ABSTRACTS

TOXICOLOGY


This report by the Medical Research Council of the clinical assessment of BAL in arsenical dermatitis confirms work already reported in Britain and U.S.A. The clinical material consisted of 44 cases, of which 41 were of the acute exfoliative type; 5% BAL in arachis oil and benzyl benzoate sterilized by heat and sealed over nitrogen was given in doses of 1-5 mg. per kilo intramuscularly into thigh or gluteal muscles. An average course was as follows: first day, 4 injections of 2 ml at 4-hourly intervals; second, third, and fourth days, 2 ml, twice daily; fifth and sixth days, 2 ml daily. Thirty-one of the 44 cases were benefited, 23 (52%) strikingly so. The average time of healing was 21 days, and subsidence of skin oedema was an early sign. Clinical relapse was sometimes seen; this responded to a second course.

The minimum dose which causes toxic signs (these appear trivial) lies between 3 and 5 mg. per kilo. In America doses 4-hourly of 3 mg. per kilo have been given on the first day to severe cases. It is considered on the basis of animal experiments, that hepatic damage but not renal damage increases toxicity, but does not contraindicate the use of both. Post-arachinamine hepatitis of infective origin is outside the scope of action of the drug. Local abscess formation at the site of injection is considered to arise from skin infection in cases of exfoliative dermatitis of long standing. Reference is made to favourable reports, published elsewhere, on the use of BAL in cases of arsenical encephalopathy and granulocytopenia. The successes reported in acute mercury poisoning and in gold intoxication are also noted. There is a valuable bibliography.

G. Brownlee.


This substance, a soluble gluscoside of BAL, was prepared from allyl glucoside by acetylation to tetra-acetyllalyl glucoside, which was 2-brominated, treated with potassium thiocacetate, and saponified. The product was not chemically pure but had less than 1% of the toxicity of BAL and prevented death in 100% of mature rabbits when given 6-5 hours after three median lethal doses of lewisite. In young rabbits (600 g.) earlier treatment was necessary. A number of other BAL derivatives were prepared, but the glucoseide proved to be the least toxic and most effective. Throughout the series toxicity decreases with increasing polarity of the molecule.

V. J. Woolley.


Lewisite (ß-chlorovinylidichloroarsine) or phenyl-dichloroarsine was applied as a drop to the dorsal skin of a rabbit and left for varying periods up to 168 hours. Some of the latter but none of the lewisite disappeared by evaporation. Immediately on application part of the arsenic became fixed in the skin that it could not be extracted by acetone, but local application of BAL made all the arsenic extractable. This effect did not follow intravenous injection of BAL, nor did BAL affect the rate at which absorption from the skin into the circulation occurred.

V. J. Woolley.


Animals were exposed, in a chamber with a capacity of 555 litres, to fume generated by striking an arc between a carbon projector rod and a lump of metallic cadmium. Electron microscope photographs showed the fume to be made up of particles of from 0.3 to 0.5 μ in diameter. The LD 50, measured in minute-mg. per cubic metre, was 500 in rats, probably about the same in mice, 2,500 in rabbits, 3,500 in guinea-pigs, 4,000 in dogs, and 15,000 in monkeys. The amount of cadmium-oxide fume retained by the lungs of the animals killed was found to be remarkably constant, and from calculations of the lung ventilation rate it was estimated to average 11% of that inhaled. From analyses made by previous investigators of the lung content of cadmium oxide in 2 men who died as the result of an industrial exposure, it was calculated that the lethal dose for man is 2,500 minute-mg. per cubic metre.

H. M. Vernon.


In order to determine the lethal dose of cadmium oxide for man, experiments were made in a Canadian factory under conditions of exposure resembling those which, in 1938, resulted in 2 deaths. They were made in the
In a number of the dogs, 2,3-dimercaptopropanol (BAL) was administered immediately after exposure or 60 minutes later. The results indicated that the more effective treatment is the largest safe dose of BAL that can be administered in the shortest possible period after exposure to cadmium inhalation. Continued treatment after the first day is probably of no value; neither is the application of a 5% BAL ointment inunction.

The early pathological changes consisted of necrosis of the lining epithelium of the lungs, particularly of the bronchioles, and of the underlying smooth muscle. Later, a marked inflammatory reaction appeared in the bronchi and bronchioles; bacterial pneumonia was only occasionally observed.


The 20 workers employed in a cadmium smelter were examined repeatedly over a period of 3 months. They had been exposed for a period of 6 months to 22 years, but had no illnesses which the plant physician believed to be due to cadmium. Air samples taken at the breathing level in the eleven operations to which these men were likely to be exposed showed in the cadmium-sulphide packaging room the average atmospheric concentration was 31 mg. of Cd per cubic metre of air, while in two other operations it was 19 and 17 mg. respectively. Many of the men were constantly covered with cadmium-sulphide or oxide powder, and they seldom wore the respirators provided or washed before eating. The only definite clinical symptom was the appearance of a yellow ring at the base of the teeth in several of the men. The ring began at the gingival margin and extended about half-way down the tooth, the colour varying from light yellow to golden brown. The ring did not appear in workers with less than 2 years’ exposure. Complaints of constipation, weakness, and headache were made by several of the men; 2 reported poor appetites, and 2 showed loss of weight, but none of these symptoms was incapacitating. The blood and urine always contained cadmium, the average per 100 g. of blood ranging from 0.010 to 0.065 mg., while the urine contained from 0.010 to 0.139 mg. per litre. In neither case did the figures show any relation to the degree of exposure. There was no anemia. The red blood cells were increased, but leucocytes were within normal limits.

