Deleading Dilemma: Pitfall in the Management of Childhood Lead Poisoning

Susana Rey-Alvarez, MD, MPH, and Theresa Menke-Hargrave, MD, MPH

From the Department of Medicine, The Children's Hospital of Boston, Harvard Medical School, Boston

ABSTRACT. Deleading is a dangerous process which, if improperly done, can result in acute lead intoxication. The following case report illustrates what happened to an already-lead-poisoned child's lead level when he was not excluded from his apartment during deleading. Supportive evidence is provided from 12 additional cases recently seen by the same pediatric practice. The issues behind the failure of these families to vacate their apartments during deleading are discussed, and the need for lead-poisoning prevention programs to address these issues, particularly that of providing alternative shelter during deleading, is stressed. Society should adequately fund such programs so that they become a reality. *Pediatrics* 1987;79:214–217; *lead poisoning, deleading.*

Lead poisoning has long been known to cause permanent brain damage.[1,2] More recently, it has been shown that even subclinical lead burdens are associated with neuropsychologic dysfunction.[3–5]

The issue of lead poisoning is indeed of nationwide importance. Recent data from the National Health and Nutrition Examination Survey provided information that approximately 675,000 (4%) children, 6 months through 5 years of age, have blood lead levels that exceed the accepted limit of 30 μg/dL.[6] Although there are many possible sources of lead that can result in poisoning, the principal source jeopardizing children is old housing paint. It has been estimated that between 40% and 50% of currently occupied houses may contain lead-based paint on exposed surfaces.[7] All of the cases reported in this paper were collected from a low-income neighborhood, but the problem of lead poisoning is not confined to poor housing as it has occurred as well in expensive homes that were renovated by their owners or by careless or inexperienced deleading crews.[8] Lead poisoning cannot be remedied without permanently removing sources of lead from the child's environment.

In 1971, both the US Congress and the Massachusetts State Legislature passed statutes addressing the need for the prevention of lead intoxication in childhood. The federal Lead-Based Paint Poisoning Prevention Act (PL 91-695) earmarked funds for screening programs and banned the use of lead in items accessible to children. The Massachusetts Childhood Lead Paint Poisoning Prevention and Control Act[9] set up an agency responsible both for screening children and for detecting sources of lead poisoning. It made the presence of lead paint in housing units with children younger than 6 years of age a violation of the sanitary code and established procedures to be followed with landlords to ensure deleading and to protect tenants from reprisal. Guidelines were developed for inspecting during and after deleading, to protect the safety and adequacy of the process. The agency was held responsible for executing these programs "to the extent permitted by appropriations."[9]

It was not until 1977, that the US Consumer Product Safety Commission banned the sale in interstate commerce of paints intended for interior and exterior residential services.[10–12]

Over the years, since these laws were passed, progress has been made in the reduction of mortality and gross morbidity from lead poisoning in childhood.[6,7] Many children have been screened for increased lead burden. However, only some of the children with positive "screens" have been treated. The following case report illustrates many of the pitfalls that beset the practitioner, the family, the landlord, and the responsible city agency on the management of the subclinical lead paint poisoning.

Received for publication Nov 18, 1985; accepted May 13, 1986.
Reprint requests to (S.R.A.) Children's Hospital of Boston, 300 Longwood Ave, Boston, MA 02115.

CASE REPORT

Anthony C. (born June 24, 1983) was a full-term black boy who weighed 3.3 kg (7 lb 5 oz) at birth. His 21-year-old mother (gravida 1, para 2, abortus 0) smoked cigarettes during her pregnancy. The baby's delivery was uneventful except for a nucal cord xl. Apgar scores were 7 at one minute and 8 at five minutes. He was enrolled at birth for primary pediatric care at his family's neighborhood health center and his mother was reliable in keeping medical appointments. Anthony C. lived with his mother and 4-year-old sister, Leslie C. in a small apartment in Boston. His maternal grandmother helped in the care of the children, keeping Leslie C. in her residence for prolonged periods of time.

