

June 28, 1962

Dr. F. Curtis Dohan
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Hospital of the University of Pennsylvania
36th and Spruce Streets
Philadelphia 4.

Dear Doctor Dohan:

I am pleased to comment on the case report included in your letter of June 26, 1962.

I have to begin by saying that the swallowing of gasoline, in the siphoning procedure to which the history referred, cannot be accepted as even barely tenable, for certain very definitive clinical reasons. Quite apart from the fact that the man had no symptoms of tetraethyllead poisoning (which could hardly have been the case, if he had ingested a significant quantity of tetraethyllead since the CNS syndrome is practically invariable and bears no resemblance to inorganic lead poisoning), the signs of absorption (stippling of the erythrocytes and high concentration of lead in the blood) do not occur in tetraethyllead poisoning. The absorption of "TEL" gives rise to no hematological evidence of any kind, while even fatal TEL poisoning is unassociated with more than an insignificant increase in the lead content of the blood (the metabolic breakdown of TEL does not result in a compound of lead that is bound by the erythrocytes).

This is not to say that your patient did not have lead poisoning, but that the source of his exposure to lead has not been identified. Incidentally one does not "frequently swallow(s) considerable amounts of gasoline" without having an acute gastronteritis, and even if one were to do so, the quantity of TEL so ingested and absorbed will almost certainly be negligible in relation to a cumulative toxic dose. Your people have simply not bothered to consider the quantitative aspects of the dosage, in arriving at their interpretation of the source of exposure. Relatively large quantities of lead must be taken by mouth regularly to cause lead poisoning (of the order of 3 to 10 mg. per day, dependent upon the compound and the length of time involved, which must be appreciable), It is not altogether uncommon to fail utterly to find the source of exposure to lead which results in intoxication. This is not often true of the adult, but amateur investigators of occupational situations arrive at the wrong answer much too easily, and this is why we have so many medico-legal controversies in these situations.

*or the source of the
absorption of TEL*

As to your primary question of the association between lead poisoning and hepatitis, I can give you no better answer than you have. I am in no position to say that this does not occur, but I can say that I have never seen it, and this is equivalent to my saying that it is rare. I consider it to be a very improbable explanation of your case.

It is not proper, of course, to jump at any conclusion, but it happens that we have seen recently two cases of lead poisoning definitely traceable to the illicit making of whisky. We have had an influx of "hillbillies" white and black, from the Kentucky and Tennessee mountain areas, and the Federal Revenue people have uncovered an astonishing number of stills. The most common type of condenser is the used automobile radiator, which, as you probably know, is heavily coated with solder. The whisky produced in such stills is quite high in its lead content and these men take on a fair volume of "moonshine".

The fact that your patient is a negro does not influence me beyond reminding me that our cases were of this race.

Returning again to the hepatitis, it seems true, from early reports, that at one time jaundice, in connection with lead poisoning, was not uncommon. There is no doubt that the disease had much more serious manifestations in that period than those seen in our day (except in children). It is possible that some of these cases were the result of toxic damage to the liver.

Please accept my thanks for satisfying my curiosity about this case, and allowing me to comment on it.

Cordially yours,

Robert A. Kehoe, M. D.

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