Most of the deleterious effects of cadmium hitherto reported have been the result of relatively brief exposures to high concentrations of the substance, but it is probable that in chronic exposures there is a rapid elimination of the cadmium from the body, and that there is no cumulative effect. The author thinks his data suggest that cadmium may not be so great an industrial hazard as has previously been reported. [It will be noted that this conclusion differs greatly from that arrived at by Paterson (see abstract in opposite column).] H. M. Vernon.


In a factory engaged in the manufacture of cadmium-faced bearings the cleaned cylinders were submerged in a well-ventilated cadmium pot at a temperature of 800° to 850° F. (428° to 456°C.), and were given other treatment with molten cadmium. Air samples taken over a period of years contained an average of 0.11 to 0.34 mg.
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of cadmium per cubic metre of air, with a maximum of 0.68 mg. The average amount present was probably about 0.1 mg. per cubic metre. Clinical records of 5 of the 8 men engaged in the work for periods of 4 to 8 years revealed complaints of fatigue, dental trouble, gastrointestinal symptoms, and, on damp days, respiratory symptoms. The blood of 2 of the men showed only 70% of haemoglobin on the Sahli scale. The mean amounts of cadmium in the urine varied from 0.1 to 0.05 mg. per litre.

H. M. Vernon.


Selenium is used in a variety of industrial operations, and in certain combinations is capable of producing acute and chronic effects. Animal experiments have demonstrated that ingestion or inhalation of selenium compounds produce damage in the liver and other organs. Reported cases of industrial or occupational selenium poisoning are not numerous, although it is to be expected that many have occurred. Cases of occupational dermatitis, and also of irritation of the upper respiratory tract and gastrointestinal disturbances, have been ascribed to selenium poisoning. The author gives an historical review of the toxicological effects of selenium, with numerous references. Selenium may be ingested, inhaled, or absorbed through the skin. Excretion may be through the lungs or in the urine and the feces.

A report is given on 5 cases of selenium intoxication arising from an industrial hazard; it indicates the need for thorough investigation in considering claims of obscure causation in workers. The patients were exposed to less than 0.2 part per million of hydrogen selenide. Predominating symptoms were nausea, vomiting, metallic taste in the mouth, alliaceous breath odour, dizziness, and extreme fatigue and weakness. Urinary excretion levels were indicative of absorption of selenium and decreased after exposure ended. Present evidence does not indicate any satisfactory method of deselenization.

Prevention of industrial selenium is dependent on an intelligent appreciation of the inherent hazards attached to the use of selenium products, and on proper engineering and medical control. It is suggested that all workers potentially exposed be examined at intervals, after a preliminary replacement examination. Careful attention should be given to complaints referable to the respiratory tract, the gastro-intestinal tract, and the central nervous system. Routine periodic examination of urine for evidence of absorption of selenium should be made.

A. Thelwall Jones.


Reference is made to the work of Madison, of Missouri, who in 1856 described cases of selenium poisoning in man and animals occurring in that State. The author considers that the poison takes particular effect on the endocrines. It is pointed out that the discovery of selenium in the urine is not, in itself, evidence of poisoning. It may occur in workers handling such materials as lead, zinc, pyrites, lime, and cement. As much as 5 mg. has been found daily in the urine for a week without the occurrence of toxic symptoms. Inorganic compounds of selenium are rapidly eliminated, so that in a week from 80 to 90% may pass out in the urine and small quantities in the feces. Organic selenium compounds behave very differently and are retained much longer.

They are found in fairly large amount in the liver, spleen, kidneys, pancreas, heart, and muscles. Smaller concentrations are found in the nervous system and the bones. Selenium may be excreted for 6 months after a period of exposure.

The author gave guinea-pigs sodium selenide by the mouth, 5 mg. daily for 20 days and then 12.5 mg. daily for 20 days. Those which survived were killed 40 days later. At necropsy the changes in the ovaries and suprarenals were marked. The suprarenal cortex was particularly affected, but in the medulla also degenerative changes were seen, which sometimes took the form of large hemorrhagic areas. The process of ovulation was disturbed. The pars anterior of the pituitary was found to contain scattered edematous or hemorrhagic areas. The thyroid was shrunken but otherwise little altered. In the pancreas changes of hypertrophic type were seen, and it appeared that the islets had been exposed to constant stimulation. The changes described were noted in all the animals examined. The author suggests that the changes in the pituitary explain many of the findings in other organs by reason of the widespread endocrine disturbance caused. He emphasizes that a high urinary output may not indicate safety since the organic compounds are more harmful, and are excreted much more slowly, than the inorganic ones.

