Lead and Hyperactivity: Lead Levels Among Hyperactive Children

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Previous work has demonstrated an association between hyperactivity and increased body lead burdens in school-age children. In the present study it is shown that within a group of hyperactive children those for whom an organic etiology is present have lead burdens lower than in those for whom no apparent cause could be found. These data lead us to reject the notion that hyperactivity per se is responsible for the acquisition of elevated lead levels, and further strengthen the suspicion that for some children lower lead level absorption may be implicated in the development of the hyperkinetic disorder.

While sufficient evidence exists to indicate that children who have been lead-poisoned can suffer neurological and behavioral residua later into their childhood (Byers & Lord, 1943; Thurston, Middelkamp, & Mason, 1955; Chisholm & Harrison, 1956; Smith, Baehner, Carney, & Major, 1963; Perlstein & Attala, 1966), little is known of children whose intoxication has gone undiagnosed and even less about children who have been subjected to low-level lead exposure (Wiener, 1970; Lin-Fu, 1972).

In a previous report (David, Clark, & Voeller, 1972) we drew attention to the existence of an association between lower level lead burdens and hyperkinesis in children. Hyperactive children as a group had significantly higher blood lead levels and higher urine lead levels after provocative chelation than a comparable group of nonhyperactive children. That hyperkinesis can be and often is a

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sequela of lead intoxication has been known since Byers and Lord's (1943) follow-up study of lead-poisoned children. The novelty in our finding was that the association between lead and hyperactivity seemed to exist not only where the lead levels were well below those ordinarily considered toxic but where there was no good reason from the children's histories to suppose that they ever were. The suspicion was thus raised that lead absorption at even relatively low levels may be implicated in the etiology of the hyperkinetic disorder.

Although we have adopted this working hypothesis, i.e., that low level lead absorption may be causally linked to hyperkinetic behavior in children, other explanations for the association present themselves. As we (David et al., 1972) and others (e.g., Lansdown, Shepherd, Clayton, & Delves, 1974) have noted, for example, the relationship can be understood as well by assuming that the children were hyperkinetic prior to acquiring a lead burden. It is entirely conceivable, then, that by virtue of being hyperactive these children might have had increased exposure to situations where lead burdens could have been acquired. It could then be said that hyperkinesis was responsible for the acquisition of higher than normal amounts of lead, rather than vice versa.

In order to evaluate this hypothesis we have categorized hyperactive children according to etiologically relevant events in their medical histories, segregating, as it were, groups of children whose hyperkinesis can with reasonable certainty be ascribed to causes other than lead — e.g., severe pregnancy and birth complications and postnatal CNS trauma. In this way we formed groups of children with similar manifest pathology but with differing potential etiologies. If indeed the hyperkinetic pathology is responsible for the greater than normal lead levels, there should be no difference between these etiologically formed groups in the lead levels they present.

The evidence available in our original report (David et al., 1972) did not support this hypothesis. Those children in whom a probable cause for hyperkinesis was present (other than lead) tended to have lead levels lower than the children in whom no apparent cause was found. However, the number of children in that sample who had a probable other cause was relatively small (N = 9). We now report a new series of hyperkinetic children that includes many more children whose hyperactivity can be ascribed to etiologies other than lead. As in our first study, children have been classified according to the likelihood that they have experienced some perinatal or postnatal insult to the nervous system. Lead levels, as well as the results of a lead exposure questionnaire, were then compared for the "etiologic" groups resulting from that classification.

**METHODS**

**Subjects**

The children studied were referred to our unit for the diagnosis and treatment of hyperactive behavior from either the Outpatient Pediatric Neurology...