Hazard of Lead Exposure in the Home from Recycled Automobile Storage Batteries

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ABSTRACT. Two families from rural areas of North Carolina had excessive lead exposure which resulted from either recycling exhausted automobile storage batteries in the home or burning the discarded battery casings for home heating. One child developed encephalopathy resulting in permanent brain damage. Decontamination efforts reduced the quantity of lead in the home environment by >50%. Rural children, previously considered to be at low risk, may in fact receive profound exposures which may go unrecognized until encephalopathy occurs. Occupational histories should be obtained from parents in order to detect children at risk from environmental toxins brought into the home on workmen's bodies and clothing. *Pediatrics* 68:225–230, 1981; *lead poisoning, lead encephalopathy, environmental toxins, storage batteries.*

Although childhood lead exposure has been classically associated with the ingestion of lead-based paint, other sources, especially automobile storage batteries, can pose a hazard from the time of original manufacture through recycling and ultimate disposal. Increased lead absorption and clinical lead poisoning of children of battery factory workers have recently been recognized[1-3]; older literature has documented the danger of using battery casings, discarded after recycling operations, as fuel for home heating.[4,5] Recently, two families from geographically separate regions of rural North Carolina have had excessive lead exposure and one child developed encephalopathy from the burning of discarded battery casings in the home. We wish to call attention again to the general hazard presented by recycling exhausted batteries or using waste products from this process in the home.

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CASE REPORTS

Family 1

There were 22 family members (Table 1) spanning four generations living in a three-bedroom house in rural eastern North Carolina. The 68-year-old great-grandfather of the index case was employed in a small-scale operation that recovered lead from exhausted automobile storage batteries, then discarded the casings. This was one of ten such operations in the county. During a three-month period, two truckloads of discarded battery castings from the operation were burned as fuel in the family's wood-burning stove. The index case was a previously normal 3-year-old girl with a one-month history of weight loss, lethargy, and vomiting. On four occasions she was evaluated for these symptoms, and each time otitis media was diagnosed. She became comatose, had a generalized seizure, and was admitted to New Hanover Memorial Hospital in Wilmington, NC, where she required intubation for mechanical ventilation. Lead encephalopathy was suspected on the basis of anemia, microcytosis, basophilic stippling of the red blood cells, and exposure history. Cerebrospinal fluid (CSF) contained 24 lymphocytes/cu mm with normal concentrations of glucose and protein. Serum glucose, calcium, and ammonia levels were normal. Capillary blood was analyzed for lead by anodic stripping voltometry,[6] and for erythrocyte protoporphyrin by hematofluorometer.[7] The level of lead in the blood was 220 μg/100 ml and the patient underwent chelation with dimercaprol (BAL) (500 mg/sq m/day) and edetate disodium calcium (1,500 mg/sq m/day).[8] She was treated for seizures with phenytoin, phenobarbital, paraldehyde, and dexamethasone. Over the next 13 days she became

TABLE 1. Profile of Lead-Exposed Members of Family 1*

Member	Sex	Age	PbB (μg/100 ml)	EP (μg/100 ml)	Hemoglobin (gm/100 ml)	Clinical and Laboratory Findings	Therapy
1	F	15 mo	84	313	9.6	BS	CaEDTA
2	F	15 mo	78	404	10.4	BS, LL	CaEDTA
3†	F	3 yr	220	...	6.0	EN, BS, NV	BAL, CaEDTA
4	M	3 yr	256	400	9.9	BS	BAL, CaEDTA
5	M	5 yr	150	202	10.6	NV	CaEDTA
6	M	5 yr	165	195	10.6	BS	CaEDTA
7	F	5 yr	59	227	12.5	BS	CaEDTA
8	M	7 yr	56	230	11.3	None	CaEDTA
9	F	9 yr	46	225	13.8	HA, NV, BS	CaEDTA
10	M	9 yr	50	167	12.0	None	None
11	F	12 yr	45	117	12.0	None	None
12	F	12 yr	36	40	12.0	None	None
13	F	14 yr	39	62	14.0	None	None
14	M	16 yr	40	29	15.0	None	None
15	M	16 yr	27	66	12.0	None	None
16	M	18 yr	...	25	16.0	None	None
17	M	18 yr	...	30	15.0	None	None
18	F	20 yr	52	20	...	None	None
19	M	20 yr	...	22	15.0	None	None
20	F	21 yr	...	33	10.0	None	None
21	F	66 yr	46	176	12.5	None	P
22	M	68 yr	79	481	9.0	BS	P