G. C. Pether.


The effect of subcutaneous injections of nickel sulphate in guinea-pigs was investigated. A dose of 10 mg. was given daily and was well tolerated, no animal dying from poisoning. Various groups of animals were killed and blood examinations carried out at intervals. No changes were noted in the chloride or calcium content of the blood. Blood sugar rose after the injections, and blood urea slightly. There was a fall in haemoglobin and an increase in red cells after fairly lengthy periods, and a slight leucocytosis. In the bone marrow there was an increase in basophil erythroblasts, metamyelocytes, and polymorphonuclear cells. At necropsy no characteristic lesions were found but the metal was identified in the brain, heart, lungs, liver, spleen, kidneys, suprarenals, and pancreas. The metal is eliminated only by the kidneys up to the fourth day. After this, it is also eliminated by the bowel but renal elimination remains greater than fecal.

G. C. Pether.


Many attempts have been made by different means to fix the fumes of mercury, but until recently no satisfactory agents have been described. In 1933 Alexeef proposed the use of active manganese dioxide as this quickly forms an amalgam with mercury. It is suggested that the active dioxide could be employed in order to protect the respiratory organs and to free contaminated spaces of fumes. In a suitable base it could be utilized to fill cracks through which mercury might escape. It was also found that non-activated manganese dioxide in various strengths of hydrochloric acid might be used with advantage. It forms an oxidized film on the surface of the mercury and as a result there is a rapid loss of surface tension. Hydrochloric acid in suitable amount and concentration assists this reaction.

G. C. Pether.

During the past 10 years (1936–45) frequent improvements have been made in working conditions to control lead exposure in a factory devoted to battery production, and as a result only 4 cases of lead intoxication have been reported among the 300 men exposed to risk. New employees are subjected to a thorough investigation, including chest radiography, blood count, and physical examination. The men are carefully instructed about personal hygiene, and potential hazards are pointed out. At monthly intervals the lead-dust in the factory air is determined at 35 to 50 points. All workers in potentially hazardous positions report periodically to the hospital, and blood checks are made at intervals of 14 to 60 days. Averages taken of the 115 men who worked more or less continuously over the 10-year period showed that there was close correlation between the stippled cell count and the percentage of haemoglobin, but there was no corresponding relation between these values and the erythrocyte counts. The curves for the concentration of lead-dust in the air showed the improvement produced after 1942 by the installation of new exhaust systems. It is considered that the maintenance of an average of not more than 5 mg. of lead per 10 cubic metres of air affords adequate control. Analyses of urine samples indicated that 0 to 150 µg. of lead per litre is the normal range for men working under controlled conditions. The range of 150 to 250 µg. is abnormal, and the workers involved should be kept under observation until the cause is identified and corrected. Whenever excretion exceeds 250 µg. steps should be taken immediately to reduce the lead intake—for example, by transferring the workers to a position of minimum exposure.


A number of known methods to determine the concentration of pyridine in the air (Schütze's titration method, colorimetric methods with chloramine, β-naphthyamine, 2,4-dinitrochlorobenzene, benzidine) were tested. The best results were obtained with a slight modification of the benzidine method, which permits the detection of 5 µg. of pyridine per litre of air and even less. Urine examination of the affected persons did not show the presence of pyridine.


Symptoms of mild chronic intoxication with pyridine vapour—intensive headache, transient dizziness, irritability, and sleeplessness—occurred in 7 workers in a chemical factory. Occasionally digestive troubles, especially nausea and vomiting, impaired memory, and lack of concentration were observed. On clinical examination no abnormalities were detected except a marked lymphocytosis in 2 of the patients. The author considers that through faulty ventilation a mild chronic intoxication with pyridine vapour and ammonia occurred in the factory over a period of 2 to 3 years. Pyridine was demonstrated in the aspirated air in concentrations of 20 to 40 µg. per litre; these should be regarded as toxic.


Carbitol is used extensively in cosmetics and industrial products, and is generally assumed to be innocuous. It is easily absorbed by the skin, and a few isolated observations by various workers suggest that it may have a toxic action. Rats failed to show ill-effects after the application of 8 to 16 ml. of carbitol per kilo daily for 2 months, so 286 rabbits were tested.

The pathological changes observed after repeated applications of the higher aldehydes of carbitol (0.16 ml. per kilo and upwards) were microscopic and functional lesions of the kidney, but smaller doses caused only functional impairment, and 0.04 ml. per kilo caused no demonstrable injuries. The results appear to indicate that for human beings the upper limit of safe daily use of the relatively uncontrolled use of products containing high concentrations of carbitol, but a daily dose of 0.04 ml. per kilo should be reasonably safe.

H. M. Vernon.


Two cases of papilloma of the renal pelvis in dye workers are reported in detail. In the first case the patient had been under treatment for a long time for multiple and massive bladder papillomata, and he was later found to have a papillary growth of the right kidney. This renal neoplasm was regarded as being a separate primary growth. In the second case the patient had what were regarded as three primary growths, a small papilloma of the bladder, a papillary growth in an atrophic left kidney, and a massive one on the right-hand side. Both renal neoplasms apparently developed after the bladder growth had been destroyed. The author states that the question of the occurrence of papillomatous disease of the upper urinary tract in dye workers has often been raised, but these cases were the first and only ones seen by him. A search of the literature has shown that only 6 other examples of pelvic papillomata in dye workers have been recorded.