During consecutive visits at 8 months and 12 months of age, Anthony C. was found to have elevated blood lead levels and free erythrocyte protophorphyrin levels, which increased from class II to class III according to the Centers for Disease Control system of classification.[13] At the 12-month health maintenance visit, Anthony C. was found to be in the 50th percentile for height, weight, and head circumference and was developmentally "on target" for age. He walked alone, threw and banged objects, had a neat pincer grasp, cooperated in dressing, and could say a meaningful word other than "mommy" and "daddy". A referral was filed with the Office of Environmental Affairs of Boston's Department of Health and Hospitals, the agency responsible for lead-poisoning prevention and treatment within Boston city limits. Home inspection detected significant lead hazard in the apartment.

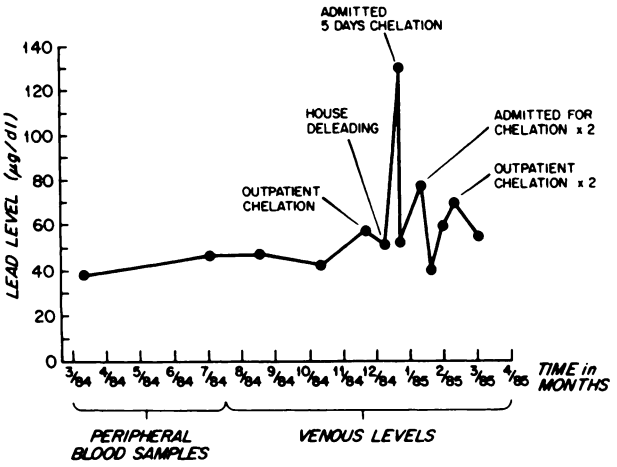

Figure. Relationship of effect of deleading to increments of blood lead values.

Because of persistently elevated venous lead levels in the moderate range, outpatient chelation was performed at Boston Children's Hospital in November 1984. House deleading began on Dec 13, 1984, and was completed in four days. The Office of Envirnomental Affairs inspector instructed Anthony's mother about the deleading procedure, including the fact that she had to find alternate housing during deleading. Having no alternate place to go, however, Mrs C. returned to her residence with Anthony every evening after the deleaders had left for the day. On Dec 19, two days after deleading was terminated, Anthony's venous lead level was found to be 130 μg/dL (Figure). He was hospitalized that same day at Boston's Children's Hospital for further chelation. He was subsequently readmitted on two occasions in January 1985. He underwent two additional outpatient chelation procedures in February of that year at his neighborhood health center. His levels, as of this writing, are still elevated in the 40 to 50 μg/dL range (class III).

Twelve other children from four different families attending the same neighborhood health center have had similar experiences to that of Anthony C. Their lead levels increased significantly following the deleading of their apartments (Table). These 12 children have also had numerous inpatient and outpatient chelation proce-

TABLE. Lead Levels and/or Free Erythrocyte Protoporphyrin (FEP) Determinations*

Name	Before Lead Abatement				After Lead Abatement			
	Age (yr)	Lead (μg/dL)	FEP (μg/dL)		Lead (μg/dL)	FEP (μg/dL)		Wk After Deleading
			Protoporphyrin/ Erythrocytes	Protoporphyrin/ Whole Blood		Protoporphyrin/ Erythrocytes	Protoporphyrin/ Whole Blood	
Family 1								
S. M.	5	40	33		56	194	462	8
D. S.	4	31	51		55	245		4
J. S.	2	30	86		54	350	1050	4
J. M.	1	49	22		72	359	1011	4
M. M.	2/12				22	20	71	8
Family 2								
J. O. M.	3		18		58	330	846	3–4
J. U. M.	5		17		39	100	263	3–4
J. A. M.	2		27		41	180	474	3–4
Family 3								
R. G.	1		25		45	43	132	3–4
M. G.	2		36		43	108	273	3–4
Family 4								
O. C.	4		32		50	60		3
G. C.	1		39		47	188		3

* S. M. from family 1 had the following values 4 weeks after deleading: lead, 34 μg/dL; 293 μg protoporphyrin per dL erythrocytes. M. M. from family 1 was exposed to lead in utero. Cord lead level was 12 μg/dL.

dures. They, however, have suffered various degrees of developmental delay secondary to lead poisoning. These families all come from low-income, minority groups. In addition to the lead poisoning in their children, they had rent increases and were threatened with eviction after lead hazards were discovered in their apartments.

