchioles occur with alveolar fibroblastic proliferation. Loss of lung surfactant has been demon-

strated. Within a few days, death from respiratory failure occurs.

Urine studies have indicated that 90 percent of the ingested paraquat is excreted in the first 24 hours. Delayed pulmonary effects appear to be the result of an irreversible process that develops long after the initial stimuli has gone.

Paraquat is poorly absorbed from the gut. Excretion data suggest that only 1 to 5 percent of the ingested material is absorbed in man. Maximal blood concentrations are reached within 4 to 6 hours after ingestion. Treatment, therefore, is primarily concerned with:

- decreasing the amount of paraquat absorbed
- perfusion of the circulating blood through charcoal columns.

Steps to decrease the amount of the paraquat absorbed include:

- gastric lavage with every precaution to avoid aspiration of gastric contents
- repeated administration of large amounts of adsorbents together with the administration of purgatives.

The ability of Bentonite, Fuller's earth and other clays to absorb bipyridyls has been studied. Fuller's earth was more effective than Bentonite. 500 ml. of a 30 percent suspension of Fuller's earth together with 5 percent magnesium sulphate should be administered after lavage.

Perfusion of the blood through charcoal columns has been advocated.[2] In addition to hemoperfusion, forced diuresis with Mannitol, hemodialysis, and corticosteroid and immunosuppresant therapy have also been recommended.[3]

Although steroids and alkaloids are given for pulmonary complications, no treatment has shown to be effective at this stage of the intoxication.

There is a simple urine test for paraquat that can provide presumptive evidence of paraquat poisonings in suspected cases exhibiting early symptomatology.[4]

[2]Smith, L.L., Wright, A., Wyatt, I., Rose, M.S. Brit. Med. J. 1974, (4):569.
[3]The Lancet Editorial, 1976, (1):1057.
[4]Goulding, R., Volans, G.N., Crome, P., Widdop, B. Brit. Med. J. 1976, (1):42.

Rodenticides

These are not particularly hazardous, but they are widely used. Children often accidentally ingest rodent tablets or baits—an event which leads to much anxiety, questions, and the need for reassurance.

Fumigants

Fumigants can present serious health hazards resulting in human illnesses and, occasionally, death. Methyl bromide, acrylonitrile, calcium cyanide, and carbon tetrachloride are the fumigants most likely to cause death if overexposure occurs. Others which can cause skin and eye injury and systemic illness include:

- sulfuryl fluoride (Vikane)
- 1,3 dichloropropene (Telone)
- 1,2 dibromo 3 chloropropane (Nemagon)
- ethylene dibromide
- formaldehyde
- chloropicrin
- phosphine
- sodium methyl dithiocarbamate (Vapam).

Methyl bromide is sold as a liquid under pressure. At atmospheric pressure, it vaporizes at 40°F to a colorless and odorless gas. Because of this property, methyl bromide should always be used in formulations which contain chloropicrin (tear gas), which serves as a warning agent. A worker who is exposed to enough of this mixture to cause tearing has also been exposed to dangerous quantities of methyl bromide.

Methyl bromide is absorbed through the lungs, skin, and mucous membranes. It can cause:

- acute poisoning, either topical or systemic, and
- chronic effects.

Acute Poisoning

Topical effects—Skin contact with the liquid or high concentrations of the vapor produces itching and prickling of the skin. This is followed by reddening and formation of vesicles and slow-healing blisters. Getting the liquid in the eyes may cause corneal ulceration.

Systemic poisoning—Symptoms usually develop 3 to 12 hours after inhalation of the vapor. Early symptoms include nausea, vomiting, dizziness, headache, blurring of

vision, and changing taste of food. These are followed by listlessness, weakness, staggering gait, and slurring of speech. In addition, the patient may complain of double vision and even temporary blindness.

In severe poisoning, the victim becomes comatose and has a high fever and respiratory embarrassment. Death is usually the result of either respiratory failure or cardiovascular collapse. Death is preceded by cyanosis, pulmonary edema, and renal failure. Muscle twitching and convulsion are not uncommon.

Chronic Effects

A papular pustular rash, not unlike acne, may develop on the face, arms, back, and chest. This is the result of repeated dermal exposures. All the symptoms and signs listed under acute effects may also appear as a result of chronic exposure. Fatigability and loss of appetite are frequent complaints. More severe chronic manifestations include a change of personality, a chronic central nervous system effect which may persist for years. Visual disturbances and locomotor impairment are common.

Treatment

First, quickly get the patient out of the contaminated atmosphere and remove all contaminated clothing. Methyl bromide can penetrate rubber gloves. Wash skin burns carefully with water. Administer a therapeutic trial with dimercaprol (BAL). If there is severe respiratory depression, give oxygen under positive pressure. Artificial respiration may be necessary. Keep the patient under observation for at least 48 hours after symptoms have subsided.

No simple laboratory tests are available for confirmation, but blood levels of bromine correlate well with the severity of the exposure.

Prevention

Methyl bromide must be applied by a closed-delivery system. All State and local requirements concerning the use of plastic sheets or tarpaulins must be followed. Guards and warning signs should be posted. The application should be done at a safe distance from inhabited structures, and under appropriate weather conditions. Animals and humans must be removed from the area to be treated.

Methyl bromide must be kept under lock except when the applicator or other responsible persons are present. The material should be stored in a cool, dry, well-ventilated building in order to avoid an explosion hazard and the possible buildup of toxic concentrations of vapors caused by leaking containers. The storage sites should be at a safe distance from populated areas and inhabited buildings.

Dinitrophenol and Pentachlorophenol

These materials are used as insecticide sprays, fungicides, and wood preservatives. They are rapidly absorbed by the gastrointestinal and respiratory tracts and the skin. They are profound stimulators, stimulating all the cells of the body by blocking oxidative phosphorylation. Body fat is the major, if not exclusive, fuel for this extra metabolism.[5] The body temperature becomes elevated and the breathing and heart rate increase rapidly. Because respiratory and cardiac stimulation do not accelerate in proportion to the increased metabolism, anoxia and acidosis develop rapidly.

Acute and Subacute Poisoning

The patient complains of marked fatigability, excessive thirst, and profuse sweating. His face is flushed. These are the result of the higher metabolic state, as is the exceptionally high fever, which may reach 110°F. The higher the fever, the more serious is the intoxication.

In such cases, tachycardia, hyperpnea, cyanosis, and muscle cramps will occur. Death, which is usually the result of respiratory or circulatory collapse, occurs within 24 hours.

In mild or subacute cases, most workers will complain of lassitude, headache, and malaise. Some, however, may have an alarming sense of excessive energy, drive, and hyperactivity. They should be warned of the dangers of overheating, because the metabolic activities of these compounds are exaggerated by heat.

[5] Shils, M.E. and Goldwater, L.J. Effect of diet on the susceptibility of the rat to poisoning by 2,4-dinitrotoluene. Arch Environ Health 8:262, 1953.

Treatment

The essential goals of treatment are:

- prompt elimination of the material and curtailment of all possible further sources of exposure,
- symptomatic treatment designed to control the high fever and its secondary consequences, such as anoxia, dehydration, and acidosis.

Gastric lavage with large amounts of sodium bicarbonate solution should be followed by saline catharsis using 15 to 30 grams of sodium or magnesium sulphate in water. To control the fever, use cold packs and alcohol sponges. Cold water enemas may be needed.

Supportive measures include:

- intravenous fluids to control dehydration and acidosis,
- oxygen and artificial respiration as required.

Following the acute phase of the intoxication, liver and renal complications may develop. These are the result of the toxic action of these materials on the renal tubules and on the liver cells.

Laboratory Confirmation

Laboratory confirmation of the intoxication is provided by the detection of high levels of dinitrophenols or pentachlorophenol in the urine and blood of the victim.

CHAPTER V
TOPICAL EFFECTS

Skin Problems Among Agricultural Workers

Skin problems accounted for 62 percent of all occupational diseases reported in the United States in 1973. The agricultural worker who is exposed to pesticides is four times more likely to develop a skin rash than the average industrial worker.

Diagnosis

Because the agricultural worker is exposed to a wide variety of agents besides pesticides, determining the cause of dermatitis is highly complex.

Dermatitis from pesticides can result from:

- exposure to primary irritants, or
- contact with contact sensitizers (allergens).

The first diagnostic consideration is to differentiate between these two types of skin rash.

Primary Irritants—Primary irritants are either absolute or relative.

Absolute irritants are usually chemicals which can cause a chemical burn or severe irritation on almost anyone's skin. The reaction occurs immediately or within an hour or so. It usually does not present a diagnostic problem.

Relative irritants are agents which can cause varying degrees of dermatitis (inflammation of the skin) according to environmental conditions. Some, like kerosene or turpentine, are more likely to cause problems on sweating skin, or under occlusive clothing and boots. All are more damaging to skin which is already abnormal (sunburn, eczema, and atopic dermatitis).

Some areas of the body are more susceptible than others. The genitalia, scrotum, and eyelids are particularly vulnerable. Thus, a worker might have dermatitis on the penis and eyelids due to contamination by materials on the hands.

The primary irritants usually produce a short term dermatitis which goes away and can be related to a known definite exposure.

The rash caused by primary irritants is more likely to be confined to the areas of the skin actually exposed to the chemical. Irritants in solution often are confined to the hands and the forearms, but relative irritants may be absorbed in clothing and boots. The rash will appear where the clothing is in closest contact with the skin—buttocks, knees, and dorsum of the feet.

Irritants dispersed in sprays or aerosols more often affect the face and neck. Powders tend to accumulate at the waistline, the collar, and tops of boots.

A primary irritant is likely to be the cause if several workers experience a rash on exposed surfaces at the same time, especially if burning or itching occurs soon after exposure. It is useful to ask the patient if he knows whether any other workers in the same area have the same problem.

When multiple cases of dermatitis follow the application of an agricultural chemical, the material or its carrier is clearly too irritating for continued use. The nurse should notify the physician as soon as possible.

Treatment—When the person is removed from further exposure, this type of dermatitis usually clears up, especially if a topical steroid cream is applied to the affected areas.

Contact Sensitizers—Substances which cause allergic contact dermatitis may affect only a few individuals who have become "sensitized" or "allergic" to the material. Even then, there may be marked differences between individuals in the degree of cutaneous reaction or severity of the clinical dermatitis.

A new product occasionally has a high potential for producing allergic skin conditions. These usually are soon recognized and taken off the market, but it is still worthwhile to ask whether a new product has been used. First reports of a hazardous substance often come from nurses or other medical personnel who are closest to actual field conditions.