* Abbreviations used are: PbB, level of lead in blood; EP, erythrocyte protoporphyrin; EN, encephalopathy; BS, basophilic stippling of red blood cells; LL, metaphyseal lead lines demonstrated by roentgenogram; HA, headache; NV, nausea and vomiting; CaEDTA, edetate disodium calcium; BAL, dimercaprol; P, penicillamine.
† Index case.

more responsive to visual and auditory stimulation and was able to sit alone in a chair and feed herself, but had severe nystagmus, could no longer speak intelligible words, and was no longer bowel or bladder trained. Three months following the illness she continues to have seizures and is profoundly retarded.

Dust samples were obtained by a vacuum collection technique[9] and analyzed by flameless atomic absorption spectroscopy using a modification of a procedure recommended by the Environmental Protection Agency.[10] Dust from a sofa adjacent to the wood-burning stove contained 13,283 ppm of lead. Dust from the kitchen floor contained 41,283 ppm of lead, which was equal to 13,555 μg of lead per square meter of floor surface. Most walls in the home were unpainted. Decontamination of the house with Calgon (use of tradenames is for identification only and does not imply endorsement by the Public Health Service or the Department of Health and Human Services) facilitated mobilization of lead, using the technique described by Milar and Mushak,[9] resulted in a reduction in the absolute quantity of lead in the house dust (Table 2). Subsequently, the contaminated wood stove has been replaced and ashes from the burned battery casings were removed from the premises.

TABLE 2. Effect of Calgon Decontamination on Environmental Dust-Lead Levels (Family 1)

Location	Predecontamination Pb		Postdecontamination Pb		% Lead Removal
	ppm*	μg/sq m†	ppm	μg/sq m	
Bedroom floor	6,808	3,323	7,753	1,528	57.8
Living room floor	5,862	4,125	5,579	1,961	52.5

* Micrograms of lead per gram of housedust (normal value < 1,000 ppm).[3]
† Micrograms of lead per square meter of floor surface (normal value < 50 μg/sq m).[9]

Family 2

An elevated blood lead level was unexpectedly found in a truck driver working in a low-exposure area of a battery recycling operation in rural western North Carolina. He was discovered to be operating an illicit battery recycling operation inside his home and was melting down the reclaimed lead on the kitchen stove. Capillary blood lead and erythrocyte protoporphyrin results are shown in Table 3. No family member was symptomatic, but there were unexplained deaths among chickens who fed where the lead waste products (dross) were discarded. X-ray fluorescence measurements[11] did not demonstrate lead on the painted surfaces of the interior walls of the house, but demonstrated some lead in the exterior surfaces of the home. The driveway was "paved" with fragments of the discarded battery casings, and soil samples taken from this area contained 12% to 13% lead by weight. Soil samples taken from the area where the dross was dis-

TABLE 3. Profile of Lead-Exposed Members of Family 2*

Member	Sex	Age	PbB (μg/100 ml)	EP (μg/100 ml)	Hematocrit	Therapy
1†	M	48 yr	72	349	45	None
2	F	41 yr	46	94	51	None
3	F	24 yr	32	23	50	None
4	F	18 yr	24	24	44	None
5	F	17 yr	43	76	41	None
6	F	7 yr	64	89	40	None
7	M	16 mo	63	252	38	CaEDTA

* Abbreviations are defined in footnote to Table 1.
† Index case.

carded measured as high as 49.2% lead by weight. The family has subsequently left the home.

