Reprinted from PROCEEDINGS OF THE SOCIETY FOR EXPERIMENTAL BIOLOGY AND MEDICINE, 1941, 46, 476-477

12033 P

Treatment of Lead Poisoning with Sodium Citrate.

SEYMOUR S. KETY AND T. V. LETONOFF. (Introduced by J. Harold Austin.)

From the Biochemical Laboratory, Philadelphia General Hospital.

In investigations undertaken by one of us (S.S.K.) it was found that sodium citrate in dilute solution exerts a powerful solvent effect on tertiary lead phosphate,[1] that citrate removes lead ion from solution by the formation of a soluble complex of extremely low dissociation,[2] and that the administration of sodium citrate to lead poisoned rats produced a significant increase in the excretion of lead.[1] The junior author (T.V.L.) independently observed that in a case of plumbism receiving potassium citrate incidental to a chloride excretion test there was a sharp drop in the blood lead level and a rise in the urinary output of lead. (Chart I. No. 7.) These observations led to the present study of the effects of the administration of sodium citrate in plumbism.

Six cases of lead poisoning seen at the Philadelphia General Hospital since August, 1940, provided the basis of this study.* All presented clinical evidence of mild to severe lead intoxication and all had abnormally high lead concentrations in whole blood on admission. Each adult received from 2 to 4 g of sodium citrate and the children from 1 to 2 g by mouth 3 times daily. Blood lead concentrations and, in 3 cases, urinary lead excretions were deter-

[1] Kety, S. S., read before the Undergraduate Medical Association, Medical School, University of Pennsylvania, April 4, 1940.

[2] Kety, S. S., to be published.

* The authors are indebted to Dr. W. Brody, Dr. E. L. Noone, the late Dr. R. H. Thompson, the late Dr. R. G. Torrey, and Dr. L. A. Wikler on whose services these patients were treated.

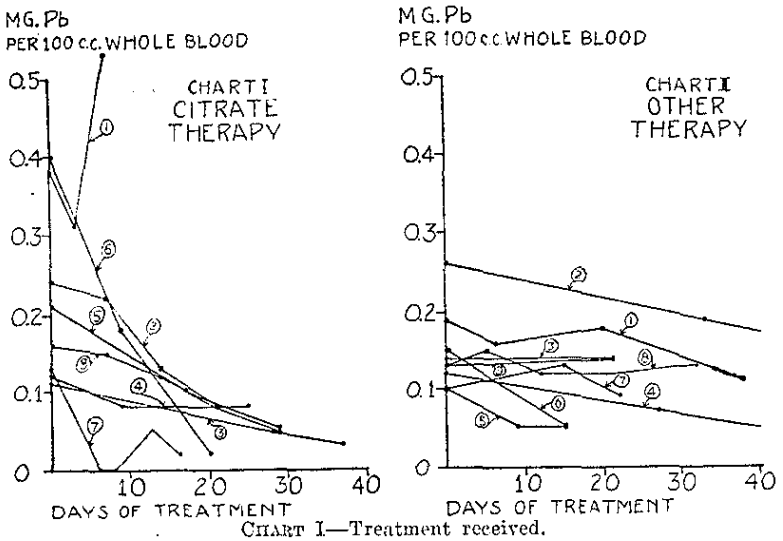


CHART I—Treatment received.

1. Sodium citrate throughout.
2. (Readmission of No. 1 after 47 days without treatment) Sodium citrate throughout, calcium carbonate 14th to 21st day.
3. Sodium citrate throughout.
4. Sodium citrate, iron and copper and thiamin chloride throughout.
5. Sodium citrate throughout, calcium gluconate 17th to 29th day.
6. Sodium citrate throughout.
7. Potassium citrate 2d and 3d days.
8. Sodium citrate throughout.

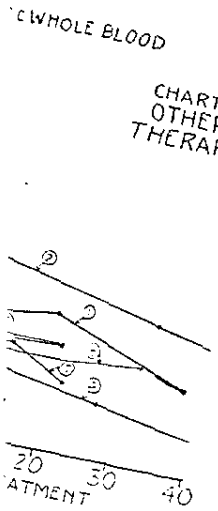
CHART II—Treatment received.

1. Disodium phosphate, cevitamic acid and oleum percomorphum throughout.
2. Disodium phosphate, diet high in phosphorus and calcium, viosterol, cevitamic acid and thiamin chloride throughout.
3. Oleum percomorphum throughout.
4. Calcium lactate and calcium phosphate throughout, cevitamic acid 9th to 14th day, cod liver oil 14th to 57th day.
5. No specific treatment.
6. Calcium phosphate 5th to 15th day.
7. Calcium chloride throughout.
8. Dihydrotachysterol throughout.
9. (Readmission of No. 8 after 10 months without treatment) Brewer's yeast, nicotinic acid and cevitamic acid throughout, monosodium phosphate and calcium carbonate 6th to 21st day.

mined before and at intervals during the course of treatment by the method of Letonoff and Reinhold[3] slightly modified.[4]

The results are presented in Chart I. There was a rapid disappearance of toxic symptoms without untoward reactions in every instance and the blood lead concentration fell to normal or nearly normal levels, (normal range as determined by this method is from 0 to 0.05 mg lead per 100 g of whole blood). Urinary excretion studies gave variable results with a trend toward increased urinary excretion of lead during treatment. In Chart II are pre-

[3] Letonoff, T. V., and Reinhold, J. G., *Ind. Eng. Chem., Anal. Ed.*, 1940, **12**, 280.
[4] Letonoff, T. V., to be published in *Ind. Eng. Chem., Anal. Ed.*



ent) Sodium citrate

oughout.
h day.

in throughout.
sterol, cevitamic

ic acid 9th to

wer's yeast,
nd calcium

it by the

l disap-
every
nearly
od is
ex-
ased
pre-

280.

sented the changes in blood lead (determined by the same method) of patients with plumbism receiving other therapy.

A controlled study is in progress with other citrate compounds to determine the means whereby lead is removed from the blood. Whatever the mechanism of action of sodium citrate, the rapid alleviation of symptoms and the drop in blood lead levels observed should make it a useful therapeutic agent and a possible prophylactic measure in lead poisoning.

The authors wish to express their gratitude for the guidance of Dr. John G. Reinhold.