anemia in plumbism. His explanation of the "shattering" of the red blood cells fits in well with the above theory.

However, there are many cases in which the exposure has continued over many years, and there is only slight anemia and no icterus present. It is believed that in such cases Rimington's [9] theory that lead may be acting as a block between protoporphyrin and iron to prevent their normal combination in the formation of hemoglobin might be a plausible explanation of this increased coproporphyrinuria.

Following this line of postulation, we believe that in the cases of S. O. and C. A. coproporphyrinuria increased because red blood cells were being destroyed by lead with resultant release of protoporphyrin, which was converted into coproporphyrin and excreted. In the case of C. S. who had chronic lead poisoning and who had been taking in large amounts of lead for many years, it is believed that the increase of coproporphyrin took place in the bone marrow and might possibly have been due to a blocking by lead during the synthesis of hemoglobin. The case of J. S. also seems to have a somewhat similar mechanism as the case of C. S.

However, as was stated previously, although many theories have been set forth, the actual mechanism of this increased coproporphyrinuria is not clearly understood. It does appear, however, in reviewing these four cases, that rather than insisting that there must be only one mechanism common to all cases of plumbism, it is wiser to consider the possibility that two mechanisms may be present, especially in view of the fact that both the clinical and the laboratory findings do vary from case to case.

While the mechanism of the coproporphyrinuria of plumbism is not definitely known, it is known that an increased excretion is seen in lead workers absorbing large amounts of lead and also in cases of acute and chronic lead poisoning.[10] It has recently been suggested that the finding of coproporphyrinuria may be a valuable aid in the prevention or early diagnosis of plumbism.[11] Kluver [12] has found the coproporphyrins to be normally present in the white matter of the central nervous system of animals, including man. This has led to speculation that the neuritic pain, headache, encephalopathy and abdominal colic present in plumbism may be explained by the coproporphyrins deposited in the tissues.

We should like to explore the effect of citric acid, as opposed to that of sodium citrate, on lead excretion in acute plumbism. We should like to determine the effect of the citrate ion as opposed to the alkalinizing effect of sodium citrate. Until such observations are complete, freedom from lead exposure, if attainable, and calcium therapy where needed, are dependable measures. Since it is not always

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10. (*a*) Maloof, C. C.: Role of Porphyrins in Occupational Diseases: I. Significance of Coproporphyrinuria in Lead Workers, Arch. Indust. Hyg. & Occup. Med. **1**:296-307 (March) 1950. (*b*) Watson.[8b]

11. de Langen, C. D., and ten Berg, J. A. G.: Porphyrin in the Urine as a First Symptom of Lead Poisoning, Acta med. Scandinav. **130**:37, 1948. Waldman, R. K., and Seideman, R. M.: Reliability of the Urinary Prophyrin Test for Lead Absorption, Arch. Indust. Hyg. & Occup. Med. **1**:290-295 (March) 1950. Maloof.[10]

12. Kluver, H.: On Naturally Occurring Porphyrins in the Central Nervous System, Science **99**:482, 1944.