

DONALD HUNTER

## POISONING BY THE NEWER METALS: BERYLLIUM, CADMIUM, OSMIUM AND VANADIUM\*

In the development of new alloys many metals, rare or unknown a few years ago, are now assuming commercial importance. So far no toxic effect has been discovered in workers handling cerium, gallium, indium, molybdenum, rhenium, titanium, tungsten and zirconium. On the contrary toxic effects are already known in the case of beryllium, cadmium, osmium, platinum, selenium, tellurium, uranium and vanadium. This review concerns the toxicity of four of these metals the use of which has been newly developed in industry.

### BERYLLIUM

Beryllium is a very light metal having about the same density as magnesium. It is hard and shiny and resembles steel in appearance and lustre. It occurs in nature as beryllium aluminium silicate or beryl,  $3\text{BeO}$ ,  $\text{Al}_2\text{O}_3$ ,  $6\text{SiO}_2$ . It was the appreciation of the properties of the alloys of beryllium which first increased its value. The principal alloy is beryllium-copper which is hard, corrosion-resistant, non-rusting, non-sparking and non-magnetic. It has a tensile strength as great as mild steel and six times that of copper. Metallic beryllium is employed as a deoxidizer in steel making. In the atomic energy industry pure beryllium rods are essential to the efficient working of the graphite pile. Industrial radiography has created a demand for pure beryllium foil for the windows of X-ray tubes: the metal permits the passage of long wave-length radiation.

Occupations involving exposure to beryllium compounds include the extraction of the metal from the ore, the preparation of beryllium steel, the manufacture and cutting of beryllium-copper

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alloys, and the making of beryllium-containing crystals for wireless apparatus. In addition exposure may occur to beryllium oxide used as a refractory for making crucibles and to beryl in the manufacture of electrical porcelain.

A considerable hazard exists in preparing and mixing the powders used as phosphors for coating the tubes of fluorescent strip lamps and certain tubes used for electric signs. In this industry the compounds involved are zinc beryllium manganese silicate, zinc beryllium silicate and beryllium oxide. The tubing is coated on the inside by pumping into it a liquid suspension of the phosphor, excess of which is then drained away. Dry mixing of the powder, spillage of the liquid suspension and brushing clean the ends of the tubes may produce considerable amounts of dust.

Symptoms attributable to exposure to beryllium compounds include conjunctivitis, irritation of the upper respiratory tract, dermatitis, subcutaneous granulomata and acute and chronic forms of lung disease (Machle, 1949). Conjunctivitis is frequently associated with dermatitis of the face. It follows exposure to soluble salts of beryllium in plants where the ore is processed or where the halides and acid salts are handled. On removal from contact the lesions readily heal. Exposure to the soluble salts may also give rise to irritation of the nose and throat, with epistaxis, and swelling and redness of the mucous membranes. Tracheo-bronchitis may develop, together with anorexia, dyspnoea and loss of weight. The symptoms and signs clear up completely within two or three weeks after exposure ceases, and no special treatment is required.

Dermatitis may be severe and may lead to sensitisation. The lesion is usually an oedematous papulo-vesicular eruption appearing mainly on the exposed surfaces. Healing occurs promptly on removal from contact and is hastened by local treatment of the lesion. Subcutaneous granulomata occur when beryllium compounds are introduced beneath the skin (Grier, Nash and Freiman, 1948). Such lesions may occur in persons who have cut themselves on broken fluorescent lamps. The granuloma may measure as much as 2.5 by 2.0 cm. in diameter. It is necessary to excise the granulomatous mass and to remove completely all beryllium from the lesion for fear that healing may be delayed.

