

The Pesticide-Exposed Worker: An Approach To The Office Evaluation

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Abstract: *Background:* Pesticide exposures have clinical, epidemiological, legal, and political ramifications that go beyond the confines of a physician's office. The examination of the employee who has been exposed to a pesticide should be undertaken in an organized and methodical manner so that specific questions of causality can be answered and treatment can be initiated.

Methods: Five representative case studies illustrate different circumstances in which a pesticide injury can be seen in the office setting.

Results and Conclusions: A wide range of pesticides is used in business and home, and the dangers of misdiagnosis, maldagnosis, underdiagnosis, and overdiagnosis are especially common given the variety of chemicals used. The exposed employee must be removed from the source of exposure. Complete decontamination is a primary concern, and patients with unstable vital signs will need to be hospitalized. A methodical office examination, however, can be carried out on many exposed employees. A detailed description of the circumstances of the exposure should be elicited, and the chemical implicated in the exposure should be researched.

A pesticide exposure is a sentinel event in the life of a patient and also suggests that other employees can be exposed. Such an exposure needs to be carefully assessed and documented, and proper treatment must be rendered. Further, the exposure can represent the first of many other exposures that might or might not be reported. Proper notification of authorities can limit exposures before they become severe. (J Am Board Fam Pract 1993; 6:33-41.)

Concerns about pesticide exposures have become common in our working society. The family physician often is the first professional to see a person who could have been exposed to pesticides. That physician must perform a thorough examination to determine whether the symptoms are due to pesticides and map the plan of treatment.

A precise diagnosis, appropriate work-up, and adequate treatment of a pesticide injury by the first physician who sees the pesticide-injured employee is important for five reasons. First, it is essential that the employee receives treatment to alleviate the signs and symptoms caused by the injury. This treatment must also be aimed at returning the employee to work, when possible, and assuring that there are no long-term residual effects. Further, any long-term residual effects must be documented so the patient can receive adequate compensation.

Second, many state public health laws require that pesticide exposures be reported in a timely manner. An accurate database must be established so state authorities can perform accurate epidemiologic studies.

Third, the exposed individual could be extremely anxious and frightened. The news media have exposed the employees and the general public to both fact and alarmist fiction about pesticides. The employee will need reassurance that he or she will probably not have any long-term residual effects from this exposure or a professional statement as to what problems the employee can expect.

Fourth, workers' compensation carriers and the workers' compensation system require timely, accurate, and complete reports. The carrier and employer also need to be assured that the employee really has a pesticide injury and is neither imagining it nor attributing symptoms from another disease process to a pesticide exposure. Workers' compensation payments for permanent and temporary disability will depend largely on the reports of the treating physician. The first examination by

Submitted, revised, 25 August 1992.

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the physician is the most important because it provides an opportunity to describe the mechanism of injury, the symptoms, and the medical history without the embellishments that often develop as time elapses.

Fifth, pesticide injuries can result in litigation in personal injury and workers' compensation courts. For forensic purposes, an acceptable medical opinion requires a reasonable medical probability of exposure that must be clearly separated from mere possibilities and speculations. Fraud must be documented very carefully, and true pesticide exposure needs to be separated from illness from other causes.

Case Reports

Five representative cases are presented to illustrate important aspects in the work-up and treatment of pesticide exposures. The patients resided in the San Joaquin Valley of California, an area of intense agriculture and pesticide use.

Case 1

A 31-year-old woman came to the office complaining of nausea, vomiting, headaches, an erythemic pruritic rash over her entire body, and a sour taste in her mouth.

This case resulted from an incident in which 5 employees were exposed and is representative of the other 4 persons. The farm laborers were working a field in June 1991 when they became ill. The supervisor later learned that an adjacent field had been sprayed the evening before with a mixture of Omite CW and Guthion (azinphos, a wide-range cholinesterase-inhibiting organophosphate insecticide). Drifting had occurred from the sprayed field into the adjacent field, where the employees worked the next morning. All 5 employees came to the physician's office with their immediate supervisor as well as the owner of the company.

The patient's blood pressure was 140/82 mmHg and her pulse was 82 beats per minute. Her physical examination was entirely normal with the exception of a macular papular rash over her entire body. Blood was drawn to measure cholinesterase activity levels and for a complete blood panel, and the patient was sent home to bathe and change her clothes.

