Leads from the MMWR

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Preventing Lead Poisoning in Young Children—United States

The CDC has issued a new statement on preventing lead poisoning in young children.[1] This statement replaces the 1978 statement,[2] which defined levels for elevated blood lead, undue lead absorption, lead toxicity, and lead poisoning. The 1985 statement is intended to serve as a guideline for lead-poisoning prevention programs in the United States.

Since 1978, investigators have reported adverse effects from low-level lead exposure on children's behavior and intelligence,[3] hemoglobin formation in red blood cells,[4] and metabolism of vitamin D.[5] These studies demonstrate that little or no margin of safety is associated with a level of 30 μg of lead per deciliter (μg/dL) of whole blood—the lowest level defined as elevated in the CDC's 1978 statement.

To be successful, a screening program designed to prevent childhood lead poisoning requires not only an acceptable and cost-effective screening procedure but also medical follow-up and means of preventing the child from future exposure to lead.[6] The erythrocyte protoporphyrin (EP) test is recommended as the screening test for lead toxicity because it can be easily performed on a drop of blood obtained from a finger prick and placed in a portable fluorometer. Since EP levels increase in both lead poisoning and iron deficiency, follow-up testing for elevated blood lead and/or iron deficiency must be done.

Some major changes in the 1985 statement compared with the 1978 statement are as follows:

1. An elevated blood lead level, which reflects excessive absorption of lead, is defined as a concentration of lead in whole blood of 25 μg/dL or greater (formerly 30 μg/dL or greater).
2. Lead toxicity is defined as an elevated blood lead level with an EP level in whole blood of 35 μg/dL or greater (formerly 50 μg/dL or greater).
3. Lead is most harmful to children between the ages of 9 months and 6 years. Ideally, all children should be screened. As more children are screened for iron deficiency by EP testing, simultaneous lead screening of these same groups becomes feasible.
4. For EP levels greater than 35 μg/dL, EP values obtained with hematofluorometers are generally lower than EP values obtained by the extraction method. Therefore, separate cut-off levels are used for classifying the urgency of medical follow-up.
5. Greater reliance is placed on the calcium disodium EDTA mobilization ("Provocative Chelation") test in determining whether a full course of chelation therapy is indicated for children with blood lead levels in the 25 to 55 μg/dL range.

The revised lead statement, *Preventing Lead Poisoning In Young Children: A Statement by the Centers for Disease Control: January 1985*, will be available on request after March 1, 1985, from Publication Activities, Center for Environmental Health, Centers for Disease Control, Atlanta, GA 30333; (404) 452-4102.

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Editorial Note: The second National Health and Nutrition Examination Survey (NHANES II, 1976-1980) found that children from all geographic and socioeconomic groups are at risk of lead poisoning.[7] An estimated 3.9% (or nearly one of 25) of the children in the United States under 5 years of age had blood lead levels of 30 μg/dL or greater—levels possibly causing adverse physiologic and neurobehavioral effects. Between 1976 and 1980, the overall mean blood lead levels dropped from 14.6 μg/dL to 9.2 μg/dL, and this corresponded with a decline in the sales of leaded gasoline during this period.[8]

Lead-based paint continues to be the major source of high-dose lead exposure and asymptomatic lead poisoning for children in the United States. Since 1977, paint produced for household use must, by regulation, contain no more than 0.06% (600 parts per million [ppm]) lead by dry weight, but some paints manufactured in the 1940s for indoor use contained more than 50% (500,000 ppm) lead. An estimated 27,000,000 households in this country remain contaminated by lead paint.[9]

Typically, symptomatic lead poisoning occurs

among children under 6 years of age living in deteriorated, pre-World War II housing. Repeated ingestion of nonfood substances has been shown to be associated with lead poisoning in young children,[10] but it is not a prerequisite for lead poisoning,[11] since children's normal mouthing behavior alone is sufficient to cause those living in contaminated homes to have high lead exposure. Lead poisoning has been reported in children whose parents moved to a city as "urban homesteaders"; the children were exposed to chips, dust, or fumes from lead-based paint when the old houses were remodeled or renovated.[10]

Other potential sources of lead exposure include the use of imported lead-glazed pottery for cooking[12] or storing food and hobbies and activities involving lead, such as working with stained glass or casting lead objects.

The highest priority for screening should be given to 12- to 36-month-old children who live in or frequently visit older, dilapidated housing, who live near lead smelters or other industrial sources of lead, or whose parents work with materials containing lead.

