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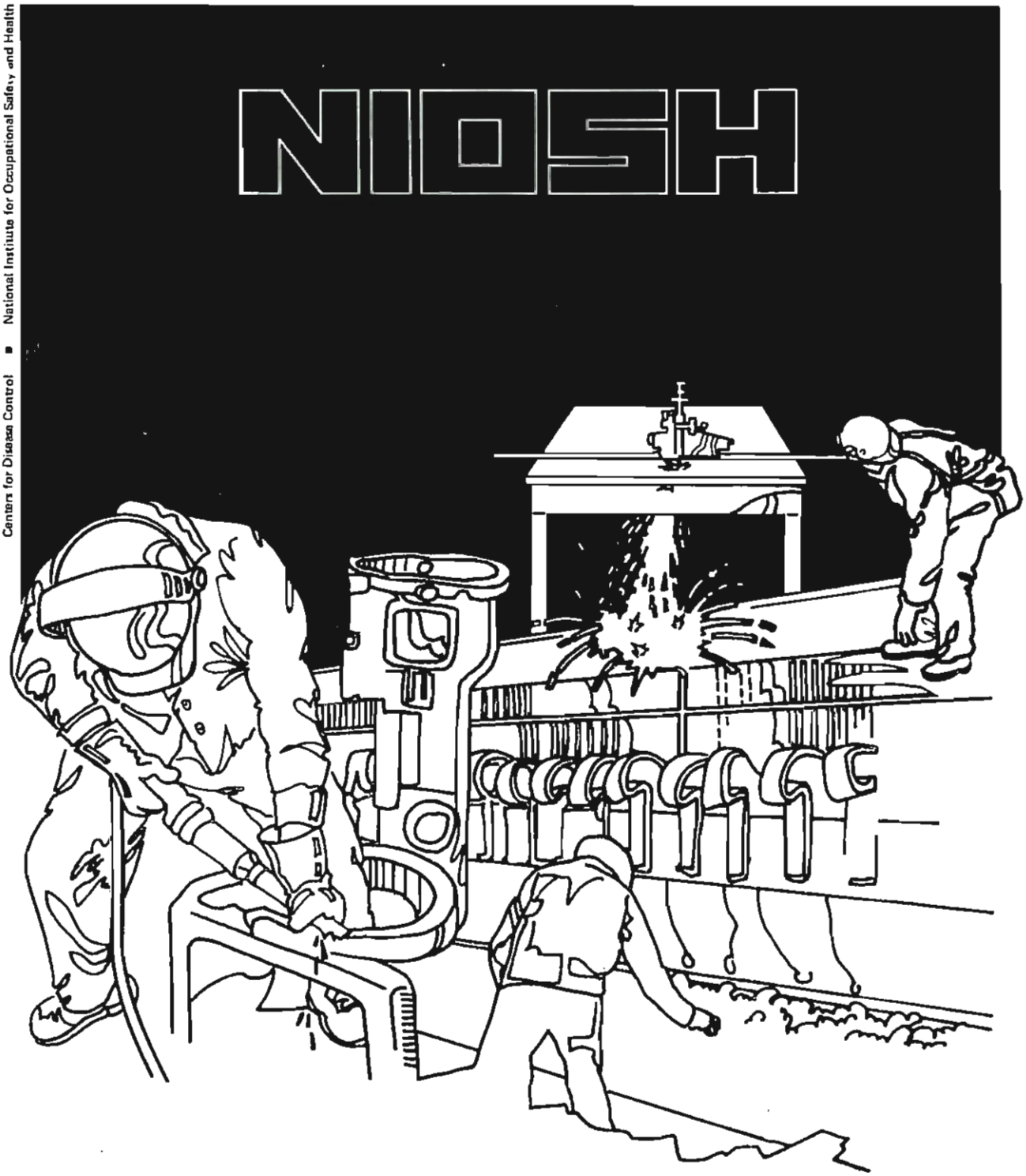

Health Hazard Evaluation Report

HETA 83-373-1501
BROWN'S BRIDGE ANIMAL HOSPITAL
GAINESVILLE, GEORGIA

PREFACE

The Hazard Evaluations and Technical Assistance Branch of NIOSH conducts field investigations of possible health hazards in the workplace. These investigations are conducted under the authority of Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 29 U.S.C. 669(a)(6) which authorizes the Secretary of Health and Human Services, following a written request from any employer or authorized representative of employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

The Hazard Evaluations and Technical Assistance Branch also provides, upon request, medical, nursing, and industrial hygiene technical and consultative assistance (TA) to Federal, state, and local agencies; labor; industry and other groups or individuals to control occupational health hazards and to prevent related trauma and disease.