DISCUSSION

Recycling lead-acid automobile storage batteries was responsible for two separate instances in which entire families received excessive lead exposure; one child developed encephalopathy. In both instances, the hazard posed by these lead recycling products was either unrecognized or denied. Both families were financially motivated to bring civilization's waste products into the home. Family 1 burned battery casings to offset the high cost of fuel to heat the home, and family 2 reclaimed lead for income supplementation. House dust obtained from family 1 was heavily contaminated and contained 66- to 82-fold more lead than noncontaminated homes previously sampled in rural North Carolina.[9] Using battery casings as fuel for home heating occurs more frequently during periods of economic stress. This was a common practice during the Great Depression of the 1930s,[4,5] as battery casings were free and burned with a hot flame. These discarded casings may be given to the poor as a well-intentioned, charitable, but misguided act.[5,12] With spiraling costs or the unavailability of conventional fuels, this practice may once again become a serious problem.

Lead reclamation operations are ideally suited for rural areas. They may be of small or large scale and be widely scattered; some may employ only a few workers. The exact number of such operations is unknown, but estimates range from 50 (C. M. Maxwell, personal communication, 1980) to several hundred. The larger of these secondary lead smelters operate in urban areas. Safety regulations may be difficult to enforce, as the existence of these operations may not always be known to local health authorities. Furthermore, attention is usually focused on the metallic lead because of its commercial value, but little attention is paid to the discarded battery casings. The residual lead coating of the casings presents a biologic hazard which usually goes unrecognized by workers.

Once accidental contamination has occurred, the quantity of lead in the home environment can be reduced but not always eliminated by the use of the decontamination techniques developed in our laboratory.[9] Following a single attempt, greater than half of the lead-containing dust was removed from the floor surfaces. The efficiency of decontamination procedures is more appropriately measured by the absolute quantity of lead remaining in the environment, rather than by the traditional parts per million measurement. The latter is highly dependent on the level of cleanliness of the home and does not accurately reflect the lead content per unit area.[9] Substantial quantities of lead may remain in the environment following a single decontamination attempt. Multiple attempts then may become necessary to reduce the lead to nonhazardous levels. Moreover, soil contamination from fallout in the proximity of the dwelling poses a hazard to young children at play. Decontamination of the external environment may prove to be a more difficult problem.

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APPENDIX

Sources of Lead for Lead Workers' Children—A Directory of Scrap Smelters*

Children of workers in hazardous industries are at heightened risk of exposure to occupational toxins.[1] Asbestos,[2] beryllium,[3] estrogenic steroids,[4] polychlorinated byphenyls (PCBs),[5] and tetrachlorodibenzodioxin (TCDD)[6] are among the chemical compounds that have been shown to cause disease in the children of industrial workers. Parental exposure, which leads to childhood disease or dysfunction, can occur prior to conception (mediated through occupational injury to the germ cells of either parent[7]), during pregnancy, or at any stage in childhood. Exposures in childhood may result from ingestion of contaminated breast milk,[8] possibly from inhalation of toxic waste gases (eg, anesthetics, solvent vapors) in parents' exhaled breath, or most commonly from ingestion of contaminated dust carried home from the workplace on parents' skin, hair, shoes, clothing, or automobiles.[1-6,9] The exposure of children to industrial chemicals will tend to be increased further when the patient's hazardous workplace is actually in the home.[10]

Lead, perhaps because of its ubiquity, has figured most prominently in reports of chemical intoxications of workers' children.[11-14] Dolcourt et al have described the fifth and sixth recorded episodes in the United States of increased lead absorption in lead workers' children. In both cases described by Dolcourt et al, children absorbed increased amounts of lead and one child developed encephalopathy as the result of exposure to small, uncontrolled lead scrap smelting operations in their homes; the exposure of the child with encephalopathy was compounded by her inhalation of fumes released by the burning of discarded lead battery casings.

Episodes such as these are not isolated—only underrecognized. The National Institute for Occupational Safety and Health (NIOSH) estimates that approximately 3,000 workers are employed in primary lead (ore) smelters and another 17,000 in secondary nonferrous metals (scrap) smelters in the United States. The risk of lead exposure in these workers' children has been reduced through implementation of federal requirements for showering and changing facilities in lead plants.[15] However, exposure will continue to occur, particularly for the children of workers in the smallest, dirtiest, and most poorly controlled lead smelters.