Screening all children for lead toxicity—including those not suspected of having been exposed to lead—is feasible, since the EP test can also be used as the screening for iron deficiency. Recently, in a nutritional assistance program, the EP test was used to screen children for iron deficiency,[13] and some Hmong refugee children were found to have lead toxicity. The source was traced to a Hmong folk remedy used for treating infants and children with fevers.

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Botulism From Fresh Foods—California

In August 1984, three cases of botulism were reported in California from two episodes in which the ill persons had eaten improperly handled food made from fresh ingredients.

EPISODE 1.—Botulism was reported in a 61-year-old Santa Cruz County woman and her 13-year-old granddaughter. The older woman had classic symptoms of bilateral ptosis, diplopia, and facial weakness; the granddaughter was less ill. Food histories revealed no recent exposures to home-canned food, but improper food handling was identified as the likely cause of illness. Three days before onset, the grandmother prepared two turkey loaves that included cereal, onion, and green pepper. One loaf was consumed without incident immediately after cooking. The other was inadvertently stored in the gas oven with the pilot light on (later measured at 32.2 °C [90 °F]), until the grandmother discovered it the next afternoon. She tasted a small portion before reheating it at approximately 150 °C (300 °F) for approximately 20 minutes and served the turkey loaf to the three other members of her household. Thirty-six hours later, she awoke with ptosis, diplopia, and facial weakness. Of the others who ate the rewarmed loaf, only the granddaughter developed symptoms. When questioned, she could not recall tasting the turkey loaf with her grandmother before reheating, but did recall eating a portion from the center of the loaf. Type A botulinal toxin was detected in the sera of both patients. Trivalent botulinum antitoxin was administered, and both recovered completely. Since the turkey loaves were completely consumed, confirmatory tests on the suspected vehicle were not possible.

EPISODE 2.—A 22-year-old Orange County man awoke at 2 AM with vomiting, blurred vision, and a "thick tongue." Symptoms progressed to total quadriplegia, then respiratory failure requiring mechanical ventilation. Forty hours before onset, he had consumed stew prepared by his roommate from fresh ingredients (including meat and unpeeled potatoes and carrots), then left overnight at room temperature. The stew was cooked in a 7-inch deep pot filled to the top and simmered for 45 minutes; the gas was then turned off and the pot left on the range. The roommate ate it hot after the initial cooking, without incident. The patient tasted it without reheating 16 hours later and complained of a bad taste. The roommate confirmed a sour taste, immediately spit it out, rinsed his mouth, and remained well. Type A botulinal toxin was detected in the patient's serum; he was treated with botulinal antitoxin and recovered after extended hospitalization.

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Adapted from *California Morbidity* (Feb 1, 1985;4), as reported by D Corzine, MD, Capitola, M Stroe, MD, Santa Cruz County Health Dept, CS Kim, MD, J Lysiak, MD, L Spurgeon, MD, J Wallace, MD, M Gallagher, Anaheim, T Prendergast, MD, Orange County Health Dept, SB Werner, MD, California Dept of Health Svcs; Enteric Diseases Br, Div of Bacterial Diseases, Center for Infectious Diseases, CDC.

Editorial Note: Because spores of *Clostridium botulinum* are ubiquitous in soil, they can contaminate fresh foods, particularly those harvested from the ground. The spores are quite heat resistant and can survive boiling for several hours. For spores to germinate and produce toxin, several conditions must be met, including appropriate temperature and pH and oxygen contents. Foodborne botulism generally results from home-canned vegetables that are contaminated with spores and are improperly prepared, thereby allowing the production of botulinal toxin. Toxin can also be elaborated in foods that are initially cooked, then held at ambient temperatures for at least 16 hours. The cases presented here are not unique, since the same mechanism of toxin production appears to have accounted for previous episodes of botulism from commercial pot pies, sauteed onions, and, in one instance, a baked potato.[1-4] These foods were cooked, allowed to stand at ambient temperatures, and consumed later without reheating. Foods heated for serving should either be eaten hot, or refrigerated and later reheated thoroughly (since the toxin is heat labile) before serving.

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Surveillance and Assessment of Alcohol-Related Mortality—United States

Reduction of morbidity and mortality associated with misuse of alcohol is a major target of the 1990 objectives for the nation.[1] Recently available Multiple Cause of Mortality tapes from the National Center for Health Statistics (NCHS) represent a tool for more comprehensive surveillance and assessment of alcohol-related mortality.[2] National multiple-cause data tapes for 1980 were analyzed (1) to evaluate completeness of mortality reporting by comparing counts of alcohol-related mortality recorded as the underlying cause with those recorded as contributing causes on the death certificate* and (2) to estimate premature mortality resulting from alcohol misuse.