HETA 83-373-1501
SEPTEMBER 1984
BROWN'S BRIDGE ANIMAL HOSPITAL
GAINESVILLE, GEORGIA

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I. SUMMARY

On July 27, 1983, the National Institute for Occupational Safety and Health (NIOSH) was requested to evaluate the cause of neurologic symptoms (shooting pains, numbness, and back pain) among workers at the Brown's Bridge Animal Hospital. Chemical exposures at the hospital included anesthetic gases and pesticides.

To determine whether health complaints were related to occupational exposures, NIOSH investigators conducted site visits at the animal hospital on August 22 and 23, 1983. NIOSH investigators inventoried pesticides in use and reviewed work practices and working conditions. We also interviewed all eight workers in the veterinary hospital. In addition, seven workers consented to physical examination and blood tests to measure cholinesterase activity.

During the site visit, NIOSH investigators noted that 22 preparations of insecticide dips, shampoos, pills, powders and sprays were in use or dispensed by the hospital. One pesticide, fenthion, was being used for an unlabeled indication: the control of fleas on dogs. Fenthion is readily absorbed through skin, highly fat soluble, and has prolonged biologic effects. No protective equipment was used to avoid skin contact during work with pesticides, except that the groomer wore gloves and a dust respirator for work with certain dips. Two workers were affected by shooting pains, muscle weakness, back pain, and numbness. One additional worker experienced shooting back pain rarely, and another had numbness and tingling of her hands and feet at night. Physical examination revealed decreased foot sensation and eye muscle weakness in one symptomatic worker. Review of medical histories failed to reveal alternate causes of these symptoms. Nerve conduction studies were all normal.

A telephone survey of the veterinarians in charge of surrounding hospitals revealed that no similar illnesses were known to exist among their workers. In addition, fenthion was not used at all, or was used much less frequently at surrounding veterinary hospitals.

A small toxicologic study triggered by this investigation tested fenthion for long-term neurotoxicity in hens. This test showed persistent effects indicating that fenthion may cause permanent neurologic damage. Further testing is indicated to confirm this finding.

Based on these findings, NIOSH concluded that a health hazard existed from exposure to fenthion pesticide at the Brown's Bridge Animal Hospital. Because of the multiple pesticide exposures, definitive proof is not possible. Fenthion has been replaced by less toxic insecticides.

KEYWORDS: SIC 0742 (Animal Hospitals), fenthion, veterinary hospitals, neurologic effects, delayed neurotoxicity, organophosphorus pesticides

II. INTRODUCTION

On July 27, 1983, the National Institute for Occupational Safety and Health (NIOSH) was requested by the president of the Brown's Bridge Animal Hospital in Gainesville, Georgia, to evaluate a potential health hazard involving two workers' neurologic symptoms of shooting pains, numbness and back pain. One worker had been diagnosed as having a polyneuropathy in April, 1983. The neurologist suspected toxic damage from a pesticide. Another worker at the animal hospital had similar symptoms plus chronic diarrhea, difficulty walking, and loss of short-term memory. NIOSH investigators made a site visit on August 22 and 23, 1983.

III. BACKGROUND

The Brown's Bridge Animal Hospital was established in 1974 in a new building by the merger of two veterinary practices. Most of the employees were hired at that time. The animal hospital specializes in the care of dogs and cats, and does some boarding of animals.

The staff consists of eight persons: two veterinarians, three veterinary assistants, one receptionist/bookkeeper, one kennel worker, and one animal groomer. The hospital is open six days a week. The workers have flexible job duties, with each one occasionally assisting in all areas of the hospital. Ten to fifteen animal surgeries are performed weekly.

Starting in September, 1982, two workers noticed they had similar health problems of back pain, shooting pains, and transitory numbness. This led to the medical evaluation of one patient, and the subsequent request for a health hazard evaluation.