W. G. Gill.


A method for the determination of halogenated hydrocarbons in blood was published by Moran (J. Industr. Hyg., 1943, 25, 243), but his method is not suitable for tissue analysis. In the procedure described here the volatile halide is removed from an oil-water mixture by combined steam distillation and aspiration, and is decomposed in a heated platinum tube. The resultant inorganic halide is absorbed in an alkaline arsenious oxide solution, and estimated by a standard procedure. The apparatus consists of a 250-ml. distillation flask with a side-arm extending to within 0.5 cm. of the bottom of the flask. It is heated by a rheostatically controlled oil bath, and the vapour and steam pass through a reflux condenser to the combustion tube of platinum, which is 30 cm. in length and has a bore of 2 mm. packed with fine platinum gauze. It leads to the halogen absorption
tube of 35-ml capacity, connected with a suction pump. Blood (oxalated) and tissue samples are stored under 15 ml. of mineral oil in stoppered tubes, which are well shaken. One hour from the time the mixture reaches boiling point is allowed for removal of the halogenated hydrocarbon from the sample. Analyses are recorded of various halogenated hydrocarbons, having boiling points ranging from 40° to 146° C., and they show an average error of only ±1.8%.

H. M. Vernon.


The present report describes improved methods for obtaining vapour samples when studying toxic effects. The tests were carried out with benzene and xylidine, as their concentration can be readily estimated by ultraviolet spectroscopy. The Beckman quartz spectrophotometer was used, and the concentrations were determined by dissolving the vapour samples in iso-octane and measuring the optical density of a 1-cm. length of solution. The most sensitive band for benzene is at 255 µ, and for xylidine 289 µ. The apparatus for producing the vapours consisted of a 30-litre pyrex bell jar connected with a compressed-air supply equipped with an oil trap and calcium chloride drying tubes to remove moisture. The glass bubbler containing the hydrocarbon to be vaporized was kept at constant temperature, and was weighed before and after the test. In another method of sampling the vapours were drawn directly from the chamber into evacuated Shepard flasks; in a third method, which was more suitable for low concentrations, two all-glass midget impingers were used in series. They contained iso-octane, and a measured volume of the chamber atmosphere was drawn through them at a rate of 0.2 litre per minute. The concentrations employed for the study of benzene ranged from 57 parts per million (p.p.m.) to 1,140 p.p.m., and of xylidine, from 50 to 100 p.p.m. The tables of test results show that the average loss of samples due to handling was less than 10%.

H. M. Vernon.


Until recently control of chromic acid mist in the workers' breathing zone above chromium-plating tanks used in the electroplating industry was effected largely by means of local exhaust ventilation, but this, while effective, results in a considerable loss of chromic acid through the exhaust system. A newer practice is the surface application of plastic chips. The essential requirements for material used in this way are: (1) the solid particles must be able to float on the surface of the chromium-plating bath; (2) the particles must not be affected by such physical conditions of heat and electricity as exist in chromium-plating tanks; (3) the material must be inert chemically to all substances encountered in the plating solution. Many plastics fulfil these requirements.

A field test conducted at the Naval Air Station, Pensacola, Florida, to determine the effectiveness of one type of plastic-chip covering is described. With a 60-minute sample, taken 8 in. above the liquid surface and 4 in. from the anode, with an exhaust ventilation rate of 170 cubic feet of air per square foot (5 m² per 1,000 cm²) of surface area, the average atmospheric concentration of chromic acid obtained with the use of spray-reducer chips was found to be 0.2 to 0.5 mg. per 10 cubic metres of air. Under exactly the same conditions but without spray-reducer chips, 45 to 50 mg. of chromic acid per 10 cubic metres of air was found. A 15-minute sample, taken under the original conditions but without the provision of exhaust ventilation, indicated an atmospheric concentration of chromic acid in the operator's breathing area ranging from 160 to 170 mg. per 10 cubic metres. Comparative results of tests carried out by the Udylite Corporation show an inverse relation between the blanket thickness of the chips and the atmospheric concentration of the chromic acid.

All tests made on electroplating plants in St. Louis under conditions where plastic chips were used as well as exhaust ventilation showed no chromic acid concentration whatever in the workers' breathing zone. Among a limited number of tests conducted at plants operating under conditions where plastic chips were used in the absence of local exhaust ventilation, was one plant where the worker's exposure to chromic acid mist varied from 19 to 30 mg. per 10 cubic metres. Such figures offer strong support to the contention that local exhaust ventilation should be provided for chromium tanks whether plastic chips are used or not.

A. J. Amor.

Industrial Physiology


The evidence for a statement made in a leading article in the British Medical Journal (1947, 1, 727) "that the inhalation of textile dusts is a steady source of death from nephritis and a group of conditions whose common factor is hyperpiesia" is examined. From critical review of the original figures—the Registrar-General's Decennial Supplement for 1931—it is found that these deaths from so-called nephritis in textile workers are in all probability due to hypertension, and can be as easily explained by natural variations in the genetic constitution of an inbred population as they can by environmental influences.