Depending on the patient's sensitization, the reaction may occur within a few hours of contact to as long as a week. Most occur within 48 hours. Redness, itching, swelling (especially around the eyes), and "water blisters" are the clues to this type of dermatitis.

Having a basic knowledge of the principal sensitizers, reading the ingredients on the labels, or checking the Physicians' Desk Reference will often help in diagnosis. Plant dermatitis is common and must be considered as a cause of allergic contact dermatitis.

Some chemicals may be both primary irritants and contact sensitizers. In addition, many agricultural compounds are dissolved in solvents such as kerosene or xylene. This creates a perfect mechanism for both primary skin damage and an allergic reaction.

Treatment—The treatment of allergic contact dermatitis includes:

- using cool compresses
- treating infections
- identifying the offending agent.

It may be necessary to refer the patient to a facility which performs patch testing.

Topical steroid creams, gels, or lotions are beneficial. Severe or extensive cases may require a short course of systemic steroids. Systemic steroid therapy should always be under the direction of a physician. It should never be given to patients who may have undetected tuberculosis or are at risk of tuberculosis.

Apart from the highly specialized area of patch testing, the laboratory has no place in the verification of topical skin effects from pesticides.

The illnesses are often a sensitivity phenomenon and are therefore not strictly dose related. Cholinesterase determinations and urinary metabolite studies are not necessary.

Effects of Pesticides on the Eyes

Eye injuries are common in agricultural workers. Topical effects can occur as the result of exposure to any of the pesticides in common use today. In addition, xylene and petroleum distillates in common use as pesticide carriers are very irritating materials. They produce a severe inflammatory response when they get into the eyes.

Eye injuries can result from:

- accidentally splashing or spilling the material into the eye
- exposure to pesticide drift
- rubbing the eyes with contaminated hands.

Sulphur, paraquat, Omite, parathion, and dieldrin are some of the most common causes of eye injuries.

Eye injuries are most common in pesticide mixers, loaders, and applicators because of their risk of exposure to the pesticide concentrate.

Damage is the result of:

- a direct irritating effect of the chemical or the vehicle
- an allergic reaction
- direct pharmacologic action on the eye, as is the case with anticholinesterase pesticides.

Conjunctivitis, corneal ulceration, uveitis and lenticular opacities are some of the lesions which occur. These chemicals have a delayed effect on visual accommodation and diminish the peripheral fields of vision.

Treatment

If there is a conjunctival infection, irrigate the eyes from the inside to the outside with large amounts of water or sterile normal saline solution. After the eyes have been thoroughly irrigated, evert first the upper and then the lower lid and clean them with a moist cotton tip to remove any debris. Then irrigate the eyes once more. Apply an eye shield and make an appointment for the patient to see the attending physician for definitive diagnosis and treatment.

CHAPTER VI
PESTICIDE EPIDEMIOLOGY

The possible effects of pesticide exposure can be divided into three categories:

- acute exposure, which produces acute poisoning and topical injuries
- chronic high exposure, which is concerned with long term effects of pesticides
- chronic low exposure or incidental exposure, which is concerned with human pesticide residue and the public's concern for the possible risks of carcinogenesis.

Those at risk in the first category are:

- pesticide workers
- members of the general public who become accidentally poisoned in the home or garden.

In the second category is the occupationally exposed worker. The third category includes the general public, who are exposed to small amounts of pesticides in water, air, food, and clothing.

The nurse is primarily concerned with the first and second categories. She is most likely to encounter acute pesticide poisoning in the clinic and hospital. She has an important role in preventing both acute and unnecessary chronic exposure to the workers in the second category.

As an individual she is probably also concerned with the third level of exposure for her own and her community's sake.

Acute Poisonings

Acute pesticide poisonings are a serious health problem in many areas of the world. The World Health Organization estimates that there are approximately 500,000 cases annually, with about a 1 percent fatality rate.

No accurate statistics exist for the United States as a whole, but reports from selected populations suggest the size of the problem.

In California in 1975, there were 503 systemic pesticide poisonings in persons employed in agriculture. Some authors, however, believe that these reported statistics represent no more than 1 percent of the number.

Incidence data is equally incomplete in Florida, a state second only to California in the amount of pesticides used. Between 1970 and 1975, pesticides were listed as a

PESTICIDE EXPOSURES, HEALTH CONCERNS AND NURSING GOALS

Pesticide Dose	Types of Exposure	Health Concerns	Nursing Goals
High	Acute Overexposure	Pesticide Poisonings Topical Injuries (Skin and Eyes)	Prevention Early Diagnosis
Intermediate	Occupational	Occupational Safety	Industrial Hygiene Occupational Health Health Education Surveillance Safety Standards
Low	Incidental	Human and Environmental Pesticide Residues	Monitoring

cause of death in 26 persons in Florida.

- Ten deaths (35 percent) were children under the age of 10.
- Males accounted for 19 cases (73 percent).
- Fifteen ingested the toxicants (57 percent).
- Three inhaled the agent (11 percent).
- Two persons died from dermal contact.

These statistics, however, tell nothing of the occupational hazards of pesticides or why these materials were in the home. The total picture is still incomplete. Information on the acute morbidity of pesticides is of extreme importance, for it is only with this knowledge that decisions can be made on future pesticide management, policies, and regulations.

Applicator and Picker Poisoning

These two syndromes have distinct clinical, epidemiologic, and public health features.

Clinically, because residues are much less toxic than pesticide concentrates, picker poisoning is of shorter duration. Cases are often multiple and the case fatality rate is low.

Illness which is the result of exposure to the concentrate is usually more severe and protracted. The case fatality rate is high.

Epidemiologically, too, there are differences. Residue intoxications occur primarily in the agricultural worker and the migrant worker through dermal exposure to the foliar residues. In this situation, the worker is not aware of the potential hazard, because he does not know that the residues are there. Occasionally, exposure may occur when workers are accidentally sprayed or come in contact with pesticide drift.

The applicator, on the other hand, should be aware of the potential hazard of the chemical which he is applying or mixing. Such persons should be acquainted with the hazards of spills and should have been taught to wear protective clothing. They are generally more informed and better trained.

Applicator poisonings occur wherever application is done. Residue poisonings occur mainly in hot, arid areas.

Preventive strategies also differ. With residue poisoning the preventive goal is to make the place of work safe. The federal government has established reentry times for different pesticides. These are the time intervals which must elapse after a crop has been treated before it is safe for an agricultural worker to enter the field. The preventive approach is very different for applicators. It focuses on the preventive potential of worker education, personal hygiene, and the wearing of protective clothing. These are skills for which the nurse is ideally suited.

Child Poisoning

Children are the group at greatest risk of accidental poisoning from pesticides which have not been securely stored or correctly

disposed of, although adults, too, are at risk if chemicals are stored in improper bottles. The home, the garden, and the local refuse dump are often the scene for the other category of acute accidental pesticide poisoning. The toddler is the person at greatest risk.

When water is added to a pesticide, the liquid often becomes milky. If this is stored in a bottle, there is a real danger that the poison will be inadvertently given to a baby.

Incorrect disposal of pesticide containers causes many fatal cases.

A 2½-year-old Mexican child died after being sprayed with what seemed to be a harmless fluid in a simple spray gun. Her father, who could speak no English, could not understand the label on the container which he brought home from the field where he had been working. There were remnants of the pesticide concentrate at the bottom of the drum. He knew that the liquid was effective against the pests in the fields, so he told his 14-year-old daughter to spray the home because of heavy roach infestation. She did this, but tragically also sprayed her younger sister who was asleep in the crib.

Transportation and storage also can be hazardous. If there is a large spill, such as might occur with a tanker collision or train crash, a highly dangerous situation can arise. In this event, a telephone call (collect) can be made to CHEMTREC, which is a special service to assist in such cases. A description of CHEMTREC service is in Appendix 5.

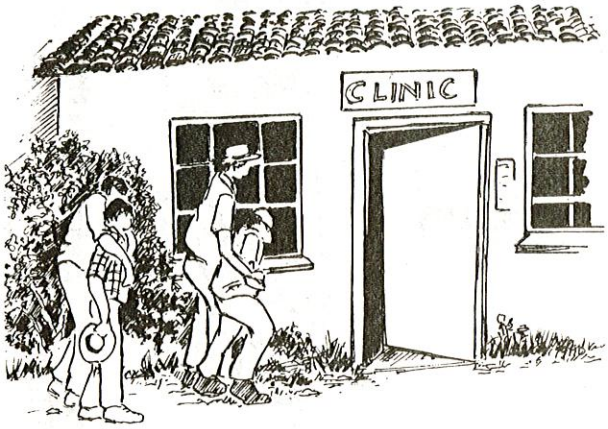
Careless unloading of pesticides can cause leaking containers. If these are stored in warehouses close to food, accidental contamination of food may occur. This is especially true during storms and floods. The general upkeep and waste disposal practices of the pesticide storehouse should be inspected regularly.

It is apparent that pesticide poisonings are a problem area for several agencies and institutions. A cooperative effort is needed to insure human safety through improved pesticide management.

CHAPTER VII
METHODS OF PREVENTION

The rural health clinic nurse and the public health nurse are in key positions to prevent pesticide poisoning.

These nurses are often the first to learn about a case. They are usually the first contact of the patient and his family. This gives them the opportunity to follow up the case.



The nurse can go to the site of the poisoning to try to retrace how the episode came about. She can visit the home to discuss safety and explain to the worker and his family how poisonings happen. Since other members of the camp will be interested, the nurse can use the incident as a teaching point.

She should also meet with crew chiefs and employers to promote pesticide safety. The nurse can meet with workers, employers, and even local government groups, civic clubs, and labor groups to explain how the occupational health hazards of the worker can be minimized with sound pesticide management. Her visibility at the place of work and in the home will facilitate her acceptance greatly. It also will allow her to see for herself the whole chain of pesticide formulation, application, and exposure.

Applicator Safety

The nurse should check for the correct use of protective clothing. She should find out whether goggles, masks, coveralls, rubber gloves and boots are provided and if they are in good repair. She should also watch for skin conditions in the workers. When abrasions in the skin occur through cuts or dermatitis, the skin loses its natural protective barrier and chemical absorption is greatly increased.

She should discuss with the operator such issues as the availability to the worker of showers at the end of the day and the opportunities for a wash and change of clothing.