IV. EVALUATION DESIGN AND METHODS

A. Environmental

NIOSH investigators inspected the job site with particular attention to the presence and usage of drugs, pesticides, and chemicals. These compounds were inventoried, and their usage was observed for work practices and possible routes of exposure. Storage practices were also evaluated. Animal surgery was also observed. A bulk sample of Spotton brand of fenthion insecticide was obtained.

NIOSH investigators selected four other animal hospitals from the telephone book and called the veterinarians. Pesticide usage information was collected, and health complaints among the hospital staffs were solicited.

B. Medical

Medical evaluation consisted of questionnaires, neurologic examinations, and blood sampling for plasma and red blood cell (RBC) cholinesterase activity levels. All eight workers completed the physician-administered questionnaire, and seven workers consented to physical examination and blood testing. The questionnaire collected demographic information, job title and duties, frequency of symptoms of acute and chronic pesticide toxicity, frequency of pesticide exposures at home and at work, and questions about general health problems associated with nerve damage.

Physical examination tested nerve function, sensation, reflexes, and motor strength.

Blood samples were collected by venipuncture. Plasma cholinesterase and RBC cholinesterase were measured by the method described by Ellman.[1]

V. EVALUATION CRITERIA

A. Environmental Criteria

As a guide to the evaluation of the hazards posed by workplace exposures, NIOSH field staff employ environmental evaluation criteria for assessment of a number of chemical and physical agents. These criteria are intended to suggest levels of exposure to which most workers may be exposed up to ten hours per day, 40 hours per week for a working lifetime without experiencing adverse health effects. It is, however, important to note that not all workers will be protected from adverse health effects if their exposures are maintained below these levels. A small percentage may experience adverse health effects because of individual susceptibility, a pre-existing medical condition, and/or a hypersensitivity (allergy).

In addition, some hazardous substances may act in combination with other workplace exposures, the general environment, or with medications or personal habits of the worker to produce health effects even if the occupational exposures are controlled at the level set by the evaluation criterion. These combined effects are

often not considered in the evaluation criteria. Also, some substances are absorbed by direct contact with the skin and mucous membranes, and thus potentially increase the overall exposure. Finally, evaluation criteria may change over the years as new information on the toxic effects of an agent become available.

The primary sources of environmental evaluation criteria for the workplace are: 1) NIOSH Criteria Documents and recommendations, 2) the American Conference of Governmental Industrial Hygienists' (ACGIH) Threshold Limit Values (TLV's), and 3) the U.S. Department of Labor (OSHA) occupational health standards. Often, the NIOSH recommendations and ACGIH TLV's are lower than the corresponding OSHA standards. Both NIOSH recommendations and ACGIH TLV's usually are based on more recent information than are the OSHA standards. The OSHA standards also may be required to take into account the feasibility of controlling exposures in various industries where the agents are used; the NIOSH-recommended standards, by contrast, are based primarily on concerns relating to the prevention of occupational disease. In evaluating the exposure levels and the recommendations for reducing these levels found in this report, it should be noted that industry is legally required to meet only those levels specified by an OSHA standard.

A time-weighted average (TWA) exposure refers to the average airborne concentration of a substance during a normal eight- to ten-hour workday. Some substances have recommended short-term exposure limits or ceiling values which are intended to supplement the TWA where there are recognized toxic effects from high short-term exposures.

A. Toxic Effects of Anesthetic Gases

In the criteria document for a recommended standard for occupational exposure to anesthetic gases, NIOSH states: "Current scientific evidence obtained from human and animal studies suggests that chronic exposure to anesthetic gases increases the risk of both spontaneous abortion among female workers and congenital abnormalities in the offspring of female workers and the wives of male workers. Risks of hepatic and renal diseases are also increased among exposed personnel. In addition, physiological function may be impaired. Effects on the central nervous system due to acute exposures to anesthetic gases have been associated with headaches, nausea, fatigue, irritability, etc." However, control procedures and work practices presented in the document should prevent the effects caused by acute exposure and significantly reduce the risk associated with long-term, low-level

exposure. A dose/response relationship for halogenated anesthetic toxicity has not been defined.[2]