L. H. Worth.


Hexaethyltetraphosphate and tetraethylpyrophosphate, used by the Germans as insecticides, have physiological and toxic effects. Hexaethyltetraphosphate, largely hydrolysed in water into simpler bodies, in doses of 1 to 3 mg. per kilo, causes hyperexcitability, twitching, salivation, and convulsions. Progressive sensitization to acetylcholine was seen in isolated frog rectus, rabbit heart, and gut. The potentiation caused by concentrations of 2 x 10⁻⁶ to 2 x 10⁻⁶ M. lasted for 6 hours despite repeated washing out. Rhythmic contractions of the gut were increased, also tone (effect initially annulled by atropine). Tetraethylpyrophosphate had similar effects, but they were more transient. In the cat anaesthetized with chloralose gradual increase of dose from 0.25 to 2 mg. per kilo caused bradycardia, lowering of blood pressure, increased salivation, bronchial secretion, convulsions, defecation, and death. The effect of peripheral vagus stimulation was only increased by 10 to 40%, but the recovery period was much prolonged. With large doses the vagus response was lost. In the cat under the influence of chloralose and atropine intravenous doses of 0.5 mg. per kilo had a well-marked
potentiating action on the response of muscles to stimulation of the popliteal nerve. Electrically a repetitive response developed with slightly asynchronous discharge. The last-mentioned dose had very little effect on blood pressure. Initial potentiation of spinal reflexes, increase of extensor tone, and convulsions were observed. Similar reactions were produced by intrathecal injection, with increased "repetitive knee-jerk," crossed extensor reflex, and mild convulsions. Violent convulsions occurred with a dose of 4 mg. per kilo and increased knee-jerk, suggesting slight absorption into the circulation. D. T. Barry.


The chief objective in the treatment of decompression sickness is the rapid restoration of normal blood supply by immediate recompression. This reduces the size of the gas emboli and of bubbles in the tissues in proportion to the pressure applied. The treatment developed by workers at the Experimental Diving Unit and the Naval Medical Research Institute, Washington, involved three basic principles: (a) the limitation of the maximal pressure applied during recompression to 65 lb. per square in., and the maintenance of this pressure for 30 to 120 minutes; (b) a prolonged recompression for periods of 12 to 24 hours at pressure levels equivalent to depths between 30 and 60 ft.; and (c) the inhalation of oxygen at pressures equivalent to 60 ft. or less in order to promote the more rapid elimination of nitrogen. This treatment was applied to 113 divers who developed decompression sickness, and in 109 of them the treatment was successful. In the other 4 symptoms recurred after treatment. Of the 75 patients given oxygen, 4 developed mild symptoms of oxygen toxicity which were relieved promptly when the oxygen was discontinued.

The divers tested had worked for 1 hour under water in a pressure diving tank at a pressure equivalent to a depth of 130 ft. After decompression according to standard Navy tables they seldom developed decompression sickness, but among those who did 107 had localized pain, 10 numbness, 10 muscular weakness, 8 visual disturbances, 4 vertigo, and 2 aphasia; there were 2 cases of unconsciousness. In about a third of the men the symptoms appeared within an hour after decompression, in half of them within 1 to 6 hours, and in the remainder, at any time up to 24 hours after decompression. H. M. Vernon.


The authors review 500 consecutive cases of impaired consciousness among pilots and aircrew. The definition includes complete loss of consciousness, with or without convulsions, sudden disturbances of awareness, amnesia, confusion, and transient giddiness. The cases are classified as neurogenic, cardiovascular, and emotional. In practice these three groups overlap, and it may be difficult to decide whether loss of consciousness is caused by epilepsy, syncope, or psychogenic factors. The neurogenic group formed just under a quarter of the cases; epilepsy was the causative factor in about half of these. The part played by fear in initiating an epileptic attack is stressed. The cardiovascular group comprised rather more than a quarter of the total. Cardiovascular inefficiency was the major causative factor, either alone or combined with contributory factors, such as infection or fatigue. The author discusses the significance of hypotension in aviators. In the Royal Air Force the lower limits of normality for systolic and diastolic blood pressures are taken as 110 and 70 mm. Hg respectively. It is considered that a single finding of hypotension according to these standards is comparatively frequent in young adults; although this condition is usually compatible with health, it indicates an increased liability to fainting. The remaining cases, which formed about a half of the total, were classified as emotional. In this group loss of consciousness was associated with anxiety and inability to stand up to the stress of flying. A combination of fatigue and anxiety was frequently present. Other aetiological factors encountered in the emotional group were panic state, hysteria, and simulation. The author considers that the history of the patient is the most important single factor in examination, and that flying should be prohibited at once for those who have had two or more recent attacks of loss of consciousness. [The unique opportunity of which the author made use in the presentation of this classic and the lucidity of style and logical presentation will make this article a classic.] R. Winfield.