When the patient was seen later that same day for an examination, her headache had decreased, and she was feeling better. Her initial blood cho-

linesterase levels were normal. Within 2 days, however, her serum cholinesterase levels began to drop, but they remained within normal levels. Her rash cleared in 1 week, and she returned to work 1 week after the incident. The patient was seen 3 weeks after the exposure, and her physical examination was entirely normal. The case was closed.

At the time of the incident the patient was lactating and had breast fed her baby while in the office waiting room at her first visit. She had taken her child with her to the fields and was breast feeding the child on breaks while working. She was told to stop breast feeding, and her baby was switched to bottle feedings.

The baby, a 4-month-old boy had no rash or any abnormalities whatsoever on physical examination. We elected not to measure his cholinesterase activity levels because of the relatively large amount of blood that would be necessary, and because results of the physical examination were normal. The child was monitored weekly by physical examination for 1 month after the exposure and findings were normal.

A report to the county health department was made using a facsimile (fax) machine, and a telephone follow-up made sure the fax was received; later a written report was sent by registered mail. The following day investigators from the Tulare County agriculture commissioner's office investigated the incident, and full cooperation was provided.

All 5 employees were removed from work until symptoms and signs had resolved. Serial cholinesterase activity levels were measured to watch for a delayed drop in erythrocyte cholinesterase activity levels. The cases were not closed on these workers until 1 month after the incident to be sure that all signs and symptoms had resolved and the patients had no residual effects.

Case 2

A 55-year-old man was sent to the office by his employer after he developed an intensely pruritic rash on his arms. The patient denied any nausea, vomiting, double vision, blurred vision, or dizziness. A complete history and physical examination were performed, the findings of which were non-remarkable. His pulse rate was 72 beats per minute, his blood pressure was 150/86 mmHg, and he had an erythemic macular papular rash, as well as secondary excoriations, covering his arms. Results

of a complete blood panel and serum and erythrocyte cholinesterase activity levels were normal.

This patient had been working with a crew of more than 30 employees, and he was the only one who developed a rash. A telephone call to the employer revealed that the only pesticide used on the grapes where this employee was working was sulphur, which had been applied about 3 weeks earlier. In questioning this employee, he admitted that he had had some problems with rashes to sulphur in other fields but had just "shined it off."

The patient was removed from work for 1 week until his rash cleared. He was treated with oral and topical steroids, and a report was made to the health department. When he returned to his job, his employer was advised to keep him away from sulfur residues in the future. The employee, however, quit and took a position with another company working among fields where sulfur had been used. The employee stated that farm labor was the only occupation in which he could earn a living. The case was closed 1 month after the exposure.

Case 3

A 26-year-old man stated he had been exposed to pesticides and wanted to be removed from work for 1 month because of this pesticide exposure. The patient complained of itching and a rash on his face but denied any other health problems. Further history revealed that the employee was pulling weeds in an irrigation ditch and was slapped in the face by the foliage as he worked.

On examination, the patient had an erythemic rash in linear streaks on his face. His history and physical examination were otherwise normal. His pulse was 82 beats per minute. His cholinesterase activity levels and results of a blood panel were normal.

A telephone call to the employer disclosed that this employee was one of a group of 20 workers who were clearing weeds in an irrigation canal. The entire area where these employees were working had never been sprayed with any pesticides or herbicides. In fact, the employer was an "organic farmer."

The examining physician went out to the area where the exposure occurred and found numerous stinging nettles. A diagnosis of stinging nettle exposure was made. The patient was given topical steroids, and he returned to work in another field.

Case 4

A 27-year-old man who was referred by his employer stated that he had been exposed to and was consequently ill from pesticides and intended to sue the employer, the pesticide manufacturer, and the examining physician. This patient had become ill, experiencing nausea, vomiting, severe pounding headaches, and dizziness every Monday when he went to work; and during the course of the week he felt progressively better. He experienced episodes of double vision but denied any rash. Physical examination was entirely normal with the exception of injected sclera and a slight tremor. His cholinesterase activity levels and the results of laboratory tests were normal.

When telephoned, his employer said that no other employee in a 4-man crew was ill, nor was the employer himself, who worked extensively around the fields. The fields had not been sprayed with any chemicals whatsoever for the 3 weeks before the patient came to the office.