She also should discuss procedures for rinsing and disposing of empty pesticide containers. Too often, they are left in the fields or are picked up and dumped into an empty lot or garbage pit, where they continue to present a poisoning potential.

She should make sure the employer knows the name of the nearest physician and hospital. She might leave her own card and tell him about the pesticide poisoning program at the clinic.

Routine cholinesterase testing is necessary for highly exposed workers such as formulators, spray rig operators, pilots, and aircraft mixers and loaders. The nurse and the employer should explore ways in which a program can be organized. Pre-employ-

ment cholinesterase determination can be built into the pre-employment physical examination.

It is customary to advise the withdrawal of the worker from continued anticholinesterase exposure if there is a 20 percent decline in these enzymes.

If a baseline level is not available, the worker should be temporarily changed to a job away from anticholinesterase pesticides if the red blood cell cholinesterase is less than 0.4 Δph/hr (Michel method).

Picker Safety

In the education of the agricultural laborer whose pesticide risk is from plant residues, field reentry regulations are most important. In addition, the nurse can help the migrant worker understand the need for:

- changing clothing
- showering after work
- laundering the clothes correctly
- keeping contaminated clothes away from living areas
- washing hands and face before smoking or eating while working in treated fields.

Parathion has been identified in the house dust of workers' homes. This is probably due to the dusty residues dropping off the workers' clothing or shoes.

One worker was hospitalized with parathion poisoning. When he went back to work a few days later, he became sick a second time after putting the same clothes on again. High concentrations of parathion were detected in the clothing.

The nurse can discuss laundering with the worker's wife. The worker's clothing should be laundered separately and subjected to several washes. Laundering of contaminated fabric three times is not effective in removing all of the residues of some pesticides (such as parathion and toxaphene).

Child Safety

The nurse should inform families about correct storage of chemicals in the home. She also should warn them about taking the child into the fields. Often it is impossible to leave the child at home, because there are no day nurseries nearby. If one is available and the mother agrees to use it, the risk that occurs from a child playing in the field while her father and mother are working would be removed.

One 5-year-old girl, for example, was hospitalized for organophosphate poisoning which she sustained while playing in the fields where her parents worked. She was poisoned as a result of eating tomatoes which had been treated earlier with phosdrin, methomyl, dimethioate and monocrotophos. The mother recognized the condition because five of her eight children had been hospitalized for parathion poisoning several years earlier.

CHAPTER VIII

ACUTE PESTICIDE POISONING VERIFICATION

Verification of acute pesticide poisonings has two aspects:

- the confirmation of diagnosis, using clinical and laboratory procedures, and
- collecting and tabulating data on confirmed pesticide poisonings to gain a fuller understanding of the problem.

Diagnosing a full-blown organophosphate illness usually poses no real problem. It appears, however, that many milder intoxications of agricultural laborers and migrant workers pass unrecognized or are not reported or satisfactorily investigated in the field. Even in the more obvious cases, laboratory confirmation is insufficiently practiced. The specific pesticide involved is rarely identified.

The verification and chemical confirmation of an acute pesticide poisoning is important to many people:

- The agricultural worker is interested, for within the process rests his assurance for just consideration and compensation for any job-related poisoning.
- Farmers are vitally interested in having safe products to use.
- Chemical manufacturers and formulators want to make a safe product and to insure that an alleged pesticide illness attributed to their product is correctly investigated and verified.

Jurisdictions, legislators, law enforcers, and insurance organizations are also served by verification of pesticide-related illnesses. The attending physician also has a professional interest, having often proceeded with vigorous treatment measures without initial laboratory confirmation.

Steps in Poisoning Verification

The four ingredients of poisoning verification are:

- a pesticide exposure history in which the symptoms complained of are compatible in time and with a known toxicity of a pesticide
- physical signs which reflect the known toxicity of the pesticide
- demonstration of significant red blood cell and plasma cholinesterase inhibition in acute organophosphate poisoning
- identification of the specific urinary pesticide metabolites and/or the intact pesticides in certain tissues of the body.

(See Chapter III for information on clinical and laboratory tests and instructions for collecting laboratory specimens.)

Many of the symptoms of acute pesticide poisoning are common to a wide variety of more frequently occurring illnesses not related to pesticides. Some of the diseases included in this list are influenza, gastroenteritis, heat exhaustion, diseases of the heart and lung, and central nervous system disorders. To distinguish mild or subacute cases of pesticide illnesses from these other conditions, an arbitrary and reasonable selection process is necessary. The following guidelines are suggested.

Apply a simple cholinesterase screening test such as the Acholest:

- to any patient who complains of having sustained a pesticide exposure
- to any worker having regular and heavy pesticide exposure
- to any patient who has three or more of the following symptoms: weakness, sweating, headache, nausea and vomiting, diarrhea, abdominal cramps, excessive tearing, salivation, bronchial secretions, shortness of breath, pains in the chest, blurring of vision, and convulsions
- to any patient with either miosis (less than 5 mm in size), muscle twitching or fasciculations, or clinical evidence of bronchial spasm or bronchial exudation
- to any patient with a pulse rate of 50 or less.

In the event of a convulsion, serum should not only be analyzed for cholinesterase inhibition but it should also be sent to a special pesticide analytical laboratory for electron capture and mass spectrometry gas chromatographic studies for organochlorine poisonings.

If the screening test is positive, the nursing and medical staff should be informed at once. The following additional steps are necessary:

- Collect 8 cc of blood in a heparinized tube for quantitation of red cell and plasma cholinesterase determinations.

- Collect 20 cc of urine for urinary pesticide studies.
- Ship both as soon as possible to a specialized pesticide analytical laboratory for more specific and precise analyses.
- Send a resume of the pertinent clinical findings and exposure history with the specimens. (Use form in Appendix 8.)
- Inform the local community health worker and public health nurse so that a field visit can be made as soon as possible.

Shipping and Notification

Even in the verification process, speed is of the utmost importance. Inform the special pesticide laboratory by telephone of the time of shipment and the expected time of arrival of the specimens and enclosures. The laboratory should complete the analyses quickly. Results should be communicated to the referral center first by telephone and then by written confirmation.

Farm worker clinics may send specimens, together with background information, to the appropriate laboratory as listed in Appendix 7.

Notice: Clinics forwarding specimens via air shipment should notify the laboratory via telephone so the shipment can be met at the airport. Flight number and date should be provided (if known) together with names and telephone numbers to be used in calling back results.

County hospitals, private clinics, industry health units and emergency rooms wishing to have samples analyzed may do so, if cost reimbursement conditions are acceptable.

If suspected cases of poisoning are being treated in rural areas distant from possible air connections to the verification laboratory, consult state departments of health or agriculture for nearby laboratory facilities which can provide blood and urine analyses.

Collection of Data

For administrative purposes, reports on pesticide poisoning should contain the following information:

1. Name of patient or patient identification number (Patient identification is needed so that followup studies can be done. Because this form is a medical record, the information will remain confidential.)
2. Sex and age of patient
3. Time and date of occurrence
4. Location of occurrence
5. Route of exposure
6. Symptoms and signs
7. Type of pesticide used:
   a) name (copy or original of label, if possible)
   b) active ingredients
   c) EPA registration number
8. Crop pesticide was applied to and target pest
9. Means by which pesticide was applied—plane, spray rig, etc.
10. Immediate first aid measures taken, medical attention provided
11. Any other facts which could be useful in analyzing the causes and effects of the poisoning.

To provide this information, duplicate the form in Appendix 8, fill it in, and furnish it with medical specimens to be analyzed. Locally used forms providing the above data may be substituted.

APPENDIX I

SELECTED CHOLINESTERASE-INHIBITING ORGANIC PHOSPHORUS PESTICIDES

Common or Trade Name	*Chemical Name*
Acephate	O,S-Dimethyl acetyl phosphoramidothioate
Acethion	O,O-Diethyl S-carboethoxymethyl phosphorodithioate
Acetoxon	O,O-Diethyl O-carboethoxymethyl phosphorothioate
Akton* or Axiom*	O,O-Diethyl O-(2-chloro-1-(2,5-dichlorophenyl) (vinyl) phosphorothioate
Alamos* or Azothoate* or Slam*	O-(p-chlorophenylazo) O,O-dimethyl phosphorothioate
Amidithion	O,O-Dimethyl-S-[[2-methoxyethyl) carbamoyl] methyl] phosphorodithioate
Amiton	O,O-Diethyl-S-(2-diethylamino) ethyl phosphorodithioate
Aphidan*	O,O-Diisopropyl-S-ethylsulfinyl methyl dithiophosphate
Aspon*	O,O,O,O-Tetra-n-propyl dithiopyrophosphate
Azethion	O,O-Diethyl S-(carbomethoxymethyl) phosphorothioate
Azinphosmethyl	O,O-Dimethyl S-[(4-oxo-1,2,3-benzotriazin-3(4H)-yl)-methyl] phosphorodithioate
Bensulide	S-(O,O-Diisopropyl phosphorodithioate) ester of N-(2-mercaptoethyl) benzenesulfonamide
Bomyl*	Dimethyl 3-hydroxyglutaconate dimethylphosphate
Bromophos ethyl	O,O-Diethyl-O(4-bromo-2,5-dichlorophenyl) phosphorothioate
Butonate	O,O-Dimethyl-(2,2,2-trichloro-1-n-butyryloxyethyl phosphonate
Carbophenothion	O,O-Diethyl-S-[[(p-chlorophenyl)thio] methyl] phosphorodithioate
Chlorfenvinphos	2-Chloro-1-(2,4-dichlorophenyl) vinyl diethyl phosphate
Chlormephos	S-Chlormethyl-O,O-diethyl phosphorothiolothionate
Chlorthion	O,O-Dimethyl 0-(3-chloro-4-nitrophenyl) phosphorothioate
Conen*	O-Butyl-S-benzyl-S-ethyl phosphorodithioate
Coroxon	O,O-Diethyl-O-(3-chloro-4-methylcoumarin-7-yl) phosphate
Coumaphos	O,O-Diethyl O-(3-chloro-4-methyl-2 oxo (2H)-1-benzopyran-7-yl) phosphorothioate
Crufomate	4-tert-Butyl-2-chlorophenyl methyl methyl-phosphoramidate
Cyanthoate	S-[[(1-Cyano-1-methylethyl)carbamoyl] methyl] O,O-diethyl phosphorothioate
Cythioate	O,O-Dimethyl O-p-sulfanoylphenyl phosphorothioate
DAEP	O,O-Dimethyl-S-2-(acetylamino)ethyl dithiophosphate
DEF*	S,S,S,-Tributyl phosphorotrithioate
Demeton	O,O-Diethyl O-(and S)-[2-(ethylthio)ethyl] phosphorothioate
Demeton methyl	O,O-Dimethyl-S-[2(ethylthio)ethyl] phosphorothioate
Diazinon	O,O-Diethyl-O-(2-isopropyl-6-methyl-4-pyrimidinyl) phosphorothioate

*Trade name.