The evaluation of any possibly toxic illness in childhood requires the ascertainment of parental occupational exposures. To assist pediatricians in evaluating possible cases of occupationally induced lead poisoning in children, the US Environmental Protection Agency and NIOSH have compiled a list of secondary lead (scrap)

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TABLE. Secondary Lead Smelters in the United States

State	City	Company
Alabama	Leeds	Interstate Lead Co, Inc
	Troy	Sanders Lead Company
Arizona	Phoenix	Alimco Smelting and Refining
California	City of Industry	Southwest Metals (Bergsoe Metals)
	City of Industry	RSR Corp (Quemetco)
	Gardena	ALCO Mining Inc
	Los Angeles	Gould, Inc
	Los Angeles	United American Metals of California
	San Francisco	ASARCO (Federated Metals)
	San Jose	Levin Metals Corp
Colorado	Commerce City	National Smelting and Refining
Florida	Jacksonville	ESB (Refined Metals) (MS&R)
	Tampa	Globe Union, Inc
	Tampa	Gulf Coast Lead Company
	Tampa	Chloride Metals
Georgia	Atlanta	Seitzinger/Evans Metals
	Atlanta	NL Industries
	Atlanta	TARACORP
	Carrolton	Southwire Corp
	Cedartown	Sanders Lead Company
	Columbus	Chloride Metals
Illinois	Chicago	Inland Metals
	Chicago	Chicago Smelting & Refining
	Chicago	Gardiner Metal Co
	Chicago	Imperial Smelting Corp
	Chicago	Suppo Smelting & Refining
	Granite City	TARACORP (NL Industries)
	Lansing	Saxon Metal Corp
	McCook, E Chicago	TARACORP (NL Industries)
	North Cook	North Chicago Smelting & Refining
	Savanna	Interstate Lead (ILCO)

TABLE—*Continued*

State	City	Company	State	City	Company
Indiana	Beech Grove	ESB (Refined Metals)	North Carolina	Charlotte	Willard Lead Products
	E Chicago	USS Lead Refining, Inc		High Point	Carolina Metals Alloy
	E Chicago	Northern Indiana Dock Co	Ohio	Canton	Canton Metals Alloys Co
	Indianapolis	RSR Corporation (Qumetro)		Cleveland	Master Metals, Inc
	Muncie	Mincon		Cleveland	River Smelting and Refining
	Whiting	ASARCO (Federated Metals)		Oakwood Village	I. Schumann Co
Kansas	Olathe	Delco Remy Battery		Wapakoneta	Ohio New and Rebuilt Parts, Inc
Kentucky	Louisville	Globe-Union Inc			
Louisiana	Baton Rouge	Schuykill Metals	Oregon	St Helens	Bergsoe Metals
	Heflin	General Battery Corp (Dixie Metals)	Pennsylvania	Altoona	A. J. Oster
				Lancaster	Lancaster Battery Co
	Shreveport	Gould Metals		Lyons Station	East Penn Manufacturing
Maryland	Baltimore	Industrial Metal Smelting		Nesquehoning	Tonolli Corp
	Baltimore	Ansam Metals		Philadelphia	Metallurgical Resources
Massachusetts	Boston	General Metals and Smelting		Philadelphia	Imperial Metal and Chemical
	Cambridge	Cambridge Smelting		Philadelphia	Acme Alloys
	Chelsea	Sam Gordon and Sons		Pittsburgh	Pittsburgh Smelting and Refining
	Somerville	Somerville Smelting			
	W Springfield	New England Smelting Works		Reading	General Battery Corp
				Throop	Marjol Battery Corp
Michigan	Detroit	Federal Alloys Corp		Temple	Prestolite (ELTRA)
	Detroit	Industrial Smelting Company	South Carolina	Gaston	Nassau Recycle
	Detroit	City Metals Refining Co, Inc		Sumter	ESB (Refined Metals)
Minnesota	St Louis Park	TARACORP (NL Industries)	Tennessee	College Grove	General Smelting and Refining
	St Paul	Gould Battery			
	St Paul	Gopher		Memphis	ESB (Refined Metals)
Mississippi	Florence	Chloride Metals		Memphis	Memphis Smelting and Refining (MS&R)
Missouri	Forest City	Schuykill Metals			
Nebraksa	Omaha	Gould, Inc		Rossville	Ross Metals, Inc
New Jersey	Jersey City	Jersey Smelting and Refining	Texas	Dallas	General Battery Corp (Dixie Metals)
	Jersey City	Alpha Metals			
	New Brunswick	Delco Remy Battery		Dallas	RSR Corp
	Newark	Federated Metals (ASARCO)		Dallas	NL Industries
	Newark	Essex Metal Alloys		Frisco	Gould, Inc
	Newark	Aetna Alloys Division		Houston	Federated Metals
	Pedricktown	NL Industries		Houston	Houston Lead Co
	Perth Amboy	E. L. Beth		San Antonio	Standard Industries
New Mexico	Socorro	CAL-WEST	Virginia	Richmond	Hyman Viener and Sons
New York	Brooklyn	Standard White Metals Corp	Washington	Seattle	RSR Corp (Quemetco)
	Brooklyn	Ney Metal Recycling, Inc	Wisconsin	Milwaukee	Crown Metal
	Brooklyn	White Metal Rolling and Stamp Co			
	Brooklyn	Belmont Smelting and Refining			
	Brooklyn	Republic Metals			
	Dewitt	Roth Bros Smelting Corp			
	Middletown	RSR Corp			
	Tottenville	Nassau Smelting and Refining			
	Wallkill	RSR			