With this knowledge the employee was questioned again, and he admitted to extensive habitual use of alcohol and illicit substances, including cannabis and cocaine. The diagnosis of cocaine and cannabis habituation and alcoholism was made, and a complete report was sent to the workers' compensation carrier.

Case 5

A 33-year-old man came to the office complaining of a "chemical reaction." He gave a 3-week history of headaches, muscle aches and pains, weakness, blurred vision, diarrhea, and lacrimation. The symptoms appeared after he had welded a pesticide spraying rig. This man's job was to repair pesticide spraying rigs, and he often had to weld broken parts. Sometimes the surfaces that he welded were contaminated with pesticides. Two pesticides that he could recall were Lannate (methomyl, a cholinesterase-inhibiting carbamate, used as a nematocide and broad-spectrum insecticide), and Carzol (chlordimeform, an acaricide and insecticide).

This man also worked on the spraying rig as a mixer-loader. Furthermore, he lived in a converted three-story water tower. His quarters were in the second and third stories and the first story was used to warehouse chemicals, including pesticides. He estimated that more than 30 different types of pesticides were stored in the building.

On examination his blood pressure was 124/88 mmHg, and his pulse rate was 78 beats per minute. The entire physical examination was normal including his skin, which showed no rashes.

The employee was removed from work pending the outcome of cholinesterase testing and a blood panel. A sample of urine was also sent to a specialized reference laboratory for a complete pesticide screening. The employee was advised to leave his current living quarters, and a complete report of this case was made to the health department. There was no specific treatment prescribed.

As a mixer-loader the employee had a baseline cholinesterase activity level established 1 year earlier. His plasma cholinesterase baseline level was 5187 mU/mL, and the exposure plasma cholinesterase level was 4287 mU/mL, 82 percent of baseline. The erythrocyte cholinesterase baseline level was 14,852 mU/mL, and the exposure cholinesterase level was 11,698 mU/mL, 78 percent of baseline. These values suggested both acute and chronic exposures. The normal laboratory values for these tests are 2700 to 8000 mU/mL for plasma and 11,100 to 17,900 mU/mL for erythrocyte cholinesterase. If baseline levels had not been available, subtle changes in the plasma and erythrocyte cholinesterase activity levels would not have been discovered.

A complete blood panel that included a complete blood count, liver panel, renal panel, and thyroid tests were normal. The urine pesticide screening revealed 4.3 µg/L of dichlorodiphenyl dichloroethylene (DDE), a metabolite of chlorophenothane (dichlorodiphenyltrichloroethane, DDT).

The employee was returned to duty 3 weeks later, when his symptoms had resolved and his erythrocyte and plasma cholinesterase activity levels had returned to 82 and 123 percent of baseline. He was then closely monitored for the remainder of the spraying season.

This employee brought in a list of 10 pesticides that the spraying company had been using in the previous year and also brought in a list of 20 chemicals that had been stored in the storage room below his living quarters. DDT, a chemical banned from use for several years, was not on the list, and the laboratory reported that the value may have been due to background levels seen in the general population rather than an acute or chronic exposure. Further environmental testing was not

performed because of circumstances outside the control of the treating physician.

This single patient experienced three exposures: chronic exposure from working as a mixer loader while using inadequate protective equipment, acute exposure by welding contaminated spraying rigs without a protective mask, and chronic exposure from living in proximity to a pesticide storage depot. Mandatory reporting prevented further exposure of this patient by alerting the inspectors.

Literature Review

Maddy and associates¹ reviewed illness, injury and death from pesticide exposure from 1949 to 1988. They found pesticide use to be extensive. 268,749,526 kg were sold in 1988 alone. Agricultural application was the largest use followed by household, home and garden, industrial, institutional, and structural use. During the 20 years of data reviewed in their report, there were 48 occupational fatalities, primarily caused by organophosphates, and 10,412 occupational illnesses and injuries.

Ellenhorn and Barceloux,² Morgan,³ and the World Health Organization (WHO)⁴ use a classification of pesticides that includes insecticides, herbicides, rodenticides, fungicides, agricultural fumigants, and structural pesticides. A certain amount of crossover occurs among these categories, but they serve to demonstrate the diversity of pesticides as chemicals (Table 1).