Common or Trade Name	*Chemical Name*
Dicapthon	0-(2-Chloro-4-nitrophenyl) O,O-dimethyl phosphorothioate
Dichlorvos	2,2-Dichlorovinyl O,O-dimethyl phosphate
Dicrotophos	Dimethyl phosphate of 3-hydroxy-N,N-dimethyl-cis-crotonamide
Diethquinalphione	O,O-Diethyl-O-(2-chinoralyl)-phosphorothioate
Dimethoate	O,O-Dimethyl S-(N-methyl carbamoyl methyl) phosphorodithioate
Dioxathion	2,3-p-Dioxanedithiol S,S-bis(O,O-diethyl phosphorodithioate)
Disulfoton	O,O-Diethyl S-[2-(ethylthio)ethyl] phosphorodithioate
DMCP	S-(p-Chlorophenyl) O,O-dimethyl phosphorothioate
Dursban*	O,O-Diethyl O-(3,5,6-trichloro-2-pyridyl) phosphorothioate
Dyfonate*	O-Ethyl-S-phenylethylphosphonodithioate
EPN	O-Ethyl O-(p-nitrophenyl) phenylphosphonothioate
Ethion	O,O,O',O'-Tetraethyl S,S'-methylene bisphosphorodithioate
Fenitrothion	O,O-Dimethyl O-(4-nitro-m-tolyl) phosphorothioate
Fensulfothion	O,O-Diethyl O-[p-(methylsulfinyl)phenyl] phosphorothioate
Fenthion	O,O-Dimethyl O-[4-(methylthio(-m-toly] phosphorothioate
Folex*	Tributyl] phosphorotrithioite
Formothion	O,O-Dimethyl S-(N-formyl-N-methylcarbomylmethyl) phosphorodithioate
Forstenon	Diethyl carbethoxydichloromethyl-phosphonate
Fostion*	O,O-Diethyl-S-(N-isopropylcarbamolymethyl) phosphorodithioate
Gardona*	2-Chloro-1-(2,4,5-trichlorophenyl) vinyl dimethyl phosphate
Hosdon*	O,O-Dimethyl-S-2(isopropylthio) ethyl phosphorodithioate
Imidan*	N-(Mercaptomethyl) phthalimide S-(O,O-dimethyl phosphorodithioate)
Inezin*	O-Ethyl-S-benzylphenylphosphonothiolate
Iodofenphos	O,O-Dimethyl-O-(2,5-dichloro-4-iodophenyl) thiophosphate
Ketothion	O,O-Diethyl S-acetonyl phosphorodithioate
Leptophos	O-(4-Bromo-2,5-dichlorophenyl) O-methyl phenylphosphonothioate
Malathion	O,O-Dimethyl dithiophosphate of diethyl mercaptosuccinate
Mecarbam	O,O-Diethyl S-[[(ethoxycarbonyl)methylcarbamoyl]-methyl] phosphorodithioate
Mecarpon	S-(N-Methoxycarbonyl-N-methylcarbamonylmethyl) dimethyl phosphonothiolothionate
Metasystox-S*+	O,O-Dimethyl S-[(2-ethylsulfinyl) isopropyl] phosphorothioate
Methyl mercaptophos	O-Methyl-O-ethyl-2-ethylmercaptoethyl thiophosphate
Methyl parathion	O,O-Dimethyl O-p-nitrophenyl phosphorothioate
Methyl phencapton	O,O-Dimethyl S-(2,5-dichlorophenylthio)methyl phosphorodithioate
Methyl potasan*	O,O-Dimethyl O-(4-methylumbelliferone) phosphorothioate

*Trade name.

Common or Trade Name	*Chemical Name*
Methyl trithion	S-(((p-Chlorophenyl)thio)methyl) O,O-dimethyl phosphorodithioate
Mevinphos	2-Carbomethoxy-l-methylvinyl dimethyl phosphate
Monitor*	O,S-Dimethylphosphoramidothioate
Monocrotophos	Dimethyl phosphate of 3-hydroxy-N-methyl-cis-crotonamide
Morphothion	O,O-Dimethyl S-(morpholinocarbonylmethyl) phosphorodithioate
Naled	1,2-Dibromo-2,2-dichloroethyl dimethyl phosphate
Naphthalaphos	N-Hydroxynaphthalimide diethylphosphate
Orthene*	O,S-Dimethyl N-acetyl phosphoramidothioate
Oxydemetonmethyl	O,O-Dimethyl S[2-(ethylsulfinyl)ethyl] phosphorothioate
Oxydisulfoton	O,O-Diethyl S-(2-(ethylsulfinyl)ethyl) phosphorodithioate
Paraoxon	O,O-Diethyl O-p-nitrophenyl phosphate
Parathion	O,O-Diethyl O-p-nitrophenyl phosphorothioate
Phencapton	O,O-Diethyl S-[[(2,5 dichlorophenyl)thio] methyl] phosphorodithioate
Phenthoate	O,O-Dimethyl S-(a-ethoxycarbonylbenzyl) phosphorodithioate
Phorate	O,O-Diethyl S-(ethylthio)methyl phosphorodithioate
Phosalone	O,O-Diethyl S-[(6-chloro-3(mercaptomethyl)-2-benzoxazolinone] phosphorodithioate
Phosphamidon	2-Chloro-2-diethylcarbamoyl-1-methylvinyl dimethyl phosphate
Phosphinon	O,O-Diethyl O-(1-(2-chloroethoxy)-2,2-dichlorovinyl) phosphate
Phosvel*	O-(2,5-Dichloro-4-bromophenyl) O-methyl phenylthiophosphate
Phoxim	Phenylglyoxyonitrile oxime O,O-diethyl phosphorothioate
Pirazinon*	O,O-Diethyl O-(6-methyl-2-propyl-4-pyrimidyl) phosphorothioate
Potasan	O,O-Diethyl O-(4-methylumbelliferone) phosphorothioate
Prophos	O-Ethyl S,S-dipropyl phosphorodithioate
Propoxon	O,O-Diethyl-S-carboethoxyethyl-phosphorothioate
Prothion	O,O-Diethyl S-carboethoxyethyl phosphorodithioate
Pyrazoxon*	O,O-Diethyl O-(3-methylpyrazol-5-yl) phosphate
Pyrazothion*	O,O-Diethyl O-(3-methylpyrazol-5-yl) phosphorothioate
Ronnel	O,O-Dimethyl O-(2,4,5-trichlorophenyl) phosphorothioate
Schradan	Octamethylphosphoramide
S-Seven*	O-Ethyl-O-(2,4-dichlorophenyl)-phosphonothionate
Sulfotepp	O,O,O,O-Tetraethyl dithiopyrophosphate
TEPP	Tetraethyl pyrophosphate
Thiometon	O,O-Dimethyl-S-[2-(ethylthio)ethyl] phosphorodithioate
Trichlorfon	Dimethyl (2,2,2-trichloro-1-hydroxyethyl) phosphonate
Trichloronate	O-Ethyl O-(2,4,5-trichlorophenyl) ethylphosphonothioate
VC-13 Nemacide	O-2,4-Dichlorophenyl O, O-diethyl phosphorothioate
Zytron	O-(2,4-Dichlorophenyl) O-methyl N-isopropylophosphoroamidothioate

*Trade name.

CROSS REFERENCE FOR SOME OF THE CHOLINESTERASE-INHIBITING ORGANIC PHOSPHORUS PESTICIDE TRADE NAMES

Aflix* ..... see formothion
Afos* ..... see mecarbam
Agrisil* ..... see trichloronate
Agritox* ..... see trichloronate
Agrothion* ..... see fenitrothion
Alkron* ..... see parathion
Alleron* ..... see parathion
Amiphos* ..... see DAEP
Anthio* ..... see formothion
Anthon* ..... see trichlorfon
Appex* ..... see Gardona+
Asuntol* ..... see coumaphos
Azodrin* ..... see monocrotophos
Basudin* ..... see diazinon
Baymix* ..... see coumaphos
Bayrusil* ..... see diethquinalphione
Baytex* ..... see fenthion
Baythion* ..... see phoxim
Betasan* ..... see bensulide
Bidrin* ..... see dicrotophos
Bilobran* ..... see monocrotophos
Birlane* ..... see chlorfenvinphos
Bladafume* ..... see sulfotepp
Bladen* ..... see parathion
Borinox* ..... see trichlorfon
Bromex* ..... see naled
Carbicron* ..... see dicrotophos
Carfene* ..... see azinphos-methyl
Cidial* ..... see phenthoate
Citram* ..... see amiton
Co-Ral* ..... see coumaphos
Corothion* ..... see parathion
Cygon* ..... see dimethoate
Cythion* ..... see malathion
Dagadip* ..... see carbophenothion
Dalf* ..... see methyl parathion
Dasanit* ..... see fensulfothion
Daphene* ..... see dimethoate
Dazzel* ..... see diazinon
Dedevap* ..... see dichlorvos
De-Fend* ..... see dimethoate
De-Green* ..... see DEF*
Delnav* ..... see dioxathion
Diazajet* ..... see diazinon
Diazide* ..... see diazinon
Diazol* ..... see diazinon
Dibrom* ..... see naled
Di-Captan* ..... see dicapthon
Dimecron* ..... see phosphamidon
Dimethogen* .... see dimethoate
Dipterex* ..... see trichlorfon
Di-Syston* ..... see disulfoton
Disyston S* ..... see oxydisulfoton
Dithione* ..... see sulfotepp
Dylox* ..... see trichlorfon
E-605* ..... see parathion
Easy Off-D* ..... see Folex*
Ectoral* ..... see ronnel
Ekatin* ..... see thiometon
Ekatin M* ..... see morphothion
Ektafos* ..... see dicrotophos
Elsan* ..... see phenthoate
Emmatos* ..... see malathion
Entex* ..... see fenthion
Equino-Aid* ..... see trichlorfon
Ethyl Parathion* . see parathion
Etilon* ..... see parathion
Etrolene* ..... see ronnel
Exothion* ..... see endothion
Filariol* ..... see bromophos ethyl
Folidol E–605* .. see parathion
Folidol M* ..... see methyl parathion
Folithion* ..... see fenitrothion
Fostion MM* .... see dimethoate
Frumin Al* ..... see disulfoton
Fujithion* ..... see DMCP
Fyfanon* ..... see malathion
Gardentox* ..... see diazinon
Garrathion* ..... see carbophenothion
Gusathion M* .... see azinphos-methyl
Guthion* ..... see azinphos-methyl
Hercules AC527* . see dioxathion
Karbofos* ..... see malathion
Klimite 40* ..... see TEPP
Korlan* ..... see ronnel
Lebaycide* ..... see fenthion
Malamar* ..... see malathion
Malaspray* ..... see malathion
Maretin* ..... see naphthalaphos
Meldane* ..... see coumaphos
Menite* ..... see mevinphos
Metasystox* ..... see demeton methyl
Metasystox-R* ... see oxydemeton-methyl
Metron* ..... see methyl parathion
Mintacol* ..... see paraoxon
MLT* ..... see malathion
Mocap* ..... see prophos
Morphotox* ..... see morphothion

*Trade name.

