Identifying and managing adverse environmental health effects: 3. Lead exposure

Margaret D. Sanborn,* Alan Abelsohn,† Monica Campbell,‡ Erica Weir§

Abstract

Lead levels in North American children and adults have declined in the past 3 decades, but lead persists in the environment in lead paint, old plumbing and contaminated soil. There are also a number of occupations and hobbies that carry a high risk of lead exposure. There is no evidence for a threshold below which lead has no adverse health effects. Blood lead levels previously considered safe are now known to cause subtle, chronic health effects. The health effects of lead exposure include developmental neurotoxicity, reproductive dysfunction and toxicity to the kidneys, blood and endocrine systems. Most lead exposures are preventable, and diagnosing lead poisoning is relatively simple compared with diagnosing health effects of exposures to other environmental toxins. Accurate assessment of lead poisoning requires specific knowledge of the sources, high-risk groups and relevant laboratory tests. In this article we review the multiple, systemic toxic effects of lead and provide current information on groups at risk, prevention, diagnosis and clinical treatment. We illustrate how the CH2OPD2 mnemonic (Community, Home, Hobbies, Occupation, Personal habits, Diet and Drugs) and specific screening questions are useful tools for physicians to quickly obtain an environmental exposure history and identify patients at high risk of lead exposure. By applying effective primary prevention, case-finding and treatment interventions for lead exposure, both the individual patient and the larger community reap the benefits of better health.

Case

A previously healthy 2-year-old girl and her mother visit their family physician because of the daughter’s 2-month history of intermittent complaints of a mild “tummy ache.” There is no associated vomiting, weight loss, or change in appetite, bowels or diet. There are no abnormal findings on physical examination. When asked about symptom onset the mother reports that it began shortly after the family started to renovate their kitchen. They live in an old farmhouse on the outskirts of town and drink water from a drilled well on the property. The physician decides to take an environmental exposure history using the CH2OPD2 mnemonic (Community, Home, Hobbies, Occupation, Personal habits, Diet and Drugs) and specific screening questions are useful tools for physicians to quickly obtain an environmental exposure history and identify patients at high risk of lead exposure. By applying effective primary prevention, case-finding and treatment interventions for lead exposure, both the individual patient and the larger community reap the benefits of better health.

Questions surrounding this case: Is the family at risk of health effects from lead exposure? Who else might be at risk? Are other laboratory tests indicated? Where can the physician get advice on the significance of the family’s blood lead levels? How should this case of lead exposure be treated?
To some extent lead is one of the small success stories of environmental health. The association of lead poisoning with cognitive impairment is well established and has resulted in the removal of lead from gasoline, paint and food cans. Despite these preventive measures, however, silent, low-level lead exposure continues to present a problem for many communities and populations. In 1997, data from the US National Health and Nutrition Examination Surveys showed that 4.4% of children in the United States had elevated blood lead levels. A later study of the Canadian population in 1994 that 5%–10% of Canadian children living in urban areas have blood lead levels exceeding 0.48 \( \mu \text{g/dL} \), even though they are not exposed to point sources. The Ontario government estimated in 1994 that 4% of children in the province still had blood lead levels above 0.48 \( \mu \text{g/dL} \); a 1992 study found that the mean level in Ontario children had fallen from 0.91 \( \mu \text{g/dL} \) in 1972 to 0.29 \( \mu \text{g/dL} \) in 1988. A study of Vancouver children using blood lead levels collected in 1989 found that 8% had elevated levels (mean 0.29 \( \mu \text{g/dL} \)). A later study of the children living in Trail, BC, the site of a lead and zinc smelter, demonstrated that 50% had an elevated blood lead level.3

