

- 690/76 Effect of Methemoglobinemia on Carbon Tetrachloride Hepatotoxicity. D. Pankow and W. Ponsold. *Toxicol. & Applied Pharmacol.* 36: 143-150, April 1976. 16 refs.

Injection of 0.8 mmol of NaNO_2/kg ip after administration of 5.2 mmol of CCl_4/kg po significantly enhanced the rise of glutamic pyruvic transaminase and leucine aminopeptidase activities in the plasma of rats over that observed after administration of CCl_4 alone. Enzyme activity was not increased by nitrite administration alone, which produced a methemoglobin concentration of about 40%. The potentiation of the CCl_4 effect was dependent on the presence of the adrenal glands. CCl_4 depresses and retards the formation of methemoglobin after nitrite administration. Similar combined effects of CCl_4 and other methemoglobin-producing compounds as aniline, nitrobenzene, m-dinitrobenzene, phenylhydrazine, and hydroxylamine hydrochloride are described.

--Author's abst.

- 691/76 Two Cases of Angiosarcoma of the Liver in Exposure to Vinyl Chloride. M. Zorica, et al. *Arhiv Hig. Rada Toks.* 26(4): 275-281, 1975. 3 refs. (Yugoslav)

Two cases of angiosarcoma of the liver in exposure to vinyl chloride are described. Both cases relate to the factory for the production and processing of PVC which started operating in 1949. The first case concerns a worker born in 1914 who worked on the polymerization of vinyl chloride for about 18 years (from 1953 to 1969), and spent the last four years in the PVC processing works. The disease became clinically manifest in April 1973 and terminated with a lethal issue the same year in August. The other case was a worker born in 1931 who worked in vinyl chloride production for about 23 years (from 1950 to 1972), five of which he spent in the hydrogen chloride works. The symptoms appeared in August 1972. The patient died in July 1973. In both cases the tumour was microscopically verified. It is assumed that the two described cases of angiosarcoma are occupationally conditioned and that they occurred as a result of exposure to vinyl chloride.

--Author's Summary in English

- 692/76 Immunological Mechanisms in the Pathogenesis of Vinyl Chloride Disease. A.M. Ward, et al. *British Med. J.* 1: 936-938, April 17, 1976. 23 refs.

Vinyl chloride (VC) disease is a multisystem disorder incorporating Raynaud's phenomenon, acro-osteolysis, thrombocytopenia, portal fibrosis, and hepatic and pulmonary dysfunction. Immunological and immunochemical investigations showed the presence of circulating immune complexes in 19 out of 28 patients with the disease and in a further two out of 30 workers exposed to VC. The immunological data were reviewed in relation to the clinical picture of the disease and to the available evidence on the metabolism of VC. The results suggest that VC disease is an immune complex disorder and that the immune response is initiated by the adsorption of VC or a metabolite on to tissue or plasma protein.

--Author's Summary

- 693/76 Exposure to Trichloroethylene Monitored by Analysis of Metabolites in Blood and Urine. O. Vesterberg and I. Astrand. *J. Occup. Med.* 18: 224-226, April 1976. 13 refs.

When neurologic effects of excessive exposure to TRI are to be prevented, analysis of TCE in blood or measurement of the excretion rate of TCE in urine seem to give valuable information. However, concerning TCE in blood the proportion thereof present as TCE glucuronide seems to be variable, rendering the use of TCE in blood as an index of the uptake somewhat uncertain. Large differences in TCE in blood occur even at similar exposure levels. As an index of exposure, TCA in urine seems to give more helpful information provided the results are evaluated judiciously.

--Cond. from text

- 694/76 Effects of 1,1,1-Trichloroethane Administered by Different Routes and in Different Solvents on Barbiturate Hypnosis and Metabolism in Mice. H.C. Shah and H. Lal. *J. Toxicol. & Environ. Health* 1: 807-816, May 1976. 25 refs.

A 24-hr inhalation of 1,1,1-trichloroethane (methylchloroform), 3,000 ppm, reduced pentobarbital hypnosis and increased hexobarbital oxidation by the 9,000 x g liver supernatant fraction in male mice. On the other hand, an ip injection of methylchloroform, 1 ml/kg, increased the duration of pentobarbital hypnosis and reduced hexobarbital metabolism by the liver microsomal enzymes. The potentiating effect of methylchloroform on pentobarbital hypnosis was diminished when it was diluted with olive oil, but was markedly enhanced when diluted with dimethylsulfoxide (DMSO) before injection. Three local applications of methylchloroform (1:1 dilution with DMSO) had an effect similar to that of inhalation on pentobarbital hypnosis.

--Author's abst.