DISCUSSION

Lead poisoning in childhood is a disease that can be prevented. Children who do not live in housing units with lead paint rarely become afflicted. In 1971, the Massachusetts Lead Poisoning Prevention and Control Act called for the establishment of a program to locate dwellings that contained dangerous amounts of lead, especially in areas where a significant number of cases of lead poisoning had been reported. The area in which these five families lived was just such an area.[14] The program was, however, made accountable only "to the extent permitted by appropriations." Since its creation in 1971, the funding and staffing of the Office of Environmental Affairs has been steadily cut.

Successive reductions in the Comprehensive Employment and Training Act Program in 1978 to 1979 and further cuts during "Block Grant" changes in 1982 have brought a staff of 100 down to its present level of 24, with 12 workers assigned to inspect homes before and during deleading and to counsel families during the process. Funding for the house-screening portion of the Massachusetts law has been nonexistent. Hence, many families with their initially normal children have moved into apartments that they did not know were dangerous to the health, intelligence, and, ultimately, employability of their children.

The fate of these 13 children illustrates what happens all too often when society opts to screen the child, not the dwelling, for lead burden. Furthermore, it demonstrates the devastating and expensive consequences when inadequate provisions exist for housing families during the deleading process. The following common problems emerged to explain why events happened as they did. (1) Parents failed to believe the dangers involved in the deleading process, despite attempts at providing adequate warning. (2) Shelter and/or funding for shelter for families during deleading was not available. (3) Existing lead legislation has serious limitations. The following two problems, although not pertinent to Anthony C. and his family, did characterize the other four families in our experience. (4) Parents failed to disclose information for fear of landlord reprisal. (5) Safety regulations are difficult to enforce when deleading is done by untrained personnel, eg, parents or landlords.

Problem 1

None of the families heeded the warnings given to them by the Office of Environmental Affairs, hospital, and health center personnel about the necessity of completely vacating an apartment during deleading. All families spent time in their dwellings while they were being deleaded. Acute increases in venous lead and free erythrocyte protophorphyrin levels, like that which occurred in December 1984 in the case of Anthony C., were observed in children from each of the families immediately following the deleading of their residences. These increases were almost certainly attributable to exposure during deleading.

Problem 2

Even if parents believe the warnings given to them about the dangers of inhabiting an apartment that is being deleaded, compliance is made extremely difficult by the unavailability both of funding for alternative shelter and of lead-free shelter facilities themselves. Neither existing lead grant monies nor Medicaid authorize payment for shelter during deleading. What free shelter exists in Boston is inadequate to meet the needs of the homeless and is unable to accommodate large families. It is not uncommon practice to keep children extra days in the hospital following completion of chelation until deleading is finished.

Problem 3

Four months after deleading, Anthony C. continues to have an unacceptable lead burden, with venous levels of 40 to 50 μg/dL. The dust in the family's carpets and upholstery constitutes one source of chronic exposure, but there are other possibilities. Existing lead legislation only mandates deleading of those areas considered accessible to a child younger than 6 years. Lead paint in areas defined as inaccessible by law, eg, more than 4 ft from the floor, can later deteriorate and a child can be at further risk. Paint peeling in hallways outside apartments, on handrails, or on house exteriors can further complicate the picture.

Problem 4

Fear of eviction and harassment by landlords is neither uncommon nor unfounded.[14] Even though the law protects tenants against such action, tenants are often unaware of the law or, if aware, they know that historically such laws have been difficult to enforce. When a landlord wrongly evicts or increases the rent or violates the housing code, it is up to the tenant, who usually has no extra money for legal fees, to initiate civil legal procedures against the landlord.

