Gasoline Sniffing and Lead Toxicity among Siblings—Virginia

Between March 1983 and December 1984, six of seven children from a family of nine developed lead toxicity from chronic sniffing of gasoline (Table 1). The children ranged in age from 10 years to 17 years; five of the six were boys. Health officials became aware of the problem after neighbors complained that the children were stealing gasoline. Attempts to control their behavior by issuing locking gas caps and providing family and individual counseling were unsuccessful. Neither the parents nor an older sister, who denied sniffing gasoline, had elevated blood lead levels.

The family lived in Virginia on an isolated lot in a rural, coastal county on the Chesapeake Bay. Despite a thorough investigation that included analyses of water, paint, and soil samples, no environmental source of lead other than gasoline could be identified. Abandoned automobiles, gardening machinery, and storage cans containing gasoline were easily accessible to the children.

One of the older boys introduced the practice to his siblings after discovering the effects of inhalation while siphoning gasoline. The children would sniff the fumes for 1-2 minutes until feeling the acute effects, which included euphoria, lethargy, loss of appetite, slurred speech, and blurred vision. These symptoms usually lasted several hours. One child reported occasional headaches and vomiting shortly after sniffing the gasoline.

Frequency of usage varied for each child, ranging from once a month to several times weekly. All the children tended to increase the frequency of sniffing during the summer months when they were out of school, and their activities were less supervised. Blood lead values obtained for three of the children during 1984 showed an increase from February through December. A similar trend during the same period was seen in the other family members who reported sniffing gasoline.

In November 1984, a physician found signs of dysdiadochokinesia (dysfunction of ability to carry out rapidly alternating movements) in two of the children, whereas the other four had normal physical examinations. After hospitalization and treatment, their blood lead levels decreased and the children were placed in supervised foster homes. Since placement, all have reportedly stopped sniffing gasoline.

Reported by J Owens, MD, L Soles, Middle Peninsula Health District, J Conover, Div of Consolidated Lab

<table>
<thead>
<tr>
<th>Sibling</th>
<th>Age</th>
<th>Blood lead, µg/dl*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10</td>
<td>Mean: 46, Range: 19.98</td>
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<tr>
<td>2</td>
<td>11</td>
<td>33</td>
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<tr>
<td>3</td>
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<tr>
<td>4</td>
<td>14</td>
<td>45</td>
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<tr>
<td>5</td>
<td>15</td>
<td>45</td>
</tr>
<tr>
<td>6</td>
<td>17</td>
<td>43</td>
</tr>
</tbody>
</table>

*Five to seven samples taken per child.

Editorial Note: Lead in gasoline is present in the form of tetraethyl lead (TEL). It is an organic compound first introduced during the 1920s as a gasoline additive because of its antiknock properties (1). After absorption through inhalation, TEL is metabolized to triethyl lead and then converted to inorganic lead (2).

Gasoline additives are a significant source of lead in the environment, and reduction of the lead content of gasoline has been associated with decreases in blood lead levels in the U.S. population (3). Recently, the U.S. Environmental Protection Agency announced, effective January 1986, a 10-fold reduction in the stan-
Recognizing and Preventing Occupational Diseases in Virginia

An estimated 20 million work-related injuries and 390,000 new work-related illnesses occur each year in this country. Yet the number of work-related injuries and diseases reported each year is much lower. In Virginia, these illnesses include the most commonly reported occupational skin diseases, repetitive motion trauma disorders, occupational lung diseases, noise-induced hearing loss, occupational cancers and a variety of other work-related disabilities. Workplace exposures may (1) cause disease, (2) act as cofactors in disease, or (3) exacerbate an underlying disease. The difficulty in accurately estimating the frequency of work-related disease is due to several factors:

1. Occupational illnesses are often clinically indistinguishable from other diseases.
2. Many occupational diseases, including pneumoconioses, chemical neuropathies, certain cumulative trauma disorders, mesothelioma and other occupational cancers, begin slowly after many years of exposure, making cause-and-effect relationships difficult to assess.
3. Workers may not recognize the symptoms as being work-related, they often do not know what substances they have been exposed to, and they may fear that reporting such problems will result in job loss.

