Texas Agricultural Extension Service
The Texas A&M University System

Physician's Guide to Pesticide Poisoning

Texas Agricultural Extension Service
The Texas A&M University System
Zerle L. Carpenter, Director
College Station, Texas

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Physician's Guide to Pesticide Poisoning

Researched and Written by

Douglass E. Stevenson
Extension Associate—Agricultural Chemicals
The Texas A&M University System

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Texas Agricultural Extension Service
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Reviewed and accepted by

Javier Ballí
Environmental Protection Agency
Extension Liaison
Dallas, Texas

Isaac Cavazos
County Extension Agent
Texas Agricultural Extension Service
Corpus Christi, Texas

Robert De Anda, III
County Extension Agent
Texas Agricultural Extension Service
Dallas, Texas

Hector Gonzalez, M.P.H.
Epidemiology Administrator
San Antonio Metropolitan Health District
San Antonio, Texas

Ventura Gonzalez
Chief Executive Officer
Vida y Salud Health Systems, Inc.
Crystal City, Texas

Fernando A. Guerra, M.D., M.P.H., F.A.A.C.P.
Director of Health
San Antonio Metropolitan Health District
San Antonio, Texas

Alfonso Holguin, M.D., M.P.H.
School of Public Health
UTHSC-Houston
San Antonio, Texas

Carlos A. Moreno, M.D.
South Central Regional Program Director
Health Education Training Centers Alliance of Texas
San Antonio, Texas

Jaqueline Shields, Ph.D.
School of Public Health
UTHSC-Houston
San Antonio, Texas

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Table of Contents

List of Figures

List of Tables

How to Use This Book

This reference gives the physician a quick guide to important sources of information on handling cases of pesticide poisoning. It is divided into four sections.

For general information on formulations, modes of action and chemical classes of pesticides, turn to Section 1.

Section 1 provides general information on pesticide formulations, targets and sites of use, modes of action, and chemical classifications.

For sources of information on pesticide poisoning and treatment, turn to Section 2.

Section 2 provides specific sources of information available to physicians on pesticide poisoning and treatment. This section shows the physician where to find specific clinical information in labels, material safety data sheets (MSDS), statements of formula, toxic inert ingredient statements, telephone emergency hotlines and clinical manuals.

For sources of information on patient management, including references to treatment in Morgan's 1989 edition of *Recognition and Management of Pesticide Poisonings*, turn to Section 3.

Section 3 provides information on patient management and on the recognition and treatment of various types of pesticide poisoning. Tables include brand names, common names, sites of use, and page references to the Morgan manual. The Morgan manual explains specific treatment for many types of pesticide poisoning and should be used as a companion to this guide.

For a list of in-depth references to pesticide toxicology and health hazards, turn to Section 4.

At the end of this guide is a bibliography of our information sources. This also is a good source of in-depth information on pesticide poisoning. For study and reference at a time less urgent than when treating a patient exhibiting symptoms of pesticide poisoning, this section may provide sources that amplify the physician's understanding of pesticide poisoning and clinical toxicology.

This manual is not a clinical guide. It is intended to complement other guides for medical evaluation and treatment of pesticide poisoning. In all cases, **sound medical judgment must prevail.** This manual cannot replace experience, training and diagnostic expertise. It is imperative that physicians fully understand the severity of pesticide health hazards and be prepared to recognize and manage this possible health problem in:

- home pesticide users;
- pesticide applicators and handlers;
- chemical manufacturing, formulating and supply workers;
- farm and ranch workers;
- foresters;
- public health workers; and
- employees of nurseries, greenhouses and agribusinesses.

Many children also are poisoning victims. They may be exposed at home and at play and work sites. Physicians must identify poisoning symptoms quickly and treat them effectively.

Foreword

Pesticide exposure occasionally results in serious illness. A manual such as this is useful to health care professionals who have little experience in recognizing the signs and symptoms of pesticide poisonings, or in treating them. Symptoms of pesticide poisoning often mimic other illnesses or are masked by them. This publication provides a quick reference, with current information on the symptomatology of pesticide exposure and the immediate management and treatment of the patient, as well as a listing of the primary pesticides used on crops grown in Texas. It can be a vital supplement to the primary reference, the Morgan manual. It will not replace the Morgan manual as a resource. However, we hope it will provide more specific information about pesticides used in Texas.

Among all the poisons that present health hazards to the public, pesticides form a comparatively small group. However, this is still a sizeable number. More than 3,000 active ingredients are present in many more registered products. EPA records show more than 100,000 separate registered pesticide products. EPA has canceled registration for more than 40,000 others in the past 5 years. The state of Texas alone has more than 11,000 separate pesticides registered by the Texas Department of Agriculture. They include insecticides, fungicides, herbicides, disinfectants and plant growth regulators.

Recent federal regulations such as the Worker Protection Standard (WPS) make a commitment to protect the health of some agricultural workers. Before WPS, state legislation in California, Texas and Arizona sought to protect farm workers from pesticide health hazards. The Texas Agricultural Hazard Communication Act makes a commitment to protect Texas farm workers but ignores people outside agriculture.

The Federal Community Right-to-Know Act sought to protect a broader segment of the public from chemical hazards. It dealt with poisons in general, not just with pesticides. EPA's pesticide label improvement program also makes an effort to protect a broader segment of the public. When compared to other health problems treated by physicians, pesticide poisoning is rare. The following information is to help physicians and health care workers in recognizing and treating pesticide poisonings. The Texas Agricultural Extension Service of The Texas A&M University System, in conjunction with the U.S. Department of Agriculture, directs educational efforts on pesticide safety through county Extension agents and Extension specialists.

The Health Education Training Centers Alliance of Texas (HETCAT) has among its goals disease prevention and health promotion, especially in the under-served border area. This manual is part of a cooperative effort by HETCAT and the Texas Agricultural Extension Service. Its purpose is to inform health care providers about pesticide health hazards. It is part of an initiative to make more pesticide information available to physicians and health care professionals. HETCAT would be interested in hearing from the users of this manual as to its usefulness. If you would like to write and give us your comments, please use the address below.

Dr. Alfonso Holguin, M.D., M.P.H.
Project Director for HETCAT
University of Texas Health Science Center of San Antonio
7703 Floyd Curl Drive
San Antonio, Texas 78225

Additional copies of this manual are available from:

Health Education Training Centers Alliance of Texas
UTHSC at San Antonio
7703 Floyd Curl Drive
San Antonio, Texas 78225
(512)614-2540

Texas Agricultural Extension Service
Agricultural Chemicals
Agronomy Field Laboratory, Room 115
College Station, Texas 77843-2474
(409)845-3849

or your county Extension agent.

General Information on Pesticides

What Are Pesticides?

A pesticide is a substance or mixture of substances intended to prevent, destroy, repel or mitigate a pest. A pesticide can also be a plant regulator, defoliant or desiccant.

Pesticide Classifications

Pesticides can be classified in four different ways: formulation; target; mode of action; and chemistry. Table 1 shows several common formulations. Table 2 shows pesticide targets or sites of use. Table 3 shows modes of action. Table 4 shows some pesticide chemistries.

Each group of pesticides has its own set of hazards. The insecticides and fumigants are usually the most toxic, but the bipyridyl herbicides also are extremely toxic.

The nerve poisons have several modes of action. The largest numbers of insecticides are cholinesterase inhibitors. Several herbicides, fungicides and plant growth regulators also are cholinesterase inhibitors. These include the organophosphates and carbamates.

Pesticides do not fit into a neat group of chemical families, although the largest numbers of pesticides are in the organophosphate group. The recent development of pesticides that look for specific biochemical targets has produced an enormous diversity in the chemistry of pest control. The second largest class of chemicals registered as insecticides includes a broadly diverse group of

Table 1. Pesticide formulations.

DRY	LIQUID	OTHER
Dusts	Emulsifiable concentrates (ECs)	Controlled release
Granular	Ultra low volume (ULV)	Repellents
Wettable powder	Tech concentrates	Attractants
Soluble powder	Flowables	Impregnated products (collars & tags)
Pellets	MECs	Predator control devices
Feed formulations	Aerosols	Animal systemics (oral, dermal, injectable, implant, feed additive)
Baits	Liquified gas/fumigants	
Fertilizer combinations	Solutions	
Water dispersable granules (WDG)	Paints	
Dry flowable (DF)		

Table 2. Target pests.

INSECTICIDES	HERBICIDES	FUNGICIDES	VERTEBRATE POISONS
Acaricide/Miticide	Algicide	Fungicide	Avicide
Juvenile hormone analog	Brush killer	Silvicide	Piscicide
Larvicide	Defoliant	Slimicide	Predicide
Ovicide	Dessicant		Rodenticide
Pediculicide	Plant growth regulator (PGR)	ANTIMICROBIALS	MISCELLANEOUS
Pheromone	Weed killer	Bactericide	Antifouling paint
Termiticide		Disinfectant	Wood protectant
		Cold sterilant	Fumigant

terpenoid compounds known collectively as pheromones. Ten years ago there were essentially four groups of insecticidal compounds. Now there are more than 15.

Herbicides present an even more bewildering diversity. They are represented in all the insecticide chemical groups and in more than 40 additional groups as well. Fungicides, antimicrobials, vertebrate poisons and other miscellaneous pest control compounds simply add to the array of commercial poisons now on the market.

Table 4 shows the wide variety of pesticides available. To treat many of them a physician must call special pesticide hotlines maintained by chemical companies to deal with poisoning and exposure emergencies.

Table 3. Pesticide modes of action.

Nerve Poisons	
Acetylcholinesterase inhibitors	Organophosphates & Carbamates
Receptor agonists	
NA^+ Channel	Chlorinated hydrocarbons/Pyrethroids
Cl^- Channel	Cyclodienes, Avermectins
Cytotoxins	Nitro & Chlorophenols, Bipyridyls
Allergens	Pyrethrins/Pyrethroids
Mitosis blockers	Thiocarbamates
Sterol inhibitors	Several groups of fungicides
Chitin inhibitors	IGRs and Organophosphate fungicides
Amino acid synthesis blockers	Sulfonylureas
Anticoagulants	Rodenticides (various chemistries)
Asphyxiants	Fumigants (various chemistries)

Table 4. Pesticide chemical structures — alphabetical order.

Acetimides	Fluorodintrotoluidine compounds	Organochlorines
Aliphatics	Halogenated hydrocarbons	Chlorinated hydrocarbons
Benzamides	Heavy metals	Chlorinated terpenes
Benzonitriles	Arsenicals	Cyclodienes
Benzothiadiazone compounds	Copper compounds	Polychlorinated ring compounds
Bipyridyls	Iron compounds	Organophosphate esters
Botanical derivatives	Manganese compounds	Phosphates
Nicotinoids	Mercurials	Phosphinates
Pyrethrins/Pyrethroids	Organotin compounds	Phosphonothioates
Rotenoids	Zinc compounds	Phosphorothioates
Rayanodines	Hexachlorocyclohexanes	Phosphorothionates
Sabadillas	Chlorinated aliphatics	Phosphorothiolates
Carbamates	Miscellaneous compounds	Phosphorodithioates
Dimethyl	Imidazolinones	Phosphoroamidates
Dithio/EBDC	Indole acid analogs	Phosphides
Heterocyclic N-methyl	Benzoic acid derivatives	Phthalates
N-methyl	Hormone-type herbicides	Pyrethroids
Oxime N-methyl	Phenoxys	Substituted ureas
Thio	Picolinic acid derivatives	Sulfonylureas
Carbanilates	Plant growth regulators	Thiophthalimides
Coumarins and Indandiones	IAA & IBA	Triazines
Cyclohexanone derivatives	Inorganic compounds (sulfur,	Triazoles
Damidozides	lime, phosphorus, etc.)	Uracils
Fluoroacetic acid compounds	Nitrophenols & Nitrocresols	

Where to Get Important Information

There are six basic sources that help physicians quickly recognize and treat pesticide poisoning. The most important of these provide information on the nature of the toxic agent, the specific cause of the poisoning, and how to treat poisoning from these compounds. Table 5 lists these sources and the page in this handbook where you can find amplified information about them.

In the order of importance and availability, these sources are labels, material safety data sheets, telephone sources, application records, statements of formula, toxic inert ingredient statements, crop sheets, and manuals on the clinical treatment and toxicology of pesticides.

Pesticide Product Labeling

A pesticide label is the single most important source of information a physician can have when treating pesticide poisoning. It contains all the information the manufacturer provides on a pesticide product. Pesticide labels are legal documents. EPA and the Texas Department of Agriculture must approve labels before a pesticide enters the market.

Labeling includes the label on the product container and package. It also includes brochures, leaflets, bulletins, manuals and any separate information available from pesticide dealers or a recognized authority.

Figure 1 shows the front panel of a category I, restricted-use pesticide manufactured by a theoretical company. Note the standard format and the various parts of the label designed to provide the physician with information.

To physicians, the label is the most important source of information on proper treatment for poisoning cases. However, labels are NOT all the same. Remember to read the label of each product.

In recent years, the EPA has adopted a standard format for pesticide labels. However, not all the information a physician needs is in one place. Instead it is scattered in several places throughout the pesticide label. Knowing where to look on the standard format of a pesticide label enables a physician to quickly find urgently needed information. Almost all of the information of value to a physician is on the front panel of a pesticide label.

Table 6 lists the various parts of a pesticide label, with the parts that are important to a physician in bold letters.

Table 7 shows a condensed list of the parts of a pesticide label important to a physician. The parts less important in treating pesticide poisoning have been omitted. To see the relative positions of these items on the label, see Figure 1 and Table 6.

There is much valuable information in the precautionary statement part of a label. This is particularly true for products with the signal word DANGER on the label. Table 8 shows information valuable to a physician contained in the precautionary statement.

Table 5. Sources of information available to physicians.

SOURCE	INFORMATION	PAGE
Labels	Best source; not always enough detail	3
MSDS	Not always available or up to date	8
Telephone hotline	Available on labels and MSDS	9
Records	Mandated but not always available; provide only product & application data	10
Statements of formula	Usually confidential; available only from manufacturer & EPA through hotline requests	12
Toxic inert statements	Statement on label; formulation data usually confidential, sometimes unknown by manufacturer	12
Other sources	Crop sheets, clinical & toxicity manuals; not always up to date	15

Table 6. Parts of a pesticide label, with sections important to physicians indicated by bold type and stars. Examples are from the label in Figure 1.

PART	EXAMPLE
Classification	Restricted use (appears at top)
Brand name	Galacticarb 15G Aldicarb Granular Insecticide
Product use	Control of Insects, Mites, and Nematodes
***Ingredient statement**	**Aldicarb 15%, common & chemical name**
Net weight	50 pounds
***Formulation**	**(associated with name 15G = granular)**
***EPA reg. & est. numbers**	**190773-213 & 190773-TX-1**
***Name & address of manufacturer**	**Galaxy Chemical**
***Signal word**	**DANGER**
Spanish - English precautions	PELIGRO PRECAUCION AL USARIO
***Emergency telephone no.**	**1-800-465-5141**
***Statement of practical treatment**	**If Swallowed... If in eyes...**
***Note to physician**	**GALACTICARB® ....(left column, bottom)**
***Precautionary statements**	**DANGER... Human & Environmental Hazards**
***Human hazards**	**Hazards to Humans & Domestic Animals**
***Route of entry**	**FATAL IF SWALLOWED.**
***Specific actions for applicators**	**Do not breathe dust...**
***Protective equipment**	**Wear long-sleeved...**
***Other precautions**	**Bathe at the end of work day...**
***Physical & chemical hazards**	**Fire hazard when used with metal feed wheel.**
***Signs and symptoms**	**Salivation, Muscle tremor, Nausea...**
***ANTIDOTE STATEMENT**	**ATROPINE SULFATE IS HIGHLY EFFECTIVE...**
Environmental hazards	Aldicarb residues...
Hazards to wildlife	This product is toxic to fish,...
General use statement	It is a violation of Federal law to use...
Reentry statement	Do not reenter...
Storage and disposal	Store unused GALACTICARB® in...

Physicians may have to sort out information on older pesticide labels. A product in a suspected poisoning case may have been packaged many years ago, before present EPA requirements took effect. Many homeowners keep pesticides on shelves for years. An older label may not have all the information a physician needs, but it will at least have the brand name, active ingredients (no toxic inerts) and signal word. It may have some of the precautionary statements, signs of poisoning, and notes to physician, but not necessarily.

There is much to learn from the label, including the product's chemical hazards, registered uses, recommended rates, compatibility with other substances, and phytotoxicity. Information on the label generally pertains to either product identification or proper product use.

Parts of a Pesticide Label

Pesticide labels have several parts. Certain statements and blocks of information must be on every pesticide label. Labels of highly toxic pesticides require additional information. Some labels require special warnings and precautionary statements. Parts of a label most important to physicians are in Table 7.

Table 7. Parts of pesticide labels important to physicians.

