hydrolysis and precipitation at the site of injection with subsequent development of peritonitis. The citrates and edetate complexes are absorbed from the injection site and show a general pattern of decreasing toxicity from the lower to higher lanthanons. Gross and histopathological examination of bone marrow (sternal), liver, kidney, spleen, adrenals, sex organs, lungs, myocardium, and mesenteric lymph nodes indicate that lesions, when found, were similar for all rare earths. There appears to be a predilection for liver and kidney. Citrates produce an acute passive congestion of lungs and liver within 4 hours after injection, which is of a transitory nature. Renal changes are found later but not consistently. In general, the citrates were more toxic than the edetate complexes. -- *Authors' summary*

1146 The Determination of Thallium in Urine. M. B. Jacobs. Am. Ind. Hyg. Assn. J. 23, 411-414 (Sept.-Oct. 1962).

A variation of the triphenylmethane dye method for the determination of thallium has been modified to be suitable for analysis of urine samples in a small laboratory. Thallium is oxidized to the thallic state by bromine water, and is reacted with methyl violet for the colorimetric measurement. A maximum of 200 micrograms thallium per liter was found in the urine of 21 exposed individuals. Urine of unexposed persons showed virtually no thallium. -- Author's abst.

1147 A Modification of the McCord and Zemp Method for the Determination of Lead in Urine. A. Frank. Am. Ind. Hyg. Assn. J. 23, 424-430 (Sept.-Oct. 1962).

In the determination of the total lead content of urine by the dithizone method a large part of the analysis time is spent in ashing. McCord and Zemp described a method using "wet ashing", which shortened the ashing time to 30 minutes. The author, by using a modification of the above method, found that no loss of lead occurred as compared with a determination carried out with a longer ashing time. This method may be used for the analysis of urine from persons suffering from lead poisoning even after treatment with EDTA or penicillamine. -- Author's abst.

1148 Toxic Properties of Dialkylnitrosamines and Some Related Compounds. D. F. Heath and P. N. Magee. Brit. J. Ind. Med. 19, 276-282 (Oct. 1962).

This paper is mainly a review, but it also includes observations which have not been published elsewhere. Some dialkylnitrosamines are valuable chemical intermediates or solvents, and their use as insecticides has been suggested. In the present paper their toxic action is reviewed. Most results refer to rats. Their main acute effect is hepatic centrilobular necrosis, though lung lesions may appear. The compounds also induce tumors in liver, lung, and kidney. One, dimethylnitrosamine, has been shown to cause kidney tumors after a single dose. The necrotic and carcinogenic doses of the compounds are closely related. Analogous formamides are much less toxic (the LD50's in rats by intraperitoneal injection of dimethyl- and diethyl-formamides are 3,800 mg./kg. and 1,740 mg./kg. and they do not cause centrilobular necrosis or tumors. Nitrosamines are oxidized in vivo and by liver preparations in vitro. Their toxic action is due to the release of powerful alkylating agents in the liver. They also inhibit protein synthesis and alkylate liver protein and ribonucleic acid. In all cases the effective agent appears to be a metabolite. The possible hazard to man in the uses of these compounds is emphasized.

1149 A Study of Acrylonitrile Poisoning in Relation to Methaemoglobin-CN Complex Formation. L. Magos. Brit. J. Ind. Med. 19, 283-286 (Oct. 1962).

Observations are recorded on methaemoglobin-CN complex formation in rats poisoned with acrylonitrile, potassium cyanide, and acetone cyanohydrin. In methaemoglobin-CN formation, the methaemoglobin level was increased by sodium nitrite. The results show that the rate of methaemoglobin-CN formation in rats killed by acrylonitrile is lower than in animals surviving potassium cyanide or acetone cyanohydrin poisoning, and much lower than in animals killed by potassium cyanide. These findings indicate that the toxicity of acrylonitrile cannot be solely due to the liberation of cyanide.