The pulmonary lesions are much more serious and disabling. The first cases of the acute disease were reported from the United States of America in 1943 by van Ordstrand, Hughes and Carmody. In 1946 Hardy and Tabershaw described cases of a chronic lung disease occurring among employees of a firm manufacturing fluorescent lamps in Salem, Massachusetts. It was the resemblance between these cases and sarcoidosis which had led to the designation Salem sarcoid. In 1949 Aub and Grier reported seven cases of acute pneumonitis in metallurgical workers exposed to the dust and fume of pure beryllium metal, and beryllium oxide. The occurrence of



such cases supports the view that the beryllium ion is toxic in its own right and that the poisonous effects of soluble beryllium salts are not to be attributed to the acid radicals. In 1949 Machle reported more than 400 cases of lung disease which had occurred in beryllium workers in the United States of America. One hundred and twenty-three of these patients showed the delayed pneumonitis known also as chronic berylliosis or pulmonary granulomatosis of beryllium workers.

The symptoms of the acute disease are those of a chemical pneumonitis. They occur during employment. In the early stages cough with blood-stained sputum is accompanied by retro-sternal pain, dyspnoea, cyanosis and loss of weight. Non-productive cough may be present for several days before dyspnoea on exertion, and later at rest becomes obvious. Rapid respiration, anorexia and marked prostration follow. Usually there is no fever but cyanosis and tachycardia are present and râles are heard over both lungs. Death may occur within two weeks. The morbid anatomy of the lungs is similar to that of acute chemical pneumonitis from other causes except that there is less evidence of necrosis. The alveoli are filled with exudate composed of oedema fluid and fibrin containing macrophages. Collections of lymphocytes and plasma cells occur in the septa. In cases where death has occurred after several weeks there are large areas of fibroblastic proliferation such as are seen in organising pneumonia. More commonly resolution begins in the third week of the illness and is complete in from five weeks to five months. Serious sequelae are unusual but attacks of the acute illness have been followed by the onset of chronic berylliosis after two years (Machle, 1949). Symptoms and signs precede radiological changes in the lungs by several weeks. Increased linear markings and a granular ground glass appearance suggesting pulmonary congestion appear first. The shadows are diffuse but may be less marked at the apices and bases owing to compensatory emphysema. Consolidation may follow. As the clinical signs disappear the lung lesion becomes granular and nodular with conglomerate masses. In from one to four months these disappear leaving residual fibrosis in some cases.

The first symptoms of the chronic disease are variable and may not appear for as long as six years after the last exposure to beryllium compounds. The majority of cases have occurred in employees in the fluorescent lamp industry, but two laboratory workers and at least ten people living in the neighbourhood of factories using beryllium compounds have been affected. Less than 5 per cent. of the persons exposed have developed the disease. This suggests that some people are unduly susceptible to the effects of absorption of small amounts of beryllium. The United States Atomic Energy Commission has investigated the contamination of the atmosphere in the neighbourhood of a plant producing beryllium oxide,

beryllium-copper and beryllium metal. During the previous ten years 1600 workers have been employed. Compared to 5 known cases of the chronic disease within the factory, 10 cases with at least one death occurred in persons living within three quarters of a mile of the plant, though they had never worked there. The atmosphere concentration of beryllium three quarters of a mile away from the factory ranged from 0.01 to 0.1 microgram per cubic metre (Eisenbud and others, 1949).

Vague ill health, slight but persistent loss of weight, weakness and lack of energy, or an unusually persistent upper respiratory tract infection may herald the onset of the disease. It may be weeks or months before abnormal signs appear in the lungs. Weakness, anorexia, and progressive loss of weight which may be as much as 20 or 30 pounds in a month become prominent symptoms and persist throughout the illness. Cough with little sputum, worse in the mornings or on exertion is constantly present and bears no relationship to the severity of the disease or to the radiographic signs. Dyspnoea is extreme and may be the presenting symptom. Tachycardia, particularly when there is cyanosis, is associated with a normal or low blood-pressure. Clubbing of the fingers develops in the later stages. Cardiac failure occurs, increasing the dyspnoea and later causing orthopnoea and oedema of the extremities. Approximately one third of the patients die while one third are permanently disabled and remain in great pulmonary distress. The rest lose their symptoms, the lung changes resolving to some extent.