INDUSTRIAL LUNG DISEASE


The solubility of quartz dust in buffered Ringer solution increases as the particle size gets smaller; whatever the size of the particles, however, there is a progressive diminution in solubility after repeated treatment with the solvent. With particles down to 1 μ there seems to be an increased rapidity of fibrous-tissue formation in the lungs of rats with diminution in particle size, although the experiments detailed here are not very conclusive. No differences in solubility or pathogenicity of racemic, dextrorotatory, or levorotatory quartz were detected. A sample of silica of extremely fine particle size was very soluble in Ringer solution, and killed rats rapidly when injected in large amounts; smaller quantities did not produce fibrosis after injection into the trachea. The addition of 1% of any of four different brands of alumina to the samples of quartz lowered the solubility of the latter to a considerable degree. Intratracheal injection of such mixtures was, however, followed by fibrosis in the lungs of rats. The silica in a sample of powdered sandstone containing 70% of quartz was only slightly soluble in buffered Ringer solution; this material, however, was almost as effective as pure quartz in producing fibrosis in the lungs of rats. On the other hand, a powdered shale containing 8% of quartz had a silica solubility between those of the sandstone and pure quartz, but produced very little fibrosis in experimental animals. Olivine, which is a magnesium silicate, had a medium silica solubility in small-particle size, and produced only slight fibrosis in the lungs of rats. It may be that siliceous dusts release soluble silica in a different manner in cells and in the test-tube, and that some dusts liberate the silicic acid in a different and more noxious state than do others. The silica released from particles of the pathogenic siliceous dusts has special...

The author observes that a diagnosis of silicosis may be confirmed only after death. If there is delay in performing necropsy ordinary methods may be useless and chemical analysis of the lungs may be required. It is not difficult to estimate the total silicatic present; gravimetric methods being superior to colorimetric ones. The solution of the problem depends more on the interpretation of the results than on the technique. If the total silicate is less than 5% of the ash, silicosis may be excluded. Lungs may still be efficient and yet contain considerable amounts of silicate, especially in the aged. Thus, in normal persons who have not been exposed to any occupational risk up to 15% of silicate in the lung ash may be found. In those known to have silicosis up to 65% has been found. It is thought that a figure above 15% indicates some morbid change.

Since so many silica-containing dusts enter the lungs, but are not derived from any material to which the individual is specifically exposed in his work, the interpretation of figures may present difficulty from the legal standpoint. Old scarred tuberculous foci in the lungs tend to collect around them any dusty material. If such a lung is ashed it may yield enough silicate to suggest the presence of silicosis, but in fact the greater part may have derived from the local lesion and there may be no question of any occupational hazard. Estimation of free silica is often more informative. This is more the task of the mineralogist than of the chemist. Expensive and specially designed x-ray plant may be required. By microanalytical methods it is possible to determine the presence of free silica even in putrefied tissue, but no exact quantitative estimations are feasible. Persons who have not been exposed to a silica hazard rarely exhibit such material in their lungs. Those handling quartz invariably have free silica particles in their lungs, yet they may not be suffering from silicosis.

It is concluded that if the total silicate in the lung ash is under 10%, and there are no quartz particles a diagnosis of silicosis may be rejected. If the total silicate is higher than this and many quartz particles are seen the findings suggest, but do not confirm, the diagnosis.

G. C. Pether.


Investigations were carried out in the Clinic for Industrial Diseases in Milan to determine the occupational hazards of glass workers. Among 265 cases investigated, evidence of silicosis was found in 5-7%; in workers handling refractory materials (potters, casters, and furnace makers) there was an incidence of 48%, some having associated tuberculosis. The morbid changes observed resembled those seen in potters suffering from silicosis, fibrosis being patchy but often confluent and widespread with marked pleural involvement. It would appear that Schramm is wrong in his contention that tuberculosis appears independently of silicosis in the glass industry. The development of silicosis is progressive; out of 95 workers in the furnaces only 35 were free from disease and most of these had worked only a short time. In those affected there was clinical evidence of pulmonary lesions after about 10 years’ exposure. Silicosis in glass workers has many points of similarity with the disease as observed in ceramic workers.

G. C. Pether.


Obliterative vascular changes produced in the lungs by silicosis are the acknowledged cause of the secondary effects on the pulmonary circulation in this disease. In order to examine their precise nature 43 unselected and consecutive cases of silicosis, uncomplicated by gross pulmonary tuberculosis, were studied at necropsy, together with an equal control series. On an average, 40 pulmonary vessels of every possible calibre were sectioned from each case, and the degree of silicosis graded into discrete nodular and massive conglomerate nodular types. Microscopy, with Weigert’s elastic tissue, proved to be more informative than naked-eye inspection. In discrete nodular disease the small vessels within the nodules were most affected (arterioles, venules, and capillaries only), but in massive conglomerate nodular silicosis vessels of all sizes were rapidly and extensively involved. These vascular changes resulted either from direct encroachment on the vessel wall by expanding nodules or nodular masses or from infiltration by the hyperemic cellular dust- and pigment-bearing granulation tissue along the nodule margins. Considerable variability of individual vascular resistance to these disruptive, occlusive, and infiltrative effects of silicosis was noted. Intravascular pressure changes were probably an important factor, evidenced by the finding of adventitial vascular hyperemia and proliferation. For the detail of the pathologic lesions reference should be made to the text.