### Table 1: Environmental exposure history of case subject using the CHOPD mnemonic

<table>
<thead>
<tr>
<th>Community</th>
<th>The 2-year-old patient and her family live on a farm. Pesticides are used on the fields surrounding the house, but they were not used recently.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Home</td>
<td>The home is an 80-year-old farmhouse. The family drinks water from a drilled well beside the house. They are currently stripping multiple layers of paint from the kitchen walls using chemical and heat paint strippers. The house is heated by a wood stove.</td>
</tr>
<tr>
<td>Hobbies</td>
<td>The mother is a stained glass hobbyist but has not worked at it recently.</td>
</tr>
<tr>
<td>Occupation*</td>
<td>The mother runs a daycare centre in the home. The father drives a truck and farms.</td>
</tr>
<tr>
<td>Personal habits</td>
<td>The patient is meeting her developmental milestones. She has a 5-year-old brother, whose teacher has complained recently that he has not been paying attention in class.</td>
</tr>
<tr>
<td>Diet</td>
<td>The family follows Canada’s food guide.</td>
</tr>
<tr>
<td>Drugs</td>
<td>The patient is taking no medications or home remedies.</td>
</tr>
</tbody>
</table>

*For a child’s environmental exposure history, the occupation question refers to workplace contaminants brought into the child’s environment.

### Sources of lead and health effects

Examples of common sources of lead exposure are listed in Table 2. Sources in the community may include a nearby lead smelter or battery recycling plant. Common sources in and around the home include interior lead paint if the home was built before 1950, contaminated soil from vehicle exhaust before 1990 or from chalking of exterior paint, and lead solder from plumbing installed before 1989. Consumer products as diverse as plastic window blinds, candle wicks, costume jewelry, and children’s knapsacks have been found to contain unacceptably high lead levels. Additional common sources include hobbies using lead (e.g., stained glass and home furniture refinishing), occupational exposure of parents, and alternative medicines and home remedies (e.g., azarcon for infant colic, and the folk remedies ghasard, greta and paylooah).4,11

In adults 20%–70% of ingested lead and nearly 100% of inhaled lead enters the blood. Children aged 9 months to 3 years are more vulnerable because they absorb lead 5–10 times more effectively than adults and have greater exposure because of their exploratory behaviour and frequent hand-to-mouth activity. Other high-risk groups include pregnant women and their fetuses and occupationally exposed workers and artisans and their families.4,13–15

Lead toxicity affects the hematologic, renal and neurologic systems. Most physicians know that lead exposure is in the differential diagnosis of microcytic anemia; lead inhibits heme synthesis and increases the rate of erythrocyte destruction.4 It is less well known that chronic, low-level lead exposure also results in glomerular and tubulointerstitial changes that lead to glycosuria, proteinuria, chronic renal failure and hypertension.13,16 A longitudinal study of renal function and lead levels in middle-aged and elderly people showed that a 10-fold increase in blood lead level predicted a decline in renal function equivalent to that caused by 20 years of aging.16 The neurotoxic effects of lead are perhaps the best known and studied.1,11,12,17 Lead disrupts the main structural components of the blood–brain barrier through primary injury of astrocytes and secondary damage to endothelial microvascu-
lature.\textsuperscript{17} Electrophysiological studies have shown decreased auditory sensitivity and visuomotor performance in children exposed to lead, as well as increased latency in brain auditory-evoked potentials. In the brain, lead-induced damage occurs preferentially in the prefrontal cerebral cortex, hippocampus and cerebellum.\textsuperscript{17} Some of the characteristic clinical features of lead exposure — distractibility, attention deficits and memory problems\textsuperscript{2,12,18,19} — are consistent with injury to these anatomical areas. Recent, compelling evidence from rat studies showed a dose-dependent relation between lead exposure and decreased gene and protein expression of N-methyl-D-aspartate receptor subunits in the hippocampus, which resulted in deficits of memory, synaptic plasticity and spatial learning in a water maze.\textsuperscript{10}