The earliest change in radiographs is a diffuse finely granular appearance homogeneously distributed throughout both lungs. The apices and bases are involved. Paratracheal masses and enlarged hilar shadows are seen in many cases. As the disease progresses fine nodulation appears on a granular background. Confluence of nodules may occur, but it is much less evident than in silicosis. Later there is lobular emphysema, particularly at the apices and bases. Signs of cor pulmonale may develop, and spontaneous pneumothorax, without pleural effusion, has occurred in a few cases. The X-ray appearances alone are not diagnostic.

The pathological findings in chronic berylliosis resemble closely those found in sarcoidosis. The lungs are grossly emphysematous with scattered fine nodules and diffuse interstitial fibrosis. Granulomata are formed within the alveolar spaces by organisation of exudate. They have a fibrinoid centre with peripheral fibrosis and varying degrees of mononuclear infiltration, together with numerous giant cells of the Langhans type. Similar lesions have been found in the skin and subcutaneous tissues, in hilar and axillary lymph nodes, and in the liver. Mid-zonal necrosis is also found in the liver.

Diagnosis depends upon an occupational history of significant exposure to beryllium compounds, a characteristic onset and course of the disease, X-ray findings and the presence of beryllium in urine



and tissues. Differentiation from sarcoidosis, chronic miliary tuberculosis, fungus infections, primary pulmonary fibrosis, diffuse nodular silicosis, miliary carcinomatosis, mitral stenosis and siderosis may be difficult. A diagnosis of chronic berylliosis can only be made by considering the whole picture together with evidence of significant exposure to beryllium compounds. Treatment is symptomatic. In patients severely affected oxygen relieves the dyspnoea. Penicillin-streptomycin aerosol therapy has relieved secondary bacterial infection but has no other beneficial effect. Attempts to increase the rate of elimination of beryllium with injections of 2:3-dimercaptopropanol (BAL) have not been successful, nor has any other therapeutic measure influenced the course of the disease.

In Great Britain in 1949 poisoning by beryllium and its compounds was added to the list of diseases prescribed under the National Insurance (Industrial Injuries) Act. In the manufacture of fluorescent lamps halophosphates which contain no beryllium must be used in place of the poisonous phosphors of the past. Preventive measures must be enforced in all industries where beryllium and its compounds are handled. Every effort must be made by engineering methods to keep the atmosphere concentrations at the lowest possible level. Protective clothing and adequate laundry services should be provided.

Workers at risk should be seen at regular intervals by a doctor and questioned about suggestive symptoms. They should be weighed at monthly intervals, and have the chest X-rayed at least once a year. Repeated medical and X-ray examinations must be made of all workers who have unexplained symptoms, particularly when these suggest disease of the respiratory system. Since subcutaneous granulomata have developed in persons who have cut themselves on broken lamps caution must be exercised in the disposal and salvage of burnt out fluorescent lamp tubes. The existence of the hazard of non-occupational beryllium poisoning in the neighbourhood of factories handling this substance presents a challenge not only to the guardians of health in industry but also to public health authorities in industrial communities in general. Indirectly, therefore, the splitting of the atom has given us new and grave responsibilities besides those connected with radio-active substances.

#### CADMIUM

Cadmium is extracted from zinc ores in the course of smelting. It is used in the manufacture of alloys and in making ceramics and cadmium vapour lamps. The rustproofing of iron and steel articles by the method of cadmium electro-plating has to some extent replaced that of coating with zinc. The principal industrial hazards

arise in the smelting of ores, the welding of alloys and the firing or welding of cadmium-plated metal.