PART	INFORMATION
Classification	Restricted or general use
Brand name	Manufacturer & product name
Statement of ingredients	Active & toxic inert ingredients: common & chemical name
EPA reg. no.	Identification of pesticide
Signal word	Relative toxicity
Emergency telephone numbers	Hotline information on treatment
Statement of practical treatment	First aid
Note to physician	Specific therapeutic instructions
Precautionary statements	Specific statements on poisoning & treatment

Table 8. Precautionary statement information valuable to physicians.

LABEL STATEMENT	INFORMATION
Signal word (restated)	Relative toxicity
Hazards to humans & domestic animals	Specific hazards and handling
Symptoms of poisoning	Signs & symptoms of overdose
Antidote statement	Antidote, if any
Physical & chemical hazards	Specific ingredient hazards

Brand name

Physicians must beware of using brand names to identify chemicals. Instead, always use active ingredient statements on labels.

Table 9 shows nearly identical brand names with very different active ingredients. Table 10 shows pesticides with very different brand names having identical active ingredients. **Physicians must not simply rely on the memory of poisoning victims or their coworkers who may give only the brand name of a pesticide product. The product label is the only way to find out what the active ingredients are.**

Classification

EPA classifies every use of every pesticide as either "general use" or "restricted." Restricted-use pesticides carry the statement at the top of the label in Figure 1. As the message at the top of the label implies, purchasers and users of restricted-use pesticides must be licensed.

Net contents and ingredient statement

The front panel of the label shows the net contents—that is, how much product is in the container. Each label also must list what is in the product. The list shows active and inert ingredients. It also shows the amount of each.

Active ingredients are the chemicals that control the target pest. They must be identified by their chemical names or official common names. Most products also have inert (inactive) ingredients. These do not act on the target pest. However, they may contribute to poisoning (see "Toxic Inerts" later in this section).

Except in the case of biological and botanical pesticides, the ingredient statement lists chemical names. Many chemical names are called by a shorter common name. Common names may be used in the ingredient statement only if they are accepted by EPA. Figure 1 shows examples of common and chemical names.

Type of pesticide and formulation

Pesticide type usually is listed on the front of the label. This short statement tells what kind of pests the product controls. Figure 1 shows an example of a soil insecticide in a granular formulation. There are many formulations.

The more common formulations have accepted abbreviations. Table 11 shows some of the abbreviations for common pesticide formulations.

Registration and establishment numbers

These numbers identify specific pesticide formulations and where they come from. The registration number identifies a specific formulation with a specific set of inert ingredients. Establishment numbers identify where a pesticide was made. These numbers are necessary in case of accidental or deliberate poisoning. These numbers also are necessary in the case of claims of misuse, faulty products or liability.

Table 9. Tersan formulations — same brand name for different active ingredients.

BRAND NAME	MANUFACTURER	ACTIVE INGREDIENT
Tersan LSR®	DuPont	zinc and maneb
Tersan SP®	DuPont	chlorineb
Tersan 1991®	DuPont	benomyl
Tersan®	DuPont	thiram

Table 10. Glyphosate formulations — different brand names, but the same active ingredients.

BRAND NAME	MANUFACTURER	ACTIVE INGREDIENTS
Accord®	Monsanto	glyphosate [41.5%]
Honcho®	Monsanto	glyphosate [41.0%]
Ranger®	Monsanto	glyphosate [28.6%]
Rodeo®	Monsanto	glyphosate [53.8%]
Roundup®	Monsanto	glyphosate [41.0%]
Roundup RT® Shuttle 100®	Monsanto	glyphosate [41.0%]
Turfgo Weed Wrangler®	UHS	glyphosate [41.5%]

Table 11. Abbreviations for common pesticide formulations.

FORMULATION	ABBREVIATION	FORM
Emulsifiable concentrate	E, EC	liquid
Wettable powder	W, WP	powder
Soluble powder	S, SP	powder
Dry flowable	DF	granule
Water dispersable granule	WDG	granule
Flowable liquid suspension	F, L	liquid
Granular	G	granule
Dust	D	dust
Ultra low volume concentrate	ULV	high % liquid
Pellet	P, PS	pellet

An EPA registration number appears on **all** pesticide labels. Most products contain only two sets of numbers. The example in Figure 1 shows EPA Reg. No. 190773-213. The first set of digits, 190773, identifies the manufacturer. The second set, 213, identifies the product. Examples of a few manufacturer or registration numbers appear in Table 12. Each time a pesticide changes hands, the manufacturer number changes to follow it. Frequently, the product number changes also. **Physicians must be aware of possible changes in registration numbers.**

The establishment number, EPA Est. No. 190773-TX-1, appears on either the label or container. The number identifies the facility that produced or formulated the product. Pesticide labels also must have the name and address of the manufacturer. A maker or distributer of a product must list its full company name and address (see Figure 1). **Physicians can use the name and address on the label to contact pesticide manufacturers in case of emergencies.**

Product use

The remaining parts of the label pertain to proper product use rather than product identification. These important parts of the label include signal words and symbols, precautionary statements, storage and disposal instructions and directions for use (see examples in Table 6).

Table 12. Registration numbers associated with pesticide manufacturers.

REGISTRANT	NUMBER
Abbott Laboratories	275
American Cynamid	241
BASF	7969
Ciba-Geigy	100
DowElanco	62719
DuPont	352
Elf Atochem	4581
FMC	279
Hoechst-Roussel	8340
ICI Americas	10182
ISK Biotech	50534
Miles	3125
Monsanto	524
Rhone-Poulenc	264
Rhom and Haas	707
Sandoz	55947
Uniroyal	400
Valent	59639

Signal word

Every label has a signal word required by the EPA. These are "DANGER," "WARNING" or "CAUTION." This word gives a signal of how dangerous the product is to humans. The signal word does not tell the risk of delayed effects or allergic reactions. The signal word appears in large letters on the front of the label—usually next to the statement, "Keep Out of Reach of Children," which is required on every product.

DANGER signals that the pesticide is highly toxic or could cause severe eye or skin injury. Highly toxic pesticides also carry the skull and crossbones symbol and the word POISON printed in red. Pesticides that can badly damage the skin or eyes may have the signal word DANGER without the word POISON. Typical statements on labels with the DANGER signal word are:

- Fatal if swallowed.
- Poisonous if inhaled.
- Extremely hazardous by skin contact—rapidly absorbed through skin.
- Corrosive—causes eye damage and severe skin burns.

WARNING signals any product that is moderately toxic. CAUTION signals any product that is slightly toxic.

Route of entry

This notice follows the signal word and tells which route of entry (mouth, skin, eyes, lungs) needs special protection.

Specific actions

In addition to route of entry, the label may list specific actions needed to prevent poisoning accidents. These include things to avoid and the kind of protective equipment to wear. Figure 1 shows several, including "Do not breathe dust."

Protective clothing and equipment

Some labels fully describe protective clothing that should be worn and equipment that should be used when handling the pesticides, or that doctors should use when handling contaminated victims. Many labels have no such message.

Other precautions

Labels often list other precautions to take. The precautions pesticide users should always take, whether or not they are stated on the label, include:

- Remove and wash contaminated clothing before use.
- Wash thoroughly after handling and before eating or smoking.
- Wash clothes daily.

First aid, note to physician, and statement of practical treatment

These statements tell physicians and emergency personnel what to do in case of pesticide poisoning. See the statement of practical treatment and the note to physician in the example in Figure 1.

All DANGER labels must include sections for first aid treatment, poison signs or symptoms, a note to physicians (or antidote), and an emergency assistance call telephone number. These are not all in the same place. See Table 6 for relative positions of each of these statements. WARNING and CAUTION labels may have only an emergency assistance call telephone number.

Physicians should advise patients with suspected pesticide poisoning to bring the pesticide label with them. This is very important. The pesticide label can provide the physician with most of the information necessary to save the life of someone suffering from pesticide poisoning. Without the label, treatment of many pesticide poisonings will be difficult.

Hazards to wildlife and the environment

Some products are classified "restricted use" because of environmental hazards alone.

General environmental statements

These statements appear on almost all pesticide labels.

Physical and chemical hazards

The physical and chemical hazard section of the label warns pesticide users of any fire, explosion or chemical hazards.

Reentry statement

Some pesticide labels contain a reentry precaution. This tells the applicator how much time must pass before people can reenter a treated area without protective clothing.

Storage and disposal

All labels give general instructions for proper storage and disposal. Table 6 and Figure 1 show examples of storage and disposal statements.

Directions for use

This section of the label gives specific information on how to use the product, including:

- Pests the product is intended to control, and crops, animals or sites to which the product may be applied.
- Precautions to take and proper equipment to use.
- Mixing and application directions, and the product's compatibility with other materials.
- When and where the product should be applied.

Any use other than that stated on the label is misuse. Misuse is the cause of many cases of accidental poisoning.

Physicians may find keys to poisoning and how it occurred by looking in the directions for use part of the label.

Material Safety Data Sheets

The Occupational Safety and Health Act (OSHA) requires businesses that store and use chemicals to maintain several records. One of these is a workplace chemical list. The other is a file of Material Safety Data Sheets (MSDS). A MSDS is a document that lists various characteristics of a chemical that may contribute to its safety in storage, transportation, use or disposal. A MSDS has the following sections (with those most important to physicians in bold type).

- **Chemical emergency call number (CHEMTREC), (800) 424-9300**
- **Identification of product**

  manufacturer's name and address, product trade name and synonyms,

  chemical name and synonyms, chemical family

- **Hazardous ingredients or mixtures**

  active ingredients, some inert ingredients, threshold limit values

- **Physical data**

  product's appearance, odor and chemical constants

- **Fire and explosion hazard data**

  information helpful to fire departments, police and other emergency response personnel

- **Reactivity data**

  product's stability, incompatibility with other materials, hazards during decomposition

- **Health hazard data**

  effects of overexposure (routes of entry, most likely route of entry, toxicity of active ingredient or LD_{50})

  emergency and first aid procedures

  note to physician (nature of poisons, patient management and supervision, antidotes, contraindications for certain pharmaceutical products)

  medical conditions aggravated by exposure

  potential carcinogen status

- **Spill or leak procedures**

  actions to take if material is released or spilled

- **Special protection information**

  help in determining how poisoning occurred, and in handling contaminated patients

- **Special precautions**

  precautions to take when handling or storing the product

- **Date of issue**

  date of the MSDS and any previous MSDS the present one supersedes

Telephone Sources

There are national, state and regional poison control center emergency numbers, as well as company telephone hotlines for chemical emergencies. Poison control centers have general clinical information readily available. Company hotlines can inform physicians about the specifics of their products. These company hotlines are the only way to get information about possible toxic effects of inert ingredients. Table 13 lists pesticide information hotline numbers. Table 14 lists some chemical company information numbers and hotlines taken

Table 13. Telephone numbers for pesticide poisoning information.

National Pesticide Telecommunication Network	(800) 858-7378
Texas State Poison Control Center	(800) 392-8548
Local Poison Control Offices:	
Amarillo	(806) 354-1100
Conroe	(409) 539-7700
Corpus Christi	(512) 881-4559
Dallas	(800) 441-0040
El Paso	(915) 533-1244
Lubbock	(806) 793-4366
Odessa	(915) 653-1231
Plainview	(806) 396-5900
San Angelo	(915) 653-6741
Tyler	(903) 531-8082
Tyler	(903) 597-8884
Wichita Falls	(817) 322-6771
Poison Control Center Toxicology	(713) 792-4300
Texas Department of Agriculture	
Right-to-Know	(512) 463-7547
Pesticide Enforcement	(800) 832-7348
Texas Department of Health	(800) 252-8239
Texas Agricultural Extension Service	(409) 845-3849

Table 14. Corporate hotlines for pesticide poisoning information.

American Cyanamid	(201) 835-3100
BASF	(800) 832-HELP
Ciba-Geigy	(800) 888-8372
Cleary Chemical	(908) 247-8000
DuPont	(800) 441-3637
DowElanco	(517) 636-4400
Elf Atochem	(215) 587-7000
Fermenta ASC	(216) 357-7070
FMC	(800) 331-3148
Grace Sierra	(408) 263-8083
Gustafson	(214) 985-8877
HACO, Inc. (Hopkins)	(608) 221-6200
Hess and Clark	(800) 424-8802
	(419) 289-9129
Hoechst-Roussel	(908) 231-4125
	(517) 636-4400
ISK Biotech	(216) 257-7070
Lesco	(216) 333-9250
Loveland Industries	(303) 356-8920
Miles	(816) 242-2582
Monsanto	(314) 694-4000
Nor Am	(302) 575-2000
PBI Gordon	(816) 421-4070
	After hours (913) 342-8783
Platte Chemical	(303) 356-4400
Rhom and Haas	(215) 592-3000
Rhone-Poulenc	(800) 334-7577
Riverdale	(708) 754-3330
Sandoz Agro, Inc.	(708) 699-1616
Scentry	(406) 248-5856
	(602) 386-6737
O.M. Scott (Scotts Company)	(513) 644-0011
Titre, Inc.	(406) 568-2914
Uniroyal	(203) 723-3670
United Horticultural	(303) 356-4400
Zeneca (ICI Americas)	(FASTMED) (800) 322-8633

from the Material Safety Data Sheets (MSDS) for their products.

Company hotlines can provide specific information about pesticide formulations, hazards and treatment. DANGER labels must all bear emergency hotline numbers. The emergency numbers on DANGER labels are the only way to get information on medical treatment for chemicals not listed in the active ingredients. Physicians should not hesitate to call these numbers for information on pesticides.

Pesticide Records Users Must Keep

When pesticide poisoning or exposure is suspected, physicians must know what pesticides have been applied. If labels and MSDS are not available, physicians can request other records.

Pesticide users, haulers, applicators, distributors, retailers and manufacturers are required by law to keep records of the materials they handle, and to

RESTRICTED USE PESTICIDE

Due to very high acute toxicity to Humans and Birds.
For retail sale to and use only by certified applicators or persons under the direct supervision of a certified applicator, and only for those uses covered by the certified applicators certification.

GALACTICARB 15G

Aldicarb Granular Pesticide

For control of certain insects, mites, and nematodes
ACTIVE INGREDIENTS: Aldicarb [2-methyl-2-(methylthio) propionaldehyde *O-(methylcarbamoyl)oxime]* 15%
Related Compounds 2%
INERT INGREDIENTS: 83%

TOTAL 100%

EPA Reg. No. 190773-213 EPA Est. No. **190773-TX-01**

NET WEIGHT 50 POUNDS

Formulated for GALAXY CHEMICAL CORP.
Bairds Pond, TX 79715

KEEP OUT OF REACH OF CHILDREN
DANGER — POISON

PELIGRO

PRECAUCION AL USARIO: Si usted no lee ingles, no use este producto hasta que la etiqueta haya sido explicada ampliamente.

TRANSLATION TO THE USER: If you cannot read English, do not use this product until the label has been fully explained to you.

For MEDICAL and TRANSPORTATION Emergencies
ONLY Call 24 hours a day 1-800-456-5141
For Product Use Information Call 1-800-465-9440

STATEMENT OF PRACTICAL TREATMENT

IF SWALLOWED: Drink 1 or 2 glasses of water and induce vomiting by touching back of throat with finger. Do not induce vomiting or give anything orally to an unconscious or convulsing person. Call a physician and follow General Advice.

IF IN EYES: Flush eyes with plenty of water and get medical attention.

IF ON SKIN: Wash thoroughly with soap and water. Remove contaminated clothing and wash before reuse.

IF INHALED: Call a physician and follow General Advice.

GENERAL ADVICE

Contact a physician immediately in all cases of suspected poisoning. Illness may be produced rapidly following overexposure to GALACTICARB® Aldicarb. If breathing stops, establish an airway and provide oxygen. Make certain to remove all sources of continuing contamination. Remove clothing and wash skin and hair immediately with large amounts of water. Transport the patient to a physician or hospital immediately and **SHOW A COPY OF THIS LABEL TO THE PHYSICIAN.** If poisoning is suspected in animals contact a veterinarian.

NOTE TO PHYSICIAN

GALACTICARB® Aldicarb is a methyl carbamate insecticide which is a cholinesterase inhibitor. Overexposure to this substance may cause toxic signs and symptoms due to stimulation of the cholinergic nervous system. These effects of overexposure are spontaneously and rapidly reversible.

Gastric lavage may be used if this product has been swallowed. GALACTICARB® Aldicarb poisoning may occur rapidly after ingestion, and prompt removal of stomach contents is indicated. Specific treatment consists of the administration of parental atropine sulfate. Caution should be exercised to prevent over-atropinization. Mild cases may be given 2 to 4 mg intravenously every 10 minutes until the patient is fully atropinized, then intramuscularly every 30 to 60 minutes as needed to maintain the effect for at least 12 hours. Dosages for children should be appropriately reduced. Complete recovery for overexposure is to be expected within 24 hours.

Narcotics and other sedatives should not be used. Further, drugs such as (pyridine-2-aldoxime methiodide) are NOT recommended unless organophosphate intoxication is also suggested.