The epidemiologic evidence of the link between low-level lead exposure in early life and later deficits in intellectual and school performance is strong. In 1987 Bellinger and associates\textsuperscript{12} conducted a prospective study involving 249 newborns to assess the relation between prenatal lead exposure and early cognitive development. At every age tested, the subjects with high prenatal exposure (umbilical cord blood lead level > 0.48 µmol/L) scored lower on the Bayley scale of infant mental development than did infants with lower prenatal exposure. In 1990 Needleman and colleagues\textsuperscript{18} conducted an 11-year follow-up study of 132 young adults first studied as primary schoolchildren. They discovered that those with high dentine lead levels (> 20 ppm) at 6–7 years of age were at a considerably higher risk of dropping out of school (adjusted odds ratio [OR] 7.4, 95% confidence interval [CI] 1.4–40.7) or of having a reading disability (OR 5.8, 95% CI 1.7–19.7) than subjects with low dentine lead levels (< 10 ppm) at that age. In 1996 Needleman and collaborators\textsuperscript{18} followed 7-year-old boys for 4 years and found an association between bone lead levels and delinquent behaviour. The subjects with high lead levels were more likely than those with low levels to have attention problems, aggressiveness, and antisocial and delinquent behaviour; raters of behaviour were blinded to the boys’ lead levels. Another large, longitudinal study, reported in 1997, found that 16% of boys who had high dentine lead levels at age 6–8 years were reading below the 12-year-old level at ages 16–18 (p < 0.001).\textsuperscript{19} These studies controlled for a large number of potentially confounding sociodemographic, familial and environmental factors.

In isolation, each of these studies demonstrates merely an association between lead levels and impaired mental development. However, the volume and consistency of the epidemiologic evidence and the strength of the prospective, longitudinal study designs, in conjunction with evidence supporting the biologic plausibility of the neurotoxicity of lead,\textsuperscript{17,20} provides persuasive evidence that low-level lead exposure results in persistent impairment of learning and other complex cognitive tasks.

### Screening for lead exposure

There are 2 main populations at risk of adverse health effects from lead exposure: children aged 9 months to 3 years and people living near point sources of lead pollution. Physicians should be aware of these point sources, such as those in the well-known cases of Trail, BC, and the South Riverdale area of Toronto.\textsuperscript{4,5,11} Information on such “hot spots” should be available from the local public health department.

Finding children with high lead levels is a more complex clinical problem. Because the early signs of lead poisoning in children manifest as subtle neurobehavioural changes that affect social interaction, physicians should consider screening for lead exposure children who present with growth failure, behavioural disorders, hearing loss, speech, language or attention deficits, developmental delay, micropolyhyposplenia or sleep problems.\textsuperscript{21} Adults presenting with glycosuria,\textsuperscript{13} unexplained neurological symptoms such as tremor, attention deficit disorder, unexplained arthralgias or headaches, or a history of occupational or hobby exposures may also require screening.\textsuperscript{14,21}

In Canada neither universal nor targeted screening for lead exposure has been practised,\textsuperscript{21} even though the estimated prevalence of elevated lead levels among children is comparable to that among children in the United States (4%–5%).\textsuperscript{1,5,6} In 1998 the US Centers for Disease Control and Prevention (CDC) issued new guidelines, adopted by the American Academy of Pediatrics,\textsuperscript{10,25} that endorse universal screening in areas where 27% or more of the housing was built before 1950 and in populations in which the proportion of 1- and 2-year-old children with elevated blood lead levels is 12% or higher. Applying these criteria to the Canadian situation, 1991 census data indicated that 20% of housing in Canada was built before 1946,\textsuperscript{4} and in 2000 the Canada Mortgage and Housing Corporation estimated that 15% of housing in Canada was built before 1950 (Virginia Salares, Canada Mortgage and Housing Corporation: personal communication, 2000). Nationally these levels are below the threshold for universal screening, but in some inner-city practices in areas with high levels of both poverty and old housing, the CDC crite-

### Questions to ask parents when screening for lead exposure in children

- In the last 6 months has your child lived in a house or apartment built before 1950?
- Are there recent or ongoing renovations in your home? Are there surfaces with peeling or chipped paint?
- Does your child or a sibling have a prior history of lead poisoning?
- Have you seen your child eat paint chips?