Overall, 75% of U.S. workers are employed in establishments with less than 500 employees. Many of these industries have no on-site medical providers, and it frequently falls on the primary care physician to recognize a condition as work-related.

Recognizing Occupational Diseases

Recognition that a patient’s illness might be work-related is frequently a good clue to the correct diagnosis. In addition, recognition allows preventive action to be initiated, determines whether other workers may be at similar risk, helps to assure that affected workers receive the compensation legally due them, and promotes the discovery of new relationships between exposure and consequent illness.

The occupational history is the most important determinant in assessing whether a medical problem is work-related. A complete occupational history has five parts:

1. A description of all current and previous jobs.
2. A listing of all chemical, physical (noise, temperature extremes), and biological hazards to which exposed, including an assessment of the amount and route of exposure and the types of workplace protective systems in effect.
3. The timing of symptoms in relation to work exposure.
4. The presence of similar symptoms among coworkers.
5. Exposure to nonwork hazards, such as cigarette smoke or chemicals used in the home, that may account for or exacerbate symptoms.

Obtaining information about the hazards of certain exposures is not always easy. The following resources are widely available to physicians and can shorten the time spent researching potential toxicity:

1. Reference books, including The Clinical Toxicology of Commercial Products, by Gleason, Gosselin and Hodge; the NIOSH publication Occupational Diseases: A Guide to Their Recognition; the NIOSH/OSHA Occupational Health Guidelines for Chemical Hazards.
2. Computerized data banks, including MEDLINE AND TOXLINE.
3. The Virginia Toxic Substances Information Bureau, at (804) 786-1763 (Virginia Department of Health) maintains an inventory of chemicals by industry and employs toxicologists to assist with data interpretation.
4. The manufacturer’s name, and often his address, are usually printed on product labels. In contacting a manufacturer, ask for the Material Safety Data Sheet on the substance of interest; this sheet contains information on the proper handling and toxicity of the chemical.
5. The regional Poison Control Center has information on most toxic substances.
6. The Hazard Communication Standard recently promulgated by OSHA will enable health professionals and workers to obtain information from employers on specific chemicals at each worksite.
Measles In Northern Virginia

Three cases of rubeola are currently being investigated in Fairfax County. All three cases are associated with a case among Christian Science attendees of a summer camp which was held at Buena Vista, Colorado in July.

Ten Virginia youths are known to have attended the camp, five as campers and five as counselors. One of these counselors, an 18 year old white male, had onset of rash illness on July 31 (this illness was later diagnosed as rubeola) and one camper, a 15 year old white male, became ill on August 7. No illnesses have been reported amongst the other eight Virginia attendees. The third patient (the only second generation case) is a noncamper sibling contact of an ill camper from California. She was visiting relatives in Fairfax County when she developed a rash on August 16. Because of their religious beliefs, none of the three patients had been immunized against rubeola.

Investigators from the Health Department are currently tracing each contact of these cases to determine immunization status and attempt to prevent further spread of the disease.

The Colorado Department of Health has closed the implicated camp on July 27 after learning on July 24 of a case of rubeola in a camper. Children from 24 states returned home, where they were advised to remain quarantined for the duration of the incubation period.

For further information about this outbreak, contact the Immunization Program at 804-786-6264.

Continued from page 1

dard allowable for lead in gasoline, from 1.1 g to 0.1 g per gallon of gasoline, and is currently considering a total ban on all lead additives (4).

Previous reports of lead toxicity from gasoline sniffing have been of American and Canadian Indians (2,5). The acute effects of inhaling gasoline, which may be caused by TEL or other volatile hydrocarbons found in gasoline, have reportedly been similar to those found in the Virginia children (6). More severe effects in those with higher blood lead levels have included seizures and acute metabolic encephalopathy (2).