That same NIOSH publication recommends a maximum exposure of 25 parts per million (ppm) nitrous oxide (time-weighted average concentration during anesthetic administration) and 2 ppm halogenated anesthetic when used alone, or 0.5 ppm when used with nitrous oxide. These recommendations are based upon available technology in reducing waste anesthetic gas levels. Previous studies in animal hospitals have shown significant reductions in halothane concentrations following introduction of vacuum scavenging systems for removal of waste anesthetic gases.[3]

B. Toxic Effects of Pesticides

1. Organophosphorus Insecticides:

Seven organophosphorus compounds were used in the animal hospital: malathion, fenthion, phosmet, dichlorvos, chlorpyrifos, crotoxyphos, and cythioate. In addition to these compounds, it should be remembered that many organophosphorus compounds have breakdown products or impurities which may be present that can be more toxic than the parent compound.

The acute toxic effects of organophosphorus pesticides are well known.[4] These effects result from impairment of acetylcholinesterase, the enzyme that degrades the neurotransmitter acetylcholine. Following inhalation, ingestion, or skin contact with high levels of organophosphates, a worker often experiences muscle weakness, nausea and vomiting, inappropriate sweating, difficulty walking, excessive salivation, chest tightness, diarrhea, and blurred vision. The mild effects of organophosphate exposure are reversible as the body slowly renews the supply of functional acetylcholinesterase. The chronic effects of organophosphate toxicity are not as well known. Memory loss, decreased ability to concentrate, and mood depression have been reported. However, the difficulty in objectively measuring these abilities and the wide variation between individuals makes the demonstration of significant impairment difficult.[5] The reversibility of these changes is also unknown.

One important chronic effect of organophosphate poisoning is organophosphate-induced delayed neuropathy (OPIDN).[6,7] This neuropathy usually occurs eight to fourteen days following an acute exposure to certain organophosphate compounds. The

neuropathy usually begins in the legs, first causing burning or tingling sensations, then causing weakness in the lower leg area and feet. The thighs and arms also become involved. Severe cases proceed to complete paralysis, impaired respiration, and death. Confusion, headache, disorientation, and altered mental and emotional states have also been reported. OPIDN has been reported following exposure to many different phosphorus-ester compounds. None of the compounds encountered at this animal hospital has been previously recognized as causing OPIDN. The nerve damage of OPIDN is usually permanent.

Fenthion, the topically-applied organophosphorus pesticide used in this animal hospital has several important toxicologic properties. It is highly fat soluble. This property allows it to accumulate in body tissues. Second, it is activated in the body. This means that the maximum effects of an absorbed dose will not appear for a few days, and will last for weeks.[8] Finally, it is readily absorbed through the skin. This combination of properties allows workers with skin exposure to build up body levels and maintain toxic symptoms for weeks. The possibility of chronic exposure causing permanent nerve damage has not been fully studied.

2. Chlorinated Hydrocarbons

Three chlorinated hydrocarbons were in use at the animal hospital: hexachlorocyclohexane (benzene hexachloride), dichlorophen, and methoxychlor. Absorption of these compounds occurs via the skin, respiratory or digestive systems. Hexachlorocyclohexane is widely used and is infrequently reported as a cause of poisoning. However, severe cases of hexachlorocyclohexane poisoning have shown convulsions, muscle spasms, and various neurologic abnormalities. Methoxychlor poisoning has not to our knowledge been reported.

3. Carbamates

Two carbamate compounds were identified in the animal hospital: carbaryl and propoxur. Absorption of carbamate compounds occurs by inhalation and ingestion, but skin absorption is highly variable, depending on the compound. Carbamates, like organophosphates, act by inactivating the enzyme acetylcholinesterase. In general, recovery from carbamate poisoning is more rapid than from organophosphate poisoning. Carbaryl is well absorbed through the skin. Toxicity in man from carbaryl poisoning includes sweating,

stomach pain, vomiting, headache, and blurred vision. High doses in hens was reported to cause a reversible neurotoxicity. Propoxur poisoning in humans has been reported as causing similar symptoms as carbaryl poisoning.

VI. RESULTS AND DISCUSSION

A. Environmental

The potential chemical exposures for the eight workers are listed in Appendix I. Although the greatest potential for exposure to most pesticides occurred in the groomer's job, this individual had no health complaints. The symptomatic workers had no unique exposures. However they were exposed to fenthion, and stated that they handled fenthion more frequently than the other workers, often getting it on their skin. Fenthion was used topically on dogs for the control of fleas.