To aid in confirmation of diagnosis, urine samples must be obtained within 24 hours of exposure and immediately frozen. Analyses will be arranged by Galaxy Chemical Corporation. Consultation on therapy can be obtained at all hours by calling the Galaxy emergency number 1-800-465-5141.

PRECAUTIONARY STATEMENTS

DANGER

HAZARDS TO HUMANS AND DOMESTIC ANIMALS

FATAL IF SWALLOWED. Causes cholinesterase inhibition. May be fatal or harmful by skin or eye contact or by breathing dust. Rapidly absorbed through skin or eyes. Do not get on skin or in eyes. Do not breathe dust. Keep away from domestic animals.

Wear long-sleeved clothing, full-length trousers, head coverings, and protective gloves when handling. Always stand up-wind from hopper when loading. Wash hands and face before eating, drinking, or using tobacco. Bathe at the end of work day, washing entire body and hair with soap and water. Change clothing daily. Wash contaminated clothing in strong washing soda solution and rinse thoroughly before reusing.

PHYSICAL AND CHEMICAL HAZARDS

Fire hazard when used with metal feed wheels or augers. Do not use with granule applicators which have metal gear feeders or augers.

SIGNS AND SYMPTOMS OF OVEREXPOSURE:

Salivation, Muscle tremor, Nausea, Watery eyes, Difficult breathing, Vomiting, Pinpoint eye pupils, Excessive sweating, Diarrhea, Blurred vision, Abdominal cramps, Weakness, Headache. In severe cases, convulsions, unconsciousness and respiratory failure may occur.

ANTIDOTE STATEMENT

ATROPINE SULFATE IS HIGHLY EFFECTIVE AS AN ANTIDOTE.
See NOTE TO PHYSICIAN.

ENVIRONMENTAL HAZARDS

Aldicarb residues may move into shallow groundwater under certain conditions. The appended Environmental Precautions Booklet should be read and understood prior to making applications. If there are any questions, contact Galaxy Chemical Corp. at 1-800-465-9440.

TOXIC TO FISH, BIRDS, AND WILDLIFE: This product is toxic to fish, birds, aquatic invertebrates and other wildlife. Birds feeding on exposed granules in treated areas may be killed. Core or incorporate granules in spill areas. Runoff from treated areas may be hazardous to fish in neighboring areas. Do not apply directly to water or wetlands. Do not contaminate water when disposing of equipment washwaters. Apply this product only as specified on this label.

GENERAL DIRECTIONS FOR USE

It is a violation of federal law to use this product in a manner inconsistent with the label.

REENTRY STATEMENT

Do not reenter treated area for 24 hours unless protective clothing, gloves and goggles are worn.

STORAGE & DISPOSAL

Store unused GALACTICARB® in original container only. Do not store in or around the home.

PESTICIDE DISPOSAL

Do not contaminate water, food, or feed by storage or disposal. Open dumping is prohibited. Pesticide wastes are acutely hazardous. Improper disposal of excess pesticide is a violation of federal law.

CONTAINER DISPOSAL

Completely empty container in application equipment. Then dispose of empty container in a sanitary landfill or by incineration, or, if allowed by state and local authorities, by open burning. If container is burned, stay away from and do not breathe or contact smoke.

Figure 1. Sample pesticide label

provide records to physicians and other emergency personnel. Physicians and health care professionals may request pesticide records to assist in diagnosis and treatment of suspected pesticide poisoning.

One law designed to protect workers by requiring employers to keep records is the Hazard Communication Standard (HCS), a rule written and enforced by OSHA. Another is The Superfund Amendments and Reauthorization Act (SARA Title III). It is sometimes known as "The Community Right-to-Know Act." It is a federal right-to-know law that affects those who produce or store hazardous chemicals. Section 311 covers the reporting of Material Safety Data Sheets. It is one physicians should know about. Under Section 311, employers must obtain and keep Material Safety Data Sheets and submit copies of each sheet, or a listing, to their local fire department, the Local Emergency Planning Committee and the State Emergency Response Committee. Household and agricultural chemicals are excluded from this rule.

There are other federal and state laws concerning the keeping of pesticide records. Some of these are the 1990 Federal Farm Bill, the Federal Worker Protection Standard, the Texas Agricultural Hazard Communication Law and Regulations, Texas Pesticide Laws and Regulations, and Texas Herbicide Laws and Regulations.

Statements of Formula

Pesticide formulations consist of a blend of ingredients. The active ingredients are usually the only ones listed on the label and MSDS. The rest are inert ingredients. These are listed for registration purposes in the confidential statement of formula. Physicians or the general public seldom, if ever, are made aware of inert ingredients in pesticides. An exception is in the EPA policy on toxic inert ingredients.

In the confidential statement of formula, EPA requires the registrant to list all ingredients that go into a pesticide formulation. These ingredients include all active and inert ingredients, the weight per batch, the percentage in the formulation, and sources of the ingredients. Table 15 shows a facsimile of EPA Form 8750-4 with a confidential statement of formula. This statement is exactly what it says—confidential. Only the registrant and EPA know what is in it. It is not available to the public, medical professionals or emergency personnel. In a medical emergency the pesticide registrant can be asked to provide information on the inert ingredients in a product, but such information is not easy to get. Sometimes it is impossible to get, particularly when formulators use inert ingredients from suppliers who do not divulge their contents and regard them as trade secrets.

Toxic Inert Ingredients

Some inert ingredients are themselves toxic. Because of concern that some inert ingredients in pesticide products might cause adverse effects to people or the environment, EPA developed a strategy for the regulation of inert ingredients. The EPA has several lists of inert ingredients used in pesticide formulations. Tables 16 and 17 show toxic inerts on EPA List 1 and List 2. List 1 contains toxic inert ingredients with known toxicities. List 2 contains toxic inert ingredients with undetermined toxicities and a high priority for testing. Only ingredients on List 1 require a warning on the front panel of the product label. The warning will be near the ingredient statement in a similar type size.

> This product contains the toxic inert ingredients (name of toxic).

EPA has been reluctant to register any formulation with inert ingredients on List 2. However, to date no toxic inert warning statement has been required for inert ingredients on List 2.

Table 15. Confidential statement of formula (CSF) for a hypothetical insecticide, Galactothion, on a facsimile of EPA Form 8750-4. Note toxic inert ingredients Rhodamine B and Phenol.

Confidential Business Information: Does Not Contain National Security Information (E.O. 12065)

Form Approved OMB No. 2070-0060. Approval expires 11-30-93

EPA — United States Environmental Protection Agency, Office of Pesticide Programs (TS-767), Washington, DC 20460

Confidential Statement of Formula

A. ☐ Basic Formulation ☐ Alternate Formulation

B. Page of

See Instructions on Back

1. Name and Address of Applicant/Registrant (Include Zip Code)
GALAXY CHEMICAL, INC.
2527 South GALAXY Drive
Bairds Pond, TX 79715

2. Name and Address of Producer (Include ZIP Code)
GALAXY CHEMICAL, INC.
2527 South GALAXY Drive
Bairds Pond, TX 79715

3. Product Name
GALAXY
Galactothion 5 EC

4. Registration No./File Symbol: 12345-10

5. EPA Product Mgr./Team No.: 15

8. Country Where Formulated: U.S.A.

7. Pounds/Gal or Bulk Density: 2

8. pH: 7.0

9. Flash Point/Flame Ext.: 143° C.

EPA USE ONLY	10. Components in Formulation (List as actually formulated into the formulation. Give commonly accepted chemical name, trade name, and CAS number.)	11. Supplier Name and Address	12 EPA Reg. No.	13. Each Component in formulation. a. Amount.	b. % by Weight	14. Certified Limits % by Weight		Purpose
	O,O-diethyl methyl phosphorothioate Related compound	Makhteshim Beer Sheva Chemical Works Ltd. 2 Park Ave. New York, NY 10016	11678-298	68.5 4.5	68.5 4.5	69 5	68.5 4.5	Insecti-cide
	Dichlorvos Related compound	AMVAC Chemical Corp. 41 E. Washington Blvd Los Angeles, CA 90023	5481-781	0.93 0.07	0.93 0.07	1.2 0.1	1.116 0.084	Insecti-cide
	Xylene	Exxon Chemical 8230 Stedman St. Houston, TX 77001		13.43	13.43	13.5	13.0	Solv-ent
	1,1,1-Trichloroethane	Tesco Chemicals 827 Central Ave. Metarie, LA 70005		5.0	5.0	6	5.0	Car-rier
	T-Mulz-O	Thompson Hayward 801 W. 21st Tulsa, OK 74107		4.5	4.5	5	4.5	Emulsi-fier
	T-Mulz-W	Thompson Hayward 801 W. 21st Tulsa, OK 74107		2.5	2.5	3.0	2.5	Emulsi-fier
	Rhodamine B	Gustafson, Inc. Box 660065 Dallas, TX 75266		0.5	0.5	0.6	0.5	Color
	Phenol	Dexol Industries 1450 W. 228th St Torrance, CA 90501	192-7484	0.07	0.07	.08	0.06	Pres-erve-ative
				17. Total Weight 100	100 %			

16. Typed Name of Approving Official: Virgil I. Pierce

18. Signature of Approving Official

19. Title: Agent

20. Phone No. (Include Area Code): (213) 446-2137

21. Date: 09/23/93

EPA Form 8750-4 (Rev. 12-90) Previous editions are obsolete.

White - EPA File Copy (original) Yellow - Applicant Copy

Table 16. EPA List 1 — Inert ingredients of toxicological concern.

- Aniline
- Asbestos
- Benzene
- 1,4-Benzenediol
- Beta-butyrolacetone
- Cadmium compounds
  - Cadmium cocoate
  - Cadmium-barium laurate
  - Cadmium 2-ethylhexanoate
  - Cadmium perborate
  - Cadmium toluate
- Carbon disulfide
- Carbon tetrachloride
- Chlorobenzene
- Chloroform
- 2,2-Dichlorovynyl dimethyl phosphate (Dichlorvos, DDVP)
- Diethylhexylphthalate
- 1,1-Dimethylhydrazine
- 1,2-Dimethylhydrazine
- Dinitro-o-cresol
- Dinitrophenol
- Dioxane
- Epichlorohydrin
- Ethylene dichloride
- 2-ethyoxy-ethanol (cellosolve)
- 2-methoxy ethanol (Methyl cellosolve)
- Ethanol ethoxy acetate
- Ethyl acrylate
- Ethylene glycol monomethyl ether (methyl cellusolve)
- Ethylene thiourea
- Ethyl methyl phenylglycidate
- Formaldehyde
- Hexachlorophene
- n-Hexane
- Hydrazine
- Isophorone
- Lead compounds
  - Lead chromate
  - Lead naphthalate
  - Lead oxide
  - Lead stearate
- Malachite green
- Mercury oleate
- Methyl-n-butyl ketone
- Methyl chloride
- Methylene chloride
- 2-Nitropropane
- Nonylphenol
- Paradichlorobenzene (PDB)
- Paraformaldehyde
- Pentachlorophenol
- Perchloroethylene
- Phenol
- o-Phenylphenol
- Propylene dichloride (1,2-Dichloropropane)
- Propylene oxide
- Pyrethrins and Pyrethroids
- Rhodamine B
- Sodium dichromate
- Sodium pentachlorophenate
- Thiourea
- Toluene diisocyanate
- Tributyltin oxide (TBTO)
- 1,1,2-Trichloroethane
- Trichloroethylene
- Tricresyl phospate
- Tri-o-cresyl phosphate

Table 17. EPA List 2 — Potentially toxic inerts/high priority for testing.

Acetonitrile	Diethylene glycol monomethyl ether
1,2,3-Benzotriazole	Diethyl phthalate
2-Benzyl-4-chlorophenol	1,1-Difluoroethane
2-Butoxyethanol	Dimethyl formamide
Butyl benzyl phthalate	Dimethyl phthalate
Butyl methacrylate	Dioctyl phthalate
Butylene oxide	Dioctyl sodium sulfosuccinate
gamma-Butyrolactone	Diphenyl ether
1-Chloro-1,1-difluoroethane	Dipropylene glycol monomethyl ether
Chlorodifluoromethane	Ethyl benzene
Chloroethane	Isopropyl phenol
2-Chlorotoluene	Mercaptobenzothiazole
p-Chlorotoluene	Mesityl oxide
p-Chloro-m-xylenol	Methyl bromide
Cresol	Methyl isobutyl ketone
o-Cresol	Methyl methacrylate
m-Cresol	Nitroethane
p-Cresol	Nitromethane
Cyclohexanone	p-Nitrophenol
Dibutyl phthalate	Petroleum hydrocarbons
2,5-Dichloroaniline	1,2-Propylene glycol 1-monobutyl ether
3,4-Dichloroaniline	Propylene glycol monobutyl ether
3,5-Dichloroaniline	Propylene glycol monoethyl ether
2,4-Dichloroaniline	Propylene glycol monomethyl ether
2,3-Dichloroaniline	Toluene
2,6-Dichloroaniline	Tolyl triazole
o-Dichlorobenzene	1,1,1-Trichloroethane
Dichloromonofluoromethane	Triethanolamine
Dichlorophene	Tripropylene glycol monomethyl ether
Diethanolamine	Xylene
Diethylene glycol monobutyl ether	Xylene range aromatic solvents
Diethylene glycol monoethyl ether	

Crop Sheets

The Texas Department of Agriculture, through the Texas Right-to-Know Law, requires that agricultural employers who qualify under the Texas Agricultural Hazard Communication Act must provide workers with pesticide safety training and give them crop sheets for the crops they work in. The crop sheet is printed in Spanish and English. It gives certain general information about pesticides used in particular crops in various regions of Texas. The crop sheet also contains specific pesticide information, such as reentry intervals. Crop sheets also list symptoms of poisoning and poison control center hotline numbers. The crop sheet can provide valuable information to physicians treating agricultural workers.

Clinical Manuals and Toxicological Guides

Among the valuable sources of information on toxicology and treatment are Hayes' three-volume set on pesticide toxicology (1991) and Morgan's manual. Another excellent source, although somewhat out of date, is Gosselin's ***Clinical Toxicology of Commercial Products*** (1984). Table 18 lists some references on diagnosis and treatment of pesticide poisoning.

Table 18. References on pesticides, pesticide toxicology, and clinical toxicology.

Dreisback, Robert H. and William O. Robertson. 1987. *Handbook of Poisoning.* Appleton and Lange: East Norwalk, CT.

Ellenhorn, Matthew J. and Donald G. Barceloux. 1988. *Diagnosis and Treatment of Human Poisoning.* Elsevier: New York, NY.

Gosselin, Robert E., Roger P. Smith and Harold Hodge, with assistance of Jeanette E. Braddock. 1984. *Clinical Toxicology of Commercial Products.* Williams and Wilkins: Baltimore, MD.

Hayes, W.J. and E.R. Laws. 1991. *Handbook of Pesticide Toxicology*, volumes 1-3. Academic Press, Inc: New York, NY.

Morgan, Donald P. 1989. *Recognition and Management of Pesticide Poisonings.* United States Environmental Protection Agency. U.S. Government Printing Office, Washington, DC.

Rumak, Barry H., ed. 1989. *The Pharmacological Basis of Therapeutics*, 7th ed. Macmillan Publishing Co.: New York, NY.

Sine, Charlotte, ed. 1993. *Farm Chemicals Handbook* (published annually). Meister Publishing Co.: Willoughby, OH.

Thomson, W.T. 1993-1995. *Agricultural Chemicals Books*, volumes 1-4. Thomson Publications: Fresno, CA.

Wagner, Sheldon L. 1981. *Clinical Toxicology of Agricultural Chemicals.* Oregon State University Press: Corvallis, OR.

Wildholz, Martha and Susan Budavari, eds. 1992. *The Merck Index.* Merck and Co., Inc.: Rahway, NJ.

Patient Management

The following section provides an overview of pesticide poisoning toxicology, recognition and management, by pesticide categories. Regardless of the product involved, there are certain precautions that must be taken with all potential pesticide exposure cases.

Acute Poisoning and Exposure Recommendations for Patient

1. If the patient is suffering from dermal exposure, wash the patient with soap and water immediately.
2. Transport the patient to the nearest doctor, hospital or clinic. A family member, friend, coworker or employer should drive. The patient must not drive.
3. Take the pesticide label to the doctor whenever possible.
4. Inform the physician of suspected exposure to pesticides.
5. The physician should request the pesticide label, Material Safety Data Sheets (MSDS) and other records. For farm workers physicians also should request crop sheets.
6. Physicians and other health professionals have the right to ask employers for any pertinent information. This includes Workplace Chemical Lists, logs, precautions, names of pesticides used, and EPA registration numbers. Above all, call a poison control center for emergency instructions. Also use the pesticide hotline on the pesticide label or MSDS.

To avoid exposure or poisoning, workers must strictly follow application and handling instructions, and take the precautions listed on labels. These precautions are frequently mandated by laws or regulations. When poisoning of farm workers, pesticide applicators, mixers, loaders or handlers occurs, it is usually because they did not follow label precautions.

