ABSTRACTS

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INDUSTRIAL TOXICOLOGY


In continuation of their previous work on the toxicity of beryllium (Arch. indus. Hyg., occup. Med., 1950, 1, 379 and 2, 25; Abstracts of World Medicine, 1951, 9, 132 and 22), the authors now describe the acute effects of the inhalation of an aerosolized aqueous solution of beryllium fluoride on dogs, cats, albino rats, albino mice, guinea-pigs, and rabbits. The experimental conditions were similar to those described in the previous papers, the concentration of beryllium fluoride was 10 mg. per c. metre in a first experiment and 1 mg. per c. metre in a second, and the period of exposure was 15 and 207 days respectively.

The effects included pulmonary changes similar to those produced by beryllium sulphate, and a macrocytic anaemia not prevented or cured by administration of vitamin B (cyanocobalamin), "letron" (a liver-stomach concentrate with a ferrous salt), or folic acid. There was also a decrease in the number of mature polymorphonuclear leucocytes and an increase in that of immature forms. A considerable amount of beryllium accumulated in the bones in a concentration exceeded only in the lungs and the pulmonary lymph nodes. Beryllium fluoride was found to be much more toxic than beryllium sulphate.

John Pemberton


There have been few reports of acute poisoning with benzene hexachloride. In the summer of 1951, 79 persons of various ages and both sexes living in the area of Carpenissi, Greece, suffered from poisoning due to the improper use of an insecticide composed of benzene hexachloride (40%) and magnesium silicate (60%), which they had sprinkled on their bedding and on the walls of their houses.

In this paper the authors describe the clinical and laboratory findings in 5 of the cases, one of which was fatal, examined in detail at the University Medical Clinic, Athens. The chief symptoms were lassitude, headache, and myalgia, followed by intestinal colic, diarrhoea, and stomatitis. Later, neurological signs appeared, including delirium, choreic and athetoid movements, tremor and convulsions, signs of upper motor neurone damage, and cerebellar signs. Optic neuritis occurred in 3 cases and one boy became blind from complete atrophy of the optic nerve. There were also electrocardiographic changes, toxic inhibition of erythrocyte maturation, an increase in blood chloride levels, a raised erythrocyte sedimentation rate, and, in the fatal case, laboratory and post-mortem evidence of liver damage. The patients were treated with "adrenal preparations", blood transfusion, vitamins B and C, and a high protein diet. Of the 79 persons affected, 18 were seriously ill and 6 died. Convalescence in most cases was very slow.

John Pemberton


Ethylene cyanohydrin is a stable material, the cyanide and hydroxyl groups being attached to different carbon atoms; it is relatively non-toxic, and there is no record of industrial intoxication. Acetone cyanohydrin on the other hand, in which the cyanide and hydroxyl groups are attached to the same carbon atom, is readily dissociated into its components, acetone and hydrogen cyanide, and it is consequently a highly toxic material. The results of animal experiments, here reported from Jefferson Medical College, Philadelphia, support the hypothesis that this toxicity is due to the release of hydrogen cyanide in vivo. The nitrite-sodium thiosulphate therapy in common use for hydrogen cyanide poisoning has proved effective in the treatment of rats poisoned with acetone cyanohydrin.

Two cases in which death was probably due to industrial intoxication by acetone cyanohydrin are described.

M. A. Dobbin Crawford

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The authors describe 4 cases of cadmium poisoning caused by an industrial process in which metallic cadmium wire was melted and blown against a rotating spindle in order to coat the latter with the metal. Only about 40% of the metal was deposited and as there was no exhaust ventilation significant amounts of cadmium contaminated the atmosphere. The 4 workers who developed symptoms of cadmium poisoning were the process operator, a maintenance welder and his assistant who worked about 10 feet (3 m.) away from the spindle, and a female clerical worker on the floor above whose desk and chair were near a ventilating duct which communicated with the workroom below in the immediate vicinity of the welding. The process began at 11.0 a.m. and was completed by 2.30 p.m., during which time about 10 lb. (4.5 kg.) of cadmium wire were used. It is believed that the operator was exposed to metallic cadmium while the other three were exposed to cadmium oxide fume generated from the airborne cadmium as it entered the welding arc. All the patients complained of cough and pain or a sense of constriction in the chest, beginning either during, or a few hours after, exposure; other symptoms were malaise, nausea, vomiting, and headache. The welder, who was most severely affected, had marked breathlessness, and this was also a feature in the operator, whose symptoms, however, were less severe.

