

Industrial Toxicology

CONGESTIVE HEART FAILURE OF RENAL ORIGIN: PATHOGENESIS AND TREATMENT IN FOUR CASES OF CARBON TETRACHLORIDE NEPHROSIS. C. K. FRIEDBERG, *Am. J. Med.* **9**:164 (Aug.) 1950.

The first of the four cases of carbon tetrachloride nephrosis presented concerned a welder whose symptoms began with nausea, vomiting and epigastric burning after he had been exposed to vats containing sulfuric acid and carbon tetrachloride. This man had advanced pulmonary edema when, after five days of treatment at another hospital, he came under the observation of the author. The pulmonary edema was undoubtedly due to the huge amount of fluids administered parenterally, 10 L. having been given in a two day period during which the damaged kidneys had excreted less than 100 cc. The risk of further overloading the circulation was recognized but apparently not completely understood. For although fluids were limited, they were not sufficiently restricted, and small intravenous infusions containing sodium were given to combat acidosis. Thus the exaggerated risk of acidosis led to an unwise compromise with the much more dangerous and imminent risk of congestive heart failure. Furthermore, the seriousness of the pulmonary edema was underestimated in treatment, and death occurred before phlebotomy could be performed.

The second patient was a mechanic who was exposed to carbon tetrachloride fumes for five hours while cleaning a machine in a small, unventilated room. Here, again, excessive administration of fluids during the first week of illness led to acute pulmonary edema which required phlebotomy for relief. However, the intake was not enormous, averaging 1,900 cc. per day for five days. Thereafter fluids, and especially fluids containing sodium salts, were relatively limited although more were given than is usually desirable. That heart failure did not recur may be attributed to two factors, severe bleeding and persistent excessive vomiting which tended to result in a static or slightly negative fluid balance.

The third patient was a 49 year old laboratory worker who was admitted nine days after inhaling carbon tetrachloride fumes. This patient presented evidence of anasarca, which was attributed in part to the excessive intravenous administration of fluids prior to admission. The fear of hypochloremia and acidosis led to the administration of sodium chloride and the precipitation of acute pulmonary edema. The patient recovered after spontaneous diuresis set in.

The fourth patient, who had been exposed to carbon tetrachloride fumes for four hours in a poorly ventilated cellar, was admitted in severe pulmonary edema which had been induced by the intravenous administration of sodium-containing fluids during the first week of the oliguria. After this was controlled there was no further evidence of heart failure on a regimen which permitted no more than 500 cc. of fruit juice orally. Although the urea nitrogen reached 226 mg. per 100 cc. of blood, spontaneous diuresis occurred at its usual time, and recovery of renal function ensued as early as in cases in which larger amounts of fluid had been given or in which efforts had been made to clear the blood stream of waste products by artificial means.

The author emphasizes that the deficient excretion of sodium and water due to primary renal disease can produce the syndrome of congestive heart failure without cardiac disease.

These cases are presented not only to reemphasize the danger of fatal pulmonary edema due to overzealous administration of massive quantities of fluids but to stress the frequency of congestive heart failure with intakes of sodium and water which are not ordinarily excessive for persons with normal hearts.

Finally, this report is designed not merely to reiterate the self-limited nature of toxic nephrosis and the merits of conservative therapy but more particularly to stress that the treatment of carbon tetrachloride nephrosis should be directed chiefly toward the prevention and control of heart failure without regard to the concomitant azotemia and almost always without regard to the associated acidosis or electrolyte disturbances.

THE ESTIMATION OF TRICHLOROETHYLENE IN BLOOD. F. H. BRAIN and P. J. HELLIWELL, *Biochem. J.* **45**:75, 1949.

New conditions are described for the quantitative production of the colored compound from trichloroethylene and pyridine in the presence of alkali. A more efficient apparatus is described for the absorption of trichloroethylene steam distilled from blood. The apparatus could be used