Maddy, et al.,¹ Ellenhorn and Barceloux,² Namba,⁵ and others^{7,8} have listed those occupations in which workers are at risk for exposure to pesticides: applicators, emergency response personnel, flaggers for aerial application, pilots, fumigators, manufacturers, mixer-loaders, and supervisors. In addition, workers in other occupations risk exposure when buildings in which they work are treated with pesticides and when pesticides drift from overflights by application aircraft. The general public risks exposure from overflights, aerosol drifts from applicators, residues on produce, spills, improper use of garden chemicals, and unauthorized entry into restricted fields and storage areas.

Morgan,³ Ellenhorn and Barceloux,² and Hayes⁹ report that the signs and symptoms of toxic reaction vary by pesticide type. Although it is beyond the scope of this article to review the

Table 1. Classes of Pesticides: A Representative List.

Class	Pest	Chemical Type	Examples
Insecticides	Insects, spiders, mites	Organophosphate, cholinesterase-inhibiting	Parathion, malathion, mevinphos (Phosdrin), chlorpyrifos (Dursban)
		Carbamate, cholinesterase-inhibiting	Aldicarb (Temik), methomyl, propoxur (Baygon), carbaryl
		Pyrethrins and pyrethroids	Allethrin, permethrin, cypermethrin
		Organochlorines	Propargite (Ornite), dicofol (Kelthane)
Herbicides (defolients)	Plants	Dipyridyls	Paraquat, diquat
		Chlorophenoxy compounds and dioxin	2,4-D, 2,4,5-T
		Nitrophenolic and nitrocresolic	Dinoseb, dinocap, dinitrocresol
		Chlorate salts, sulfonyleureas, atrazine	Sodium, potassium
Rodenticides	Mice, rats, gophers	Anticoagulants	Warfarin, coumafene, pindone
		Metal phosphides	Zinc, aluminum phosphide
		Miscellaneous	Sodium fluoroacetate, strychnine, Vacor, Red squill
Fungicides	Fungi, molds	Dithiocarbamates	Thiram, ziram
		Organochlorines	Hexachlorobenzene, quintozone
		Dicarboximides	Captan, captafol, folpet
		Miscellaneous	Chlorothalonil, benomyl
Fumigants	Insects, molds, fungi	Halogenated hydrocarbons	Carbon tetrachloride, ethylene dibromide, DBCP, methyl bromide
			Vertebrates
		Sulfur and phosphorus	
	Structural pesticides	Insects, molds, fungi, mites, vertebrates	Organochlorines
Wood preservatives			Pentachlorophenol, arsenicals, creosote, borates, copper, and zinc naphthalenate
Nematicides	Nematodes	Hydrocarbons	DBCP
Molluscicides	Mollusks		Metaldehyde
Avicides	Birds	4-Aminopyridine	Avitrol
Piscicides	Fish	Rotenone	Noxfish
Ovicides	Eggs	Chlordimeform	Ovatoxin

signs and symptoms of each class of pesticide, certain complaints are universal; vertigo, fatigue, nausea, vomiting, rash, or blurred vision should lead the clinician to suspect pesticide poisoning if there is a complaint or possibility of exposure.

Hayes,⁹ Ellenhorn and Barceloux,² and others^{3,4} have suggested several factors to consider when assessing a pesticide exposure: dose, contact time, chemical type, chemical name, and route of exposure. Dose can vary by concentration, both in the delivery container and as the chemical comes in contact with the person. For example, a concentrated chemical can become highly diluted when atomized for spray application. Dose also varies with contact time, i.e., the time from the actual exposure to the time of complete decontamination. Chemical type determines which organ system or systems are affected; for example,

organophosphates affect the neuromuscular junction. The route of exposure can be dermal, respiratory, gastrointestinal, or any combination of the three.

Errors in diagnosis are common. He and colleagues,¹⁰ writing in the People's Republic of China, reviewed 573 cases of acute pyrethroid poisoning reported in the Chinese medical literature from 1983 to 1988. In this series, a number of misdiagnoses, maldiagnoses, and inappropriate treatments were exposed. Specifically, several patients were overdiagnosed as having severe pyrethroid poisoning when they only had mild cases. In addition, the authors described several cases in which pyrethroid poisoning was misdiagnosed as organophosphate pesticide poisoning and atropine was inappropriately prescribed. In one case, a patient actually died of atropine overdose.