After exclusion of all contributing degenerative cardiovascular disease, such as coronary atheroma and hypertension, over half the present series (26 out of 43 cases) were found to have right ventricular hypertrophy. Right heart failure supervened commonly in massive conglomerate nodular silicosis (11 out of 23 cases), but of the 20 cases of discrete nodular disease there was only 1 in which right-sided failure was regarded as of major aetiological importance in causing death; this was a case of bilateral pulmonary arterial thrombosis. The lung parenchyma between the nodules underwent fibrotic changes and ischemic necrosis; in massive conglomerate disease, anemic cavitation occurred also. Discrete nodular disease, despite varying degrees of central necrosis, appeared to be self-limited. The death of the cells was related to a combination of factors: the age of the silicotic lesions, direct toxic action of the free silica, proliferation of fibroblasts, progressive deposition of collagen, and disruption of nourishing capillaries. However, the full role of vascular changes in stimulating pulmonary fibrosis was uncertain. Pseudo-cavities were observed in emphysematous areas and were caused by contraction of the silicotic fibrous mass and disruption of marginal alveoli, with hyperinflation following bronchiolar obliteration. The deleterious effect on the right heart of variations in pulmonary intravascular pressure which resulted from the sum of these ischaemic lesions was accounted for on the basis of Visscher’s theory of decrease in the coronary vascular pressure gradient.

J. L. Lovibond.

Simple pneumoconiosis of coal-workers is a focal disease but differs from classical silicosis. The black spots seen throughout the lungs vary from microscopic size up to 5 mm in diameter. They may be soft or hard on palpation, depending on the amount of fibrosis, which is less dense than in typical silicosis and is rarely concentric. These areas are associated with emphysema, which is the most important feature of the disease. Radiographic reticulation is found and corresponds with these focal lesions but gives no reliable indication of the degree of associated emphysema. [The illustrations are very good.]

H. E. Harding.


Suspensions of powdered bagasse proved very toxic when injected intravenously into rabbits or into the trachea of rabbits or guinea-pigs. Similar suspensions which had been autoclaved or sterilized by formaldehyde were relatively innocuous. The lesions produced by the unsterilized suspensions were scattered areas of necrosis containing polymorphonuclear leucocytes, usually foreign-body giant cells, fragments of mycelium, and a peripheral proliferation of fibroblasts. Cultures from the powdered bagasse yielded a variety of organisms. Injection of suspensions of pure cultures of these organisms showed that a type of Aspergillus fumigatus produced lesions closely resembling those of the original dust. In these experiments, the bagasse fibre appeared to act as a rather inert material, and the acute inflammatory process was due to micro-organisms, probably fungi, attached to the bagasse.

[It is not clear that these experiments throw any light on the changes occurring in the lungs of some workers who handle bagasse.]

H. E. Harding.


White rats were made to inhale particles of aluminium during a period of increased respiration produced by rolling them in a revolving box containing the powder. The aluminium powder was that used in making metallic paint, and consisted of flat irregular plates about 5 to 8 µ thick and 5 to 25 µ long covered with a thin film of stearin. The metal was about 99 5% pure. Most of the inhaled particles stayed within bronchi, but some penetrated to the alveoli, where they were taken up by hypertrophied phagocytes. Within these cells the small plates of aluminium were slowly but steadily disintegrated during 8 to 20 weeks, until they were broken down into spherical bodies of a uniform diameter of 0.5 to 1 µ. The process of disintegration of a metallic sheet was irregular and proceeded along lines which sometimes appeared to correspond with lines of incomplete fracture. The breaking up of the metallic plates appeared to occur within the cell, and no particles were observed free in the alveoli; it was not possible, however, to demonstrate aluminium in the cell cytoplasm, which retained a normal appearance throughout.

The authors suggest that the mode of breaking of aluminium sheets may be determined by the presence of metallic impurities—iron, silicon, titanium, manganese, and vanadium—between the crystals of aluminium.

H. E. Harding.


This is a preliminary report on an experiment started in 1945, in which exposure to air-borne grinding dust for 8 to 10 months produced a faint diffuse dust pigmentation in the lungs of guinea-pigs and white rats. Histologically there were scattered inactive dust cells in the lungs without evidence of fibrosis. Some of the animals were deliberately infected with tuberculosis, but showed no acceleration of the spread of this disease as a result of inhalation of the dust.

Observation of grinders, burners, and welders in the foundry industry showed a lack of clinical symptoms and an absence of conglomerate shadows in radiographs, although nodulation was present in many. The incidence of tuberculosis was lower than the usual and when infection did appear it often progressed very slowly. Workers seldom complained of shortness of breath, even though radiographs of their lungs showed definite, evenly distributed fine nodulation. Radiographic stippling may appear after a comparatively short period of exposure (2 to 6 years in some cases); once it appears it alters very little over the course of several years despite continued exposure to dust.

The author considers that the radiographic appearances in these men are produced by siderosis, and are not due to silicosis. [It is undoubtedly true that much of the radiographic picture in foundry workers is produced by siderosis, but, in England at least, silicosis in not uncommon in these workers.]

H. E. Harding.


The author recounts 2 case-histories previously described (J. industr. Hyg., 1947, 29, 145) in which diffuse, not nodular, fibrosis was found in the lungs of workmen exposed to the fumes liberated during the fusion of bauxite in the manufacture of cordum. Chemical examination of the lungs showed a high total silica, only a small proportion of which was identifiable by x rays as quartz, mullite, or feldspar.