Murfotox* ..... see mecarbam
Muscatox* ..... see coumaphos
N–2790* ..... see dyfonate
Nankor* ..... see ronnel
Neragan* ..... see bromophos ethyl
Neguvon* ..... see trichlorfon
Nialate* ..... see ethion
Niram* ..... see parathion
Nitrox* ..... see methyl parathion
No Pest* ..... see dichlorvos
Novathion* ..... see fenitrothion
Nuvacron* ..... see monocrotophos
Nuvanol* ..... see fenitrothion
Orthophos* ..... see parathion
Ortho Phosphate Defoliant* .... see DEF*
Panthion* ..... see parathion
Parathene* ..... see parathion
Parawet* ..... see parathion
Partron M* ..... see methyl parathion
Perfekthion* ..... see dimethoate
Pestan* ..... see mecarbam
Pestox III* ..... see schradan
Phosdrin* ..... see mevinphos
Phosfene* ..... see mevinphos
Phoskit* ..... see parathion
Phosphopyran* ... see endothion
Phosvit* ..... see dichlorvos
Phytosol* ..... see trichloronate
Prolate* ..... see imidan
Rabon* ..... see Gardona+
Rampart* ..... see phorate
Rawetin* ..... see naphthalaphos
Resistox* ..... see coumaphos
Rhodiatox* ..... see parathion
Rogor* ..... see dimethoate
Roxion* ..... see dimethoate
Ruelene* ..... see crufomate
Ruphos* ..... see dioxathion
Sapecron* ..... see chlorfenvinphos
Solverex* ..... see disulfoton
Soprathion* ..... see parathion
Spectracide* ..... see diazinon
Stathion* ..... see parathion
Sumithion* ..... see fenitrothion
Supona* ..... see chlorfenvinphos
Systox* ..... see demeton
Sytam* ..... see schradan
Tamaron ..... see Monitor*
Tanone* ..... see phenthoate
Tartan* ..... see cyanthoate
Task* ..... see dichlorvos
Tekwaisa* ..... see methyl parathion
Terracur P* ..... see fensulfothion
Tetrachlorvinphos . see Gardona*
Tetraethyl Pyrophosphate . see TEPP
Tetram* ..... see amiton
Tetron* ..... see TEPP
Thimet* ..... see phorate
Thiocron* ..... see amidthion
Thiodemeton .... see disulfoton
Thiophos ..... see parathion
Thiotepp ..... see sulfotepp
Tiguvon* ..... see fenthion
Timet ..... see phorate
Trichlorphon .... see trichlorfon
Trimetion* ..... see dimethoate
Trinox* ..... see trichlorfon
Trithion* ..... see carbophenothion
Trolene* ..... see ronnel
Tugon ..... see trichlorfon
Valexon* ..... see phoxim
Vapona* ..... see dichlorvos
Vaponite* ..... see dichlorvos
Vapotone* ..... see TEPP
Viozene* ..... see ronnel
Volaton* ..... see phoxim
Zithiol* ..... see malathion
Zolone* ..... see phosalone

*Trade name.

SELECTED CHOLINESTERASE-INHIBITNG CARBAMATE PESTICIDES

Common or Trade Name	*Chemical Name*
Aldicarb	2-Methyl-2-(methylthio) propionaldehyde 0-(methylcarbamoyl) oxime
Banol	6-Chloro-3,4-xylyl methylcarbamate
Baygon	0-Isopropoxyphenyl methylcarbamate
Bufencarb	3-(1-Methylbutyl) phenyl methylcarbamate and 3-(1-Ethylpropyl) phenyl methylcarbamate (3:1)
Butacarb	3,5 Di-tert-butylphenyl methylcarbamate
Carbaryl	1-Naphthyl N-methylcarbamate
Carbofuran	2,3-dihydro-2,2-dimethyl-7-benzofuranyl methylcarbamate
Dichlormate	3,4-and 2,3-Dichlorobenzyl methylcarbamate
Dimetilan	3-Hyrdroxy-N,N, 5-trimethylpyrazole-1-carboxamide dimethylcarbamate
Dioxacarb	0-1,3-Dioxolan-2-ylphenyl methylcarbamate
Ficam	2,2-Dimethyl-1,3-benzodioxol-4-y1-methylcarbamate
Formetanate (hydrochloride)	M-[((Dimethylamino)methylene) amino phenyl methylcarbamate] (hydrochloride)
Landrin	3,4,5-Trimethylphenyl methylcarbamate and 2,3,5-Trimethylphenyl methylcarbamate
Matacil	4-(Dimethylamino)-m-tolyl methylcarbamate
Mesurol	4-(Methylthio)-3,5-xylyl methylcarbamate
Methomyl	5-Methyl N-[(methylcarbamoyl) oxyl] thioacetimidate
Mexacarbate	4-(Dimethylamino)-3,5-xylyl methylcarbamate
Mobam	4-Benzothienyl methylcarbamate
Oxamyl	Methyl N',N'-dimethyl-N-[(methylcarbamoyl)oxy]-1-thiooxamidate
Pirimicarb	2-(Dimethylamino)-5, 6-dimethyl-4-pyrimidinyl dimethylcarbamate
Promecarb	3-methyl-5-isopropylphenyl methylcarbamate
Thiofanox	3,3-Dimethyl-1-(methylthio)-2-butanone 0-[(methylamino)carbonyl] oxime

CROSS REFERENCE FOR SOME OF THE CHOLINESTERASE-INHIBITING CARBAMATE PESTICIDE TRADE NAMES

A 363 ..... see Matacil
Ambush ..... see Aldicarb
Aminocarb ..... see Matacil
Aphox ..... see Pirimicarb
Arprocarb ..... see Baygon
Bay 9010 ..... see Baygon
B-37344 ..... see Mesurol
Bay 39007 ..... see Baygon
Bay 44646 ..... see Matacil
Bay 70142 ..... see Carbofuran
Bendiocarb ..... see Ficam
Blattenex ..... see Baygon
Bux ..... see Bufencarb
Carbamult ..... see Promecarb
Carbanolate ..... see Banol
Carpolin ..... see Carbaryl
Carzol ..... see Formetanate (Hydrochloride)
CIBA 8353 ..... see Dioxacarbe
Curaterr ..... see Carbofuran
D-1221 ..... see Formetanate (Hydrochloride)
D-1410 ..... see Oxamyl
Dicarol ..... see Formetanate (Hydrochloride)
Dowco 139 ..... see Mexacarbate
Draza ..... see Mesurol
Elocron ..... see Dioxacarb
ENT 27164 ..... see Carbofuran
ENT 27300 ..... see Promecarb
EP 316 ..... see Promecarb
EP 332 ..... see Formetanate (Hydrochloride)
Famid ..... see Dioxacarb
FMC 10242 ..... see Carbofuran
Furadan ..... see Carbofuran
G-22870 ..... see Dimetilan
G-13332 ..... see Dimetilan
Hexavin ..... see Carbaryl
IPMC ..... see Baygon
Karbaspray ..... see Carbaryl
Lannate ..... see Methomyl
Maz ..... see Mexacarbate
MCA-600 ..... see Mobam
Mercaptodimethur see Mesurol
Metalkamate ..... see Bufencarb
Methiocarb ..... see Mesurol
Metmercaptron ... see Mesurol
Minacide ..... see Promecarb
Mos 78 ..... see Mobam
MXMC ..... see Mesurol
NC 6897 ..... see Ficam
NIA 10242 ..... see Carbofuran
Nudrin ..... see Methomyl
OMS 716 ..... see Promecarb
Ortho 5353 ..... see Bufencarb
PHC ..... see Baygon
Pirimor ..... see Pirimicarb
PP 062 ..... see Pirimicarb
Propoxur ..... see Baygon
Ravyon ..... see Carbaryl
Romate ..... see Dichlormate
Rowmate ..... see Dichlormate
Schering 34615 ... see Promecarb
Schering 36056 ... see Formetanate (Hydrochloride)
Sendran ..... see Baygon
Septene ..... see Carbaryl
Sevin ..... see Carbaryl
Sirmate ..... see Dichlormate
Snip ..... see Dimetilan
Sok ..... see Banol
Suncide ..... see Baygon
Temik ..... see Aldicarb
Tendex ..... see Baygon
Tricarnam ..... see Carbaryl
UC 9880 ..... see Promecarb
UC 21149 ..... see Aldicarb
UC 22463 ..... see Dichlormate
UC 7744 ..... see Carbaryl
Unden ..... see Baygon
Vydate ..... see Oxamyl
Yaltox ..... see Carbofuran
Zectron ..... see Mexacarbate

APPENDIX 2

PESTICIDE TOXICITY

Because most pesticides destroy unwanted organisms, they obviously are toxic materials. Some pesticides are much more toxic than others. Severe illness may result when only a small amount of one type of pesticide has been ingested; with another type, a large amount may be ingested with no serious effects.