The authors also described many cases of maldagnosis in which heat stroke, respiratory infection, and food poisoning were inappropriately diagnosed as pyrethroid exposure and poisoning.

A careful history and physical examination with supporting laboratory tests are essential. Saunders, et al.¹¹ reported an outbreak of dermatitis caused by Omite CR (propargil, a miticide and acaricide) exposure among 198 orange pickers employed by a packing house. The authors emphasized careful documentation of re-entry times, i.e., the interval between spraying a field and when a crew can safely enter the field. These data assist researchers to determine re-entry times or authorities to fine applicators or gang bosses when applicable.

The importance of reporting exposures to health authorities is highlighted by the work of O'Malley and McCurdy.¹² These authors described an incident in which a group of 30 migrant field workers employed by a grape grower in Madera County, California, were exposed to Phosalone (benzophosphate), a cholinesterase-inhibiting organophosphate used as an acaricide, molluscicide, and fruit and nut insecticide. This group complained of gastrointestinal and constitutional symptoms, among others. Many symptoms resolved soon after examination, but 4 patients with severe sinus bradycardia were hospitalized with persistent problems for several days. All but 16 had moderate to severe inhibition of both plasma (nonspecific) cholinesterase and erythrocyte (specific) cholinesterase levels.

The authors called attention to other cases of Phosalone poisoning that could have been underdiagnosed because the signs and symptoms closely mimic gastrointestinal disease. They suggested that the rate of poisoning among agriculture workers can represent sentinel health events: co-workers should be examined to determine whether only 1 field worker or a group has been poisoned. Because subacute poisoning, as well as pesticide poisoning, can mimic many other diseases, field workers with poisoning could be underdiagnosed.

Peoples and Maddy¹³ reviewed an event in which a 120-person grape-picking crew suffered exposure in 1979. The employees were required to enter the field before the 30-day safety interval elapsed, and the entire group became ill as a result of exposure to Torak (dialifor) and Zolone (phosalone). The management of the cases became diffi-

cult because of the many symptoms and signs. Although clinical response was prompt, many exposed workers developed mild symptoms that persisted for weeks after the incident. Agricultural investigators found high levels of both offending chemicals on the leaves of the grapes the employees had been harvesting. The blood chemical levels of the work crew were also high. The exposed workers had a broad-symptom complex that included weakness, one-sided head pressure, nausea, vomiting, tightness of the chest, and blurred vision. Three workers were admitted to the hospital. The authors pointed out that the California reporting system resulted in the other employees being examined, and further illness and injury were avoided by decontamination and removal of the workers from exposure.

Discussion

Given the great amounts of pesticides used in California and the large numbers of crops grown there, the experience of California can be easily applied to other states and countries. The data presented by Maddy, et al.¹ were the result of an extensive reporting system used in California since 1949. Other states have not had systems in place as long, or their reporting system might not be as stringent. It is believed, therefore, that their data most probably reflected the experience of workers around the world.

The work-up of a pesticide-exposed employee or an employee who thinks he or she has been exposed to pesticides needs to be done in a methodical and thorough manner (Table 2). It is important to remove the worker from the location of exposure immediately and to keep that worker away until symptoms have completely resolved and the environment in which the exposure occurred is rendered safe. Workers must be kept out of the fields or buildings until the proper re-entry times have elapsed, spilled chemicals are cleaned up safely, and the area completely decontaminated.

Decontamination of workers should be carried out immediately. Ideally, worker decontamination should occur on the job and should involve a thorough bathing, including washing hair, and a change of clothing. Contaminated clothing becomes hazardous waste and must be discarded in accordance with local regulations.

An exposed worker should be sent to the emergency department for treatment or hospitalization

Table 2. Office Work-up of the Pesticide-Exposed Worker.