NOTE: Special attention should be given to children suffering from pesticide poisoning. This can occur through direct contact with or ingestion of household chemicals. It also can occur through exposure to residues in the house, on lawns or in gardens. It may occur through contact with family members who are farm laborers when workers come in from the field. Or it may occur through contact with family members who have applied lawn or garden pesticides. It may occur when contaminated clothing is washed with the family laundry. The NAS (1993) report on pesticides shows that acute and chronic toxicity thresholds can be lower for children than for adults. Antidote recommendations and dose levels for children also may be different than for adults.

For detailed information on treatments consult the Morgan (1989) manual. Gosselin (1984) also may prove useful in diagnosis, treatment and therapy.

Organophosphates (insecticides)

Toxicology Acetylcholine accumulation is caused by irreversible phosphorylation of the acetylcholinesterase enzyme.

Chemical Effects Cholinergic junctions produce muscarinic effect on smooth muscles and gland cells, causing muscle contractions and secretions.

Nicotinic junctions produce excitatory effects on skeletal muscles and autonomic ganglia, but can weaken or paralyze the end plate cells.

Brain effects include sensory and behavioral disturbance, incoordination and depressed motor function.

Respiratory depression and pulmonary edema are usual causes of death.

Exposure

Reported cases of pesticide poisoning involving children are usually organophosphate poisonings. Routes of exposure include inhalation, ingestion and dermal absorption.

Symptoms/Signs

Symptoms develop within 12 hours of exposure (average onset is within 4 hours). The most important symptoms are headache, nausea, dizziness, muscle twitching, weakness, hypersecretion, miosis and pulmonary edema. Other symptoms are anxiety, restlessness, tremor, incoordination, vomiting, abdominal cramps, diarrhea, sweating, salivation, tearing, rhinorrhea, bronchorrhea, blurred or dark vision, chest tightness, wheezing, productive cough, tachycardia, hypertension, sinus arrest, toxic psychosis, confusion, bizarre behavior, unconsciousness, incontinence and convulsions.

A constant exposure at low doses can cause persistent anorexia, weakness and malaise. Acute ingestion may cause prolonged paralysis of the head, neck, limbs and thorax muscles.

In some cases organophosphates can be stored in fat cells for prolonged periods. Neurotoxicity in this case can cause weakness, paralysis and paraesthesia of the extremities, predominantly of the legs, persisting for weeks to years.

Laboratory

Do not wait for laboratory confirmations if there are strong clinical indications of organophosphate poisoning. Test for low cholinesterase levels in plasma or red blood cells. There are various tests available (Michael, Nabb-Whitfield, Ellman-Boehringer). A 25 percent or more depression is generally regarded as an exposure/poisoning.

It is important to document baseline or preexposure levels. Many persons have a cholinesterase level that will test normal but is actually lower and should be considered a case of poisoning. When in doubt, draw two samples 4 weeks apart. A significant change between the two levels suggests poisoning.

Treatment

1. If necessary, clear the airway and administer oxygen (gastric lavage may be necessary, as well as cardiac and respiratory mechanical support and monitoring).
2. Administer atropine sulfate IV.

   Adults and children 12 years and older:

   0.4-2.0 mg every 15 minutes until atropinization is achieved. Repeat doses for 2 to 12 hours or longer as necessary.

   Children under 12:

   0.05 mg/kg of body weight, repeated every 15 minutes until atropinization is achieved. Repeat dosage is 0.02-0.05 mg/kg of body weight (Morgan, p. 6).
3. Draw a heparinized blood sample.
4. Pralidoxime (protopam, 2-PAM) may be necessary if severe respiratory or neuromuscular compromise exists.

   Adults and children 12 years and older:

   1-2 gm/minute IV (no more than 0.2 gm/minute).

   Children under 12:

   20-50 mg/kg of body weight. Repeat in 1 to 2 hours, then in 10- to 12-hour intervals. (Do NOT use for probable carbamate poisoning.)
5. Observe the patient for 72 hours.

6. If ingested, gastric lavage is necessary to prevent central nervous system (CNS) depression.

- Intubate, aspirate and lavage.
- Remember to protect the airway.
- Use large orogastric tube.
- Lavage with activated charcoal in isotonic saline.
- After lavage, instill activated charcoal with a cathartic.

Adults and children 12 years and older:

50-100 gm/300-800 ml water

Children under 12:

15-30 gm/100-300 ml water

Table 19. Common organophosphate insecticides used in Texas.

NORTHEAST

BRAND*	CHEMICAL	CROPS	MORGAN (1989)
Bidrin	dicrotophos	cotton	pp. 1-11
Counter	terbufos	corn	pp. 1-11
Cygon	dimethoate	corn, watermelons, melons, wheat, sorghum	pp. 1-11
Di-syston 8	disulfoton	corn	pp. 1-11
Diazinon	diazinon	blueberries, melons, coastal bermudagrass	pp. 1-11
Dylox	trichlorofon	coastal bermudagrass	pp. 1-11
Guthion	azinphosmethyl	cotton, peaches, pecans	pp. 1-11
Lorsban	chlorpyrifos	corn, sorghum	pp. 1-11
Malathion	malathion	melons, coastal bermudagrass	pp. 1-11
Parathion	ethyl parathion	cotton, wheat, sorghum	pp. 1-11
Pencap-M	methyl parathion	cotton, wheat, southern peas	pp. 1-11
Phosdrin	mevinphos	melons	pp. 1-11

SOUTHEAST

BRAND*	CHEMICAL	CROPS	MORGAN (1989)
Counter	terbufos	corn, sorghum	pp. 1-11
Cygon	dimethoate	wheat, watermelons	pp. 1-11
Diazinon	diazinon	watermelons, peanuts, blueberries, coastal bermudagrass	pp. 1-11
Dylox	trichlorfon	coastal bermudagrass	pp. 1-11
Guthion	azinphosmethyl	cotton, pecans	pp. 1-11
Lorsban	chlorpyrifos	pecans, sorghum, peanuts	pp. 1-11
Malathion	malathion	pecans, squash	pp. 1-11
Orthene	acephate	cotton	pp. 1-11
Parathion	ethyl parathion	cotton, wheat, sorghum	pp. 1-11
Pencap	methyl parathion	wheat, soybeans, rice, cotton	pp. 1-11
Trithion	carbofenthion	watermelons	pp. 1-11
Zolone	phosalone	pecans, peaches	pp. 1-11

Table 19, continued.

SOUTH /LOWER RIO GRANDE VALLEY

BRAND*	CHEMICAL	CROPS	MORGAN (1989)
Counter	terbufos	sorghum, corn	pp. 1-11
Cygon	dimethoate	sorghum, melons, tomatoes, watermelons, kale, kohlrabi, collards, mustard greens, Swiss chard	pp. 1-11
Di-syston	disulfoton	peanuts, cauliflower	pp. 1-11
Diazinon	diazinon	turnips, onions, collards, kale, kohlrabi, mustard greens, Swiss chard	pp. 1-11
Guthion	azinphosmethyl	broccoli, peppers, cotton, peaches, sugarcane	pp. 1-11
Lorsban	chlorpyrifos	sorghum, corn, citrus	pp. 1-11
Metasystox-R	oxydemetonmethyl	squash, cucumbers, melons, watermelons	pp. 1-11
Monitor	methamidophos	cauliflower, melons, cabbage, peppers, watermelons, broccoli	pp. 1-11
Orthene	acephate	peanuts, lettuce	pp. 1-11
Pencap-M	methyl parathion	cotton	pp. 1-11
Phosdrin	mevinphos	turnips, spinach, celery, lettuce	pp. 1-11

WEST TEXAS AND HIGH PLAINS

BRAND*	CHEMICAL	CROPS	MORGAN (1989)
Bidrin	dicrotophos	cotton	pp. 1-11
Cygon	dimethoate	alfalfa seed, sorghum	pp. 1-11
Lorsban	chlorpyrifos	alfalfa seed, sorghum	pp. 1-11
Monitor	methamidophos	melons, cabbage	pp. 1-11
Orthene	acephate	peppers	pp. 1-11
Parathion	ethyl parathion	alfalfa, cotton	pp. 1-11
Pencap-M	methyl parathion	alfalfa, onions	pp. 1-11
Phosdrin	mevinphos	melons, watermelons, peppers	pp. 1-11
Thimet	phorate	sugar beets	pp. 1-11

*All brand names are registered trade names.

Carbamates (insecticides)

Toxicology

Acetylcholine accumulation is caused by reversible carboxylation of the acetylcholinesterase enzyme.

Chemical Effects

Cholinergic junctions produce muscarinic effects on smooth muscles and gland cells, causing muscle contractions and secretions.

Nicotinic junctions produce excitatory effects on skeletal muscles and autonomic ganglia that can cause twitching and also weaken or paralyze end plate cells.

Brain effects include sensory and behavioral changes, incoordination and depressed motor function.

Note: Unlike organophosphate acetylcholine accumulation, carbamates dissociate more readily, which:

- limits the duration of poisoning;
- produces a greater span between symptom production and lethal dose; and
- invalidates blood CHE fluids.

Exposure

Inhalation, ingestion, dermal. Excreted by the kidneys and liver.

Symptoms/Signs

Early symptoms are malaise, muscle weakness, dizziness and sweating. Other symptoms include headache, salivation, nausea, vomiting, abdominal pain, diarrhea, miosis, incoordination, slurred speech, dyspnea, bronchospasm, chest tightness, pulmonary edema, blurred vision, muscle twitching, spasms, convulsions and cardiac complications.

Laboratory

Unless blood is tested within 1 to 2 hours after exposure, cholinesterase levels will not be depressed.

Urine analysis should be done for N-methyl carbamate metabolites.

If clinically strong for acute poisoning, DO NOT WAIT for laboratory confirmation.

Treatment

1. Clear airway. Oxygen and gastric lavage may be necessary, as well as cardiac and respiratory mechanical support and monitoring.
2. Administer atropine sulfate IV.

   Adults and children 12 years and older:

   0.4-2.0 mg every 15 minutes (until atropinization).

   Children under 12:

   0.05 mg/kg of body weight every 15 minutes (until atropinization).

   DO NOT USE PRALIDOXIME.
3. Draw a heparinized blood sample.
4. Take a urine sample for metabolites.
5. Observe the patient for 72 hours.
6. If ingested, gastric lavage is necessary to prevent CNS depression.
   - Intubate, aspirate and lavage.
   - Remember to protect the airway.
   - Use large orogastric tube.
   - Lavage with activated charcoal in isotonic saline.
   - After lavage, instill activated charcoal with a cathartic.

     Adults and children 12 years and older:

     50-100 gm/300-800 ml water.

     Children under 12:

     15-30 gm/100-300 ml water.

Table 20. Common carbamate insecticides used in Texas.

NORTHEAST

BRAND*	CHEMICAL	CROPS	MORGAN (1989)
Furadan	carbofuran	corn, sorghum	pp. 12-24
Sevin	carbaryl	blueberries, peaches, pecans, sorghum, southern peas, watermelons, wheat, coastal bermudagrass	pp. 12-24
Temik	aldicarb	peanuts	pp. 12-24

SOUTHEAST

BRAND*	CHEMICAL	CROPS	MORGAN (1989)
Furadan	carbofuran	corn, rice, sorghum	pp. 12-24
Ordram	molinate	rice	pp. 12-24
Sevin	carbaryl	squash, pecans, watermelons, corn, peaches, rice, coastal bermudagrass, blueberries	pp. 12-24

SOUTH/LOWER RIO GRANDE VALLEY

BRAND*	CHEMICAL	CROPS	MORGAN (1989)
Furadan	carbofuran	sorghum, corn	pp. 12-24
Lannate	methomyl	peppers, onions, tomatoes, lettuce, cucumbers, corn, cauliflower, cabbage, broccoli, spinach, celery, mustard greens, squash, kale, collards, turnips, Swiss chard, kohlrabi, watermelon	pp. 12-24
Sevin	carbaryl	peanuts, lettuce, mustard greens, kale, collards, peppers, peaches, sorghum, turnips, Swiss chard, kohlrabi	pp. 12-24
Temik	aldicarb	peanuts, citrus	pp. 12-24

WEST

BRAND*	CHEMICAL	CROPS	MORGAN (1989)
Furadan	carbofuran	alfalfa, sugar beets	pp. 12-24
Lannate	methomyl	alfalfa, grapes, watermelons, cabbage, peppers, potatoes, seed sorghum	pp. 12-24
Sevin	carbaryl	alfalfa, grapes	pp. 12-24

*All brand names are registered trade names.

Organochlorines (insecticides)

The organochlorines are persistent chemicals and build up in human tissue. Use of these products is much more common outside the United States. Physicians may have to treat persons who were exposed to organochlorines in other countries. Agricultural laborers who have worked in more than one country may have been exposed to some of these chemicals. Symptoms of poisoning from these chemicals may occur many months after exposure.

In the United States, the use of most organochlorines has been banned for between 10 and 20 years. Almost none of these products exist in channels of trade in the United States. However, partly used containers of these materials may be tucked away in homes, closets or sheds.

Mexico recently canceled most uses of organochlorines, but inventories still exist in channels of trade and on farms. Organochlorines are still widely used in many countries of Central and South America, Africa, the Caribbean, and Southeast Asia.

Toxicology

Causes interference with fluxes of cations across the nerve cell membranes.

Chemical Effects

Myoclonic jerking (nerve cell membrane interference causes neuronal irritability), pulmonary gas exchange interference (metabolic acidosis), and myocardial arrhythmias are common effects.

High concentrations induce hepatic enzyme activity, biotransformation of steroid hormones, porphyria cutanea and aplastic anemia. Lipophilic compounds are likely to be excreted in maternal milk.

Exposure

Ingestion (gastrointestinal absorption), dermal, inhalation of aerosols.

Symptoms/Signs

Early onset symptoms of these chemicals are similar to those of some organophosphates (check symptom section on organophosphates or the Morgan manual for further description). Convulsions begin 48 hours after exposure and continue for days.

Other symptoms are sensory disturbance hyperesthesia, paresthesia of face and extremities, headache, dizziness, nausea, vomiting, incoordination, tremor, mental confusion, myoclonic jerking, and tonic-clonic convulsions.

Prolonged chronic exposure causes weight loss, tremor, muscle weakness, involuntary eye movement, chest and joint pain, skin rash, slurred speech, and mental changes.

Respiratory metabolic acidosis can cause death.

Laboratory

Blood gas chromatographic exams and urinary metabolites analyses should be performed. These are available through universities, poison control centers and private labs.

Treatment

Observe the patient for toxicosis, sensory disturbances, incoordination, slurred speech and involuntary motor activity (convulsions).

Take seizure precautions.

Give oxygen plus an anticonvulsive.

Gastric lavage may be necessary.

Monitor and assist pulmonary ventilation and cardiac status.

Table 21. Common organochlorine insecticides formerly or presently used in Texas

BRAND*	CHEMICAL	U.S. REG.	MEX. REG.	INT'L. REG.	USE PATTERNS	MORGAN (1989)
Aldrite, Aldrin	aldrin	No	No[1]	Yes	soil insects, field crops, industry	pp. 17-24
BHC	benzene hexachloride	No	No[2]	Yes	livestock, ornamentals	pp. 17-24
Chlordane	chlordane	No	Yes	Yes	termites, industry, soil, insects	pp. 17-24
Kepone	chlordecone	No	No[1]	Yes	ants	pp. 17-24
Acaralate	chlorobenside	No	Yes	Yes	mites, citrus fruits, nuts	pp. 17-24
Acaraben	chlorobenzilate	No	Yes	Yes	mites, citrus fruits, nuts	pp. 17-24
Mitox	chloropropylate	No	Yes	Yes	mites, citrus fruits, nuts	pp. 17-24
DBCP, Fumazone, Nemagon, Nemafume, Nemaset	dibromochloropropane	No	No[1]	Yes	soil insects, soil diseases, nematodes	pp. 138-141
Rhothane	DDD	No	Yes	Yes	fruits, vegetables	pp. 17-24
DDT	DDT	No	Yes	Yes	cotton, houseflies, mosquitos, public health, vector control	pp. 17-24

Table 21, continued.