B. Medical

1. QUESTIONNAIRE RESULTS

All eight workers answered the questionnaire. The history of acute symptoms of cholinesterase inhibition and the prevalence of chronic poisoning symptoms are given below. If listed below, the worker experienced the acute symptom at least once during the 12 months preceding the questionnaire. In addition, none of the workers reported the presence of diabetes mellitus, chronic renal failure, or a family history of similar neurologic disorders. None of the workers reported drinking more than eight drinks per week.

HISTORY OF ACUTE PESTICIDE POISONING SYMPTOMS IN THE PRECEDING 12 MONTHS

	Number of Workers Affected
Headaches	5
Shortness of breath or chest tightness	5
Unusual weakness	3
Nausea or vomiting	3
Inappropriate sweating	2
Difficulty walking	1
Excessive salivation	1
Blurred vision	0

HISTORY OF SYMPTOMS OF CHRONIC TOXICITY IN THE PRECEDING 12 MONTHS

	Number of Workers Affected
Parasthesias	5
Decreased recent memory	2
Impaired concentration	1
Unusual depression	1

The index workers were defined as those two workers who had multiple neurologic symptoms. The first worker had experienced the gradual onset of low back pain, bilateral leg pain, generalized parasthesias, and leg muscle weakness. The second worker had experienced the gradual onset of difficulty walking, depression and low back pain. Following the initial interviews on August 23, 1983, both of the index workers have reported by telephone that they have had worsening of their symptoms. The second worker has had the onset of continuous painful parasthesias on the soles of both feet, worsened by walking. The first worker has noticed a rapid decrease in short-term memory, requiring frequent referrals to clinic notes to remember client names, etc.

2. PHYSICAL EXAMINATION

Physical examination of the seven employees revealed few clinical findings. The second worker, affected with the most severe symptoms, had demonstrable weakness of the ocular muscles and was unable to maintain upward gaze. This person also had a sensory neuropathy causing decreased sensation to pinprick below the left knee.

3. LABORATORY RESULTS

Results of the blood tests show that none of the seven workers tested fell outside of the reference range for cholinesterase activity. These results should be interpreted with caution, however, because the biologic effect of pesticide toxicity is dependent on the degree of inhibition of the individual's baseline enzyme activity: an individual can be symptomatic and still have results within the normal limits.

CHOLINESTERASE ACTIVITY

Worker	A	B	C	D	E	F	G
RBC Cholinesterase	3.00	4.20	3.20	3.70	3.20	3.90	3.20
Plasma Cholinesterase	6.50	4.50	4.00	3.90	3.70	3.70	5.20

RBC Cholinesterase given in IU/ml (Reference range 3.00-5.00)

Plasma cholinesterase given in mIU/ml (Reference range 2.50-7.10)

The job title of the workers is not listed to preserve confidentiality.

4. TELEPHDNE SURVEY

Four veterinarians in the surrounding community were surveyed by telephone. In general, chemical use in their practices was similar to the Brown's Bridge Animal Hospital with the exception of fenthion. Two of the veterinarians did not use fenthion, and two of the veterinarians used it rarely, with stringent precautions. These precautions included isolation of the dog from the owner for 24 hours after fenthion application, and not allowing the dog to travel home in an enclosed car with the owner during this period. None of the other veterinarians complained of any neurologic symptoms, nor were they aware of any health problems among their employees.

5. LABORATORY FOLLOWUP

As a result of this investigation, Dr. Robert Metcalf of the University of Illinois initiated a small neurotoxicity study dermally exposing three chickens to five milligrams per kilogram per day of fenthion. Although the experiment was designed to last 60 days, pesticide exposure had to be stopped after 18 days due to severe illness and weight loss in the exposed chickens. Over the next 32 days, the chickens failed to recover normal walking ability. This suggests that permanent nerve damage may result from chronic exposure. Further studies are planned to see if these results can be reproduced.

VI. RESULTS AND DISCUSSION

The two index workers at the animal hospital both meet the definition of polyneuropathy; that is, they have had abnormalities in two or more peripheral nerves. The veterinarian has symptoms dominated by low back

and leg pain, generalized parasthesias, and loss of short-term memory. The veterinary assistant has mild ataxia, loss of short-term memory and painful parasthesias on the bottoms of both feet. Two additional workers had a single neurologic symptom: tingling hands and feet at night or shooting back pain. These two workers were not considered cases due to the rare, intermittent symptoms. It is possible that they have early or mild symptoms of nerve damage.

