DAVIDOFF, H. **Accidental Poisoning by Vinyl Chloride: Report of Two Cases.** *Canadian Med. Ass. J.* 1960, Apr. 16, v. 82, No. 16, 828–30. [12 refs.]

2 similar rapidly fatal cases, the second proved by circumstances to have been caused by vinyl chloride, illustrate dangers when safety rules are disregarded and toxicological difficulties of proof of poisoning from volatile gases. Also called chloroethylene or chloroethene, $CH_2 = CHCl$ is an explosive, heavy and volatile gas; BP = 14°C. In enclosed production of polyvinyl resin, polymerization is not completed and, before a tank is opened, residual vinyl chloride is pumped into another recovery tank. The residual gas contains 90% vinyl chloride, 8·5% CO_2, vinyl acetate, trichloroethylene, N and argon.

The first patient entered a tank to clean it, after the usual safety tests. Air was continuously exhausted and replaced, but the air hose afterwards appeared defective. He did not wear the required safety belt and line. Nor was the required rescue apparatus immediately available when, 10 minutes later, he failed to answer. The rescuer could not detect vinyl chloride. The second patient was found beside an open valve 20 minutes after he had entered a pit outside the recovery tank to open this water outlet valve from this tank. One rescuer closed the valve, was affected by escaping gas, became giddy, saw circles, crawled away shouting and recovered quickly; another, wearing a respirator, removed the body. Both of the men who died had often done these tasks.

At autopsy, performed 3 and 8 hours later, no vinyl chloride was found. It may have been expelled by artificial respiration. The pathological findings in both were cyanosis, conjunctival burns, congestion of internal organs, especially lungs and kidneys, and failure of the blood to clot. Though not diagnostic, these findings were observed in experiments with vinyl chloride and are being published. The first patient, aged 21, also showed slight heart and kidney disease. He may have fainted before being asphyxiated. Vinyl chloride rapidly induces narcosis and, in dogs, causes cardiac abnormality. It is considered one of the least dangerous of chlorinated hydrocarbons; the MAC is 500 p.p.m.

M. Patricia Fitzsimons

PETRINATI, L., SCANSETTI, G., CAPELLARO, F., GAIDO, P. C. & RASETTI, L. Indagini sull'esposizione cronica al meta-amino-fenolo e para-nitro-etilbenzolo. **[Investigations on Chronic Exposure to Meta-Amino-Phenol and Para-Nitro-Ethyl-Benzol]** *Rass. Med. Indust.* Rome. 1959, Nov.–Dec., v. 28, No. 6, 437–46. [18 refs.] English summary.

The authors examined 18 persons, 12 of whom were exposed occupationally to risk of poisoning by meta-aminophenol, and 6 by paranitroethylbenzol, whose ages ranged from 24 to 51, and the length of time at such work from 2 to 7 years. All the patients showed definite clinical signs, with cyanosis especially of the lips a prominent symptom and almost all had hypotension when examined at the end of the shift; in 9 patients when examined after 1 day's holiday, *i.e.*, about 32 hours after ending the shift there was no material difference from the original findings. The patients mostly complained of asthenia often accompanied by headache (in both groups) and 2 showed grave and 4 marked cyanosis. Methaemoglobin levels did not differ much in the 2 kinds of risk and 1 day's holiday was sufficient to reduce the MetHb level to nearly normal but in 3 patients even though this was within normal limits, cyanosis was unchanged even after 1 week's absence.

The authors discuss carefully this persistence of cyanosis with normal MetHb levels and suggest that it is due to slowing down of the peripheral circulation due to the diminished oxygenation of the blood. The free erythrocytic and the urinary coproporphyrin behaved in the opposite manner, *i.e.*, they rose after absence from work. It thus appears that the toxic product causing the methaemoglobinaemia may also affect the enzyme systems synthesizing porphyrin.

W. K. Duncombe

PALLADE, Sulamith, LONDON, Micaela, ROVENTA, Ana & POPOVICI, Carmen. La tossicità di un concime largamente usato in agricoltura: Il superfosfato. **[Toxicity of a Largely Used Agricultural Fertilizer: Superphosphate]** *Med. d. Lavoro.* 1960, Jan., v. 51, No. 1, 49–58, 5 figs. [25 refs.] English summary (8 lines).

This report comes from the section of Industrial Hygiene of the Institute of Hygiene and Public Health of Rumania. In it the authors point out that one of the very important components of superphosphate is fluorine which sometimes during manufacture has led to severe symptoms of mottled teeth, digestive disturbances, bony changes, atrophic rhinitis, and ulceration of the nasal mucosa occasionally going on to perforation of the septum after prolonged contact. However the toxic action of the fertilizer is due not only to its F content but to the particular solubility of the F salts and to the physical state of the fertilizer. When given intragastrically to rabbits superphosphate is much more toxic than the apatite from which it is made; the urinary elimination of F gives a precise and convenient indication of the yield to F, but after administration of non-lethal doses to rabbits there was some retention of F. In these animals there appears to be some potentiating action of P on the F ions as regards toxicity and after 2 doses of superphosphate (90 mgm. F/kem.) there was a marked reduction in the activity of the blood phosphatase but not in cholinesterase though *in vitro* this was also reduced.

The authors conclude that in contrast to what has been thought hitherto superphosphate has a definite toxic action and repeated exposure of workers to small doses may have some effect. Hence the MAC should be established, bearing in mind not only the F content but also the complex composition of the product.

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