Chronic gasoline sniffing can result in significant lead toxicity, which may go undetected until severe medical problems arise. Besides providing medical care for lead toxicity, healthcare providers need to understand the social and cultural factors influencing young people to abuse chemicals and drugs (5).

CDC recommends that all children between 9 months and 6 years of age be screened for lead toxicity, defined as a blood lead level of 25 µg/dl or greater and an erythrocyte protoporphyrin (EP) level of 35 µg/dl or greater. The most common source of lead in lead poisoning is lead-based paint. As evidenced by this report, older children and adolescents are also at risk of lead toxicity from different sources of lead in the environment (7).

References

Revision of the Case Definition of Acquired Immunodeficiency Syndrome

Patients with illnesses that, in retrospect, were manifestations of acquired immunodeficiency syndrome (AIDS) were first described in the summer of 1981 (1,2). A case definition of AIDS for national reporting was first published in the MMWR in September 1982 (3,4). Since then, the definition has undergone minor revisions in the list of diseases used as indicators of underlying cellular immunodeficiency (5-8).

Since the 1982 definition was published, human T-cell lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV) has been recognized as the cause of AIDS. The clinical manifestations of HTLV-III/LAV infection may be directly attributable to infection with this virus or the result of secondary conditions occurring as a consequence of immune dysfunction caused by the underlying infection with HTLV-III/LAV. The range of manifestations may include none, nonspecific signs and symptoms of illness, autoimmune and neurologic disorders, a variety of opportunistic infections, and several types of malignancy. AIDS was defined for national reporting before its etiology was known and has encompassed only certain secondary conditions that reliably reflected the presence of a severe immune dysfunction. Current laboratory tests to detect HTLV-III/LAV antibody make it possible to include additional serious conditions in the syndrome, as well as to further improve the specificity of the definition used for reporting cases.

The current case definition of AIDS has provided useful data on disease trends, because it is precise, consistently interpreted, and highly specific. Other manifestations of HTLV-III/LAV infections than those currently proposed to be reported are less specific and less likely to be consistently reported nationally. Milder disease associated with HTLV-III/LAV infections and asymptomatic infections may be reportable in some states and cities but will not be nationally reportable. Because persons with less specific or milder manifestations of HTLV-III/LAV infection may be important in transmitting the virus, estimates of the number of such persons are of value. These estimates can be obtained through epidemiologic studies or special surveys in specific populations.

Issues related to the case definition of AIDS were discussed by the Conference of State and Territorial Epidemiologists (CSTE) at its annual meeting in Madison, Wisconsin, June 2-5, 1985. The CSTE approved the following resolutions:

1. that the case definition of AIDS used for national reporting continue to include only the more severe manifestations of HTLV-III/LAV infection; and
2. that CDC develop more inclusive definitions and classifications of HTLV-III/LAV infection for diagnosis, treatment, and prevention, as well as for epidemiologic studies and special surveys; and
3. that the following refinements be adopted in the case definition of AIDS used for national reporting:
   a. In the absence of the opportunistic diseases required by the current case definition, any of the following diseases will be considered indicative of AIDS if the patient has a positive serologic or virologic test for HTLV-III/LAV:
      (1) disseminated histoplasmosis (not confined to lungs or lymph nodes), diagnosed by culture, histology, or antigen detection;
      (2) isosporiasis, causing chronic diarrhea (over 1 month), diagnosed by histology or stool microscopy;
      (3) bronchial or pulmonary candidiasis, diagnosed by microscopy or by presence of characteristic white plaques grossly on the bronchial mucosa (not by culture alone);
      (4) non-Hodgkin’s lymphoma of high-grade pathologic type (diffuse, undifferentiated) and of B-cell or unknown immunologic phenotype, diagnosed by biopsy;
      (5) histologically confirmed Kaposi’s sarcoma in patients who are 60 years old or older when diagnosed.
   b. In the absence of the opportunistic diseases required by the current case definition, a histologically confirmed diagnosis of chronic lymphoid interstitial pneumonitis in a child (under 13 years of age) will be considered indicative of AIDS unless test(s) for HTLV-III/LAV are negative.
   c. Patients who have a lymphoreticular malignancy diagnosed more than 3 months after the diagnosis of an opportunistic disease used as a marker for AIDS will no longer be excluded as AIDS cases.
   d. To increase the specificity of the case definition, patients will be excluded as AIDS cases if they have a negative result on testing for serum antibody to HTLV-III/LAV, have no
Federal Regulations Related to Animal Rabies Vaccines