Alcohol-related mortality attributable to specific

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Table 1—Number of deaths and ratio of deaths to physicians' reports of underlying causes, by alcohol-related causes of mortality—United States, 1980.*

Cause of death	ICD-9-CM§	No. deaths†			Ratio of deaths to underlying causes
		Underlying cause	Contributing causes	Total	
Alcohol dependence	303	4,351	13,911	18,262	4.2
Alcoholic cirrhosis¶	571.0-571.3	12,951	2,223	15,174	1.2
Alcohol Abuse**	305.0	893	3,903	4,796	5.4
Excessive blood alcohol	790.3	11	1,351	1,362	123.8
Alcoholic cardiomyopathy**	425.5	650	129	779	1.2
Alcohol psychosis	291	454	309	763	1.7
Alcohol gastritis	535.3	84	30	114	1.4
Alcohol polyneuropathy**	357.5	4	9	13	3.3

*Includes nonresident deaths.
†Because more than one condition may appear as contributing cause for any one death, the numbers in the "contributing causes" and "total" columns are not mutually exclusive.
§The International Classification of Diseases, 9th Revision, Clinical Modification.
¶Includes all types of alcoholic liver disease (ie, fatty liver, alcoholic hepatitis, etc).
**New diagnostic categories in ICD-9-CM.

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diagnoses has been recorded on death certificates as either the underlying or contributing cause of death (Table 1). For example, 85% of the 15,174 mentions of alcoholic cirrhosis as a cause of death in 1980 were recorded as the underlying cause of death; a similar pattern was seen with alcoholic gastritis, alcoholic cardiomyopathy, and alcoholic psychosis. In contrast, although alcohol dependence was listed as a contributing cause of alcohol-related mortality in 13,911 deaths, it was only recorded as the underlying cause of death on 4,351 death certificates, 24% of the total alcohol dependence-associated deaths.** A similar pattern was seen with the third leading cause of alcohol-related mortality, alcohol abuse, which was listed as the contributory cause in 3,903 (81%) of 4,796 deaths.

Estimates of premature mortality[3] associated with alcohol misuse among persons aged 1 year through 64 years are based either on underlying cause or on contributing cause (Table 2). Alcohol dependence and alcoholic cirrhosis, the two major reported causes of alcohol-related mortality, account for substantial years of potential life lost (YPLL) due to alcohol use.** When the average YPLL per death is examined, however, it is apparent that deaths resulting from the acute effects of alcohol account for relatively more mortality in younger persons. For example, an average of 29.1 years of life were lost for each death associated with excessive blood alcohol levels, in contrast with 14.4 years lost for each death from alcoholic cirrhosis.

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Editorial Note: For two reasons, these data conservatively estimate total mortality and premature mortality associated with alcohol misuse. First, only causes of death presumed to be specific for alcohol have been included, while other deaths resulting from liver disease, but not attributed specifically to alcohol misuse (eg, other cirrhosis, ICD-9-CM 571.4-571.9), are excluded. Second, deaths from injuries (eg, motor vehicle incidents, homicides, suicides, fires, and falls), many of which may be alcohol-related, have been excluded.[4,5] More precise monitoring of the effectiveness of efforts to reduce morbidity and mortality associated with alcohol misuse will require more complete reporting on death certificates or alternative sources of mortality data.

*An algorithm is used by NCHS to assign underlying and contributing causes from those causes listed on the death certificate.

**The two patterns are not mutually exclusive. For a given death where the underlying cause is alcoholic cirrhosis, for example, alcohol dependence may be a contributing cause. While underlying causes can be added across conditions, contributing causes and total counts cannot be added.

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Table 2—Years of potential life lost (YPLL) and average YPLL per reported death, by alcohol-related cause of mortality—United States, 1980.

Cause of death	No. deaths			YPLL			Average YPLL per reported death*
	Underlying cause	Contributing causes	Total	Underlying cause	Contributing causes	Total	
Alcohol dependence	3,436	10,340	13,776	52,831	154,228	207,059	15.0
Alcoholic cirrhosis	10,159	1,435	11,594	149,605	17,183	166,788	14.4
Alcohol abuse	797	3,395	4,192	17,368	83,738	101,106	24.1
Excessive blood alcohol	11	1,262	1,273	269	36,719	36,988	29.1
Alcoholic cardiomyopathy	518	96	614	7,782	1,420	9,202	15.0
Alcohol psychosis	344	205	549	5,683	2,993	8,676	15.8
Alcoholic gastritis	69	26	95	1,249	444	1,693	17.8
Alcohol polyneuropathy	3	6	9	9	43	52	5.8

*Average YPLL equals the total YPLL divided by the reported number of persons 1 year through 64 years old who died from each cause.