A blood lead test should be ordered if the parents answer Yes to any of these questions.
ria are met and universal screening may be appropriate. For example, 1991 census data revealed that 47% of dwellings in the former City of Toronto were built before 1946, as compared with 20% in Ontario as a whole.\(^2\)

For children living in lower-risk areas, the CDC recommends “targeted screening based on risk-assessment during specified pediatric visits.”\(^3\) A comparison study on the cost-effectiveness of lead screening strategies in the United States suggests that risk assessment followed by a blood lead test in high-risk children is the least expensive strategy.\(^4\) In clinical practice, this risk assessment can be done by asking parents 4 questions that target common, specific sources of lead exposure (see box on preceding page).

The first question reflects the fact that the age of the housing is now considered the single most important environmental marker of lead poisoning in a community.\(^5\) The 4 questions were validated in a large study (\(n = 4678\)) and were found to have a sensitivity of 75% and a specificity of 49% for detecting a blood lead level greater than 0.48 \(\mu\)mol/L.\(^6\) A smaller study using similar questions found a sensitivity and specificity of 75% and 39% for detecting the same blood lead level.\(^7\) Both studies showed better sensitivities for detecting higher levels.

Only a blood level is a valid and reliable measure of current lead exposure.\(^8\) Summer, when lead exposure tends to peak, is a good time to test children’s levels.\(^9\) The half-life of lead in blood is 30 days, so a blood level is useful if the exposure occurred within 3 months. If the exposure occurred beyond that time, a zinc protoporphyrin test (half-life 68 days) may be helpful.\(^10\) However, this test has no significant correlation with blood lead levels, and false-positive results can occur in children with iron deficiency anemia.\(^11\) Measuring lead levels in urine is not recommended for screening,\(^11,12\) and neither is measuring lead levels in hair samples, which has a reported sensitivity of only 57%.\(^13\)

The blood lead level should be less than 0.50 \(\mu\)mol/L, with adverse health effects likely occurring at greater levels. Levels between 0.30 and 0.50 \(\mu\)mol/L are above the Canadian average\(^4\) and should prompt a detailed exposure history and search for environmental sources of lead exposure. Any blood level of 0.50 \(\mu\)mol/L or higher requires repeat measurement immediately and in 3–6 months; in some cases immediate referral or treatment is also required. Table 3 summarizes the actions required by primary care physicians according to blood lead level.

### Table 3: Recommended actions to be taken by primary care physician according to blood lead level\(^11,12\)

<table>
<thead>
<tr>
<th>Blood lead level, (\mu)mol/L</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.30—&lt; 0.50</td>
<td>Level is above average in Canadian population: take an exposure history</td>
</tr>
<tr>
<td>0.50—0.75</td>
<td>Repeat blood test now and in 3–6 mo; take full exposure history; contact local public health department with the exposure history to obtain advice on home remediation; counsel parents on exposure reduction and home remediation</td>
</tr>
<tr>
<td>&gt; 0.75—1.00</td>
<td>Refer patient to a pediatrician for lead poisoning assessment; consult a community or environmental medicine specialist about exposure reduction and home remediation</td>
</tr>
<tr>
<td>&gt; 1.00—2.10</td>
<td>Refer patient immediately to a pediatrician for specialist treatment; consider chelation therapy</td>
</tr>
<tr>
<td>&gt; 2.10</td>
<td>Urgent referral and individualized case management are required; admission to hospital may be necessary</td>
</tr>
</tbody>
</table>

Prevention

There is no evidence for a threshold below which lead has no adverse effects. The precautionary principle when applied to lead exposure means that any reduction in exposure is beneficial to health. In addition to identifying people with elevated blood lead levels, physicians play an important role in educating all patients to minimize their exposure to lead. This has important economic consequences because of
the well-established negative relation between lead levels and intelligence quotient (IQ) and adult earning power. In the United States calculations of the societal costs of cognitive impairment caused by lead have shown that an economic benefit of $2.5 billion per annual birth cohort would accrue from reducing population lead levels in children by only 0.05 µmol/L. It has also been calculated that an average drop of 5 points in IQ across the population would double the number of people with an IQ below 70.