BRAND*	CHEMICAL	U.S. REG.	MEX. REG.	INT'L. REG.	USE PATTERNS	MORGAN (1989)
Telone D-D, Vidden D	dichloropropane & dichloropropene	No	No[1]	Yes	soil fumigant, nematodes, insects, soil diseases	pp. 17-24
Telone II	1,3-dichloropropene	Yes	Yes	Yes	soil fumigant, soil insects, nematodes, soil diseases	pp. 17-24
C-17	dichloropropene & chloropicrin telone	Yes	Yes	Yes	soil fumigant, insects, nematodes, soil diseases	pp. 17-24
Kelthane	dicofol	Yes	Yes	Yes	mites, fruits, nuts, vegetables, cotton	pp. 17-24
Dieldrin	dieldrin, dieldrite	No	No[1]	Yes	cotton, rangeland pests, soil insects	pp. 17-24
Pentac	dienochlor	No	Yes	Yes	soil insects	pp. 17-24
Thiodan	endosulfan	Yes	Yes	Yes	cotton, fruits, vegetables	pp. 17-24
Endrin	endrin	No	No[1]	Yes	small grains, tobacco, orchards	pp. 17-24
Perthane	ethylan	No	Yes	Yes	fruits, nuts, ornamentals, vegetables	pp. 17-24
Heptachlor	heptachlor	No	Yes	Yes	industrial, seed treatment, soil insects, termites	pp. 17-24
Lindane	lindane (gamma isomer of hexchloro-cyclohexane)	Yes	Yes	Yes	fruits, nuts, planter box seed treatment, vegetables	pp. 17-24
Dechlorane	mirex	No	No[1]	Yes	ants	pp. 17-24
Marlate	methoxychlor	Yes	Yes	Yes	fruits, nuts, livestock, vegetables	pp. 17-24
Moth Crystals	paradichlorobenzene	Yes	Yes	Yes	clothing, fabric, hides	pp. 17-24
Strobane, Toxakil, Toxaphene	toxaphene	No	No[2]	Yes	cotton, livestock, vegetables	pp. 17-24

*All brand names are registered trade names.
[1]Canceled in Mexico after 3 January, 1991 (**Catalogo Oficial**, 1994).
[2]Canceled in Mexico after 3 January, 1994 (**Catalogo Oficial**, 1994).

Biological Insecticides (pyrethrins, nicotine)

Toxicology

Pyrethrins are botanical insecticides with very low mammalian toxicity. They lack environmental persistence and break down rapidly. Commercial pyrethrum is a powder seldom found outside industry. Pyrethrum extract is the usual commercial form. The extract is a preparation of pyrethrins in an organic solvent. The solvent may be methanol, acetone, kerosene, fuel oil, or any other petroleum distillate. Formulations exist as extract alone or in combination with other insecticides. They come in human and veterinary pharmaceuticals, in liquid concentrates, and as aerosols such as "bug bombs." Frequently, the carriers and propellants are more toxic than the pyrethrins themselves.

Chemical Effects

These substances may cause dermal or respiratory allergic reactions--in some cases anaphylactic.

Exposure Dermal, inhalation (pulmonary mucous membranes).

Symptoms/Signs Symptoms include asthma reactions, allergic rhinitis, contact dermatitis, and cholinesterase symptomatology if the biological insecticide was combined with organophosphates or carbamates.

Laboratory Varies among biological insecticides.

Treatment

1. Administer antihistamines.
2. Treat asthmatic symptoms.
3. Treat chronic dermatitis with steroids.
4. Flush eyes with clean water and saline solution.
5. Treat other toxic effects of organophosphates, carbamates and other insecticides.
6. Treat nicotinic poisoning by washing the patient with soap and water. The patient also may need pulmonary ventilation.

Table 22. Common biological insecticides used in crops in Texas.

NORTHEAST

SUBCATEGORIES	BRAND*	CHEMICAL	CROP	MORGAN (1989)
biological	Dipel	*Bacillus thuringiensis*	sweet potatoes	pp. 32-33
elemental sulfur	Dusting Sulfur	sulfur	peaches	pp. 48-49
pyrethroids	Ammo	cypermethrin	cotton, onions	pp. 34-36
	Asana	esfenvalerate	pecans, watermelons	pp. 34-36
	Ambush, Pounce	permethrin	pecans, vegetables, mushrooms	pp. 34-36
	Karate	lambdacyhalothrin	cotton	pp. 34-36
	Baythroid	cyfluthrin	cotton	pp. 34-36
	Scout	flucythrinate	cotton	pp. 34-36

SOUTHEAST

SUBCATEGORIES	BRAND*	CHEMICAL	CROP	MORGAN (1989)
elemental sulfur	Dusting Sulfur	sulfur	peaches	pp. 48-49
organochlorine	Thiodan	endosulfan	squash	pp. 17-24
	Marlate	methoxychlor	livestock, vegetables, fruits, mosquitoes, black flies	pp. 17-24
pyrethroids	Ammo	cypermethrin	cotton	pp. 34-36
	Pydrin	fenvalerate	cotton	pp. 34-36
	Scout	tralomethrin	cotton, soybeans	pp. 34-36
	Ambush, Pounce	permethrin	soybeans	pp. 34-36

Table 22, continued.

SOUTH/LOWER RIO GRANDE VALLEY

SUBCATEGORIES	BRAND*	CHEMICAL	CROPS	MORGAN (1989)
inorganic salt	Kocide	copper hydroxide	peppers	p. 103
	Tri-Basic $CuSO_4$, C-O-C-S, Fixed Copper	cupric, oxychloride, sulfate	peppers, tomatoes	p. 103
organochlorine	Kelthane	dicofol	citrus	pp. 17-24
	Acaraben	chlorobenzilate	citrus	pp. 17-24
	Thiodan	endosulfan	cucumbers, tomatoes	pp. 17-24
	Dipel	*Bacillus thuringiensis*	collards, lettuce, kale, celery, mustard greens, cabbage, broccoli, turnips, spinach, Swiss chard, kohlrabi, cauliflower	pp. 32-33
pyrethroids	Ambush, Pounce	permethrin	spinach, turnips, tomatoes, collards, mustard greens, kale, peppers, lettuce, broccoli, turnips, celery, cauliflower, Swiss chard, kohlrabi	pp. 34-36
	Pydrin	fenvalerate	squash, cabbage	pp. 34-36
	Asana	esfenvalerate	tomatoes, cabbage, cucumbers, squash	pp. 34-36
	Ammo, Cymbush	cypermethrin	cotton, onions	pp. 34-36

WEST

SUBCATEGORIES	BRAND*	CHEMICAL	CROP	MORGAN (1989)
inorganic	Dusting Sulfur	sulfur	grapes	pp. 48-49
inorganic salts	Kocide	copper hydroxide	peppers	p. 103
organochlorine	Endocide, Thiodan	endosulfan	watermelons, peppers, cabbage	pp. 17-24
pyrethroids	Ambush, Pounce	permethrin	cotton, melons, cabbage	pp. 34-36
	Asana	esfenvalerate	cotton, peanuts	pp. 34-36
	Pydrin	fenvalerate	cotton, melons, potatoes	pp. 34-36
	Ammo, Cymbush	cypermethrin	onions	pp. 34-36

*All brand names are registered trade names.

Rodenticides and Predator Control Agents

Toxicology

Effects vary with the type of rodenticide and the organ affected. These products are usually in the form of baits, powders and dusts. Principal types are anticoagulants, inorganics, convulsants, and a variety of compounds including single feeding and cumulative poisons.

Anticoagulants (coumarins and indandiones) depress the hepatic vitamin K-dependent synthesis of substances essential to blood clotting--prothrombin (factor II) and factors VII, IX and X. Warfarin (a coumarin) and chlorphacinone (an indandione) can be absorbed across the skin. However, this occurs only under extraordinary conditions. Ordinarily, poisoning by these agents occurs through ingestion.

Inorganic rodenticides include yellow phosphorus, zinc phosphide and thallium sulfate. Symptoms depend upon which organ is affected.

Zinc phosphide, a common rodenticide used in orchards against voles and meadow mice, liberates phosphine gas when acted upon by moisture. The effects may manifest themselves as damage to the liver, lungs, heart and central nervous system, if the patient survives the initial shock from ingestion.

The convulsants are named for the symptoms they produce rather than for a common mode of action. Strychnine causes violent epileptiform convulsions by direct excitatory action on cells of the central nervous system. The fluoroacetates are latent inhibitors requiring metabolic conversion in the liver to fluorocitrate, which poisons critical enzymes of the Kreb's cycle and impairs cellular respiration.

Substituted urea rodenticides are specific poisons for b cells of the pancreas. Pyriminil (Vacor®) is a substituted urea compound with high mammalian toxicity. It is a single-dose rodenticide no longer registered in the United States. However, home stocks of this product still exist and several suicides and accidental poisonings have resulted from this compound. Severe effects have resulted from very small doses. In addition to its effects on the pancreas, pyriminil also affects glucose metabolism and produces pronounced neurotoxic effects. The exact mechanism of action of substituted urea rodenticides remains uncertain.

Chemical Effects

Inorganic compounds such as phosphorus and zinc phosphide are highly reactive and corrosive to tissues. Phosphorus may burn skin.

Exposure

Ingestion, dermal, mucous membranes.

Symptoms/Signs

Anticoagulants:

Coumarins -- Initial symptoms include increasing pallor, weakness, back pain, abdominal pain, pain in the extremities and vomiting. Later symptoms include nosebleed, bleeding of mouth and gums, massive bruises, hematoma of knee and elbow joints, hematuria, epistaxis and circulatory failure.

Indandiones -- Initial symptoms may range from practically none to pallor, weakness, nausea, severe hematuria, epistaxis and bleeding from mouth and gums. Later symptoms include depression, rapid and labored respiration, accelerated pulse, hemorrhage, bleeding into the thoracic cavity, and circulatory failure.

Inorganics produce a variety of symptoms. Phosphorus severely burns the skin, mucous membranes and other tissues with which it comes in contact. Early symptoms include lethargy, restlessness and irritability. Vomiting and diarrhea usually ensue.

Zinc phosphide--This substance is much less irritating to skin than phosphorus, but it is very irritating to respiratory mucosa. Inhaling dust may produce pulmonary edema. Symptoms include nausea, vomiting, excitement, chills, chest tightness, dyspnea, and cough that may progress to pulmonary edema. Severe symptoms include delirium, convulsions, coma, shock from toxic myocardiopathy, jaundice, tetany, anuria and hemorrhage. Treatment for phosphide poisoning is discussed in the "Fumigants" section of this manual. Also consult Morgan (1989), pp. 136-141.

Convulsants:

Sodium fluoroacetate--This substance produces cardiac arrythmia progressing to ventricular fibrillation. Effects on the nervous system are expressed as tonic-clonic convulsions, spasms and rigor.

Strychnine--Strychnine produces tightness and twitching of the muscles, especially those in the face and neck. Movements may be abrupt. Vomiting may occur. Generalized convulsions occur within 15 to 30 minutes after ingestion. They may be clonic at first but quickly become tonic. Convulsions

become progressively severe. Patients remain conscious until respiratory stoppage produces anoxia and cyanosis. Convulsions may be violent enough to cause compression fractures of the vertebrae. Legs are adducted and extended, feet curved inward. Foam gathers at the mouth. Eyes protrude and pupils dilate. The pulse may be difficult to detect.

Substituted ureas:

Pyriminil--Pyriminil produces lassitude, anorexia, constipation and abdominal bloating in the early stages. Later it may produce painful paresthesia with numbness of the extremities and difficulty in walking. Progressive autonomic and peripheral polyneuropathy is characterized by orthostatic hypotension and greatly diminished response to pinprick. Later effects also include diabetes as a result of the effect on b cells of the pancreas.

Treatment Treatments vary with the nature of the agent, and are described in Morgan (1989), pp. 115-130.

Table 23. Rodenticides and predator control agents used in Texas.

RODENTICIDE TYPE	BRAND*	CHEMICAL	SITE/PEST	MORGAN (1989)
botanical	Gopher Getter	strychnine	pocket gophers	pp. 123, 125
	Dethdiet, Rodine	red squill	rats, mice	pp. 126 -128
coumarin	Decon, Tox-Hid, Warfacide	warfarin	rats, mice	pp. 115 -119
	Fumarin, Fumasol, Krumkil, Ratfin, Rat-A-Way, Tomarin	coumafuryl	rats, mice	pp. 115 -119
	Havoc, Talon, Ratak, Volak	brodifacoum	rats, mice	pp. 115 -119
cyanide	M-44, Coyote Getter	sodium cyanide	coyotes	pp. 143 -145
fluoroacetate	Compound 1080	sodium fluoroacetate	coyotes	pp. 123 -124
indandione	Ramik, Gold Crest, Diphacine, Promar	diphacinone	rats, mice, squirrels	pp. 115 -119
	Rozol, Drat, Quick, Lepit, Saviac	chlorphacinone	rats, mice	pp. 115 -119
substituted urea	Vacor, PNU, Tracking Powder	pyriminil	rats, mice	pp. 126 -128
thiourea	Antu, Anturat, Bantu, Keyais, Rat-Trak, Rat-Tu	α-naphthyl thiourea	Norway rats	pp. 126 -127
vitamin D-related	Quintox	cholecalciferol	rats, mice	p. 129
zinc phosphide	ZP Bait	zinc phosphide	rats, mice	p. 12

*All brand names are registered trade names.

Fungicides

Toxicology Effects vary with fungicide type and the organ affected. These products are usually in the form of dusts, powders and granules.

Chemical Effects Fungicides are often dermal sensitizers; some also have systemic manifestations

Exposure Dermal, mucous membranes.

Symptoms/Signs

Common symptoms include skin lesions, scarring, conjunctivitis, keratitis, pyrexia, corneal opacities, hepatomegaly, porphyria, cellulitis, weakness, anorexia and methemoglobinemia.

Laboratory

Recommendations vary among different fungicides.

Treatment

Specific treatment varies, depending upon the chemical family of the product. General treatment recommendations are as follows:

1. Wash the exposed area.
2. Lavage and induce vomiting with syrup of ipecac if the fungicide has been ingested.
3. Administering 3-8 gm q.i.d. of cholestyramine will accelerate elimination.
4. Treat porphyria symptoms.
5. Monitor liquids and cardiac functions.

Table 24. Common fungicides used in Texas.

NORTHEAST

SUBCATEGORIES	BRAND*	CHEMICAL	CROPS	MORGAN (1989)
benzimdazole	Apl-luster, Arbotect	thiabendazole	sweet potatoes	p. 113
	Benlate	benomyl	melons	p. 112
benzonitrile	Bravo	chlorthalonil	melons, peanuts	p. 95
chlorinated hydrocarbons	Terrachlor	PCNB	peanuts	pp. 17-27
chlorophenoxy	Bayleton	triadimefon	wheat	p. 113
ethlenebisdithiocarbamate	Dithane, Manzate	maneb, mancozeb	peanuts, watermelons	pp. 100-101
organotin	Du-ter	triphenyltin	pecans	p. 108
pthalimide	Captan	captan	peaches	p. 102

SOUTHEAST

SUBCATEGORIES	BRAND*	CHEMICAL	CROPS	MORGAN (1989)
benzonitrile	Bravo	chlorthalonil	squash	p. 112
chlorophenoxy	Bayleton	triadimefon	wheat	p. 113
dicarboximide	Captan	captan	peaches	p. 102
ehtylenebisdithiocarbamate	Dithane, Manzate	mancozeb	wheat, squash	pp. 100-101
elemental sulfur	Dusting Sulfur	sulfur	peaches	pp. 48-49
organotin	Du-ter	triphenyltin	pecans	p. 108
trizole	Tilt	propiconazole	rice, wheat	None (Call CIBA-GEIGY 1-800-888-8372)

Table 24, continued.

SOUTH/LOWER RIO GRANDE VALLEY

SUBCATEGORIES	BRAND*	CHEMICAL	CROPS	MORGAN (1989)
benzimidazole	Benlate	benomyl	celery, melons, peaches, watermelons	p. 112
chlorinated isophalic acid	Bravo	chlorthalonil	celery, cauliflower, cabbage, broccoli, melons, cucumbers, peanuts, onions, tomatoes, squash, watermelons	p. 95
chlorinated hydrocarbon	Terraclor	PCNB	peanuts	pp. 17-27
chlorophenoxy	Bayleton	triadimefon	cucumbers, squash, melons	p. 113
dicarboximide	Captan	captan	peaches, spinach	p. 102
dimethylaniline	Ridomil, Apron	metalaxyl	melons, cucumbers, onions, sorghum, squash, spinach, watermelons	p. 113
diozadiazole	Rovral	iprodione	onions	p. 112
elemental sulfur	Dusting Sulfur	sulfur	peaches	pp. 48-49
ethylenebisdithiocarbamate	Dithane	menab	celery, cauliflower, cabbage, broccoli, watermelons, lettuce, cucumbers, onions, collards, mustard greens, kale, Swiss chard, kohlrabi, tomatoes, turnips, spinach, celery, melons	pp. 100-101
	Dithane, Manzate	mancozeb	squash	pp. 100-101
organotin	Torque, Vendex	fenbutatinoxide	citrus	pp. 108-109

WEST

SUBCATEGORIES	BRAND*	CHEMICAL	CROPS	MORGAN (1989)
benzimidazole	Benlate	benomyl	watermelons, grapes	p. 112
carbamate	Topsin M	thiophanatemethyl	peanuts	pp. 96-101
chlorinated hydrocarbon	Terrachlor	pentachloronitrobenzine	peanuts	pp. 17-27
chlorinated isopthalic acid	Bravo	chlorthalonil	watermelons, melons, onions, potatoes, peanuts	p. 95
chlorophenoxy	Bayleton	triadimefon	grapes, melons, sugar beets	p. 113
dimethyl aniline	Ridomil	metalaxyl	melons	p. 113
ethylenebisdithiocarbamate	Dithane	maneb	watermelons, onions, potatoes, peanuts	pp. 100-101
	Manzate	mancozeb	onions	pp. 100-101
organotin	Triple Tin	triphenyltin hydroxide	sugar beets	pp. 108-109
pthalimide	Captan	captan	grapes	p. 102

*All brand names are registered trade names.