Problem 5

The process of deleading, particularly when improperly done, can create a highly accessible source of lead. Small particles of the element can be released into dust which can be inhaled or ingested if people are not excluded from the working area. If furniture and carpeting are not adequately covered, such microparticles penetrate the upholstery and the carpet pile in such a way that they become an ongoing source of lead.[15] Deleading improperly done by a child's parent and a landlord's use of the highly dangerous technique of burning off paint, as it has been reported elsewhere,[8,16] were the causes for acute lead poisoning in two of our families. Inskip and Atterbury[17] reported that, during sanding or burning of old paint, lead deposits in the environment at a rate of 5 to more than 100 $mg/m^2/h$. When this information is compared with the balanced studies of Ziegler and Edwards[18] which show that retention starts when lead intake in children is greater than 5 mg/kg of body weight per day, the enormous hazard created by burning and sanding becomes apparent.[18]

As the damage from low-level lead poisoning becomes more obvious, the mandate for prevention becomes clearer. Effective lead programs must address the needs of families during the deleading project, and funding should be provided to make the program happen. In the 1980s, the management of lead poisoning will need to look beyond the use of ethylenediaminetetraacetic acid and *d*-penicillamine to preserve, create, and fund the structures necessary to safely remove lead from a child's environment.

ACKNOWLEDGMENTS

We thank Anne Wilson and Ronald Jones from the Office of Environmental Affairs for their cooperation, Dr Marta Killner for sharing one of these families with us, and Dr Bernard Guyer, Dr Judith Palfrey, and Dr John Graef for their advise and encouragement.

REFERENCES

1. Rutter M: Raised lead levels and impaired cognitive/behavioral functioning: A review of the evidence. *Dev Med Child Neurol* 1980;22(suppl):1
2. Lin-Fu JS: What price shall we pay for lead-poisoning in children? Children Today. US Department of Health, Education, and Welfare publication No. (HSA) 79-5144. Government Printing Office, 1979
3. Needleman H, Gunnoe C, Leviton A, et al: Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N Engl J Med* 1979;300:689
4. Yule W, Lansdown R, Millar IB, et al: The relationship between blood lead concentrations, intelligence and attainment in a school population: A pilot study. *Dev Med Child Neurol* 1981;23:567
5. Bellinger DC, Needleman HL: Lead and the relationship between maternal and child intelligence. *J Pediatr* 1983;102:523
6. Mahaffey KR, Annest JL, Roberts J, et al: National estimates of blood lead levels: United States, 1976–1980. *N Engl J Med* 1982;307:573
7. Lin-Fu JS: Children and lead. *N Engl J Med* 1982;307:615
8. Fishbein A, Anderson K, Sassa S, et al: Lead poisoning from "do-it-yourself" heat guns for removing lead-based paint: Report of two cases. *Environ Res* 1981;24:425–431
9. Massachusetts General Code, *Lead Poisoning Prevention and Control Act*, chapter III, §190–199, 1971. Amended 1973, 1974, 1975, 1978, 1979
10. US Consumer Project Safety Commission: Regulation of products subject to other acts under the Consumer Products Safety Act, 16 CFR §1145
11. Lead-containing paint and certain consumer products bearing lead-containing paint, 16 CFR §1303
12. Hazardous Substance and Articles, administration and enforcement regulations, 16 CFR §1500
13. Preventing lead poisoning in young children: A statement by the Centers for Disease Control January 1985. Atlanta, US Department of Health, Education, and Welfare, 1985
14. Community Health Improvement Program of the Harvard School of Public Health: Casas sin Plomo Final Report (grant 72 090 7098 2 30) Boston, ACTION Office of Policy and Planning, 1982, pp 1–19, appendices
15. Milar CR, Mushak P: Lead contaminated housedust: Hazard, measurement and decontamination, in Chisolm JJ Jr, O'Hara DM (eds): *Lead Absorption in Children: Management, Clinical and Environmental Aspects*. Baltimore, Urban and Schwarzenberg, 1982, pp 143–152
16. Feldman RG: Urban lead mining: Lead intoxication among deleaders. *N Engl J Med* 1978;298:1143–1145
17. Inskip M, Atterbury N: The legacy of lead-based paint: Potential hazards to do-it yourself enthusiasts and children, in *Heavy Metals in the Environment*. Edinburgh, CEP Consultants, Ltd, 1983, vol 1, pp 286–289
18. Ziegler EE, Edwards BB, Jensen RL, et al: Absorption and retention of lead in infants. *Pediatr Res* 1978;12:29