Toxicologists use a simple animal toxicity test to rank pesticides according to their inherent toxicity. Before any pesticide can be registered, the manufacturer must provide the results of these tests to EPA. The LD_{50} (the dosage level of a toxic chemical which is lethal to 50 percent of a population of test animals) is measured in terms of:

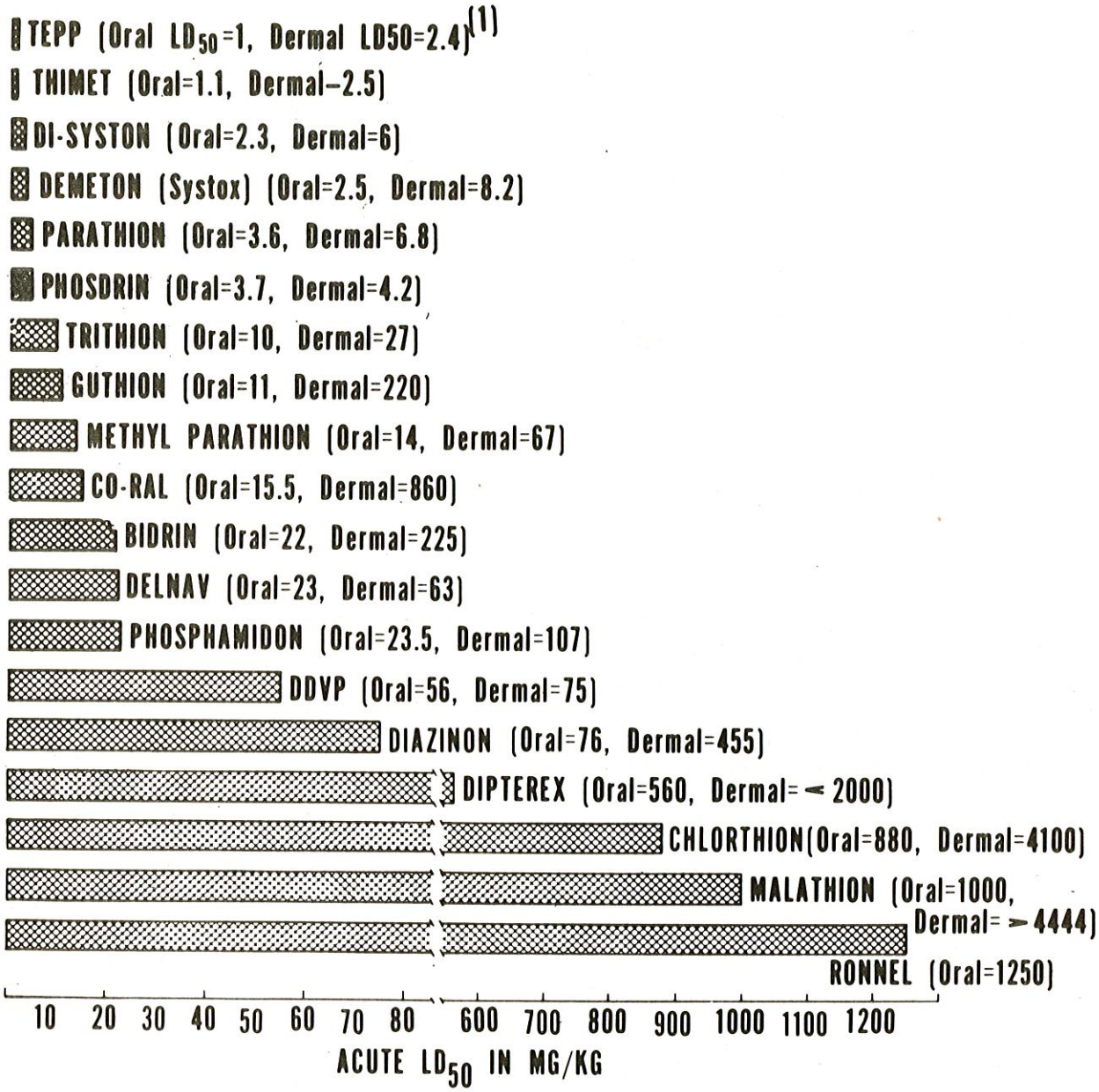
- oral toxicity (material is fed to rats)
- dermal toxicity (material is applied to the skin of rats)
- respiratory toxicity (material is inhaled).

In this way an arbitrary toxicologic ranking has been obtained for the organophosphate and organochlorine pesticides. The materials on the top of the list are the most toxic, and those at the bottom are the least toxic. The size of the dose is the most important single item in determining the safety of a given chemical. Actual statistics of human poisonings correlate reasonably well with these toxicity ratings. Health personnel will be able to get some idea of the probable severity of the poisoning being treated by referring to these figures.

The amount of pesticide required to kill an adult male can be correlated with LD_{50}:

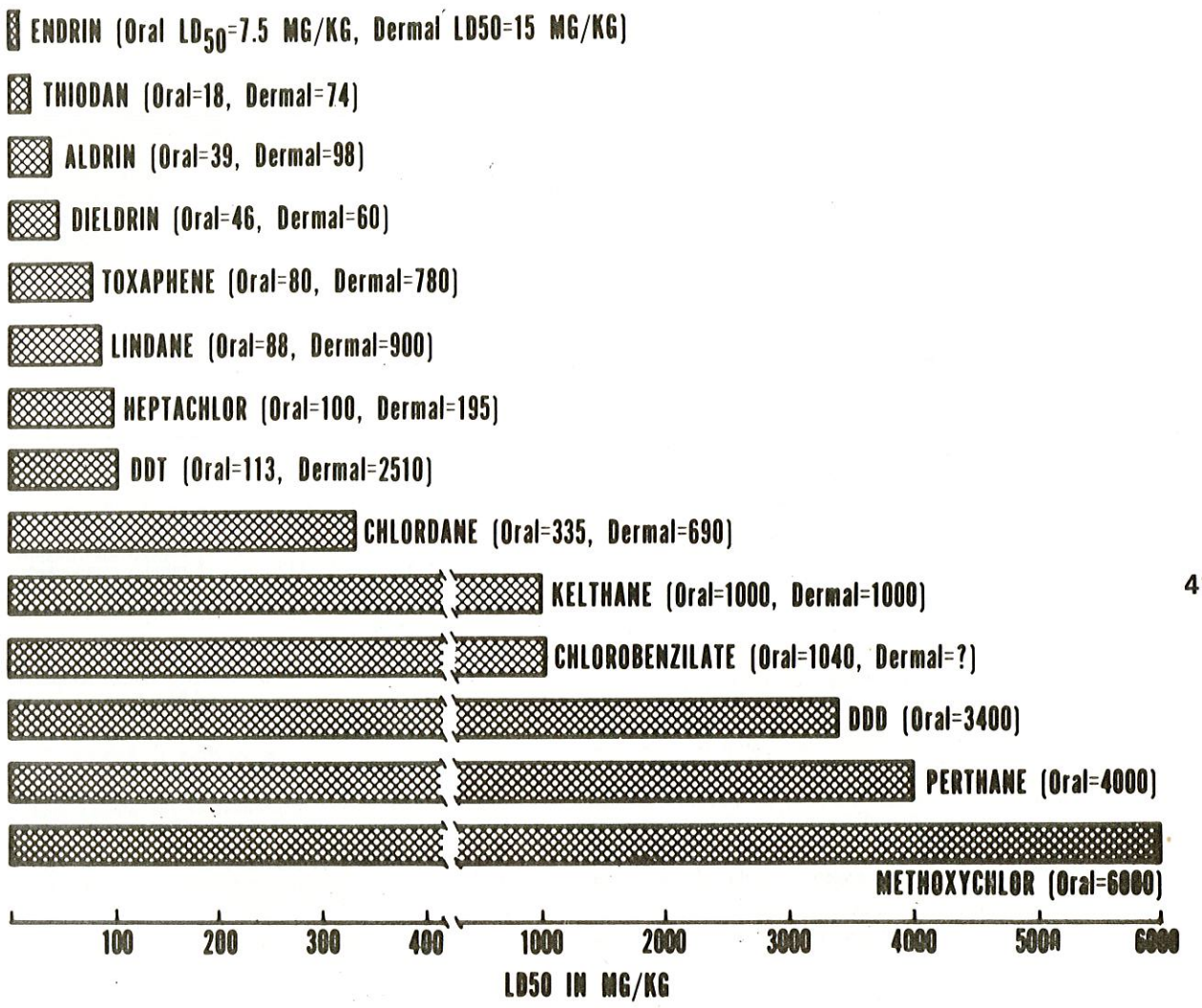
Acute Oral LD_{50}	Material which will kill an adult male
5	a few drops
5 - 50	a pinch to a teaspoonful
50 - 500	a teaspoonful to a tablespoonful
500 - 5,000	a 1 ounce to 1 pint
5,000 - 15,000	1 pint to 1 quart

Oral ingestions are more toxic than respiratory inhalations and these are more toxic than dermal absorptions. In addition, there are individual physical and chemical differences in a chemical which render the material more likely to produce poisoning. Thus, parathion changes to the more toxic "paraoxon" with high temperatures. Ethyl parathion is more toxic than methyl parathion, yet there is no great difference in their oral toxicity. Work exposure is usually dermal and that is why many more illnesses are seen in workers exposed to ethyl parathion than those exposed to methyl parathion.



Acute Oral and Dermal Toxicity Values for Some Organophosphate Pesticides.

(Prepared by the Bureau of Occupational Health, State of California Department of Public Health. Copied with permission.)



Acute Oral and Dermal Toxicity Values for Some Chlorinated Hydrocarbon Pesticides.

(Prepared by the Bureau of Occupational Health, State of California Department of Public Health. Copied with permission.)

APPENDIX 3

ACHOLEST T.M.

(Cholinesterase Test-Paper)

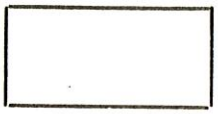
METHOD FOR USING ACHOLEST TEST PAPER

Equipment

1. ACHOLEST Cholinesterase Test-Paper, Bottle I.

Test Strip

2. Four slides.
3. 0.1 ml. pipette (0.01 ml. graduations)
4. Comparative color strips, Bottle II.



Comparative Strip

5. Watch or stop-watch.

Procedure: Place 0.05 ml. of non-hemolyzed plasma* on each of two thoroughly cleaned slides (avoid traces of acid or alkali). The plasma is to be quickly separated from the blood sample inasmuch as blood cells present in the plasma interfere with the reaction. With a pair of clean, dry scissors, cut one-half of a strip (2 x 1 cm.) of Acholest Test Paper from Bottle I, and one-half of a strip of control paper from Bottle II. Using tweezers, place the untouched half of each strip on each slide containing the plasma and cover with a second thoroughly cleaned slide, using gentle, even pressure several times in order to ensure complete saturation of the test and control papers and prevent mottling. To prevent evaporation of fluid, do not separate the slides until the test has been completed. Record the moment of contact of the test and control papers with the plasma as the beginning of the test.

