Residential Deleading: Effects on the Blood Lead Levels of Lead-Poisoned Children

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ABSTRACT. Acute elevations of venous blood lead levels (PbB) are periodically reported in children with chronic lead poisoning, during deleading of their houses. To evaluate this phenomenon 114 preschool children who entered the Massachusetts Childhood Lead Poisoning Prevention Program case management system during 1984 and 1985 were retrospectively studied. PbB increased from a mean (±SE) of 1.76 ± 0.03 μmol/L (36.4 ± 0.6 μg/dL) prior to deleading to 2.03 ± 0.07 μmol/L (42.1 ± 1.5 μg/dL) during deleading ($P < .001$). Among 41 subjects for whom deleading was done by dry scraping and sanding, the mean mid-deleading PbB was higher than the pre-deleading PbB by 0.44 ± 0.12 μmol/L (9.1 ± 2.4 μg/dL). However, when deleading was done by covering or replacement of painted surfaces in the residences of 12 subjects, mid-deleading PbB decreased 0.11 ± 0.12 μmol/L (2.25 ± 2.4 μg/dL) ($P < .005$). In a subset of 59 subjects who had no chelation therapy and were available for follow-up 250 ± 14 days after completion of deleading, PbB had decreased from 1.72 ± 0.04 μmol/L (35.7 ± 0.9 μg/dL) to 1.24 ± 0.04 μmol/L (25.5 ± 0.9 μg/dL) ($P < .001$). The long-term effect of deleading is a significant reduction in PbB. However, deleading resulted in a significant, albeit transient, increase in PbB. *Pediatrics* 1991;88:893–897; *lead poisoning, prevention, control, children.*

In spite of efforts to limit the amount of lead in the environment, childhood lead poisoning remains a widespread problem. The second National Health and Nutrition Examination Survey estimated that between 1975 and 1980, 1.5 million children in the United States had blood lead levels greater than 1.17 μmol/L (24 μg/dL).[1] In a 1988 report to Congress, the Agency for Toxic Substances and Disease Registry estimated that as many as 12 million children younger than 7 years of age are exposed to potentially toxic levels of lead paint in their homes.[2] Lead paint, particularly if it is deteriorated, is the most dangerous source of lead readily available to small children. The successful management of childhood lead poisoning requires the identification and removal of the source of lead[3] and there is general agreement that vigorous abatement of lead paint hazards should continue.[4] Recently, however, concern has also been raised regarding the potential exacerbation of lead poisoning during the paint removal or "deleading" process.[5–7] Case reports indicate that family members and workers are at risk of becoming lead poisoned during deleading.[8–10]

The present study was undertaken to evaluate the extent of this problem, to identify potential risk factors associated with increased venous blood lead levels (PbB) during deleading, and to evaluate the progress of children living in abated housing.

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PROTOCOL

The Massachusetts Department of Public Health's Childhood Lead Poisoning Prevention Program provides both medical and environmental follow-up of lead-poisoned children. Most poisoned children are identified through screenings done by primary health care providers and are entered into the program's case management system. The blood lead levels of children entered into the case management system are reviewed. Because contamination of finger-stick samples by surface lead frequently occurs, all screening (capillary) tests showing elevated blood lead levels are subsequently confirmed by venous tests analyzed at the State Laboratory by Delves' Cup Method[11] for atomic absorption spectrophotometry. The laboratory participates in the Centers for Disease Control proficiency testing pool for lead and erythrocyte protoporphyrin. For the purposes of this study only venous PbB were considered.

The housing unit of each poisoned child was examined by trained state inspectors using x-ray fluorescence or sodium sulfide. At the time of this study, Massachusetts statutes required that property owners remove or permanently cover any paint with a lead content greater than 1.2 mg/cm^2 which was loose and peeling or present on chewable surfaces below 4 ft. When violations of these statutes were identified, the property owner was required to make necessary repairs. State lead inspectors regularly instructed property owners and parents on deleading procedures. The importance of removing children during the deleading process and cleanup following deleading including washing surfaces with trisodium phosphate was stressed. However, these instructions were not uniformly followed and no mechanism was in place to provide shelter to the families. Periodic inspections were done during the deleading process and the method of deleading was recorded. Upon completion of the deleading, the unit was inspected and, if no violations remained, repainted and declared safe. The dates of initiation and completion of the deleading process were recorded.