Revised regulations setting uniform age and dosage requirements for vaccinating animals against rabies were recently issued by the government agency that regulates animal vaccines, the Animal and Plant Health Inspection Service (APHIS) of the United States Department of Agriculture. Effective July 15, 1985, rabies vaccine should be administered to animals at three months of age or older with a repeat dose one year later. Subsequent vaccinations are given based on the duration of immunity established for the particular vaccine used.

The requirement that inactivated rabies vaccines should be administered intramuscularly at one site in the thigh has been deleted. Inactivated vaccines proven effective with other-than-intramuscular administration will contain label and enclosure recommendations for administration by other routes. This means that unless label recommendations state otherwise, the vaccine must still be given intramuscularly at one site in the thigh.

The recommendation for annual rabies revaccination in high risk areas has also been deleted. Animals vaccinated with products known to confer immunity for more than one year have not been found more susceptible to rabies during the period of immunity than those revaccinated annually.

Dog Rabies Reported

An astute veterinarian in Staunton examined a 10 year old mixed border collie in early June. The dog was showing ataxia, pupils of unequal size and did not close its mouth. There were no signs of aggression or seizure activity. Suspicious of rabies, the veterinarian questioned the owner about the possibility of the dog having had an encounter with a wild animal. The owner finally remembered a fight between the dog and a raccoon when they were in Swoope, Virginia approximately two weeks previously. The dog was sacrificed and tested and found to be rabid. The dog’s owner and his son received rabies post-exposure prophylaxis, but because of the veterinarian’s early suspicions, none of the veterinary hospital personnel received an exposure requiring treatment.

This is the first rabid dog to be reported in Virginia since 1982 when two rabid dogs and 10 rabid cats were reported for the year. Although rabies occurs rarely in domestic animals, these are the species responsible for a disproportionate number of human exposures. It behooves all who are involved in animal care and rabies control to have a high index of suspicion for rabies when handling animals.
Cases of selected notifiable diseases, Virginia, for the period August 1 through August 31, 1985

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<td></td>
<td>This Month</td>
<td>Last Month</td>
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<tr>
<td>Measles</td>
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<tr>
<td>*Bacterial</td>
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<tr>
<td>Hepatitis A (Infectious)</td>
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<tr>
<td>B (Serum)</td>
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<tr>
<td>Non-A, Non-B</td>
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<tr>
<td>Salmonellosis</td>
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<td>Gonorrhea</td>
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<td>Rocky Mountain Spotted Fever</td>
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<td>Rabies in Animals</td>
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<td>Kawasaki’s Disease</td>
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<tr>
<td>Other: Acquired Immunodeficiency Syndrome</td>
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<td>13</td>
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</tbody>
</table>

**Counties Reporting Animal Rabies:** Augusta 2 raccoons; Fauquier 3 raccoons, 1 skunk; Rockingham 1 raccoon, 1 skunk; Shenandoah 1 raccoon; Warren 1 gray fox; Arlington 1 fox; Fairfax 1 bat; Scott 1 skunk; Hanover 1 bat.

**Occupational Illnesses:** Carpal tunnel syndrome 11; Pneumoconiosis 11; Hearing loss 6; Silicosis 5; Asbestosis 4; Dermatitis 2; Mesothelioma 2

*other than meningococcal

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