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IN REPLY REFER TO:

April 30, 1971

Dr. Robert A. Kehoe
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Dear Dr. Kehoe:

Many thanks for your letter of April 7. I would, in the first place, have to agree with you and say that clear-cut clinical symptoms of lead poisoning in children are virtually always associated with blood lead levels of approximately 80 µg Pb/100 G whole blood or greater. Exceptions to this would be children with severe anemia or prior brain damage due to other causes in which the complexities introduced by the presence of multiple diseases makes the assignment of symptoms often difficult. In such children, I would be inclined to depend more upon the response to chelation therapy and also on the response in terms of ALA or coproporphyrin excretion in reaching a decision. With respect to the very mild symptoms of poisoning which are subjective in nature, I feel that we are on much less firm ground in dealing with children than you are in dealing with adults because of the difficulties in assessing minor changes in behavior and gastrointestinal function in a two-year old child. I am also in good agreement with you on the point that the risk of acute symptomatic lead poisoning increases markedly as blood lead levels rise above approximately 80 µg Pb/100 G whole blood, but that the actual occurrence of symptoms and the severity of symptoms cannot be correlated precisely with blood lead levels above the 80 µg level. There is, of course, in children the additional epidemiologic factor of the very large dose that can result from the ingestion of paint chips. This is quite different, I think, from the industrial situation in which, barring breaks in environmental control techniques, a relatively constant level of exposure might be anticipated. Not so in the child in whom we find blood lead levels can rise at a precipitous rate within a period of one to two months from a relatively safe level to a level associated with symptoms. This must certainly be due to the very high dose which such children may ingest. It is for this reason that we set a level of action for preventive purposes well below the 80 µg level. I would suspect that blood lead levels between 40 and 50 µg Pb/100 G whole blood are highly suggestive of the probability that a child has an additional abnormal source of lead and that given sufficient time and continued pica, he may absorb enough lead to cause clinical lead poisoning. In Baltimore, we are fortunate in that the Health Department will take preventive action on the basis of the diagnosis of "increased lead absorption" which the Health Department now defines as a confirmed blood lead level >50 µg Pb/100

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G whole blood. I believe that in the minds of most pediatricians, almost no distinction is made between the terms "increased lead absorption" and "lead poisoning". This and the very high dose of lead that children may ingest is at the root of most of the confusion. There is also great confusion among pediatricians because most are forced, at present, to base their actions on inaccurate analytical results. I might state parenthetically that reports of lead poisoning in adults with blood lead levels in the 40-70 µg Pb/100 G whole blood range have always raised my suspicion that the analytical results were low. In any event, I heartily agree that accurate analytical results and techniques constitute one of the main stumbling blocks to the adequate management of lead poisoning and its prevention today. At the moment, I am trying to take on this unglamorous, but I fear, necessary task.

The area of great uncertainty with respect to potential adverse effects of lead is, in my mind, the range of lead absorption associated with blood lead levels in the 50 to 80 µg Pb range. This is a range associated with metabolic alterations in heme synthesis. While the disturbance in heme synthesis is, with respect to the formation of red blood cells, clearly reversible, it is also possible but not proven, that the disturbance in heme synthesis may reflect metabolic derangements in other tissues and particularly in the nervous system which may not be reversible. And so there is in the minds of many the question of whether some subtle effects will occur in the developing young child that are not manifested by the usual clinical indices. This is an area which I hope to investigate in the coming years. Pediatricians in particular are sensitive to the possibility of subtle, deleterious sub-clinical effects in the child. This stems largely from their experience with various inborn errors of metabolism, such as phenylketonuria and galactosemia in which symptomatic control is insufficient and metabolic control is essential, if the patient is to have a good prognosis. Likewise, it appears in young children that measles, even in the absence of post-measles encephalitis, can be associated with injury to the brain. And so I think it is proper in pediatrics that we concern ourselves with the sub-clinical aspects of disease processes. I do hope that this answers some of the questions posed in your recent letter.

Yours sincerely,

J. Julian Chisolm, Jr., M.D.

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