The last PbB available *prior* to the initiation of deleading was defined as the pre-deleading PbB. The PbB determined *during* the deleading process was defined as mid-deleading PbB. If more than one mid-deleading PbB was obtained the mean value of these levels was used. For those children who received any chelating agents, mid-deleading PbB obtained after initiation of the therapy were not considered. The post-deleading PbB was the first level obtained *following* the successful completion of deleading. The follow-up PbB was the *last* available PbB obtained more than 100 days after completion of deleading while the subject resided in the deleaded residence.

The PbB determination immediately prior to the initiation of deleading was selected as the baseline PbB. To assess the stability of PbB prior to environmental deleading, subjects' records were reviewed for PbB determined before the pre-deleading level. Subjects residing in the same house who did not have chelation therapy and had two venous PbB samples obtained prior to deleading were selected for this purpose.

DATA COLLECTION AND ANALYSIS

To be included in this study subjects had to meet the following criteria: be younger than 6 years of age; have entered the case management system between January 1984 and December 1985; have a confirmatory PbB >1.2 μmol/L (25 μg/dL) obtained prior to deleading; have had at least one PbB determination during deleading and one PbB determination post deleading; successful completion of the deleading process in compliance with Massachusetts statutes; continual residence in the deleaded unit before and after deleading.

To identify risk factors associated with increases in PbB, we studied the change in PbB subsequent to deleading (mid-deleading − pre-deleading PbB) in relation to methods of deleading, and the subjects' age at initiation of deleading. Paired and unpaired Student's t tests were used for comparison. Linear regression, calculated by least squares, was used for correlation. Values are expressed as mean ± SE. Values are significant at $P < .05$.

RESULTS

Of the 1065 children who met the age and case management system entry criteria, 484 were excluded because there was no record of a confirmatory venous PbB ≥1.2 μmol/L (25 μg/dL) taken prior to deleading, 267 were excluded because they moved, and 200 were excluded because the work was not complete or did not comply with the statute.

The final patient population studies was 114. The subjects' ages ranged from 11 to 72 months (median = 24 months), and 52 (46%) were boys.

Lead Levels

The mean (±SE) PbB before, during, and after deleading are illustrated in Fig 1. The mean (±SE) mid-deleading PbB was 2.03 ± 0.07 μmol/L (42.1 ± 1.5 μg/dL) compared with a pre-deleading PbB of 1.76 ± 0.03 μmol/L (36.4 ± 0.6 μg/dL) ($P < .001$). In 17 (15%) subjects mid-deleading PbB were more than 50% higher than pre-deleading levels. Five (4%) subjects sustained greater than 100% increases in PbB and in two children PbB were increased greater than 200% during deleading. The first mid-deleading PbB were determined 63 ± 4 days after the pre-deleading levels. Environmental deleading was accomplished in 98 ± 7 days.

Post-deleading PbB for all subjects, determined 49 ± 8 days after completion of deleading, was 1.62 ± 0.05 μmol/L (33.5 ± 1 μg/dL), significantly lower than the mid-deleading PbB ($P < .001$) and pre-deleading PbB ($P < .02$). Forty-two of the subjects, however, underwent chelation therapy between the determination of mid-deleading and post-deleading PbB with one or more of the following agents: dimercaprol, calcium disodium edetate, and D-penicillamine.

To determine the effect of deleading alone, a subset of 59 children who had no chelation therapy was studied. These subjects continued to reside in the deleaded unit. Follow-up PbB were determined 250 ± 14 days after the deleading work was completed. The change in PbB for this group is illustrated in Fig 2. Pre-deleading and mid-deleading PbB were comparable (1.72 ± 0.04 μmol/L [35.7 ± 0.9 μg/dL] and 1.71 ± 0.04 μmol/L [35.5 ± 0.8 μg/dL], respectively). Post-deleading PbB were significantly lower than the pre-deleading and mid-deleading PbB (1.5 ± 0.05 μmol/L [31 ± 1 μg/dL]; $P < .001$). Follow-up PbB were 1.24 ± 0.04 μmol/L (25.5 ± 0.9 μg/dL), significantly lower than post-deleading PbB ($P < .001$).