Herbicides

Chemical Effects Effects depend on the adjuvants (stabilizers, penetrants, safeners, surfactants) used in the product or the other pesticides mixed with the product.

Exposure Eyes, dermal, mucous membranes, ingestion.

Symptoms/Signs Symptoms include skin lesions, scarring, conjunctivitis, keratitis, pyrexia, corneal opacities, hepatomegaly, porphyria and other serious CNS symptoms. Carbamate herbicides produce symptoms similar to those caused by carbamate insecticides.

Laboratory Tests are generally not available to confirm human absorption; determine recent exposure from the patient's occupation.

NOTE: Some herbicides, such as chlorophenoxys, have lab urine and blood procedures that are useful for assessing the magnitude of the poisoning.

Treatment Treatment for carbamate herbicide poisoning should follow the suggestions given for carbamate insecticides. General treatment guidelines for other herbicides are:

1. Wash the skin, flush the eyes and treat toxicosis.
2. Gastric lavage may be necessary.
3. Support with IV solutions.
4. Control body temperature with physical means.
5. Pulmonary and cardiac monitoring may be necessary, and the patient may need oxygen continuously to reduce anoxia.
6. Anticonvulsive therapy may be necessary.
7. For bipyridyl (diquat/paraquat) poisoning administer Bentonite and Fuller's Earth. Consult Morgan (1989), pp. 76-82, for specific patient management recommendations.

Note: Listen for bowel sounds; ileus may occur.

Table 25. Common herbicides used in Texas.

NORTHEAST

SUBCATEGORIES	BRAND*	CHEMICAL	CROPS	MORGAN (1989)
acetamide	Dual, Bicep	metoxachlor + atrazine	corn	pp. 83-88
	Enid	diphenamid	sorghum, sweet potatoes	pp. 83-88
acetanilide	Lasso	alachlor	corn, peanuts	pp. 83-88
alkyl cyclohexene	Poast	sethoxydim	blueberries	pp. 83-88
bipyridyl	Grammoxone	paraquat	blueberries, cotton, sweet potatoes	pp. 76-82
	Diquat	diquat	alfalfa seed, aquatic vegetation	pp. 75-82
benzoic acid + chlorophenoxy	Weedmaster	dicamba + 2,4-D	coastal bermudagrass	pp. 83-88
benzoic acid derivative	Banvel	dicamba	wheat, coastal bermudagrass	pp. 83-88
chlorophenoxy	2,4-D	2,4-D	wheat, coastal bermudagrass	pp. 63-67
	Butyrac	2,4-DB	peanuts	pp. 63-67
chloro-s-triazine	Princep	simazine	blueberries	pp. 83-88
dichlorobenzoic acid	Amiben	chloramben	sweet potatoes	pp. 83-88
dinitroaniline	Surflan	oryzalin	blueberries, peaches	pp. 83-88
	Treflan	trifluralin	peanuts, watermelons, southern peas	pp. 83-88
	Prowl	pendimethalin	peanuts	pp. 83-88
organophosphate	Roundup	glyphosate	sweet potatoes, blueberries, peaches, pecans	pp. 1-11
phthalates	Dacthal	DCPA	southern peas, sweet potatoes	pp. 83-88
pyridazinone	Solicam	norflurazon	blueberries	pp. 83-88
sulfonylurea	Glean	chlorsulfuron	wheat	pp. 83-88
triazine	Aatrex	atrazine	corn, sorghum	pp. 83-88
	Princep	simazine	corn, orchards	pp. 83-88
	Pramatol	prometon	fence lines, equipment, yards, ditch banks	pp. 83-88
	Bladex	cyanzaine	corn	pp. 83-88
	Sencor	metribuzin	alfalfa, soybeans	pp. 83-88
	Lexone	metribuzin	alfalfa, soybeans	pp. 83-88
trichlorpicolinic acid	Grazon	picloram	coastal bermudagrass	pp. 83-88

Table 25, continued.

SOUTHEAST

SUBCATEGORIES	BRAND*	CHEMICAL	CROPS	MORGAN (1989)
acetamide	Dual	metolachlor	corn, sorghum, soybeans	pp. 83-88
	Stampede	propanil	rice	pp. 83-88
acetanilide	Lasso	alachlor	corn, sorghum	pp. 83-88
alkyl cyclohexene derivative	Poast	sethoxydim	blueberries	pp. 83-88
benzoic acid + chlorophenoxy	Weedmaster	dicamba + 2,4-D	coastal bermudagrass	pp. 63-67
bipyridyl	Grammoxone	paraquat	blueberries, orchards	pp. 76-82
chlorophenoxy	2,4-D	2,4-D	wheat, coastal bermudagrass	pp. 63-67
	Butyrac	2,4-DB	peanuts	pp. 63-67
chloro-s-triazine	Aatrex	atrazine	corn, sorghum	pp. 83-88
	Princep	simazine	blueberries	pp. 83-88
dichlorbenzoic acid	Amiben	chloramben	squash	pp. 83-88
dinitroaniline	Treflan	trifluralin	watermelons, peanuts	pp. 83-88
	Surflan	oryzalin	peaches, blueberries	pp. 83-88
diphenylether	Blazer	acifluorofen	soybeans	pp. 83-88
imidazolinone	Scepter	imazaquin	soybeans	pp. 83-88
organophosphate	Roundup	glyphosate	corn, peaches, pecans, blueberries	pp. 1-11
	Prefar	bensulide	squash, watermelons	pp. 1-11
pyridazinone	Solicam	norflurazon	blueberries	pp. 83-88
sulfonylurea	Glean	chlorsulfuron	wheat	pp. 83-88
	Ally	metsulfuron, methyl	wheat	pp. 83-88
thiadiazin	Basagran	bentazon	rice, soybeans	pp. 83-88
thiocarbamate	Bolero	thiobencarb	rice	pp. 83-88
trichloropicolinic acid	Grazon	picloram	coastal bermudagrass	pp. 83-88

SOUTH/LOWER RIO GRANDE VALLEY

SUBCATEGORIES	BRAND*	CHEMICAL	CROPS	MORGAN (1989)
acetanilide	Lasso	alachlor	corn, peanuts	pp. 83-88
benzoic acid	Bavel	dicamba	sugarcane	pp. 83-88
chlorophenoxy	Butyrac	2,4-DB	peanuts	pp. 83-88
dimethylurea	Karmex	diuron	citrus, sugarcane	pp. 83-88
dinitroanaline	Treflan		cauliflower, cotton, turnips, sugarcane, grapes	pp. 83-88
dinitrotoluidine	Surflan	oryzalin	peaches	pp. 83-88
organophosphate	Roundup	glyphosate	corn, citrus, peaches	pp. 1-11
sulfonamide	Betasan, Prefar	bensulide	lettuce	pp. 83-88
triazine	Aatrex	atrazine	corn, sorghum, sugarcane	pp. 83-88
	Princep	simazine	noncrop	pp. 83-88
	Caparol	prometryn	cotton	pp. 83-88
	Evik	ametryn	sugarcane	pp. 83-88
uracil	Hyvar	bromacil	citrus	pp. 83-88

Table 25, continued.

WEST

SUBCATEGORIES	BRAND*	CHEMICAL	CROPS	MORGAN (1989)
benzene	Sulfonamid, Prefar	bensulide	onions	pp. 83-88
benzonitrile	Buctril	bomoxylnil	seed sorghum	pp. 83-88
bipyridyl	Gramoxone	paraquat	potatoes	pp. 76-82
carbanilate derivative	Betanal	penmedipham	sugar beets	pp. 83-88
chlorophenoxy	2,4-D	2,4-D	barley, wheat	pp. 63-67
cyclohexones	Poast	sethoxydim	sugar beets	pp. 83-88
dinitroaniline	Treflan	trifluralin	cotton, watermelons, grapes, potatoes	pp. 83-88
	Surflan	oryzalin	grapes	pp. 83-88
diphenyl ether	Goal	oxyfluorfen	onions	pp. 83-88
methyl sulfonate	Nortron	ethofumesate	sugar beets	pp. 83-88
organophosphate	DEF	DEF	cotton	pp. 1-11
	Prefar	bensulide	melons, onions	pp. 1-11
sulfonylurea	Ally	metsulfuron methyl	wheat	pp. 83-88
	Glean	chlorsulfuron	wheat	pp. 83-88
triazine	Sencor	metribuzin	potatoes	pp. 83-88
thiocarbamate	EPTAM, GENEP	EPTC	alfalfa, sugar beets	pp. 83-88

*All brand names are registered trade names.

Solid or Dissolved Arsenicals

Toxicology The metal and nonmetal physical properties of arsenicals have reversible combination effects on tissue proteins and enzymes. Arsenicals compete with phosphates, and cause injury to nerve cells, blood vessels, liver, kidney and other tissues.

Chemical Effects Chemical effects depend on the substance's biochemical transformation mechanisms (vascular dilation).

Exposure Mucous membranes, dermal, ingestion.

Symptoms/Signs **Acute:** Within 1 hour there is a garlic odor in breath and feces; mouth, pharynx and esophagus inflammation; burning abdominal pain; thirst; vomiting; and diarrhea. Poisoning may result in renal injury, CNS disorders, cardiovascular and liver damage, anemia, leukopenia, thrombocytopenia, circulatory failure and death.

Chronic: In chronic exposure there are more prominent dermal signs; hyperkeratosis; hyperpigmentation; dermatitis; subcutaneous edema of face; edema of eyelids and ankles; loss of nails or hair; stomatitis; weight loss; peripheral neuropathy; liver injury; EKG anomalies; anemia; skin cancer; lung cancer; rarely encephalopathy.

Laboratory At 24 hours perform Gutzeit and Reinsoit tests on urine sample.

Treatment

1. Wash skin and hair with copious amounts of soap and water.
2. Gastric lavage and intubation may be necessary.
3. Hydrate with IV fluid if necessary.
4. Cardiac monitoring is important.
5. Administer dimercaprol (BAL) as recommended in Morgan (1989), p. 60.

Table 26. Arsenical herbicides.

INORGANIC ARSENICALS	ORGANIC ARSENICALS
Arsenic acid	Cacodylic aid
Sodium arsenite	Methane arsenic acid
Copper arsenites	Monosodium methane arsonate
Calcium arsenite	Monoammonium methane arsonate
Zinc arsenates	Calcium acidmethane arsonate

Arsenicals are sometimes used in combination with other pesticides because of their binding effect. They are mostly used as defoliants, herbicides and insecticides in the form of powders or solutions. They were used in cotton, potato and tomato production, and as wood preservatives and ant killers. Arsenic acid ceased to have application in cotton after cotton processors were forced to dispose of waste water as toxic waste because of arsenic residues. Arsenicals have very few applications outside of ornamental home and garden treatments to control crabgrass. Farm workers from Central America and South America, where arsenicals are still widely used, may have high residues of arsenic in their bodies.

Treatment for the arsenical insecticides and herbicides is described in Morgan (1989), pp. 54-62.

Fumigants

Toxicologists continue to group together, under the category of fumigants, several groups of compounds that have little in common except toxicity to one or more pests and relatively high vapor pressures. Some of the compounds are gases at room temperature. Others are liquids or solids. Their activity against pests depends on their vapors.

Toxicology

The general toxicology of the fumigants varies with the type of compound. The toxicity of many of the compounds is a physical property and follows Ferguson's principle, being lethal at thermodynamic activities between 0.1 and 1.0. Others are volatile chemical poisons and are active at thermodynamic levels far below 0.1.

Chemical Effects

Effects vary with the chemical nature of the fumigant. Some have no effects. Others produce strong irritation of eyes, skin and upper respiratory tract.

Exposure

Dermal, inhalation, mucous membranes, ingestion.

Symptoms/Signs

Symptoms vary with the product. A few symptoms include headache; nausea; muscle aches; irritation of eyes, nasal and pharyngeal passages; pulmonary edema; lassitude; mental disorientation; convulsions; hemolysis; cyanosis; and coma.

Laboratory

Will vary among fumigants.

Treatment

Eleven general steps in treating poisoning by fumigants are listed in Morgan (1989), pp. 138-141. They are as follows:

1. Flush contaminating fumigants from the skin and eyes with copious amounts of water or saline for at least 15 minutes. Some fumigants are corrosive to the cornea and may cause blindness. Specialized medical treatment should be obtained promptly. Contact with skin may cause blistering and deep chemical burns. Absorption of some fumigants across the skin may be sufficient to cause systemic poisoning in the absence of fumigant inhalation. For all these reasons, decontamination of eyes and skin must be immediate and thorough.
2. Remove the victim to fresh air immediately. Even though initial symptoms and signs are mild, keep the victim quiet in a semi-reclining position. Minimum physical activity limits the likelihood of pulmonary edema.
3. If the victim is not breathing, clear the airway of secretions and resuscitate with positive-pressure oxygen apparatus. If this is not available, use chest compression to sustain respiration. If the victim is pulseless, employ cardiac resuscitation.
4. If pulmonary edema is evident, there are several measures available to sustain life. Medical judgment must be relied upon, however, in the management of each case. The following procedures are recommended:

   (NOTE: CHECK FUMIGANT LABELS FOR ANY CONTRAINDICATIONS TO DRUGS.)

   - Put the victim in a sitting position with a backrest.
   - Use intermittent and/or continuous positive-pressure oxygen to relieve hypoxemia. (Do not give oxygen at greater concentrations or longer periods than necessary, because it may exaggerate fumigant injury to lung tissue. Monitor arterial pO_2.)
   - Slowly administer furosemide, 40 mg, or sodium ethacrynate, 50 mg, intravenously, to reduce venous load by inducing diuresis. Consult package insert for additional directions and warnings.
   - Slowly administer morphine in small doses (5 -10 mg), intravenously, to allay anxiety and promote deeper respiratory excursions.
   - Administer aminophylline (0.25-0.50 gm) slowly, intravenously. Consult package insert.
   - Digitalization may be considered, but there is a serious risk of arrhythmias in an anoxic and toxic myocardium.
   - Tracheostomy may be necessary in some cases to facilitate aspiration of large amounts of pulmonary edema fluid.
   - Epinephrine, atropine and expectorants are generally not helpful, and may complicate treatment.
   - Watch for recurrent pulmonary edema, even up to 2 weeks after the initial episode. Limit the victim's physical activity for at least 4 weeks. Severe physical weakness usually indicates persistent pulmonary injury. Serial pulmonary function testing may be useful in assessing recovery.

5. Combat shock by placing the victim in the Trendelenburg position and administering plasma, whole blood, and/or electrolyte and glucose solutions intravenously, with great care to avoid pulmonary edema. Central venous pressure should be monitored continuously. Vasopressor amines must be given with great caution, because of the irritability of the myocardium.

6. Control convulsions. Seizures are most likely to occur in poisonings by methyl bromide, hydrogen cyanide, acrylonitrile, phosphine and carbon disulfide.

   - Establish pulmonary gas exchange at the best possible level by administering oxygen by continuous positive-pressure ventilation.
   - In poisoning by cyanide or acrylonitrile, proceed directly with antidotal therapy (see Morgan, pp.143-44).
   - Control convulsions caused by other agents with careful IV injection of diazepam: 5-0 mg in adults and children over 12 years; 0.25-0.40 mg/kg of body weight in children under 12 years. (See Morgan, p. 21.) Repeat dosage in 4 to 6 hours if necessary. CAUTION: Be prepared to maintain pulmonary ventilation mechanically, and to manage hypotension and cardiac arrhythmias. Alternative or supplemental anticonvulsive therapy is discussed in the reference cited.
   - In methyl bromide poisoning, it may be necessary to give benzodiazepines or barbiturates orally for days or weeks after the poisoning to control involuntary motor activity. Consult package inserts for appropriate dosages.

7. If a fumigant liquid or solid has been ingested less than several hours prior to treatment, quantities remaining in the stomach must be removed as effectively as possible by gastric intubation, aspiration and lavage, after all possible precautions have been taken to protect the respiratory tract from aspirated gastric contents.

   - Put in place a cuffed endotracheal tube prior to gastric intubation. Administer oxygen, using a mechanical ventilator if respiration is depressed.
   - Lavage the stomach with a slurry of activated charcoal in saline or water. Leave a volume of the slurry in the stomach with an appropriate dose of sorbitol as a cathartic (for dosages, see Morgan, p. 8).
   - If treatment is delayed and if the patient remains fully alert, administer activated charcoal and sorbitol orally (for dosages, see Morgan, p. 8). Repeated administration of charcoal at half or more of the initial dosage every 2 to 4 hours may be beneficial.
   - Do not give vegetable or animal fats or oils, which enhance gastrointestinal absorption of many of the fumigant compounds.