In 1923 Legge reported three cases of cadmium poisoning, one of them fatal, in men in a paint factory where ingots of cadmium were melted during a period of three hours in a poorly ventilated room. All three men complained of dryness of the throat, headache and nausea. The urine was coloured brown. A necropsy on the man who died showed hyperaemia of the bronchi, gastro-intestinal tract and kidneys. In 1942 Nasatir reported a fatal case. Death occurred on the fifth day after exposure to cadmium fume caused by burning off, with an oxy-acetylene flame, deposits of metal containing a high percentage of cadmium. The symptoms consisted of a feeling of constriction of the chest, increasing dyspnoea and cough which became much worse before death.

In 1944 Spolyar and others wrote an extensive report on cases of cadmium poisoning resulting from flanging operations on cadmium-plated pipe. The resulting exposure to cadmium oxide fume resulted in five cases including one death. On the basis of the 59 cases reported up to that date the mortality rate of industrial cadmium poisoning appears to be 15 per cent. In 1945 Johnstone reported the case of a young Mexican labourer who was sent to hospital following the use of an oxy-acetylene torch on the inside walls of a furnace in which cadmium residues had been recovered from scrap metal. The patient was extremely ill with severe dyspnoea and exhaustion and he gave a history of headache, cough and pain in the chest. The temperature rose to 104° F, the pulse to 140 and the respiratory rate to 50. Patchy signs appeared in the chest and bronchopneumonia was revealed by X-rays. Cyanosis and increasing respiratory distress preceded death. At necropsy the lungs showed confluent bronchopneumonia.

In 1944 Ross described mass poisoning due to cadmium oxide fume affecting 23 workers. Finely divided cadmium dust, from a cadmium recovery chamber, became ignited owing to red hot cigarette ash carelessly dropped by one of the workers. In a few minutes the cadmium dust became incandescent and emitted clouds of cadmium oxide fume. The victims complained of irritation of the eyes, headache, vertigo, dryness of the throat, cough, constriction of the chest and weakness of the legs. Three hours later a set of delayed effects was observed. These included shivering, sweating, nausea, epigastric pain and dyspnoea. No case was fatal.

When cadmium-coated metal is heated dangerous quantities of cadmium oxide are formed and volatilised. It has therefore been suggested that cadmium-coated metal should be labelled. While this measure is effective for large pieces it is somewhat difficult to ensure that small objects so coated are labelled (Fairhall, 1946).



## OSMIUM

Osmium occurs among the platinum group of metals as the alloy osmiridium. It is the densest of all known substances being nearly three times as heavy as iron. Osmiridium is exceptionally hard and is therefore used for the tips of the gold nibs of fountain pens. It is also used for electrical contacts, as a catalyst in the preparation of synthetic ammonia, and for measuring the rapidity of explosion of gun-cotton. Osmium itself is innocuous but the volatile osmium tetroxide, commonly called osmic acid, is slowly formed on exposure of the spongy metal to air. Osmium tetroxide has a very irritating odour resembling bromine, which attacks the nose and eyes. The vapour has a sudden vigorous effect on the mucosa of the nose, pharynx and bronchi likened by workmen to the kick of a mule. If fairly high concentrations are inhaled there is a sense of momentary constriction of the chest and inability to breathe, the aftermath of which may persist for 12 hours. Deville and Debray (1859), the first chemists to make an extensive study of osmium compounds, described how the vapour of osmic acid attacked their eyes. One of them had conjunctivitis which interfered with vision for some weeks.

Raymond (1874) reported a fatal case of poisoning which occurred in a Paris workman. The vapour of osmium tetroxide had given rise to capillary bronchitis followed by broncho-pneumonia. Necropsy revealed purulent bronchitis and confluent broncho-pneumonia. Brunot (1933) exposed himself to the vapour of osmic acid and found that a smarting sensation in the eyes was followed by lachrymation and the appearance of haloes around bright lights. He then exposed rabbits until they died of broncho-pneumonia. Necropsy showed dark red consolidation with scattered purple areas in the lungs. The bronchi were filled with pus. The kidneys showed cloudy swelling. He also placed one drop of one per cent. osmium tetroxide in water into the conjunctival sacs of rabbits. After 24 hours there was oedema and swelling of the eyelids with profuse purulent discharge and a metallic brown stain on all parts of the conjunctiva. After 48 hours there were superficial ulcers in the conjunctiva and the cornea was milky and semi-opaque. Ten days after the exposure the acute stage had subsided, but there was corneal opacity and a pannus began to form which after one month slowly extended towards the centre of the cornea.