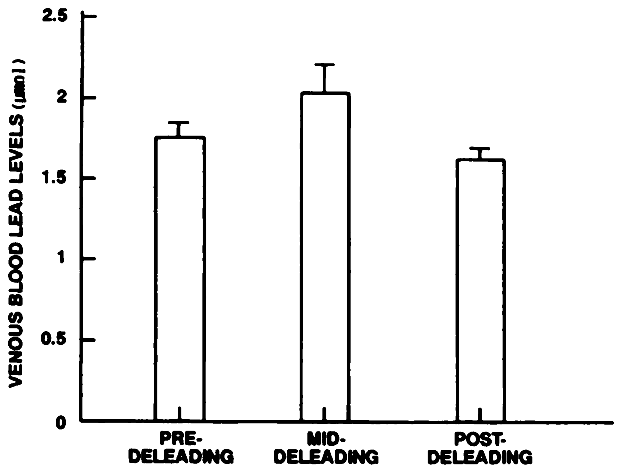

Fig 1. Venous blood lead levels (PbB) (mean ± SE) in 114 children with lead poisoning, before, during, and after deleading. Mid-deleading PbB were significantly higher than pre-deleading PbB ($P < .001$). Post-deleading PbB were significantly lower than mid-deleading PbB ($P < .001$).

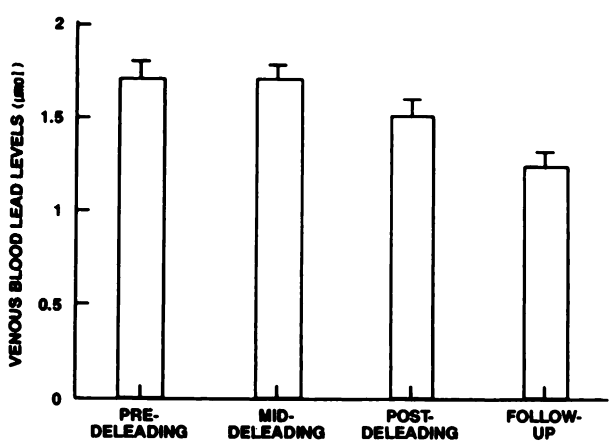

Fig 2. Venous blood lead levels (PbB) in 59 children with lead poisoning following residential deleading, who were available for long-term follow-up and had no chelation therapy. Post-deleading PbB were significantly lower than mid-deleading PbB ($P < .001$) and the follow-up PbB were significantly lower than post-deleading PbB ($P < .001$).

The stability of PbB prior to deleading was studied in 32 subjects who had had two venous samples drawn prior to deleading. The interval between these two sample was 73 ± 23 days. The pre-deleading PbB of these subjects was 1.74 ± 0.05 μmol/L (36.0 ± 1.1 μg/dL), comparable with their PbB drawn earlier (1.7 ± 0.06 μmol/L [35.4 ± 1.3 μg/dL]; $P > .5$).

Method of Deleading

The records of 80 (62%) subjects described the methods used to delead residences. Dry scraping and sanding, used in 41 cases, was associated with an increase of 0.44 ± 0.12 μmol/L (9.1 ± 2.4 μg/dL) in mid-deleading PbB compared with pre-deleading levels. By comparison, the PbB of the 12 subjects whose homes were deleaded by replacing or permanently covering painted surfaces decreased by 0.11 ± 0.12 μmol/L (2.25 ± 2.4 μg/dL) during deleading ($P < .005$). The homes of 17 children were deleaded using a combination of two or more methods. Heat guns, in combination with other methods, were used to strip paint in 5 cases. In 4 children's homes propane torches were used to soften paint prior to scraping. In all cases in which torches were used the children sustained very high increases in PbB. Mid-deleading PbB were 1.72 ± 0.52 μmol/L (35.7 ± 10.8 μg/dL), or 98% higher than pre-deleading PbB.

Age

There was no significant correlation between the subject's age and changes in mid-deleading PbB ($r = .06$, $P > .5$). However, when the children were grouped according to age, those aged 19 through 30 months (n = 52) experienced an increase in mid-deleading PbB over pre-deleading levels of 0.39 ± 0.14 μmol/L (8.04 ± 2.8 μg/dL) compared with 0.1 ± 0.11 μmol/L (2.0 ± 2.3 μg/dL) in the group younger than 19 months of age (n = 20) and 0.23 ± 0.09 μmol/L (4.81 ± 1.9 μg/dL) in 31- through 72-month-old children (n = 42). These differences are not statistically significant.

DISCUSSION

The removal of lead sources including lead in water, soil, paint, and dust from the environment of poisoned children has been the classic means of protecting them from further exposure.[12] When a child's home is shown to be painted with lead-based paint, abatement usually involves the removal or covering of the paint or the replacement of the painted surfaces with fresh stock.