The diagnosis of polyneuropathy can often be strengthened by clinical examination and laboratory nerve testing. In this case, decreased leg sensation and painful hyperesthesia of the left sole was found in the veterinary assistant on August 23, 1983. Nerve examination findings can change over time; additional information could be gained by repeated examinations over time. Laboratory nerve testing is most helpful for the detection of demyelinating diseases. In this case, no abnormalities were found.

There are many causes of polyneuropathy: systemic diseases such as diabetes mellitus, deficiency of vitamin B_{12}, alcoholism, and toxic damage from pesticides to name a few of the causes. A review of medical and environmental histories for the two index cases failed to identify a possible cause for their nerve damage except for their contact with pesticides.

A review of the human effects of pesticides indicates that acute, chronic, and delayed neuropathy can occur. The acute effects have been discussed above and are reversible. The chronic effects have been recognized most often in workers in pesticide manufacturing plants and application processes. These chronic effects include peripheral nerve damage and central nervous system effects. Delayed neuropathy has been recognized following exposure to certain organophosphorus compounds--hence the name organophosphate-induced delayed neuropathy (OPIDN). OPIDN has not been considered a chronic effect because previous investigations have focussed on acute exposures which usually exhibit acute toxic symptoms and are followed by eight to 14 days of normal nerve function. After this grace period, the nerve disease produced by axonopathy becomes apparent and results in permanent nerve damage, which is fairly stable in severity. OPIDN has been considered separately from other chronic pesticide toxicity because 1) it has a unique time course, 2) it is only caused by organophosphorus compounds, 3) it spares the central nervous system.

In this case, the index workers have had long term, intermittent exposures to a wide variety of pesticide compounds. Most of the compounds are widely used in similar situations without known problems. Our attention was directed to fenthion because 1) this usage is unusual, 2) fenthion is highly fat soluble, allowing for build-up,

and 3) little information is available on human toxicity associated with fenthion. Whether the symptoms here represent chronic pesticide toxicity or OPIDN is unknown. In this setting with multiple potential exposures, it is not possible to single out exposure to fenthion epidemiologically. Subsequent studies, as described under laboratory follow-up above, have supported our suspicion that fenthion is the cause. It is possible that exposures to other compounds caused additive or synergistic neurologic effects.

VII. RECOMMENDATIONS

Fenthion should not be used for unapproved indications. Under no circumstances should skin contact be allowed. Accidental skin contamination should be removed promptly by washing with soap and water.

Work practices for handling pesticides should be improved. Skin contact should be avoided, and any pesticide on the skin should be promptly removed with soap and water. Precautions should also be taken to avoid inhaling pesticide dust or mist. Storage of pesticides should be in secure solid containers. Loose bags should be closed securely and placed in solid containers.

The anesthesia machine which is leaking should be repaired. Levels of anesthetic should be measured in the operatory during sugery. If these levels are above those recommended by NIOSH, a scavenging device for waste anesthetic gas should be installed.

Rabies prophylaxis immunization should be offered to all employees. Booster shots should also be scheduled. Tetanus immunization should also be kept current.

VIII. REFERENCES

1. Ellman GL, Courtney KD, Andres V, Jr., Featherstone RM. A New and Rapid Colorimetric Determination of Acetylcholinesterase Activity. Biochem Pharmacol 1961; 7: 88-95.

2. National Institute for Occupational Safety and Health. Criteria for a recommended standard--occupational exposure to waste anesthetic gases and vapors. Cincinnati, Ohio: National Institute for Occupational Safety and Health, 1977. (DHEW publication no. (NIOSH) 77-140).

3. Dressen DW, Jones GL, Brown J, Rawlings CA. Monitoring for Trace Anesthetic Gases in a Veterinary Teaching Hospital. JAVMA 1981; 179: 797-9.

4. Hayes WJ, Jr. Pesticides Studied in Man. Baltimore, Maryland: Williams and Wilkins, 1982.

5. Rodnitzky RL, Levin HS, Mick DL. Occupational Exposure to Organophosphate Pesticides, A Neurobehavioral Study. Arch Env Health 1975; 30: 98-103.