McLaughlin, Milton, and Perry (1946) recorded the effects of osmium tetroxide in seven men engaged in refining osmiridium. The hazard arose from the fine mist or spray containing osmic acid which came off from the reaction vessels when the ore containing the precious metals dissolved in aqua regia. The men exposed had smarting of the eyes with lachrymation and sometimes frontal or orbital headache. They saw haloes round lights, one man saying they were green in the centre and red round the outside. At the height

of the symptoms they were unable to read or to see the images on a cinema screen. The eyes were always normal by the next day. Only one man complained of cough and expectoration. No chronic or cumulative effects were noted. Histologists who use osmic acid as a stain for myelin and fat are familiar with headache which comes on if the bottle is left unstoppered on the bench. Because of the blackening of osmium tetroxide in contact with oil and fat an aqueous solution of osmic acid was at one time used for taking finger prints. The method was given up because it was soon realised that its use in contact with the skin caused dermatitis.

Preventive treatment of osmic acid poisoning consists of the proper ventilation of reaction vessels and other apparatus giving off osmium tetroxide. Clearly the vapour of this substance must not be allowed to enter the atmosphere of the workroom.

## VANADIUM

The ores of vanadium are patronite, vanadium sulphide, carnottite, potassium uranyl vanadate, and vanadinite, a lead vanadium oxide. In countries where the ores are not found petroleum residues form the main source of supply. More than 20 tons of vanadium pentoxide are recovered annually from soot which collects in the boilers and smoke stacks of ships burning Venezuelan and Mexican fuel oil. About 95 per cent. of the world's supply of vanadium is consumed in the manufacture of special alloy steels. Vanadium pentoxide is used as an oxidising catalyst in the conversion of naphthalene to phthalic anhydride and is replacing platinised asbestos in the contact process for the manufacture of sulphuric acid.

In 1911 Dutton described vanadium poisoning in a plant where vanadium ore was ground. The clinical picture as he recorded it has never been confirmed. It included loss of appetite, anaemia, emaciation, diarrhoea, dimness of vision, melancholia, dry paroxysmal cough, haemoptysis and albuminuria. Vanadium was found in the urine, faeces and saliva. He thought that pulmonary tuberculosis was apt to supervene upon the bronchitis caused by vanadium. In 1938 Molfino showed that vanadium anhydride has a strongly irritant action on the respiratory tract of animals. It is absorbed, and excreted in the urine. In 1939 Symanski made a study of 19 cases which occurred in a metallurgical works in Germany. He claimed that it is only the pentoxide of vanadium which gives rise to a toxic dust. He observed severe conjunctivitis often with suppuration. There was chronic bronchitis, with a feeling of constriction in the chest, persistent cough, profuse expectoration, and sometimes haemoptysis.

Balestra and Molfino (1942) reported respiratory symptoms in labourers working over petroleum ash containing vanadium. Wyers (1946) described ten cases of respiratory disease among ninety workers making vanadium pentoxide. These men had complained of



shortness of breath, pain in the chest, palpitation on exertion and paroxysmal cough rarely with haemoptysis. On examination they showed greenish-black discolouration of the tongue, tremor of the fingers and arms and rhonchi throughout both lungs. Three of them developed pneumonia of which one died. Four showed dust reticulation in X-rays of the lungs. Wyers concluded that colds and pneumonia were more frequent than in the general population but was not able to produce statistical evidence in support of this.