Examination of stack fumes and of settled dust in the plant showed that most of the silica was amorphous and not crystalline. Animal experiments with these two dusts were initiated by Gardner, but results in guinea-pigs are not yet available. Experiments are also proceeding on the effects on animals of mixtures of quartz or vitreous silica and aluminium oxide. A mixture of 1% of an electrically fused alumina and 99% quartz had produced no fibrosis within a month after intraperitoneal injection into guinea-pigs and intravenous injection into rabbits.

H. E. Harding.
ENVIRONMENT


Although during the last quarter of a century smoke production has diminished and its ill effects on health have lessened, smoke pollution still persists to a harmful extent in many large towns. It was estimated that 2,000,000 to 2,500,000 tons of tarry and carbonaceous matter were deposited over the country in 1945 together with 5,000,000 tons of oxides of sulphur and 500,000 tons of grit and ash. The ill effects of smoke pollution of the atmosphere may be direct, from the inhalation of chemical impurities, or indirect, from reduction in the effective sunshine. Under certain atmospheric conditions smoke assists in the destruction of life itself. The deleterious effects are most marked when fog and frost occur in a smoky town in the winter, resulting in a high concentration of grit, tar, and sulphur. For weeks afterwards this sends up the incidence of and the mortality from pneumonia and bronchitis. Figures showing the relation of fog to the mortality from respiratory diseases in some Yorkshire towns suggest that association of mist with low temperature is probably not very detrimental to health unless there is smoke pollution as well: the experience of Glasgow is that keen frost, fog, and still air occurring together are a harmful combination, mere cold without the fog not causing any increase in the fatalities from respiratory diseases. The amount of sickness that smoke-fogs cause is much greater than can be measured by crude vital statistics, the irritant effect on the mucous membrane of the respiratory tract being especially felt by elderly people subject to bronchial catarrh who are the first to fall ill when a smoky fog arrives. Apart from its action when associated with fog, smoke accounts for some part at least of the higher mortality rates for pneumonia and bronchitis in children under 5 years of age in the Midland industrial area of Britain. Its influence in depriving towns of sunshine is demonstrated by the low sunshine average in the years 1930-2 in Bolton (975 hours), Manchester (983), and Burnley (1,000), with Blackpool's 1,368 hours. The loss of ultra-violet rays, commonly 50%, may rise to 100%. Caryl Thomas.


A Government Commission studied the methods used to ventilate various metal works in which carbon monoxide escaped into the air. Figures are given in mg. per litre of air, and it is shown that some kinds of fuel and certain processes are more difficult to handle than others. During and since the war there has been a steady reduction in the carbon monoxide concentration in these works. In the severe winters workers may be tempted to reduce the ventilation, which should be controlled by the supervisory staff. G. C. Pether.

ABSTRACTS


An electrostatic dust sampler for use in industrial hygiene has been described by Barnes and Penney (J. Industr. Hyg., 1938, 20, 3) and has been commercially developed. The present author has modified and improved this precipitator, and its operating voltage has been increased by the development of an oil-immersion coil which will provide voltages up to 13,500. Increased dependability of the electrical system has been secured, and easy changing of the vibrator points has been achieved by the use of a totally enclosed vibrator unit. A flexible head ensures greater sampling flexibility. A manometer to obtain greater accuracy in measuring flow rates and a simplified voltmeter which reliably indicates the performance of the instrument have been designed.

H. M. Vernon.

GENERAL


The only source of information on age-distribution of diseases among the British civil population is contained in the reports of the Department of Health for Scotland, and these have been used in this analysis. The two groups studied were men and single women in the years 1936-7. The incidence of accidents in women increases with age, whereas the incidence in men tends to decline with age. The author points out that this is accounted for by increased liability to fractures among women over 45. There is not enough data available at present to determine how far this can be ascribed to differences in social habit and how far to differences in physique, but it is not due to differential fragility of bone. There is a striking difference between men and single women in relative age incidence of pulmonary tuberculosis. Among the women the distribution shows a sharp peak at the younger age groups 20 to 30; in men the distribution is spread over youth and middle life, and shows a secondary rise in the 50 to 55 age group. The author suggests that this disparity arises from industrial risks—such as silicosis among miners, to which men are alone exposed—and that if these risks were removed the male distribution might well become comparable with the female. Clinical experience would not lead one to agree with this. This male distribution is general and does not appear to be related to particular industries, such as mining.

The incidence of adenitis, appendicitis, diphtheria, dermatitis, measles, mumps, sore throats, scarlet fever, and "ulcers, carbuncles, boils, and abscesses" declines with advancing age; there is, however, a marked rise with age in the incidence of arteriosclerosis, asthma, bronchitis, cerebral hemorrhage, erysipelas, hernia, myocarditis, neoplasms, phlebitis, valvular diseases of the heart, and varicose veins. Diseases showing an uncertain trend include conjunctivitis, hemorrhoids, influenza, benign neoplasms, peptic ulcers, pneumonia, sinusitis, and tuberculosis. K. M. A. Perry.