The process of deleading lead-painted buildings may cause a substantial increase in ambient lead levels. Increased ambient lead has been implicated in lead poisoning in deleaders[9] and their children.[10] Feldman,[9] describing lead poisoning in two deleaders, stated "in the effort to protect children from lead paint, those removing it are falling victim."

The present study evaluates the impact of environmental deleading on lead levels of those children who are intended to benefit from the process. Our data indicate that the overall effect of deleading is a significant decrease in lead levels. However, the process of deleading may produce a significant if transient elevation in blood lead levels. Those elevations are more likely to occur when dry scraping or sanding is used and the children are not removed from the home during the process.

Increases in PbB in lead-poisoned children have been the subject of several case reports.[5-7] Two previous reports described increases in PbB in children subsequent to renovation of their older homes. Rabinowitz et al[13] reported a significant rise in blood lead levels in a cohort of children without lead poisoning whose homes were renovated. Wolf and Hicks,[14] using a case control study, reported similar findings. In both reports PbB were measured in capillary blood collected by finger-stick. Such samples may result in falsely elevated PbB due to contamination with lead-laden dust particularly at times when remodeling is in progress. The present study is a longitudinal follow-up of children with lead poisoning before, during, and after deleading, in whom only venous PbB were used.

The review of PbB prior to the beginning of deleading in 32 subjects indicates that PbB were stable over at least an average of 73 days when neither chelation therapy nor deleading was used. By contrast, PbB during deleading underwent marked changes, usually increasing considerably over pre-deleading levels. The PbB of 39 subjects increased more than 1 SD above the pre-deleading level, while 20 subjects experienced a decrease in PbB level of more than 1 SD below the pre-deleading level. In 17 (15%) of the subjects, PbB increased 50% above pre-deleading levels. In no subject was there more than a 50% decrease in mid-deleading levels compared with pre-deleading levels.

The difference in the rise in PbB during deleading for children in different age groups is inconclusive and requires further study. Children aged 18 through 30 months seem more likely to sustain a dramatic increase in PbB during deleading. This may be the result of increased hand-to-mouth activity, and thus higher exposure to ambient lead in toddlers, as suggested in a previous report.[15]

Despite the immediate hazard associated with deleading described in this study, the overall long-term effect of deleading is positive and results in a significant decrease in PbB in lead-poisoned children. The decline in PbB after deleading in the present study was greater than that found by Moel et al[16] in 74 children with lead poisoning who were followed up without systematic control of lead in their environment.

CONCLUSIONS

Because the homes of lead-poisoned children are often the primary source of lead, deleading is an essential measure for the prevention of further lead exposure. The consequence of deleading is positive and results in a decline of PbB in children with lead poisoning. However, deleading is associated with a significant, transient elevation of PbB in many children. It is most dangerous if accomplished with the use of torches, sanding, and dry scraping. In light of the significant increase in PbB during residential deleading, Massachusetts statutes were revised in 1988. Dry abrasive blasting, on-site use of methylene chloride, and the use of propane torches were banned. Other methods of lead paint removal, which may be performed only by licensed contractors, are strictly regulated. Residents' exposure to dust and fumes generated during deleading is further limited by regulations that limit access to the work site to certified deleaders until the area has been cleaned and found safe by a licensed lead paint inspector. Measures such as these, which limit the child's exposure to lead during deleading, should be strictly imposed.

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NOCTURNAL CORTISOL, THYROID STIMULATING HORMONE, AND GROWTH HORMONE SECRETORY PROFILES IN DEPRESSED ADOLESCENTS

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Abstract. Twelve depressed adolescents and 12 controls matched for age, sex, Tanner stage, time of menstrual cycle (females), weight, and time of year assessed were studied over 3 nights. Measurements for cortisol, thyroid stimulating hormone, and growth hormone were made on serum collected at 10 p.m., 12 midnight, 1 a.m., 2 a.m., 3 a.m., 4 a.m., and 6 a.m. in eight pairs and every 20 minutes from 8 p.m. to 7 a.m. in four pairs. Cortisol secretion did not significantly differentiate the groups. Thyroid stimulating hormone secretion was significantly elevated in the depressed group at one time point. Growth hormone secretion significantly differentiated the two groups at most time points, and the depressed adolescents significantly hypersecreted growth hormone (area under the curve). Implications for the diagnosis, etiology, and treatment of adolescent depression are discussed. *J Am Acad Child Adolesc Psychiatry.* 1991;30.3:407–414. Key Words: cortisol, thyroid stimulating hormone, growth hormone, adolescent, depression.