Physicians can receive advice about reducing lead exposure and home remediation from the local public health department. The Canada Mortgage and Housing Corporation booklet Lead in Your Home is also a useful handout for patients. The comparable US document is another excellent resource. The module on lead toxicity of the Agency for Toxic Substances and Disease Registry and the resource manual on the health risks from lead and their prevention from Toronto Public Health have excellent practical information and are recommended as quick office references.

Lead paint should be covered over with latex paint or removed with chemical stripper using appropriate personal protection, including gloves and a respirator. Removal by heat stripping or sanding mobilizes the lead and increases exposure. The local public health department can offer advice on the safest way to deal with old lead paints.

To reduce lead in drinking and cooking water, use only the cold water tap and run the water for 30–60 seconds or until the water is as cold as it gets. An inexpensive reverse osmosis filter (e.g., Brita) is also effective in removing lead from drinking water. Hobbyists, such as stained-glass workers, can reduce personal and family exposure to lead by wearing gloves and respirators, washing hands after working with lead and ensuring that their work area is well-ventilated, frequently vacuumed and off-limits to children in the house.

The questions answered

Is the family at risk of health effects from lead exposure? Who else might be at risk?

In the case described earlier the mother and both children are found to have blood lead levels that may be associated with adverse health effects (Table 4). The children in the home daycare are also at risk.

<table>
<thead>
<tr>
<th>Family member</th>
<th>Blood lead level, µmol/L*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>First test</td>
</tr>
<tr>
<td>2-year-old daughter (case)</td>
<td>0.98</td>
</tr>
<tr>
<td>5-year-old son</td>
<td>0.87</td>
</tr>
<tr>
<td>Mother</td>
<td>0.77</td>
</tr>
<tr>
<td>Father</td>
<td>0.31</td>
</tr>
</tbody>
</table>

Note: ND = not done.

*Health effects are likely to occur at a blood lead level of 0.50 µmol/L or higher; mean level in Canadian children is 0.29 µmol/L.

Are other laboratory tests indicated?

Further tests, including a complete blood count and renal function test, would be appropriate for the family’s 2 children. The girl is found to have a mean corpuscular volume of 80 fL.

Where can the physician get advice on the significance of the family’s blood lead levels?

The physician should contact the local public health unit about the significance of the lead levels. The health unit can also provide detailed information on cleaning up the home. A pediatrician with experience in managing lead poisoning should be consulted regarding the girl, and a community medicine specialist (e.g., the medical officer of health) or an environmental health specialist could help assess the sources and extent of the lead exposure.

How should this case of lead exposure be treated?

The parents should be advised to remove the children from the house immediately while clean-up is done. All surfaces should be scrubbed with a general all-purpose cleaner twice and then weekly and the house vacuumed thoroughly. (Trisodium phosphate [TSP] is no longer recommended for clean-up; more recent advice on lead remediation suggests that a general all-purpose cleaner is just as effective as TSP in cleaning up lead dust in the home.)

The children’s follow-up blood lead levels measured 2 years later were still above average (data not shown), which suggests continuing exposure, perhaps in the drinking water. The physician should advise the parents to have their drinking water tested for lead and to replace the plumbing or use a reverse osmosis filter if the lead level is elevated.

[Further detailed exposure history questionnaire is available on the Ontario College of Family Physicians Web site (www.cfpc.ca/ocfp/index.html) — click on “Exposure History Sheets in MS Word” in the scrolling menu located in the middle of the page). The different components (Community, Home and Hobbies, Occupation, Personal habits, Diet and Drugs) can be printed on coloured paper for easy identification in patient charts. The questionnaire may be given to a patient to complete at home and bring to the next appointment for review and interpretation.]

Competing interests: None declared.

Contributors: Dr. Sanborn conceived of and drafted the article. Drs. Campbell, Abelsohn and Weir contributed substantially to the conception and design of the article and to the acquisition of data. All authors contributed to the revising of the manuscript and approved the final version.

References


Additional resources

For physicians


For patients


Correspondence to: Dr. Margaret D. Sanborn, Chesley Medical Clinic, Box 459, 33 Second St. SE, Chesley ON N0G 1L0; msanborn@dghc.on.ca

Articles to date in this series