Inflammation of the fauces and nasopharynx was noted in all 4 patients and in 3 the conjunctivae were congested as well. In chest radiographs taken after exposure there was an increase in the vascular markings in all 4 cases, compared with similar radiographs taken before exposure. The electrocardiogram showed sinus bradycardia in 3 cases and in the case of the welder a P-R interval up to 0.24 second. Vital capacity, which was reduced in each case, returned to normal during the recovery period, which varied from one week to 6 weeks.

Urinary excretion of cadmium continued for periods varying from 3 to 7 weeks, depending on the severity of the symptoms.

Although the respiratory system was affected many of the symptoms were not primarily respiratory. It is suggested that cadmium inactivates cholinesterase by combining with the sulphhydryl portion of the molecule; this would account for the bradycardia, heart block, abdominal pain, nausea, vomiting, and sense of constriction in the chest which were noted in all the cases.

After this incident exhaust ventilation was fitted to the plant and an airline helmet was provided for the operator. In spite of these precautions air samples taken in the breathing zone of the operator, but outside his helmet, revealed a concentration of cadmium in air which was about 11 times the maximum allowable (0.1 mg. per cubic metre of air); 10 feet away from the operator the concentration was a little above the maximum allowable.

W. K. S. Moore

INDUSTRIAL LUNG DISEASE


It would appear, if statistics are to be believed, that the incidence of bronchial carcinoma is higher in patients suffering from asbestosis, siderosis, and the effects of chrome inhalation than in controls, whereas the incidence in patients suffering from silicosis is no higher. Pneumoconiosis occurs in dockers handling grain and in boiler scalers, and the present authors, from the Chest Clinic, Hull, describe 26 cases of bronchial carcinoma occurring in men working in these trades. Of the 26 patients (20 of whom were grain dockers), 14 were seen in 1951-52, a period when the number of deaths from bronchial carcinoma among the general population of Hull was on the increase. There are 5,000 dockers in Hull and only half of these are exposed to dusts; among the other half, and this is probably significant, not one case of bronchial carcinoma was seen. The authors believe that exposure to dust is an aetiological factor, but they admit that the unknown factor responsible for the increased incidence in the general population also played some part. In 5 of their patients there was histological evidence of pneumoconiosis but only in one (a boiler scaler with obvious silicosis) was there radiological evidence of this condition. There were no non-smokers or heavy smokers among their patients.

Paul B. Woolley


Writing from the Cumberland Infirmary, Carlisle, the author discusses the natural history of the condition conveniently termed “farmer’s lung”; and briefly reviews the literature. The symptoms of this disorder are breathlessness, cough, cyanosis, and slight pyrexia, accompanied by the presence of widespread crepitations in the lungs, and they make their appearance soon after exposure to the dust of mouldy hay. The x-ray appearances show increased lung markings, with additional soft shadowing. Spontaneous recovery is usual in about 2 months, but there may be residual emphysema in some cases. It is reputed among farmers occasionally to have caused sudden death, and also to be the cause of broken-windedness in horses. An enquiry showed that most rural doctors are familiar with the disease, especially in north-west England where it is common, but as it usually resolves spontaneously cases are not often seen in hospital.

In the discussion of aetiology and differential diagnosis, distinction is made between farmer’s lung and asthma caused by mouldy hay dust; the former condition usually develops within 24 hours of exposure, and presents as a mild acute pulmonary oedema without cardiac distress. Rigor and vomiting are common at the onset. Recovery from the condition, both clinically and radiologically, is gradual, and may take up to 3 months. In some of the author’s cases moulds were isolated from the sputum and often corresponded with those found in the hay...

