not statistically significantly different and concluded: “Both fingerstick lead screening tests, CPb and FP, had excellent correlation with the confirmatory VPb [venous lead].” Table 4 of their article also shows false-positive rates for the capillary microtainer method (CPb) of 36.2% and 65% at cutoffs of 15 and 25 μg/dL, respectively. The + 7.59 μg/dL positive bias was for the capillary microtainer method, not for FPDC. The large bias is acceptable for the clinical studies because (as indicated in my previous letter) “the venous confirmations, were drawn up to 6 weeks after the screening samples.” The accuracy of ±4 μg/dL for PT results are determined on the same samples.

Further evidence of the usefulness of filter-paper-based blood lead screening in childhood lead poisoning is supported by two new additional studies published in the peer-reviewed literature [5, 6].

References

A Nonoccupational Source of Mercury Intoxication

To the Editor:
The sources of exposure to mercury and its compounds are numerous, polymorphic, and often insidious. The fortuitous observation of an increased urinary mercury excretion of 19 μmol/mol creatinine (34 μg/g creatinine) in a gendarme led us to search for the source of exposure, there being no indication of an occupational exposure to mercury.

Increased excretion of mercury was confirmed in a second sample taken by his general practitioner. A measurement of urinary Hg was then suggested to all the members of the family.

His two sons had values [0.73 and 1.13 μmol/mol creatinine (1.3 and 2 μg/g)] within the expected range for a nonoccupationally exposed population [<2.8 μmol/mol creatinine (<5 μg/g)]. Surprisingly, however, their mother showed a urinary mercury of 46 μmol/mol creatinine (82 μg/g). In early December 1995 the gendarme and his wife were referred to the clinic of industrial toxicology to elucidate the source of exposure. The urinary mercury of the wife, who was not occupationally exposed to mercury, was still high, 52 μmol/mol creatinine (93 μg/g) but her blood mercury was not (5.5 μg/L).

Neither subject had complaints or signs of mercury intoxication. A detailed investigation was then carried out.

Their diet was unremarkable. According to their statement, they did not consume any classical or homeopathic drugs or herbal teas, nor had they undergone dental treatment—although even if they had, this hardly could have explained such a high urinary excretion in the mother. No object containing mercury (e.g., thermometer, precision instrument) had been broken in the home, either recently or earlier (such breakage would have been expected also to contaminate the sons, unless the object had been broken in a room not frequented by them). There was no indication of the use of lightening cream or soap or exposure to painting or pesticides containing mercury. They did not practice spiritism.

After almost 1 h of interview, we finally found the clue. “Are you sure you don’t take any drug? The spray you may use when you have throat pain or the drops you put into your eyes or your nose are also drugs.” Finally, the woman took out of her handbag a small bottle containing drops prepared by a pharmacist that she used daily for a few years. She smoked and the smoke irritated her nasal mucosa. Every day she used a few drops of this “mixture.” The husband used the same drops but less frequently. We contacted the pharmacist, who delivered the preparation without any medical prescription, and learned that it contained 300 mg/L of borate phenyl mercury.

We prescribed 2 g of dimercapto-succinic acid [1] to be taken orally by the woman, whose subsequent Hg excretion in 24-h urines was 83.4 μmol/mol creatinine (148 μg/g). By 2.5 months later, her urinary excretion of Hg had decreased to 24.9 μmol/mol creatinine (44 μg/g); by 8 months after the withdrawal of the nose drops, the urinary concentration of mercury was 2 μmol/mol creatinine (3.8 μg/g).

Reference

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