smelters and related facilities in the United States (Table). Pediatricians should realize in reviewing the list that the smallest, potentially most hazardous smelters are also the most transitory and thus may not be included. Conversely, some smelters on the list may currently be inactive. In any event, however, pediatricians may readily obtain current information on potentially hazardous exposures in any industrial plant in the United States from NIOSH's Division of Surveillance, Hazard Evaluations and Field Studies by phoning (513) 684-2427.

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WILLIAM DOUGLASS ON THE FIRST REPORTED CASES OF SCARLET FEVER IN NEW ENGLAND, 1736

In 1736, William Douglass (1691–1752) of Boston, wrote an excellent account of an epidemic of a "new" disease which he called *angina ulcusculosa*, yet his description of this "new epidemical eruptive fever" was the first adequate description of scarlet fever in English.[1] Douglass's account was published twelve years before John Fothergill (1712–1780),[2] the English physician, published his classic account of both diphtheria and scarlet fever, although Fothergill failed to differentiate between the two conditions.

Douglass wrote:

> The first attack is somewhat of a *chill* or shivering; soon after follows *Head ake* or some other versatile *spasmodick pains*, as pain in the back, joints, side, etc; a vomiting or nausea, or in some constitutions, which are not easily provoked to vomit, only a certain uneasiness or sickness at Stomach; at the same time the *Uvula*, but chiefly the Tonsils, were tumified, inflamed and painful, with some white specks, then follows a flush in the Face and some *miliary eruptions* therewith a benign *mild fever*, the same efflorescence soon after appears on the neck, chest and extremities; the 3rd or 4th Day, Eruption is at the hight and well defined with fair intervals; the flushing goes off gradually with a general *itching*; and in a Day or two more the *cuticle* scales or peels off, especially in the extremities: At the same time the cream coloured sloughs or specks in the *Fauces* become loose and [are] cast off . . . The tongue from the beginning is furr'd as in a *Mercurial Ptyalism,* urine high coloured. . . in the whole course of the Distemper [there is] a very *great prostration* of strength and faintness. . .[and a] loss of *en bon point*. As in the Measles there is a peculiar smell, so in our Distemper the *effluvia* from the Patient have a proper smell; in children as if troubled with *Worms*, in grown Persons the rancid smell of foul Bed Linnen.
>
> This is a Real History of the distemper as *it appeared* in *Boston New-England*, taken clinically from the life and not copies. There is no stroak or cause but which I can vouch by real not imaginary cases. It is founded only upon observations,. . . that is upon the Symptoms that appeared in the course of this Epidemical disease, it must therefore be of permanent truth.

Noted by T.E.C., Jr, MD

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