Steps	Specifics
Remove from exposure	1. Remove patient from area where exposure occurred
Decontamination	1. Bathing from head to toes with soap and water 2. Segregation and safe disposal of contaminated clothing, wash water, towels, etc.
Transport to emergency department if patient shows hypertension, bradycardia, or shock	1. Appropriate emergency care
Exposure history	1. Contact with employer 2. Material safety data sheet (MSDS) 3. Emphasis on past exposures and drug and alcohol history 4. Thorough discussion of how exposure occurred
Physical examination	1. Thorough standard physical examination
Laboratory studies	1. Complete blood count, renal panel, liver panel, and urinalysis 2. Cholinesterase levels, plasma and erythrocyte 3. Chest radiograph, electrocardiogram as indicated 4. Blood and/or urine chemical levels if indicated and interpretable
Treatment	1. Specific antidote if available 2. Symptomatic treatment
Research on pesticide used (information aids documentation if the research could be done prior to the physical examination and treatment)	1. MSDS (material safety data sheets) 2. Reference texts (Table 3) 3. Poison control centers 4. Specific telephone numbers stated on labels
Work status	1. Off work until signs and symptoms are resolved and laboratory values return to normal
Appropriate reports to health authorities	1. Record documentation in clinic notes
Follow-up	1. Serial examinations 2. Serial vital signs 3. Serial laboratory tests In organophosphate poisonings, repeat cholinesterase tests until levels reach pre-established base line or plateau in the normal range 4. Serial documentation of work status
Case closure	1. Permanent and stationary, reach preinjury state of health 2. Careful documentation of residual impairments or disabilities

if there is evidence of hypotension, shock, bradycardia, or respiratory distress. Many exposed workers who do not exhibit these findings usually do not need to be hospitalized and can be treated on an out-patient basis.

Case 1 illustrates several important points. The patient came to the office accompanied by her supervisor and by the owner of the company. The supervisor and the owner had already researched and found out the chemicals to which the employee had been exposed. The employee was initially examined, had blood drawn to measure cholinesterase activity levels, and was then sent home for decontamination. If hypotension or bradycardia had been found, or if the patients were otherwise dramatically symptomatic, they would have been sent to the emergency depart-

ment or admitted to the hospital. They were doing well, however, their pulse rates were normal, respiration rates were normal, and they could be cared for on an out-patient basis.

Although the basic presenting complaint of all the cases reviewed in this article was that of a "pesticide exposure," only in the last case were there elements of the classic SLUD syndrome associated with pesticide exposures (salivation, lacrimation, urination, diarrhea). Therefore, if a clinician waits for those classic signs, many pesticide exposures will be missed.

A thorough history and physical examination of an exposed patient is extremely important. The physician must document precisely the chemicals to which the employee has been exposed and should obtain the material safety data sheet

(MSDS) on these chemicals. Federal law requires that an employer make available to the employee material safety data sheets. Sometimes, however, an employee can be exposed to pesticides through circumstances outside the control of the employer, such as occurred in case 1. If possible, the physician should contact the person who applied the chemicals to get the proper chemical names and application dates.

It is helpful for the examining physician to gain some background on the pesticides in question. Table 3 presents a short list of references that are useful for physician and clinic offices.

The physician should get a thorough history of all toxic substance exposures including drugs and alcohol. Physical examinations should include particular attention to vital signs. Any rashes, abnormal pupil size, and neurological findings should be carefully documented.

Laboratory tests should be oriented toward immediate diagnosis, as well as long-term follow-up care. Although electrocardiograms and chest radiographs should be reserved for those who have respiratory symptoms or cardiac dysrhythmia, a complete blood count and renal and liver panels should be done on all pesticide-exposed persons to monitor hepatorenal and bone marrow changes.

Cholinesterase activity levels should be measured for all individuals in whom exposure to organophosphates or carbamates is suspected. Reporting serial levels can help document an exposure and recovery, even if the results are reported in the normal range.¹⁴

Whether to perform urine tests for the exposed chemicals needs to be handled on a case-by-case basis. If the offending chemical has not been identified and if the employer, insurance company, or government body needs to have those data for epidemiological or legal reasons, then it is advisable to perform urine pesticide screening. These tests, however, are rarely helpful in the acute diagnosis and treatment of pesticide poisoning because

it can take 2 to 3 weeks to get the results from a reference laboratory.

Many states require that pesticide exposures be reported. It is important to make a telephone or fax report immediately to alert the authorities that other exposed workers may yet be in the area and to record on the clinic notes that such a report was completed. Furthermore, follow-up reports through the mail are often required.

