

Lead Industries Association

60 EAST 42ND STREET

NEW YORK 17, N.Y.

ANDREW FLETCHER, PRESIDENT
H. C. BROWNELL, VICE PRESIDENT
J. A. MARTINO, VICE PRESIDENT
M. M. ZOLLER, VICE PRESIDENT
ROBERT LINOLEY ZIEGFELD,
SECRETARY-TREASURER

MANFRED BOWDITCH
DIRECTOR OF HEALTH AND SAFETY

April 9, 1957

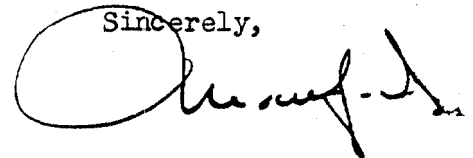
Robert A. Kehoe, M.D., Director
Kettering Laboratory
University of Cincinnati
Eden Avenue
Cincinnati 19, Ohio

Dear Bob:

Herewith is a reproduction of several abstracts culled from
the current issue of the British Journal of Hygiene.

Having in mind your masterful disquisition in the January
22, 1955 issue of the J.A.M.A., I wonder if you might feel inclined to
pen a few transient lines to Dr. Perrault and his pals.

Sincerely,



MB:GW

Enclosure



VE 8 02694

GROTH, O. & RIGNÉR, K. G. Ny blyförgiftningsrisk. [A New Risk of Lead Poisoning] *Nordisk Hyg. Tidskr.* 1956, v. 37, Nos. 9/10, 238-41.

The English summary appended to the paper is as follows:—

"Two cases of severe lead poisoning are described occurring in workers at a small workshop producing lead glass reflectors. The clinical picture was typical in both cases. Lead and corprophyrin were found in the urine. Blood examination showed basophilic stippling of the red cells and a transient thrombocytopenia.

"On inspection of the workshop the arrangements for protection against lead dust were grossly inadequate. The personal hygiene was also below the standard required for lead workers.

"These cases illustrate that the occupational risk can be great when new methods are used or new products are manufactured in small workshops, especially when knowledge of the risks involved is lacking."

LEBOCQ, J. & GUYOTJEANNIN, C. Le dosage de l'urée sanguine dans la prévention du saturnisme professionnel. [The Amount of Urea in the Blood with regard to the Prevention of Occupational Lead Poisoning] *Arch. Malad. Professionnelles.* Paris. 1956, Sept.-Oct., v. 17, No. 5, 466-9.

The authors propound the idea that persons with an abnormal level of urea in the blood are more predisposed to sustain lead poisoning if exposed to a lead hazard. The abnormal level, however, may be due to some passing phase in metabolism; free sweating may be a cause, or abnormal protein diet, especially if associated with alcoholism. Attention to diet may easily abate the abnormal level. An industrial doctor, examining for work entailing a lead hazard, should proceed with caution. A refusal to pass the individual may label him with an inferior capacity for employment and do him harm in the labour market. And the same applies to removal from the hazard to safer work. The doctor must proceed carefully, pass the person and keep a watch on his further health.

Notes on 24 instances are quoted: some with exposure to lead and an abnormally high level of urea which became normal under a dietary; some with high blood pressure or chronic nephritis with no abnormal level of urea; some with lead exposure but no signs of lead absorption, but an abnormally high urea level; some intended to face a lead hazard who are found to have an abnormally high level; and some with lead absorption and a high level. What should be regarded as a dangerous limit is left undecided.

E. L. Collis

PERRAULT, M., TRUHAUT, R., KLOTZ, B., BOUDÈNE, C., DREUX, C., CLAVEL, B. & CHAIN, F. Sur l'efficacité de l'E.D.T.A. calcique dans l'intoxication saturnine professionnelle. [The Efficiency of CaEDTA in Occupational Lead Poisoning] *Arch. Malad. Professionnelles.* Paris. 1956, Sept.-Oct., v. 17, No. 5, 423-9. [29 refs.] Discussion 470-72.

An acute case of occupational lead poisoning is described. The man, who was employed in making accumulators, had been handling for a year a mixture containing lead. He had paroxysms of abdominal pain and suffered from constipation, severe headaches and pains in the legs. CaEDTA was given by intravenous perfusions, with rapid removal of all pains and feelings of malaise. The perfusion was given over a period of 2-3 hours twice a day for 4 days and this course repeated after an interval of 3 days.

The headaches and joint pains persisted until the second course. Punctate red cells soon disappeared from the blood and large amounts of lead were excreted in the urine.

The action of CaEDTA is considered at length. It is a chelating agent which means that it captures lead or other metals from the tissues, converting them into soluble and non-toxic salts; these salts are promptly carried to the kidneys to be excreted. Lead in the soft tissues and blood is first chelated; then lead stored in the bones passes into the blood where it is chelated in turn. Metals which may be chelated are calcium, copper, lead, mercury, iron and chromium. In order to avoid chelation of normal calcium, leading to hypocalcaemia and tetany, the calcium salt is used instead of the sodium salt. The lead excreted in the urine may reach 4 mgm. in a child and 10 mgm. in an adult per day. The authors prefer intravenous administration, since the contents of the stomach may neutralize some of the chelating power before the drug reaches the blood.

In discussion, a plea is entered for oral administration in less acute cases to patients in hospital but treated as ambulatory cases. Possibly EDTA might be used prophylactically for workers unavoidably exposed to a lead hazard. EDTA is far less toxic than BAL; indeed no injurious side effects have been seen. Its possible value in other conditions besides plumbism has yet to be assessed. A successful ambulatory case is instanced.

E. L. Collis

MAYNARD, L. S. & FINK, S. The Influence of Chelation on Radiomanganese Excretion in Man and Mouse. *J. Clin. Investigation.* 1956, Aug., v. 35, No. 8, 831-6, 3 figs. [12 refs.]

Experiments were carried out on 9 human patients and on mice. ^{56}Mn (half-life 2.59 hours) and ^{52}Mn (half-life 5.8 days) were used as tracers in the investigations. Mn was administered intravenously. It was found that while Mn excretion in human beings is mainly faecal, pre-chelation with EDTA results in its almost exclusive elimination in the urine for about 24 hours. After this time it again becomes faecal. Experiments on mice indicated that the Mn excretion curves were divided into fast and slow components, and that pre-chelation with EDTA not unexpectedly increased the fast component. The authors suggest that free and bound forms of Mn exist in the body and that these may account for the observed behaviour of Mn.

D. G. Harvey