

August 2, 1948

Dr. Herbert K. Abrams,
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Department of Public Health,
2002 Acton Street,
Berkeley 2, Calif.

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Dear Doctor Abrams:

The best answers I can give you to the questions raised in your letter of July 27 will be found in publications of one kind or another which have been made by myself and my associates over a number of years. Accordingly I am sending you a group of reprints which I believe will be satisfactory for your purposes. I am also sending you a report with which you and your associates should be familiar if you are not already. A committee of the American Public Health Association, of which I am chairman, undertook to set down, in a carefully and critically edited manner, the principal facts concerning occupational lead exposure. I think it is fair to say that no such collection of information is available elsewhere in the English language. There is nothing in this report that is open to very serious question or that is actually controversial. The facts, as stated, are facts substantiated by adequate information.

To answer several of your questions specifically, our observations on the use of BAL in lead poisoning (covered, in part, by a reprint being sent to you) have quite clearly demonstrated that this material influences the distribution of lead in the body quite dramatically. Promptly after administration of BAL the lead concentration in the blood drops sharply, while that in the urine increases many fold. This whole response occurs quickly and is over quickly, the end result being that a few milligrams of lead are lost from the body more than would have been without such treatment. Unfortunately, BAL cannot be administered continuously or even repeatedly over a considerable period of time, and therefore this procedure accomplishes little or nothing. We are studying the possibility of the use of a less toxic analogue of BAL, which might be administered continuously or quite frequently, and thus bring about a significant loss of lead from the body. Up to the present time, however, having studied a series of more than 20 cases of acute episodes of lead intoxication, we are quite sure that this brief period of deleading is of no practical importance in the treatment of the disease. Incidentally, the use of BAL has no apparent influence on the course of the illness. The symptoms of an acute episode of intoxication are neither increased or decreased, so far as we can see, by the administration of BAL. Therefore, this treatment is of physiological interest, rather than of practical importance. Our advice to physicians is to treat episodes of lead poisoning symptomatically. The episode of colic can be controlled by orthodox anti-spasmodic drugs, including intravenous administration of calcium gluconate. Other

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forms of treatment by means of diet and by the administration of agents which are supposed to influence "mobilization" or "demobilization" of lead are quite without avail, since, with the exception of BAL, there is no agent known which has any material influence on the distribution of lead within tissues of the body or on its excretion. Unfortunately, the evidence for "mobilization" and "demobilization" in relation to the calcium metabolism is based on false premises, and ~~has~~ have no effect upon human lead poisoning.

The diagnosis of lead poisoning is discussed adequately and comprehensively in the report of the American Public Health Association, which I am sending to you. It would be impossible to answer your questions briefly and effectively in this letter, since the questions raised relate to comprehensive physiologic information which cannot be given in a few statements without appearing to be dogmatic. However, I will make one statement. The practicing physician should undertake to make a diagnosis in suspected cases of lead poisoning by means of a comprehensive clinical study of his patient. The diagnosis of lead poisoning simply cannot be made by some short-cut laboratory procedure, and only an ignorant or stupid physician will try to make it by such means. The results of the laboratory examinations merely answer the question of whether the individual has had significant lead exposure within the very recent past. They tell nothing concerning the presence or severity of his illness, which can only be determined by careful physical examination. The differential diagnosis must be made on essential clinical evidence.

I trust that these comments will be helpful to you, and wherein they do not agree with some of the more orthodox notions expressed in some of the earlier literature, you may rest assured that they are backed up by adequate clinical and experimental evidence. Not all of this has been published, but we are preparing articles for publication which will establish the truth of my statements beyond any reasonable doubt.

Cordially yours,

Robert A. Kehoe, M. D.

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