* Heparinized, not citrated or oxalated tubes may be used for collection.

The Acholest Test Paper and the control paper, when dry, are similarly egg yolk colored. When exposed to the plasma, the Acholest Test Paper turns green, gradually developing into a yellowish color, after passing through various tones of green-yellow. The control paper, however, upon contact with the blood plasma, turns immediately yellowish with no further change in color. The plasma cholinesterase activity is measured by the time required for the Acholest Test Paper to reach the color of the control paper. For accurate comparison of the color, it is recommended that the test be performed on a white background with diffused light.

From the moment of contact of the plasma and Acholest Test Paper to the point when the comparative tone of color has been reached, the following time values have been established:

Minutes	Activity of Plasma Cholinesterase
Below 5	"increased"
5 - 20	"normal"
20 - 30	"suspicious"
30 and longer	"decreased"

Precaution: Acholest Test Paper should be stored away from light in tightly closed containers to protect it from moisture and chemical vapors. **Avoid contact with fingers. Plasma to be used should be free of cellular components. Always place Acholest Test Paper on the plasma, never drip plasma on the Test Paper; this similarly applies to the control strip.**

Readings should always be carried out under the same light conditions and room temperature. (Room temperature differences in the range from 68° F (19° C) to 75° F (25° C) do not influence the reliability and accuracy of Acholest Test Paper). Also hemolysis of a minor degree does not affect the accuracy of Acholest Test Paper. **It is important, however, to note that pH changes of the plasma might interfere with the accuracy of Acholest Test Paper independent of the plasma cholinesterase activity.** Such pH changes might be the result of alkali or acid cleansing materials on test equipment or of undue storage of plasma which might have led to degradation or decay (e.g. protein fractions). However, as a rule, storage of the plasma up to seven days at a temperature of 20° C or below (refrigeration of plasma is desirable), does not interfere with the accuracy of Acholest Test Paper.

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E. FOUGERA & CO., INC.
HICKSVILLE, NEW YORK 11802

APPENDIX 4

LABORATORY METHODS

Cholinesterase Determination

The four laboratory techniques most commonly used for quantitative expressions of these enzyme activities are the electrometric (Michel), the titrimetric (pH stat), the colorimetric (Ellman) and gas chromatographic (Cranmer) methods.

1. Michel method (pH meter)—Plasma and red cells are incubated with acetylcholine for one hour. The drop of the pH is due to the formation of acetic acid and is directly proportional to the cholinesterase activity. Normal values: Plasma 0.53 - 1.24 ΔpH units and Red Blood Cells 0.57 - 0.98 ΔpH units.

2. pH Stat (titrimetric)—The plasma and red cells are incubated with acetylcholine for 3 minutes and the acid formed is titrated with a base. The amount of base used is directly proportional to the cholinesterase activity in the blood sample. Normal values: Plasma 3.6 - 6.8 μM/ml/min and Red Blood Cells 11.1 - 16.0 μM/ml/min.

3. Ellman method (colorimetric)—Plasma and red cells are incubated for 10 minutes with acetyl thiocholine and the resultant thiocholine produces a yellow color in the presence of 5:5-dithiobis-(2-nitrobenzoic acid). The concentration of the yellow complex is directly proportional to the amount of cholinesterase present. Normal values: Plasma 5.8 - 16.6 M-SH/ml/3 min.

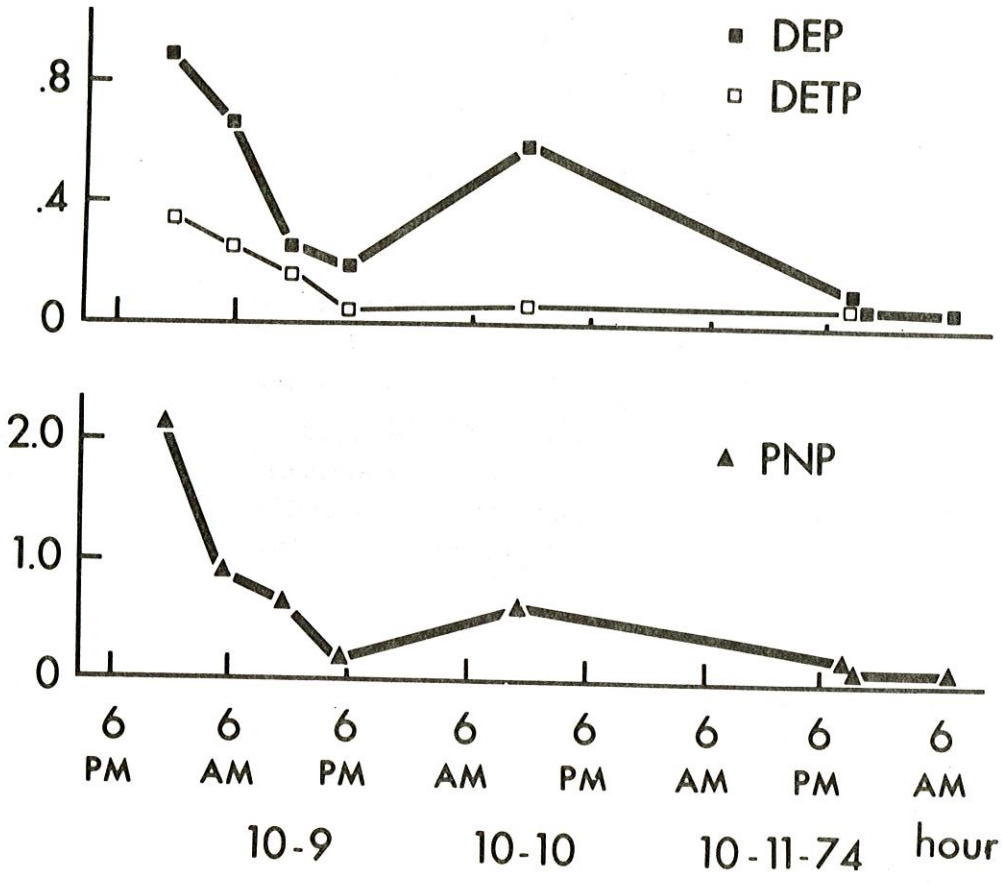
4. GLC method (chromatographic)—Plasma and red cells are reacted for 30 minutes with a compound that is similar to acetylcholine. The product formed, dimethyl butanol, is quantitated using a gas chromatograph. Normal values: Plasma 2.1 - 4.6 μM/ml/min and Red Blood Cells 8.2 - 11.8 μM/ml/min.

Urinary Pesticide Metabolite Data

The organophosphates are metabolized in man to produce two major types of pesticide metabolites in urine. These are the phenolic metabolites (e.g. nitrophenols and halogenated phenols) and the alkyl phosphate metabolites (namely, diethyl thiophosphate (DETP), diethyl phosphate (DEP), dimethyl thiophosphate (DMTP) and dimethyl phosphate (DMP). The carbamates, the other major group of anticholinesterase pesticides, are metabolized to non-halogenated phenols which are excreted in urine.

In poisoning cases the information from both types of metabolites is informative. Take ethyl parathion as an example. This is a diethyl thiophosphate pesticide and the excretion of the diethyl phosphate moiety in the urine is related to the parent compound. Parathion is oxidized to the more toxic product paraoxon, and it is this which is largely responsible for illness. Paraoxon is reflected by the excretion of diethyl phosphate (DEP) so that high concentrations of the oxon derivative are seen in the urine in poisoned victims. Paranitrophenol is the phenolic moiety and the identification of this in urine facilitates the specific diagnosis of the pesticide involved in the exposure, for paranitrophenol is only found in the urine following exposure to parathion and two other pesticides being used today (EPN and chlorthion).

Both the alkyl phosphate and phenolic metabolites have been found to be excellent indices of exposure if this exposure is a significant one, such as occurs in occupational and accidental exposure. The following chart illustrates the sequential excretion of these metabolites expressed as concentrations of the metabolites per milliliter in a parathion poisoning case.



Intact Pesticide Studies

The intact pesticide may be identified in blood, in other body tissues, and in gastric washing by electron capture gas chromatography using a flame photometric detector.

APPENDIX 5

CHEMTREC

CHEMICAL TRANSPORTATION EMERGENCY CENTER

For assistance in any transportation emergency involving chemicals (in the continental U.S.)

PHONE: Day or Night—Toll-Free

***800 — 424-9300**

*** Add long-distance access number if required**

483-7616 in District of Columbia

For calls originating outside the continental U.S.: **202—483-7616**—Washington, D. C.

CHEMTREC

WHAT IT IS

CHEMTREC stands for Chemical Transportation Emergency Center, a public service of the Manufacturing Chemists Association at its offices in Washington, D. C.

CHEMTREC provides immediate advice for those at the scene of emergencies, then promptly contacts the shipper of the chemicals involved for more detailed assistance and appropriate follow-up.

CHEMTREC operates around the clock—24 hours a day, seven days a week—to receive direct-dial toll-free calls from any point in the continental United States through a wide area telephone service (WATS) number, 800-424-9300 (483-7616 for calls originating within the District of Columbia; 202-483-7616 for calls originating outside the continental U.S.).

Shippers, including MCA members and non-members, are notified through pre-established phone contacts providing 24-hour accessibility, via information operators, or through cooperation of fire and police services.

As circumstances warrant, the National Transportation Safety Board or appropriate offices of other agencies may be notified.

CHEMTREC's capabilities have been recognized by the Department of Transportation, and a close and continuing relationship is maintained between CHEMTREC and the Department.

WHAT IT IS NOT

Because chemicals find so many uses and have such a wide range of characteristics, there is much need for information about them—composition and purity, physical and chemical properties, effects on people and the environment, sources of supply, etc. It is important to understand that CHEMTREC *is not* intended and *is not* equipped to function as a general information source, but by design is confined to dealing with chemical transportation emergencies. Drivers should not call CHEMTREC on problems other than chemical cargo emergencies.

MODE OF OPERATION

CHEMTREC's number has been widely circulated in professional literature distributed to emergency service personnel, carriers, and the chemical industry, and has been further circulated in bulletins of governmental agencies, trade associations, etc.

Shipping documents of participating companies are requested to include the following: "For help in chemical emergencies involving spill, leak, fire or exposure, call toll-free 800-424-9300 day or night."