In order to prevent industrial poisoning by vanadium compounds mechanisation and enclosure of all dusty processes must be strictly enforced.

*London Hospital,  
London, England.*

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## SADRŽAJ

### OTROVANJE NOVIJIM METALIMA: BERILIJEM, KADMIJEM, OSMIJEM I VANADIJEM\*

Razvijanjem novih legura mnogi metali, koji su do pred nekoliko godina bili rijetki ili nepoznati, postaju važni za trgovinu. Dosada se nije ustanovio toksični učinak kod radnika, koji rukuju cerijem, galijem, indijem, molibdenom, renijem, titanijem, volframom i cirkonijem. Naprotiv, znade se za otrovnost berilija, kadmija, osmija, platine, selena, telura, urana i vanadija. Ovaj prikaz obrađuje toksikologiju ovih četiriju metala, koji se odnedavna upotrebljavaju u industriji.

Berilij kao metal upotrebljava se za desoksidaciju u proizvodnji čelika. Na široku upotrebu nailazi u industriji u smjesi s ostalim metalima. Mnogo se upotrebljava u proizvodnji fluorescentnih svjetiljaka. U industriji svjetiljaka upotrebljavaju se ovi spojevi berilija: cink-berilij-mangan-silikat, cink-berilij silikat i berilijev oksid. Kod ljudi, koji su izloženi djelovanju berilijevih legura, opažen je konjunktivitis, iritacija gornjeg respiratornog trakta, dermatitis, potkožni granulomi i akutna i kronična oboljenja pluća. Simptomi akutnog oboljenja pluća su isti kao i kod kemijskog pneumonitisa.

Većina slučajeva kroničnog oboljenja pluća javlja se kod radnika u industriji fluorescentnih svjetiljaka. Oboljenje počinje ovim znakovima: neodređena boležljivost, stalan gubitak na težini, opća slabost, a katkad se javlja i uporna infekcija gornjeg respiratornog trakta. Prije pojave abnormalnih znakova na plućima mogu proći nedjelje i mjeseci. Najranije promjene u rentgenogramu pluća su difuzne fine granulacije, koje su homogeno raspoređene na obje strane pluća. Kad oboljenje napreduje, pojavljuju se u pozadini fine sitne nodulacije. Noduli se spajaju, ali je to mnogo manje vidljivo nego kod silikoze. Kasnije se razvija emfizem. Dijagnoza se može postaviti samo na osnovu rentgenskog nalaza. Prikazana je terapija i prevencija oboljenja uzrokovanih berilijem.

Kadmij se upotrebljava kod proizvodnje amalgama i za izradu keramičkog materijala i svjetiljaka s kadmijevim parama. Opisani su simptomi oboljenja uzrokovanih kadmijem. Kod smrtnih slučajeva utvrđena je bronhopneumonija, a kod ostalih javljali su se simptomi dispnoe i kašlja. Radnici su se žalili na glavobolju i bol u grudima.

Osmij se upotrebljava u smjesi s iridijem za vrškove naliv-pera, za električne kontakte, kao katalizator i t. d. Sam osmij je neškodljiv, ali je vrlo štetan osmijev tetroksid. Pare osmijeva tetroksida djeluju na sluznice nosa, ždrijela i dušnika. Ako se udišu velike koncentracije, dolazi do osjećaja časovitog stezanja u grudima i nesposobnosti disanja. Opisana su oštećenja oka.

Vanadij se upotrebljava za proizvodnju specijalnih legura. Vanadijev pentoksid služi kao oksidativni katalizator. Vanadij oštećuje pluća. Otrovani se žale na kratkoću daha, bol u grudima, lupanje srca kod napora i na kašalj. Tipična je zelenkasto-crna boja jezika. Od ostalih simptoma spominje se drhtanje prstiju i ruku.

Opisane su preventivne mjere za sprečavanje otrovanja novim metalima.

Londoniska bolnica,  
London, Engleska

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