Treatment can vary. There are few specific antidotes to toxic agents. Most employees exposed to pesticides do not need specific treatment because simply removing the individuals from exposure will cause their symptoms to resolve. Topical and oral steroids and antihistamines can relieve rashes; headaches and dizziness might be best left untreated, although the patient often needs reassurances that the symptoms will resolve. Serial laboratory tests and examinations might be required for several weeks to months following the exposure to document resolution or persistence of problems.

Regular reports to workers' compensation carriers on the employee's work status are necessary to assure that temporary disability payments are paid to the employee. The treating physician must document when a case is closed and the exposed worker has reached a preinjury state of health so employers and workers' compensation carriers can close the case. Documentation of permanent disability or impairment is also important so appropriate disposition of an employee's workers' compensation claim can be made. If the family physician is not comfortable in making such definitive statements, appropriate consultation or referral should be arranged.

Summary

Workers who come to the office complaining of a pesticide exposure require a thorough examination with special attention paid to careful documentation. Exposed workers can be affected by the barrage of information and misinformation about pesticides reported by the media, and they have many legitimate concerns. Information gathered by a careful and detailed work-up can be helpful in releasing the worker to a safe work environment and in reassuring the worker that there will be no long-term impairment or disability. If the worker suffers from long-term impairment and disability, documentation to

Table 3. Suggested Office Reference Texts.

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- Ellenhorn MJ, Barceloux DG. *Medical Toxicology. Diagnosis & treatment of human poisoning*. New York: Elsevier, 1987.
- Hayes WJ. *Pesticides Studied in Man*. Baltimore: Williams & Wilkins, 1982.
- Morgan DEP. *Recognition and Management of Pesticide Poisonings*. 4th ed. Washington, DC: Environmental Protection Agency, 1989.
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substantiate the employee's claim is important. Epidemiological and legal investigations to improve worker safety rely on accurate and timely reports from the initial treating physician.

I am grateful to the Huffington Library of the American Academy of Family Physicians for research assistance and to Frederic Rieders, PhD, for reviewing the manuscript.

References

1. Maddy KT, Edmiston S, Richmond D. Illness, injuries, and deaths from pesticide exposures in California 1949-1988. *Rev Environ Contam Toxicol* 1990; 114:57-123.
2. Ellenhorn MJ, Barceloux DG. *Medical toxicology. Diagnosis and treatment of human poisoning*. New York: Elsevier, 1988.
3. Morgan DP. *Recognition and management of pesticide poisonings*. 4th edition. Washington, DC: Environmental Protection Agency, 1989.
4. *Safe use of pesticides: third report of the WHO Expert Committee on Vector Biology and Control*. Technical report series no. 634, Geneva: WHO, 1979.
5. Namba T. Cholinesterase inhibition by organophosphorus compounds and its clinical effects. *Bull World Health Organ* 1971; 44:289-307.
6. *Pesticides: health aspects of exposure and issues surrounding their use. Continuing education seminar for health personnel course, syllabus and manual*. Berkeley: State of California, Department of Health Services, Hazard Evaluation Section, 1988.
7. Warnick SL, Carter JE. Some findings in a study of workers occupationally exposed to pesticides. *Arch Environ Health* 1972; 25:265-70.
8. Lessenger JE, Riley N. Neurotoxicities and behavioral changes in a 12-year-old male exposed to dicofol, an organochloride pesticide. *J Toxicol Environ Health* 1991; 33:255-61.
9. Hayes WJ. *Pesticides studied in man*. Baltimore: Williams & Wilkins, 1982.
10. He F, Wang S, Liu L, Chen S, Zhang Z, Sun J. Clinical manifestations and diagnosis of acute pyrethroid poisoning. *Arch Toxicol* 1989; 63:54-8.
11. Saunders LD, Ames RG, Knaak JB, Jackson RJ. Outbreak of Omite-CR-induced dermatitis among orange pickers in Tulare County, California. *J Occup Med* 1987; 29:409-13.
12. O'Malley MA, McCurdy SA. Subacute poisoning with phosalone, an organophosphate insecticide. *West J Med* 1990; 153:619-24.
13. Peoples SA, Maddy KT. Organophosphate pesticide poisoning. *West J Med* 1978; 129:273-7.
14. Coye MJ, Barnett PG, Midtling JE, Velasco AR, Romero P, Clements CL, et al. Clinical confirmation of organophosphate poisoning by serial cholinesterase analyses. *Arch Intern Med* 1987; 147: 438-42.