An emergency reported to CHEMTREC is received by the Communicator on duty, who records details in writing and by tape recorder. The Communicator then attempts to determine the essentials of the problem (as detailed on the left column of this page under "USER GUIDANCE"). This is to enable him to provide the best available information on the chemical(s) reported to be involved, thereby giving specific indication of the hazards and what to do (as well as what not to do) in case of spills, fire, or exposure as the immediate first steps in controlling the emergency. Information on the various chemicals, as furnished by the producers, is within easy reach. Trade names and synonyms of chemical names are cross-referenced for ready identification by whatever name is given.

CHEMTREC's Communicators are not scientists. They are chosen for their ability to remain calm under emergency stresses. To preclude unfounded personal speculation regarding a reported emergency, they are under instructions to abide strictly by the information prepared by technical experts for their use.

Having advised the caller, the Communicator proceeds immediately to notify the shipper by phone. The known particulars of the emergency thus relayed, responsibility for further guidance—including dispatching personnel to the scene or whatever seems warranted—passes to the shipper.

Although proceeding to the second stage of assistance becomes more difficult where the shipper is unknown, the Communicator is armed with other resources to fall back on. For example: Concerning radioactive materials, CHEMTREC can call on the Energy Research and Development Administration (ERDA). (Formerly Atomic Energy Commission).

Identification of product and shipper is important. Shipping papers are carried by truck drivers, and in engine or caboose of trains. Car and truck numbers and carrier names can be useful in tracing unknown cargoes.

Mutual aid programs exist for some products, whereby one producer will service field emergencies involving another producer's product. In such cases, initial referral may be in accord with the applicable mutual aid plan rather than direct to the shipper. Arrangements of this sort are established on chlorine through the Chlorine Institute and on pesticides through the National Agricultural Chemicals Association.

The former has CHLOREP, the Chlorine Emergency Plan, in which the nearest producer responds to a problem. NACA has a Pesticide Safety Team Network of some 40 emergency teams distributed throughout the country. CHEMTREC serves as the communication link for both programs.

In Canada, the Canadian Chemical Producers' Association operates a Transportation Emergency Assistance . Program (TEAP) through regional teams prepared to give phone and field response.

Many individual companies have well organized response capabilities, for their own products, some of which preceded CHEMTREC by several years. This program does not seek to displace these, but rather collaborates with them and enhances their effectiveness. CHEMTREC's single telephone number affords this opportunity.

BACKGROUND

MCA is a trade association of chemical manufacturers, large and small, representing more than 90% of the production capacity for basic industrial chemicals in the United States and Canada. It has long been active in programs to improve the safety of chemical shipping containers, both package and bulk units, and their reliability in handling and shipment, thereby minimizing failures and leakage of contents under extraordinary stress. Such efforts continue unabated.

Nevertheless, despite precautions taken, train derailments, truck upsets and collisions, and barge accidents, do occur. Such emergencies deserve to be handled as well as possible to minimize the consequences to life and property.

Emergency services—fire and police—are normally well prepared to cope with common materials, including certain flammables such as fuel oil and gasoline. Too often they are at a disadvantage when chemicals are encountered, especially since "what should be done"—and of equal importance, "what should *not* be done"—in the early stages may bear so heavily on the outcome. They need accurate and clearly understandable information to help them evaluate the situation and act with proper precautions for their own safety, as well as for the protection of the general public.

Realizing that personnel of chemical producers possessed the necessary expertise, officials of concerned Federal departments approached MCA. A study was undertaken by industry safety, packaging, and transportation specialists. After thorough consideration, it was concluded that a single center, nationwide in coverage and accessible to all through a single telephone number, would be the most expeditious arrangement—for contact with it and for feedback from it. Following review and confirmation by the industrial specialists of MCA's technical committees, CHEMTREC as now in operation was authorized.

CHEMTREC was established and continues as a voluntary project of the chemical manufacturing industry, wholly supported through the Manufacturing Chemists Association. It became operational on September 5, 1971.

USER GUIDANCE

CHEMTREC can usually provide hazard information warnings and guidance when given only the NAME OF THE PRODUCT and the NATURE OF THE PROBLEM. For more detailed information and/or assistance, or if product is unknown, attempt to provide as much of the following information as possible:

Name of caller and call back number
Location of problem
Shipper or manufacturer
Container type
Rail car or truck number
Carrier name
Consignee
Local conditions

FOR MORE INFORMATION

Questions regarding the operation of CHEMTREC should be directed to: Manager, Chemical Transportation Emergency Center, 1825 Connecticut Avenue, N.W., Washington, D. C. 20009. Phone: 202—483-6126.

APPENDIX 6

COMMUNICATIONS CHECKLIST FOR HEALTH PROFESSIONALS

√ Attending Physician

√ Family Members

√ Employers

√ Hospital Emergency Room

√ Ambulance Service

√ Health Department

√ Local, County or State Police

√ Landowner

√ Pesticide Analytical Resource Laboratory

√ EPA Resource (if necessary)

√ Chemtrec (if necessary)

APPENDIX 7

LABORATORY AND EPA REGIONAL OFFICE LOCATIONS

Pesticide Verification Laboratory	State Service Area	EPA Region	EPA Regional Pesticide Branch Chief
Robert Altman, M.D., M.P.H. Project Director Epidemiologic Studies Program New Jersey State Dept. of Health John Fitch Plaza P. O. Box 1540 Trenton, New Jersey 08625 (609) 292-7608 Off-hour number: (609) 392-2020	ME VT NH MA RI CT NJ NY PR VI	I II	A. Charles Lincoln, Chief EPA, Pesticide Branch John F. Kennedy Bldg. Boston, Massachusetts 02203 (617) 223-5126 Stanley Fenichel, Chief EPA, Pesticide Branch 26 Federal Plaza, Room 1005 New York, New York 10007 (212) 264-8356
	DE DC MD PA VA WV	III	Nelson Davis, Chief EPA, Pesticide Branch Curtis Building 6th and Walnut Streets Philadelphia, Pa 19106 (215) 597-9869
Dr. Ana Barquet Dept. of Epidemiology and Public Health University of Miami School of Medicine P. O. Box 520875 Miami, Florida 33152 (305) 547-6972 Off-hour number: (305) 235-6280	AL FL GA KY MS NC SC TN	IV	Roy Clark, Chief EPA, Pesticide Branch 345 Courtland Street, N.E. Room 204 Atlanta, Georgia 30308 (404) 257-3222
E. Gomes 152 E. Stenger San Benito, Texas 78586 (512) 399-5352 Off-hour number: (512) 399:3455	AR LA NM OK TX	VI	Norman E. Dyer, Chief EPA, Pesticide Branch 1201 Elm Street 1st International Bldg. Dallas, TX 75270 (214) 749-7126

	IL IN MI MN OH WI	V	Mitchell Wrich, Chief EPA, Pesticide Branch 230 S. Dearborn St. Chicago, IL. 60604 (312) 353-2192
Donald P. Morgan, M.D., Ph.D. Project Director Epidemiologic Studies Program University of Iowa Oakdale Campus, AMRF Oakdale, IA 52319 (319) 353-5558 Off-hour number: (319) 338-8474	IA KS MO NE	VII	John Wicklund, Chief EPA, Pesticide Branch 1735 Baltimore Ave. Room 249 Kansas City, MO 64108 (816) 374-3036
	CO MT ND SD UT WY	VIII	Ivan Dodson, Chief EPA, Pesticide Branch Lincoln Tower Building 1860 Lincoln Street Suite 900 Denver, CO 80203 (303) 837-3926
	AZ CA GU HI NV	IX	Jake McKenzie, Chief EPA, Pesticide Branch 100 California Street Room 340 San Francisco, CA. 94111 (415) 556-3352
Dr. Darrell Brock Acting Project Director Epidemiologic Studies Program Bureau of Laboratories Department of Health & Welfare Statehouse Boise, Idaho 83707 (208) 384-2233	AK ID OR WA	X	Robert Poss, Chief EPA, Pesticide Branch 1200 6th Avenue Room 11-C Seattle, WA 98101 (206) 442-1090

APPENDIX 8

PESTICIDE POISONING EXPOSURE HISTORY
(MEDICAL RECORD)

Name of Patient _____
Age _____ Race _____ Sex _____
Address _____
Date of incident _____ Location of incident _____
Taken to _____ Hospital or Clinic
Date of Admission _____
Address _____
Person to contact with results _____ Telephone No. _____

EXPOSURE HISTORY: (Circle Appropriate Information)
Type of pesticide exposure: Ingestion Dermal Inhalation
Was this episode due to: Accidental Exposure Suicide Occupational Exposure
Name of pesticide involved _____
EPA Registration No. _____
Active ingredients _____
Crop pesticide was applied to and target pest _____
Time of last pesticide exposure of patient: Hour _____ Date _____

If Occupational Exposure patient was: (Circle)
Applying Pesticides:
Aerially Ground spray (hand) Spray rig (mechanical)
Or:
Loading Picking crops Mixing Thinning crops
Other _____

SYMPTOMATOLOGY DATA (Please Circle Appropriate Information if Present)
Weakness Sweating Nausea Vomiting Diarrhea
Abdominal Cramps Excessive Tearing Excessive Salivation
Excessive Bronchial Secretions Shortness of Breath Pains in Chest
Blurring of Vision Convulsions Other _____

SIGNS (Please circle appropriate information if present)
Miosis (less than 5 mm) Muscle twitching Muscle fasciculations
Bronchial spasms Bronchial exudation
Cholinesterase Screening Test: Positive Negative
Other _____

SPECIMEN COLLECTION

	Date	Time	
1. Heparinized blood			Collected before 2-PAM Administration? YES NO Atropine Administration? YES NO
2. Urine			
3. Other			

(over)

ADDITIONAL COMMENTS: Such as–

- important details relating to victim's pesticide exposure
- others affected and how
- other damage caused by occurrence.

SHIPPING

Send specimens and this form to one of the following laboratories:

Robert Altman, M.D., M.P.H.
Project Director
Epidemiologic Studies Program
New Jersey State Dept. of Health
John Fitch Plaza
P. O. Box 1540
Trenton, New Jersey 08625
(609) 292-7608

Dr. Ana Barquet
Dept. of Epidemiology and Public Health
University of Miami School of Medicine
P. O. Box 520875
Miami, Florida 33152
(305) 547-6972

E. Gomes
152 E. Stenger
San Benito, Texas 78586
(512) 399-5352

Donald P. Morgan, M.D., Ph.D.
Project Director
Epidemiologic Studies Program
University of Iowa
Oakdale Campus, AMRF
Oakdale, Iowa 52319
(319) 353-5558

D. Darrell Brock
Acting Project Director
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