8. Intravenous infusions of glucose are valuable in limiting the hepatotoxicity of many substances. Monitor central venous pressure to avoid precipitating, or aggravating, pulmonary edema by fluid overload. The victim should be watched closely for indications of delayed or recurrent pulmonary edema, and for bronchopneumonia. Fluid balance should be monitored, and urine sediment should be checked regularly for indications of tubular injury. Measure serum alkaline phosphatase, LDH, ALT, AST and bilirubin to assess liver injury.

9. Hemoperfusion over activated charcoal has been used in managing a case of carbon tetrachloride poisoning with apparent success. An extraction efficiency of about 80 percent was demonstrated for the system employed (Schwarzbeck, A. and W. Kosters, *Arch. Toxicol.*, 35:207-211, 1976). It is possible that other fumigant compounds would be effectively removed from blood by this method.

10. Extracorporeal hemodialysis may be needed to regulate extracellular fluid composition if renal failure occurs. It is probably not very effective in removing lipophilic fumigant compounds from blood, but is, of course, effective in controlling extracellular fluid composition if renal failure occurs.
11. Morgan mentions certain specfic measures for poisonings by particular fumigants (naphthalene, methyl bromide, carbon tetrachloride, hydrogen cyanide). Refer to the Morgan page reference in the fumigant list on page 49. Morgan provides insufficient or no information on dazomet (Basamid), phosphine (Phostoxin, aluminum phosphide, zinc phosphide) and sulfuryl fluoride.

Naphthalene

Toxicology

Ingestion, intensive or prolonged inhalation, or dermal contact with treated fabric may cause hemolysis in persons deficient in glucose-6-phosphate dehydrogenase. Secondary renal tubular damage may ensue from excretion of the naphthol, other aphthalene metabolites, and products of hemolysis.

Naphthalene produces selective but reversible damage to the nonciliated bronchiolar epithelium. If ingested, naphthalene may produce local irritation of the gastrointestinal tract.

Convulsions and coma may occur, particularly in children. In infants, high levels of hemoglobin, methemoglobin and bilirubin in the plasma may lead to encephalopathy (kernicterus). Some individuals exhibit dermal sensitivity to naphthalene.

Chemical Effects

Naphthalene is a solid, white hydrocarbon, the main constituent of moth balls. It is packaged in ball, flake or cake form as a moth repellent. It sublimes slowly. It has a sharp, pungent odor that is irritating to eyes and upper respiratory passages.

Symptoms/Signs

High concentrations produce headache, dizziness, nausea and vomiting. Acute poisoning also may produce ptosis and clear red secretions around the eye. Chronic toxicity causes weight loss and loss of appetite.

Laboratory

Naphthalene is converted mainly to alpha naphthol in the body and promptly excreted in conjugated form in the urine. Alpha naphthol can be measured by gas chromatography.

Treatment

Morgan (1989, p. 141) provides the following specific suggestions for treatment of naphthalene toxicosis.

- Naphthalene toxicosis caused by vapor inhalation usually can be managed simply by removing the individual to fresh air. Contaminated skin should be washed promptly with soap and water. Contaminated eyes should be flushed with copious amounts of clean water. Irritation may be severe, and if it persists, should receive medical attention.
- If solid naphthalene has been ingested and retained less than several hours prior to treatment, and if the patient is fully alert, the stomach should be emptied. Administer syrup of ipecac, followed by several glasses of water. Dosage for adults and children older than 12 is 30 ml; dosage for children under 12 is 15 ml. When vomiting subsides, give activated charcoal and sorbitol (see Morgan, p. 8). If the patient is obtunded or excited, do not give ipecac, but take steps to protect the airway, then aspirate and lavage the stomach with a slurry of activated charcoal. Leave charcoal and sorbitol in

the stomach before withdrawing the tube (see previous reference). Repeated administration of charcoal every 2 to 4 hours may be beneficial.

If treatment is delayed more than several hours, administer as much activated charcoal orally as the patient will tolerate. Include sorbitol in the charcoal slurry unless diarrhea has already commenced.

Examine the plasma for evidence of hemolysis: a reddish-brown tinge. Examine the blood smear for "ghosts" and Heinz bodies. If present, monitor red blood cell count and hematocrit for anemia, and urine for protein and cells. Measure direct- and indirect-reacting bilirubin in the plasma. Monitor fluid balance and blood electrolytes. If possible, monitor urinary excretion of naphthol to assess severity of poisoning.

Paradichlorobenzene

Toxicology

Liver injury and tremor may occur following ingestion of large amounts. Although accidental ingestions, especially by children, have been fairly common, symptomatic human poisonings have been rare. Other stereoisomers of dichlorobenzene are more toxic than the para-isomer. Some nervous system effects have been observed through excitation of axonic transmission. Ingestion has produced slight increases in the weight of liver and kidneys and a slight focal necrosis and cirrhosis of the liver. However, evidence shows paradichlorobenzene partitions into fat at 10 to 40 times the rate it concentrates in liver or kidneys.

Liver necrosis observed with paradichlorobenzene is proportional to the degree of covalent binding of active metabolites to liver proteins. Paradichlorobenzene has induced some porphyria. It also has produced increased urinary coproporphyrin excretion.

Studies of chronic effects have shown mutagenicity and carcinogenicity in experimental animals. These included renal tubular cell adenocarcinomas, hepatocellular carcinomas and pheochromocytomas.

Chemical Effects

Paradichlorobenzene is the active principle in commercial "moth crystals" and many home garden formulations designed to control wood boring insects. It is solid at room temperature, and is now widely used as a moth repellant, air freshener, and deodorizer in homes and in public facilities. It is commonly placed in public rest rooms in the form of "toilet cakes" or "urinal cookies." The vapor is only mildly irritating to the nose and eyes.

Exposure

Inhalation, dermal, ingestion.

Symptoms/Signs

Symptoms depend upon the amount and route of exposure. They include mild irritation to nose and eyes; headache; weakness; muscle twitches; tremors; loss of equilibrium; horizontal and vertical nystagmus; rapid, labored breathing; and coma. Reversible eye ground changes and systemic changes have been observed.

Laboratory

Paradichlorobenzene is metabolized mainly to 2,5-dichlorophenol, which is conjugated and excreted in the urine. This product can be measured chromatographically.

Treatment

Treatment for paradichlorobenzene toxicosis is outlined above in Morgan's general treatments for fumigants (pp. 137-143).

Carbon Tetrachloride

Toxicology

Carbon tetrachloride is toxic to the central nervous system and liver. It is less toxic than chloroform as a central nervous system depressant, but is much more severely hepatotoxic, particularly following ingestion. Liver cell damage is apparently caused by a free radical generated in the process of initial dechlorination. Kidney injury also occurs; sometimes this is exaggerated by jaundice. Cardiac arrhythmias, progressing to fibrillation, may follow inhalation of high concentrations of carbon tetrachloride or ingestion of the liquid.

Carbon tetrachloride impairs the NADPH-dependent oxidative enzymes in liver microsomes by causing irreversible damage to cytochrome P-450. It does not act as a competitive inhibitor. In the liver, carbon tetrachloride produces elevated levels of glutamicoxaloacetic transaminase and aldolase (commonly used in following the clinical course of human patients poisoned by the compound).

Centrolobular necrosis of the liver is the lesion most characteristic of poisoning by carbon tetrachloride. The necrosis progresses cell by cell. Electron microscopy reveals vesiculation of the rough endoplasmic reticulum, clumps of tangled, smooth membranes, and vacuolization of the Golgi apparatus. It also reveals loss of polysomes and accumulation of fat.

Definite renal tubular lesion, including tubular necrosis and deposition of calcium, have been observed regularly. Mitochondria and not endoplasmic reticulum appear to be the primary subcellular site of carbon tetrachloride toxicity in the kidney.

Chemical Effects

Carbon tetrachloride is a colorless liquid with a sweetish odor. It is nonflammable and noncorrosive. It is inert generally but is decomposed by water at high temperatures. It is not particularly irritating at low concentrations, but in higher concentrations gives a suffocating sensation.

Exposure

Inhalation, oral, dermal. Carbon tetrachloride is readily absorbed by tissues, including the linings of the respiratory and digestive tracts. It also will pass through the skin.

Symptoms/Signs

Symptoms of carbon tetrachloride poisoning may include giddiness, sleepiness and some dizziness. When ingested there may be some increase in peristalsis. There is respiratory excretion (indicated by odor of the breath as well as by chemical analysis). Skin contamination may produce erythemia as well as signs of carbon tetrachloride respiratory excretion. Alcohol consumption increases the toxic effects of carbon tetrachloride. Acute symptoms include kidney and liver failure, narcosis and gastroenteritis.

Laboratory

Many halocarbons, including carbon tetrachloride, can be measured in blood by gas chromatography. Some, including carbon tetrachloride, can be measured in the expired air as well.

Treatment

Morgan (p. 142) outlines specific treatment for carbon tetrachloride poisoning. Several treatment measures have been suggested to limit the severity of hepatic necrosis. Neither the effectiveness nor the safety of any of these measures has been established.

- Inhalation of oxygen at 1 or 2 atmospheres for 2 hours twice daily may have some value.
- Oral administration of tocopherol (vitamin E) in doses of several hundred milligrams per day has been suggested on the grounds of its action as a free radical scavenger.

Oral administration of N-acetyl cysteine (Mucomyst) may be worthwhile as a means of reducing free radical injury. Dilute the proprietary 20 percent product 1:3 in soda pop, and give about 3 ml/kg body weight of the diluted solution as a loading dose. Give half of this dosage every 4 hours after the loading dose for a total of 17 doses. (This dosage schedule is used for acetaminophen poisonings.) Administration via duodenal tube may be necessary in a few patients who cannot tolerate Mucomyst.

Hemoperfusion over activated charcoal should be considered. It was apparently effective in one carbon tetrachloride poisoning. (See Schwarzbeck, A. and W. Kosters, *Arch. Toxicol.*, 35:207-211, 1976.)

Carbon Disulfide

Toxicology

Carbon disulfide vapor is only moderately irritating to upper respiratory membranes, but it has an offensive "rotten cabbage" odor. Acute toxicity is due chiefly to effects on the central nervous system. Long-term occupational exposures have been shown to accelerate atherosclerosis, leading to ischemic encephalopathy, myocardiopathy and gastrointestinal dysfunction. Toxic damage to the liver and kidneys may severely limit the function of these organs.

Chemical Effects

Impurities give carbon disulfide a foul smell. However, in the pure form it has a sweetish odor.

Exposure

Inhalation, ingestion, dermal. Carbon disulfide is readily absorbed from the respiratory and gastrointestinal tracts.

Symptoms/Signs

Symptoms demonstrate the effects of carbon disulfide on the nervous system. They include drowsiness, motor weakness, flaccid paralysis, nerve tenderness, staggering and stumbling as though drunk, extreme thirstiness, loss of appetite, behavioral changes, rigidity and tremor (parkinsonism), and choreatic movments. Inhalation of high concentrations for short periods has caused headache, dizziness, nausea, hallucinations, delirium, progressive paralysis and death from respiratory failure. More prolonged exposure to lesser amounts has lead to blindness, deafness, paresthesia, painful neuropathy and paralysis.

Laboratory

Carbon disulfide can be measured in urine by gas chromatography, but the test is not generally available. A qualitative test for carbon disulfide metabolites in urine (based on their reducing properties) is used for monitoring occupational exposure (Djuric D., N. Serducki and I. Burkes. "Iodine-azide test on urine of persons exposed to carbon disulfide." *Brit. J. Indus. Med.*, 22:321-3, 1965).

Treatment

Morgan (p. 143) suggests the following treatments for carbon disulfide poisoning:

Mild poisonings by inhalation may be managed best by no more than careful observation, even though sensory hallucinations, delirium and behavioral aberrations can be alarming.

Severe poisonings may require the following specific measures:

- If manic behavior threatens the safety of the victim, diazepam, 5-10 mg in adults, 0.2-0.4 mg/kg of body weight in children, administered slowly and intravenously, may be helpful as a tranquilizer. Give as much as is necessary to sedate the patient. Do not give catecholamine-releasing agents such as reserpine and amphetamines.

In severe poisonings by carbon disulfide, pyridoxine hydrochloride (vitamin B_6) may have some antidotal action against the neurotoxic effects. Its value is theoretical; neither its effectiveness nor its safety has been tested in carbon disulfide poisonings. The usual dosage in other poisonings (Isoniazid) has been 5 gm in a 10 percent solution, given slowly and intravenously, or included in a 1-liter intravenous solution of 5 percent glucose. When the victim can swallow, pyridoxine hydrochloride can be given orally in daily doses as high as 25 mg/kg body weight. There is probably little value, and possibly some hazard, in extending the treatment beyond 1 or 2 weeks.

Hydrogen Cyanide

Toxicology

Hydrogen cyanide gas causes poisoning by inactivating cytochrome oxidase, the final enzyme essential to mammalian cellular respiration. The cells of the brain appear to be the most vulnerable to cyanide action. Similar color of the retinal arteries and veins may be a useful sign of cyanide poisoning; it is due to failure of reduction of hemoglobin as blood perfuses poisoned tissues. Cyanide poisoning does not produce cyanosis but leaves the venous blood fully oxygenated and the patient pinker than normal.

The cyanide ion has essentially the same toxicity, regardless of the route by which it is absorbed. Whether the route of exposure is oral or respiratory, victims experience an almost instantaneous collapse and cessation of respiration. After poisoning, the heart may continue for some time after respiration stops. While respiration is active, the venous blood remains oxygenated and the patient's color florid. In fact, this condition may persist if death is sudden.

Accidental poisoning may occur if an applicator remains in a fumigated space or accidentally triggers an M-44 Coyote Getter, or if someone blunders unknowingly into a contaminated environment.

Chemical Effects

The toxicity of hydrogen cyanide, simple cyanide salts, and other compounds such as cyanogen and acrylonitrile depends upon their ability to yield the cyanide ion. Hydrogen cyanide has the characteristic sharp smell of bitter almonds. The ability of acrylonitrile to destroy human epidermis is most likely a local effect of the unmetabolized molecule. It is a curious fact that blood levels in fatal cyanide poisoning usually are higher following ingestion (3.2-160 ppm) than following inhalation (0.5-15 ppm).

Acrylonitrile is biotransformed in the body to hydrogen cyanide. Toxicity and mechanisms of poisoning are essentially the same as have been described for cyanide, except that acrylonitrile is irritating to the eyes and to the upper respiratory tract.

Exposure

Inhalation, ingestion, dermal.

Symptoms/Signs

Unconsciousness and death may occur immediately following inhalation of a high cyanide concentration, respiratory paralysis being the principal mechanism. Lesser exposures cause a constriction and numbness in the throat, stiffness of the jaw, salivation, nausea, vomiting, dizziness and apprehension. Worsening of the poisoning is manifest as violent tonic or clonic convulsions. Trismus and opisthotonos occur. Paralysis follows seizure activity. Incontinence is characteristic. The skin remains pink. Fixed, dilated pupils, bradycardia, and irregular gasping respiration (or apnea) are typical of profound poisoning. The heart often continues to beat after breathing has stopped. A bitter almond odor to the breath or vomitus may be a clue to poisoning, but not all individuals are able to detect this odor.

Laboratory

Cyanide ion from cyanide itself or from acrylonitrile can be measured in whole blood and urine by an ion-specific electrode or by colorimetry. The upper limit in whole blood among nonexposed nonsmokers is about 0.02 mg per liter; it is 0.04 mg per liter in smokers. Symptoms may appear at levels above 0.10 mg per liter. Urine cyanide is usually less than 0.30 mg per liter in nonsmokers, but as much as 0.80 mg per liter in smokers. Thiocyanate, the metabolite of cyanide, also can be measured in blood and urine. It is usually present in plasma at levels less than 4 mg per liter in nonsmokers, but up to 12 mg per liter in smokers. Urine thiocyanate is usually less than 4 mg per liter in nonsmokers, but may be as high as 17 mg per liter in smokers.

Treatment

Morgan (pp. 143-145) makes the following specific suggestions for treating cyanide poisoning:

Poisonings by hydrogen cyanide and acrylonitrile gases or liquids are treated essentially the same as poisoning by cyanide salts. Because cyanide is so promptly absorbed following ingestion, treatment should commence with prompt administration of antidotes, deferring gastric evacuation (in ingestion poisonings) until antidotes have been administered.

Table 27. Morgan's recommended dosages of supplemental sodium nitrite and sodium thiosulfate based on hemoglobin level.

Initial hemoglobin concentration gm/100 ml	Supplemental volume of 3% sodium nitrite ml/kg	Supplemental volume of 25% sodium thiosulfate ml/kg
14.0	0.25	1.25
13.0	0.21	1.05
12.0	0.17	0.85
11.0	0.13	0.65
10.0	0.09	0.45
9.0	0.05	0.25
8.0	0.00	0.00
7.0	0.00	0.00

Morgan states, "Although various cobalt salts, chelates, and organic combinations have shown some promise as antidotes to cyanide, they are not generally available. None have been shown to surpass the nitrite-thiosulfate regimen in effectiveness."

