months. Unfortunately, in the very population in which the Centers for Disease Control's recommendation of combining vaccines at 15 months might make the most sense at first—namely, patients in public health clinics, where the cost saving can be important—the needs of these parents may be the greatest for the additional visit at 18 months. Immunizations should not drive the well-child visit schedule.

Perhaps this recommendation by the Centers for Disease Control to omit the 18-month child health supervision visit will stimulate us to examine just what should be the content of this visit and, more importantly, its effectiveness and for whom. In the meantime, I do not want to see this visit discontinued.

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Pediatric Lead Poisoning in 1987: The Silent Epidemic Continues

Great gains in the understanding and control of childhood lead poisoning have been made since this disease was first diagnosed in the early years of this century.[1,2] Efforts of pediatricians, public health officials, and enlightened regulators have brought about substantial reductions in the lead content of paint, food, milk, water, air, and, most recently, gasoline.[3,4] Significant decreases in children's blood lead levels and in the incidence of lead poisoning have resulted.[4,5] Few American pediatricians trained in the past decade have ever seen a child with the coma, papilledema, and convulsions of acute lead encephalopathy.

Despite these advances, the silent epidemic of childhood lead poisoning is not yet over. Data from the Second National Health and Nutrition Examination Survey, conducted from 1976 to 1980, indicated that 9.1% of preschool children in the United States—a total of 1.5 million children—had blood lead levels of 25 μg/dL or more.[6] Among black preschool children, the prevalence of increased lead absorption was 24.5%.[6]

Furthermore, a series of epidemiologic studies using increasingly sensitive biologic markers has established that lead is toxic to children at levels of exposure considered safe only a decade ago.[3] Lead is now recognized to cause a syndrome of "subclinical poisoning." This syndrome affects adversely the functioning of erythrocytes, peripheral nerves, kidneys, the immune system, bones, and the CNS. All of these effects may occur in the absence of clinically evident signs or symptoms.

The hematologic dysfunction in subclinical lead poisoning involves disruption of heme biosynthetic enzymes. Inhibition of δ-aminolevulinic acid dehydrase begins at a blood lead level of 5 to 10 μg/dL.[7] Inhibition of ferrochelatase, resulting in elevation of erythrocyte protoprophyrin, becomes detectable in children at a blood lead concentration of 15 μg/dL.[8]

Renal toxicity in subclinical lead poisoning involves enzyme inhibition in the proximal tubular lining cells.[8] At blood lead levels below 25 μg/dL, lead inhibits activation in those cells of vitamin D.[9]

Neuropsychologic dysfunction in subclinical lead poisoning has been reported by Needleman et al.[10] In asymptomatic children with elevated lead burdens, corresponding to blood lead levels of 25 to 45 μg/dL, Needleman et al found that mean verbal IQ was 4.5 points lower than in similar children with lesser lead burdens. This deficit remained evident when results were adjusted for parental education and socioeconomic status. A recent reanalysis of these data by the US Environmental Protection Agency has corroborated these findings.[11]

Further evidence for the subclinical neurotoxicity of lead comes from two European studies. Winneke et al[12] found a mean difference in verbal IQ (after correction for socioeconomic factors) of 4.6 points between asymptomatic children with elevated lead burdens and a comparison group. Smith et al[13] found significant differences in IQ and reading ability among three groups of school-aged children with mean blood lead levels at examination of 14.4, 11.9, and 11.4 μg/dL. In a recent review of these studies, Rutter considered it "highly implausible" that the consistent decrements in IQ observed in them could have arisen by chance.[14] He concluded that daily exposure of children to lead in the modern urban environment causes a mean IQ decrement of 2 to 5

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points.[14] That conclusion, taken together with the results of the Second National Health and Nutrition Examination Survey, has profound implications for the health of American children.[6] Although a mean IQ deficit of 2 to 5 points may appear insignificant, Needleman et al[15] have demonstrated that a downward shift of this magnitude is associated with a threefold increase in the number of children with IQ scores below 80 and a threefold reduction in the number with IQ scores above 125. In the population of American children with elevated blood lead levels, the aggregate effect of this silent loss will be immense. The attainment foregone and the societal disruption engendered are incalculable.

The statement on childhood lead poisoning by the American Academy of Pediatrics' Committee on Environmental Hazards[16] in this issue was stimulated by these considerations. It is the first comprehensive statement on lead poisoning from the Academy in more than a decade. It deserves the careful attention of every pediatrician and other health professional who cares for children. Its major recommendations are (1) that screening of preschool children for lead and for predisposing iron deficiency should be widespread; (2) that screening should begin with measurement of erythrocyte protoporphyrin at 9 to 15 months of age, coincident with determination of hematocrit; (3) that vigorous abatement of all lead hazards should continue; and (4) that reporting of lead poisoning should be mandatory in all states.

This statement complements the new *Statement on Preventing Lead Poisoning in Young Children* released in 1985 by the Centers for Disease Control.[17] The Centers for Disease Control has determined that prevention of subclinical lead poisoning in children requires downward revision in the definition of increased lead absorption. An elevated blood lead level is defined now by the Centers for Disease Control as a whole blood lead concentration of 25 μg/dL or greater (formerly 30 μg/dL or greater). Lead toxicity is defined as a blood lead level of 25 μg/dL or greater in conjunction with an erythrocyte protoporphyrin level of 35 μg/dL or greater (formerly 50 μg/dL or greater). The Academy concurs in these definitions. It is reasonable to anticipate, as additional information develops on the toxicity of lead at low levels of exposure, that these definitions will be revised downward further.

The major tragedy of subclinical lead poisoning is that its neurologic consequences are permanent and irreversible. Although vigorous chelation can reduce morbidity and mortality, no therapy can replace dead neurons.[18] It is self-evident, then, that the major effort of pediatricians must be directed toward prevention. Prevention of lead poisoning will continue to be accomplished through individual and community action aimed at abating all sources of exposure to lead.[19]

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