6. Metcalf RL. Historical Perspective of Organophosphorus Ester-Induced Delayed Neurotoxicity. Neurotoxicology 1982; 3: 263-7.

7. Abou-Donia MB. Organophosphorus Ester-Induced Delayed Neurotoxicity. Ann Rev Pharmacol Toxicol 1981; 21: 511-48.

8. Merrill DG, Mihm FG. Prolonged Toxicity of Organophosphate Poisoning. Critical Care Medicine 1982; 10: 550-1.

IX. AUTHORSHIP AND ACKNOWLEDGEMENTS

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X. DISTRIBUTION AND AVAILABILITY OF REPORT

Copies of this report are currently available upon request from NIOSH, Division of Standards Development and Technology Transfer, 4676 Columbia Parkway, Cincinnati, Ohio 45226. After 90 days, the report will be available through the National Technical Information Service (NTIS), 5285 Port Royal, Springfield, Virginia 22161. Information regarding its availability through NTIS can be obtained from NIOSH Publications Office at the Cincinnati address. Copies of this report have been sent to:

1. Brown's Bridge Animal Hospital
2. NIOSH, Region IV
3. OSHA, Region IV

For the purpose of informing affected employees, copies of this report shall be posted by the employer in a prominent place accessible to the employees for a period of 30 calendar days.

APPENDIX I

EXPOSURES BY JOB TITLE

This table lists the potential chemical exposures of the workers at the animal hospital by job title. Potential exposures are defined as use on at least a biweekly basis of a compound in liquid or powder form. Actual exposure is defined as skin contact or breathing the dust, mist, or fumes. For some jobs with occasional exposures, the unusual exposures are listed separately. The information in the table was obtained from workers or by observation by NIOSH personnel.

Groomer: The animal groomer worked in the kennel area and was primarily concerned with bathing animals using dips and shampoos, and trimming hair. In addition, the groomer worked in the kennels around caged animals.

Adams Anticrawl	spray	pyrethrins 0.5% piperonyl butoxide 0.1% n-octyl bicycloheptane dicarboximide 0.166% propoxur 1.0%
Adams Flea Off	spray	pyrethrins 0.1% piperonyl butoxide 1.5% n-octyl bicycloheptane dicarboximide 0.5% isopropyl alcohol 97% bisbutenylenetetrahydrofurfural
CEVA seleen	shampoo	selenium disulfide
Ectocide	dip	malathion 57% concentrate
Gamma Rx	dip	hexachlorocyclohexane
Mitaban	dip	amitraz emulsion 19%
Mycodex	shampoo	lauramide DEA 5% Polyethylene glycol "600" distearaate piperonyl butoxide 0.5% pyrethrins 0.05%
Mycodex	shampoo	sodium lauryl sulfate 12%
Paramite	dip	phosmet
Ritter's Flea	powder	carbaryl 2.8% methoxchlor 0.25%
Sendran	dip	propoxur 8%

Kennel Worker: The kennel worker took care of the caged animals and cleaned their cages. Occasionally he would spray the kennel area with a pesticide spray.

Vetkem yard	spray	chlorpyrifos 6.7%
Adams Flea Off	spray	Ingredients listed above
Sevin	dust	carbaryl
Unknown brand	spray	chlordane

Secretary/Book-keeper: This worker did clerical work in the reception area and occasionally handled animals.

Adams Anticrawl	spray	Ingredients listed above
Adams Flea Off	spray	Ingredients listed above

This worker also had rare exposure to:

Paramite	dip	phosmet
Spotton	liquid	fenthion 20% topical liquid

Veterinarians and Veterinary Assistants: These workers handled animals, performed surgery, and occasionally applied pesticides.

Adams Anticrawl	spray	Ingredients listed above
Adams Flea Off	spray	Ingredients listed above
Spotton	liquid	fenthion 20% topical liquid
Ritter's Flea	powder	Ingredients listed above
Waste anesthetic	gas	halothane methoxyflurane

In addition, the veterinary assistants would occasionally dip animals and be exposed to:

Gamma Rx	dip	hexachlorocyclohexane
Mitaban	dip	amitraz emulsion 19%
Paramite	dip	phosmet
Sevin	dust	carbaryl