This is a general account of the pathology and histology of pneumoconiosis due to pumice dust, based on the post-mortem findings in 4 cases, which are not individually described. The middle zones of both lungs were mainly affected, the apices escaping as a rule. Two types of macroscopic appearance were found—a linear fibrosis, corresponding to the radiological "reticulation", and a massive fibrosis. Unlike other forms of silicosis, the stage of nodulation seems to be entirely absent; the author considers that this may be associated with the low content of free silica (185%) in pumice dust. This may also account for the long latent period, perhaps 20 to 30 years, before symptoms develop. Tuberculosis was not found in association with pumice-dust silicosis. Pleural fibrosis was usually present, and also marked enlargement of the mediastinal lymph nodes, but there was no evidence of bronchiectasis, although there was generalized emphysema.

Histologically, the condition did not seem to differ greatly from other forms of silicosis; widespread connective-tissue thickening interspersed with mineral deposits and without extensive cellular infiltration was characteristic. There was no focal emphysema. The bronchial mucosa was greatly swollen and infiltrated with masses of cells and phagocytes containing mineral particles; a true obliterating granulomatous bronchitis was present. Local circulatory obstruction appears to lead eventually to extensive small arterio-venous shunts, and this phenomenon may explain the pulmonary venous congestion and deficient oxygenation characteristic of silicosis. Pumice dust, containing little free silica, does not quickly set up a local irritant process, but is drained into the lymph nodes and blood stream, with involvement of other organs, including the pleura, and alteration in the blood plasma (dysproteinenaemia). Massive fibrosis is not considered to follow bronchial obstruction, but to be due to the arterio-venous lesions already mentioned.

L. G. Norman


Silicosis due to pumice dust affects organs other than the lung, the multi-visceral involvement which occurs justifying, in the authors' opinion, the designation "silicotic disease". Histological changes found post-mortem in the liver, spleen, and kidneys in 4 cases are described. The liver showed a diffuse reticulo-endothelial hyperplasia; there were widespread and abundant deposits, of a microcrystalline mineral, mainly concentrated in the histiocytes of the portal spaces and the Kupffer cells. The authors consider that the dust is carried to the liver and deposited in crystalline form throughout the process. In one case there was also a diffuse amyloidosis. There was moderate enlargement of the spleen, with hyperplasia of the reticular elements of the pulp, reduction in the number of follicles, and a moderate degree of congestion. Deposits of microcrystalline siliceous material were present, while numerous small infarcts were observed in one of the 4 cases. There was no siderosis. The kidneys showed a widespread deposit of microcrystalline material, less marked than in the liver and spleen, lying mainly in the capillaries of the glomeruli and cortical connective tissue. There was also an early generalized fibrosis, particularly of the glomerular vascular network, with congestion and cellular deposits in and around the tubules. The latter changes were probably associated with alteration in the blood protein levels, mainly due to abnormal excretion of serum albumin.

L. G. Norman

Conference on Silicosis

A conference on silicosis and occupational chest diseases, jointly sponsored by the McIntyre Research Foundation, of Toronto, Canada, and the Saranac Laboratory, of Saranac Lake, New York, has been arranged for Monday, Tuesday, and Wednesday, February 7, 8, and 9, 1955, in the Town Hall at Saranac Lake.

The papers to be presented in the five full sessions will all report on original work conducted or sponsored by either the McIntyre Research Foundation or the Saranac Laboratory. In addition there will be papers presented by guest lecturers.

Doctors, scientists, and business men concerned with the problems of occupational chest diseases in all parts of the United States, Canada, and foreign countries are invited to attend.

The business arrangements including reservations will be handled by Norman R. Sturgis, Jr., and the treasurer will be Clarence L. Wagner, both of the Trudeau-Saranac Institute staff. All communications concerning the conference should be addressed to Mr. Sturgis, Saranac Laboratory, Saranac Lake, New York.