1. If the victim is an adult:
   - Administer oxygen continuously. If respiration fails, maintain pulmonary ventilation mechanically.
   - Administer amyl nitrite (perles) by inhalation for 15 to 30 seconds of every minute, while a fresh solution of 3% sodium nitrite is being prepared.
   - As soon as the solution is available, inject intravenously 10 ml of 3% sodium nitrite solution over a 2- to 4- minute interval, keeping the needle in place. CAUTION: Monitor pulse and blood pressure during administration of amyl nitrite and sodium nitrite. If systolic blood pressure falls below 80 mm Hg, slow or stop nitrite administration until blood pressure recovers.
   - Follow sodium nitrite injection with an infusion of 50 ml of 25% aqueous solution of sodium thiosulfate administered over a 10-minute period. Initial adult dose should not exceed 12.5 gm.

- If symptoms persist or recur, treatment by sodium nitrite and sodium thiosulfate should be repeated at half the dosages given in the previous paragraphs.
- Measure hemoglobin and methemoglobin in blood. If more than 50 percent of total hemoglobin has been converted to methemoglobin, blood transfusion or exchange transfusion should be considered, because conversion back to normal hemoglobin proceeds slowly.

2. If the victim is a child:
   - Give amyl nitrite, oxygen and mechanical respiratory support as recommended for adults.
   - The following dosages of antidotes have been recommended by C.M. Berlin (*Pediatrics*, 46:793-796, 1970): Children who weigh more than 25 kg should receive adult dosages of sodium nitrite and sodium thiosulfate. Children who weigh less than 25 kg should first have two 3- to 4-ml samples of blood drawn and then, through the same needle, receive 10 mg/kg (0.33 ml/kg of 3% solution) of sodium nitrite injected over a 2- to 4-minute period. Following sodium nitrite, administer an infusion of 1.65 ml/kg of 25% sodium thiosulfate at a rate of 3 to 5 ml per minute. At this point, determine the hemoglobin content of the pretreatment blood sample. If symptoms and signs of poisoning persist or return, give supplemental infusions of sodium nitrite and sodium thiosulfate based on the hemoglobin level (Table 27). These recommended quantities are calculated to avoid life-threatening methemoglobinemia in anemic children. They are aimed at converting approximately 40 percent of circulating hemoglobin to methemoglobin. If possible, monitor blood methemoglobin concentrations as treatment proceeds.

Chloropicrin

Toxicology

Chloropicrin is severely irritating to the upper respiratory tract, eyes and skin. Inhalation of an irritant concentration sometimes leads to vomiting. Ingestion could be expected to cause a corrosive gastroenteritis. Chloropicrin is lethal in 10 minutes at 2000 mg/m^3. It is intolerable at 50 mg/m^3. It causes lacrimation and eye irritation at 2 mg/m^3. The odor can be detected at 7.3 mg/m^3. The threshold limit value is 0.7 mg/m^3.

Chemical Effects

Chloropicrin is a colorless, slightly oily liquid with an intense odor. It is sold as a grain fumigant and soil fumigant. It is also mixed with other fumigants to increase its effectiveness or as a warning agent in the case of 98% methyl bromide formulations. Chloropicrin was used as a chemical warfare agent during World War I. Even then, its main value was its irritating properties.

Exposure

Inhalation, mucous membranes, dermal, ingestion.

Symptoms/Signs

Common symptoms of chloropicrin exposure are eye, nose and throat irritation and inflammation; lacrimation; coughing; sore throat; and vomiting. Others include vertigo, headache, nausea and fatigue. Some persons exposed to chloropicrin reported dizziness, drowsiness, wheezing, blurred vision, skin irritation, headache, and a bad taste in the mouth.

Laboratory

There are few laboratory procedures to confirm poisoning by chloropicrin.

Treatment Treatment is symptomatic. Follow steps 1 through 11 beginning on page 36. Then take the following measures to maintain pO_2 above 60 mm Hg (Morgan, pp. 138-141).

- Administer 60 to 100% oxygen.
- Intubate and ventilate mechanically.
- Maintain positive and expiratory pressure breathing.
- Hayes (1991, vol. 1, p. 676) suggests: "Fluid balance must be maintained; use of a diuretic may be required. Steroids may be administered on a short-term basis (2 to 4 days) to decrease the inflammatory response of the lung."

Methyl Bromide

Toxicology Respiratory distress may not occur for 4 to 12 hours after exposure. Methyl bromide sometimes induces pulmonary edema, hemorrhage, or a confluent pneumonia. It is a central nervous system depressant and has prounounced effects on the CNS. It also may cause convulsions.

Chemical Effects Methyl bromide is colorless and nearly odorless, but is severely irritating to the lower respiratory tract. If liquid methyl bromide contacts the skin, severe burning, itching and blistering occurs. Skin necrosis may be deep and extensive.

Exposure Inhalation, dermal, eye.

Symptoms/Signs Early symptoms of acute poisoning include headache, dizziness, nausea, vomiting, tremor and ataxia. Repeated prolonged exposures have led in some cases to a long-lasting syndrome of ataxia, incoordination, muscle weakness and areflexia. One case of recurrent myoclonic seizures required treatment for 5 years following methyl bromide exposure.

Laboratory Methyl bromide yields inorganic bromide in the body; the anion is slowly excreted in the urine (half-life in the body is about 12 days). The serum from persons having no exceptional exposure to bromide usually contains less than 1 mg bromide ion per 100 ml. The possible contributions of medicinal bromides to elevated blood content and urinary excretion must be considered, but if methyl bromide is the exclusive source, serum bromide exceeding 5 mg per 100 ml probably means some absorption, and 15 mg per 100 ml is consistent with symptoms of acute poisoning. Inorganic bromide is considerably less toxic than methyl bromide; serum concentrations in excess of 150 mg per 100 ml occur commonly in persons taking inorganic bromide medications. In some European countries, blood bromide concentrations are monitored routinely in workers exposed to methyl bromide. Blood levels higher than 3 mg per 100 ml are considered a warning that personal protective measures must be improved. A bromide concentration higher than 5 mg per 100 ml requires that the worker be removed from the fumigant-contaminated environment until blood concentrations decline to less than 3 mg per 100 ml.

Treatment Morgan (pp. 140-142) states: "In methyl bromide poisoning, it may be necessary to give benzodiazepines or barbiturates orally for days or weeks after the poisoning to control involuntary motor activity. Consult package inserts for appropriate dosages. If given very soon after life-threatening exposure to methyl bromide, there may be some theoretical value in administering dimercaprol (BAL) in vegetable oil intramuscularly. For adults, give 3-5 mg/kg and repeat every 6 hours until four to six doses have been given. Neither the effectiveness nor the safety of this treatment has been tested in methyl bromide poisoning. CAUTION: Dimercaprol may cause troublesome side effects (hypertension, tachycardia,

nausea, headache, paresthesia, pain, lacrimation, sweating, anxiety and restlessness). Although usually not so severe as to preclude treatment, these effects may require antihistamine therapy."

1,3-Dichloropropene

Toxicology

Dichloropropene and dichloropropane are strongly irritating to the skin, eyes and respiratory tract. Bronchospasm may result from inhalation of high concentrations. Liver, kidney and cardiac toxicity is probably similar to that produced by carbon tetrachloride. It also produces nontumorigenic lesions of the nasal mucosa and changes in the morphology of renal and hepatic tissues.

Chronic effects include mutagenicity but not carcinogenicity. It is weakly mutagenic on liver microsomes. Formulations of 1,3- dichloropropene form mutagenic oxidation product. Chronic toxicity and oncogenicity studies have generally produced negative results.

Chemical Effects

Dichloropropene is a colorless to straw-colored liquid. It is severely irritating to skin, eyes and upper respiratory tract. It also is extremely flammable with a flash point of 92 degrees F (25 degrees C).

Exposure

Inhalation, dermal, eyes.

Symptoms/Signs

Exposure to low concentrations produces fatigue; desensitization or loss of the sense of smell; headache; and chest discomfort. Low concentrations may not immediately irritate eyes, skin or respiratory passages.

Exposure to higher concentrations produces strong irritation of mucous membranes, chest discomfort, headache, weakness and unconsciousness.

Laboratory

Dichloropropene and dichloropropane, like many halocarbons, can be measured in blood by gas chromatography. Like some other halocarbons, they also can be measured in the expired air.

Treatment

Follow steps 1 through 11 beginning on page 36.

Labels provide the following precautionary note to physicians: "Because rapid absorption may occur through lungs if product is aspirated, and cause systemic effects, the decision to induce vomiting or not should be made by a physician. Ir lavage is performed, endotracheal and/or esophageal control is suggested. Danger from lung aspiration must be weighed against toxicity when considering emptying the stomach."

Dibromochloropropane

Toxicology

Dibromochloropropane is irritating to skin, eyes and the respiratory tract. Exposure produces slight to moderate CNS depression. Eye damage has resulted from repeated exposure to the vapors. Liver and kidney damage are prominent features of acute poisoning.

Chronic exposure to relatively low concentrations has led to permanent sterility of workers in a manufacturing plant, by causing diffuse necrosis of seminiferous

tubule cells. Because it is much less odiferous than ethylene dibromide, exposure of workers to toxic concentrations of DBCP is more likely.

Men with chronic exposure to DBCP were mildly to severely oligospermic with some totally azoospermic. An analysis of semen from DBCP-exposed workers showed positive correlation between length of exposure to DBCP and the extent of reduction in sperm production. DBCP-exposed men also produce serum levels of follicle-stimulating hormone (FSH) and luteinizing hormone (LH).

Other chronic effects include gastric squamous carcinoma, papilloma, and tumors of the stomach, lung and nasal cavity.

Chemical Effects Dibromochloropropane produces respiratory irritation at vapor concentrations of 60 ppm or higher.

Exposure Inhalation, dermal, eye.

Symptoms/Signs Symptoms include headache, nausea, vomiting, ataxia and slurred speech, as well as severe and continuous cramping, epigastric and right upper quadrant pain, fever, anorexia, nausea, vomiting and diarrhea.

Laboratory Dibromochloropropane, like many halocarbons, can be measured in blood by gas chromatography.

Treatment Follow steps 1 through 11 beginning on page 36.

Phosphine (Aluminum Phosphide)

Toxicology Phosphine gas is only slightly irritating to the respiratory tract, but is at least as toxic systemically as hydrogen cyanide. Used to treat storage spaces, it is slowly released by hydrolysis of solid aluminum phosphide (phostoxin). Mechanisms of toxicity are not well understood. Pulmonary edema is a common cause of death.

Chemical Effects Phosphine is a colorless gas. Aluminum phosphide is usually formulated as a white pellet. The odor is said to resemble that of carbide or decaying fish. The odor threshold of the gas is about 2 ppm, but because of odorous impurities produced from metal phosphides, the odor threshold for the pesticide is about 0.02 ppm.

Phosphine is spontaneously flammable if a trace of P_2H_4 is present. It combines violently with oxygen and halogens.

Exposure Inhalation, ingestion.

Symptoms/Signs Morgan (1989) lists the principal manifestations of poisoning as fatigue, nausea, headache, dizziness, thirst, cough, shortness of breath, paresthesia and jaundice. Additional poisoning symptoms listed by Hayes (1991) include lassitude, immobility followed by deepened restlesssness, ataxia, pallor, epileptiform convulsions, apnea and cardiac arrest.

Laboratory There are no practical tests for absorbed alkyl oxides, aldehydes or phosphine that would be helpful in the diagnosis of poisoning.

Treatment Morgan gives general guidelines for treating phosphine poisoning (pp. 138-141). He includes this special emphasis: "Control convulsions. Seizures are likely to occur in poisonings by phosphine."

Sulfuryl Fluoride

Toxicology

Sulfuryl fluoride (Vikane® Gas Fumigant) is highly toxic. It is colorless and odorless. Therefore, it has no warning properties. It has an acute oral LD_{50} in rats of 100 mg/kg. Its effect on humans depends on the concentration and the length of exposure.

The mortality curve for acute inhalation exposure is very steep. Only a small margin exists between lethal and non-lethal exposures. The time/concentration relationship holds true: the higher the concentration, the faster the effect. It was not teratogenic in animal studies. Some signs of maternal toxicity (less weight gain and increased water consumption) have been observed. Decreased fetal body weight indicates fetotoxicity.

Chronic, long-term, daily exposure may produce some tissue damage even at relatively low concentrations (less than 30 ppm). Fluorosis of the teeth may occur when humans are chronically exposed. Workers who frequently come into contact with sulfuryl fluoride gas can have their urine checked for fluoride.

Mutagenicity and carcinogenicity are unknown and currently under investigation.

Chemical Effects

Sulfuryl fluoride gas has a very low vapor pressure (BP is -67 degrees F). Skin or eyes exposed to the liquid are injured by freezing. It is packaged as a liquified compressed gas. At low concentration it is not irritating to mucous membranes and gives no warning of its presence. It can penetrate most fibrous or porous materials. Its toxicity and penetrating qualities make it an excellent broad spectrum fumigant.

Exposure

Inhalation, eyes, dermal. Human ingestion is highly unlikely since sulfuryl fluoride liquid turns to a gas at -67 degrees F.

Symptoms/Signs

Persons exposed to sulfuryl fluoride may show little evidence of intoxication at first. Initial effects will probably be on the central nervous system, with slow speech and gait evident. Exposure to high concentrations causes respiratory irritation, pulmonary edema, nausea, abdominal pain, CNS (central nervous system) depression, slowing of movements and speech, and numbness in the extremities. Exposures to progressively higher concentrations produce convulsions, tremors and strychnine-like muscular rigidity.

Laboratory

Urine samples may be analyzed for total fluorides. However, the interpretation of these levels requires a base line (pre-exposure level) to determine if increased fluorides are actually due to sulfuryl fluoride poisoning.

Treatment

Follow steps 1 through 11 beginning on page 36. **There is no known antidote.** Clinical observation is essential. Treatment is based on the clinical judgment of the physician and the individual reaction of the patient.

The manufacturer of sulfuryl fluoride (Vikane® Gas Fumigant) gives more treatment information in bulletins and labels, as well as an emergency telephone number: **(517) 636-4400.**

The manufacturer provides the following general information for physicians: "If the patient is removed to fresh air and put at rest, central nervous system symptoms and signs will be the first to appear. It is essential to keep such an individual at bed rest for at least 24 hours. Clinical observation should be directed at the pulmonary, hepatic and renal systems. A post mortem finding in one fatality attributed to Vikane was pulmonary edema, with death attributed to cardiorespiratory failure. Convulsions may ensue with respiratory arrest being a terminal event. Assisted respiration may be necessary."

Table 28. Common fumigants used in Texas.

CHEMICAL TYPE	BRAND*	CHEMICAL	SITE-PEST	MORGAN (1989)
aldehyde	Acritet, Carbacryl	acrolein (acrylaldehyde)	space fumigant	pp. 138-141
carbon disulfide	Grain Fumigant, Premium Grain Fumigant	carbon disulfide	stored product pests	pp. 138-143
chlorinated hydrocarbon	Borer Control, Moth Crystals, Moth TEK, Deodorizer Rose, Para Moth	paradichlorobenzene	lilac borers, peach borers, lesser peach borers, clothes moths, apple borers	pp. 134-141
	Vertifume, Grain Fumigant, Premium Grain Fumigant	carbon tetrachloride	stored product pests	pp. 138-142
	Telone II, Telone C-17, D-D, Vidden-D	dichloropropene & dichloropropane	soil pests	pp. 138-141
chloropicrin	Larvacide, Picfume, Telone C-17	chloropicrin	stored product pests, soil pests	pp. 138-141
cyanide	Cyanogas, Cyanamid Fumigant, Cyclon, M-44 Coyote Getter	hydrogen cyanide	stored product pests, coyotes	pp. 143-145
halogenated hydrocarbon	Brom-o-gas, Brom-o-sol, Dowfume MC-2, Dowfume MC-33, Terr-o-gas	methyl bromide	stored product pests, drywood termites, soil pests, wood boring beetles	p. 142
	DBCP, Fumazone, Nemagon, Nemafume, Nemaset	dibromochloropropane	soil pests	pp. 138-141
	Bromofume, EDB, Nephis	ethylene dibromide	soil pests	pp. 138-141
hydrocarbon	Moth Balls, Moth Flakes	naphthalene	clothes moths	pp. 138-142
inorganic	Vikane	sulfuryl fluoride	drywood termites, stored products, wood boring beetles	pp. 138-141
	Grain Fumigant, Premium Grain Fumigant	sulfur dioxide	stored product pests	pp. 138-141
oxide	ETO, Oxyrane	ethylene oxide	space fumigant	pp. 138-141
phosphorus compound	Phostoxin, Fumitoxin	aluminum phosphide (phosphine gas)	stored product pests	pp. 138-141

*All brand names are registered trade names.

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