ABSTRACTS

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TOXICOLOGY


The LD 50 of beryllium lactate given intravenously to mice and rats is 0.34 mg. and 0.54 mg. per kg. bodyweight respectively. All the other salts examined had about the same toxicity except sodium beryllium malate, which is much less toxic—possibly owing to its slow breakdown. When given subcutaneously or intraperitoneally in doses of 5 mg. per kg. these salts cause no fatalities, owing to local fixation of the beryllium to tissue proteins. After intravenous injection, death is due to narcosis of the liver, in the sinusoids of which beryllium can be determined histologically. It also causes degenerative changes in the red pulp, but not in the Malpighian bodies, or the spleen. V. J. Woolley.


Hitherto, owing to the scarcity of supplies, little original work has been done in Italy or any other continental European country to determine the clinical value of British anti-lewisite (BAL). As it is now more widely available, the author reviews the literature on this material, with particular reference to its use in industrial medicine.

In arsenic poisoning BAL is effective because of the greater affinity of arsenic for its -SH group than for the thiol group in the tissues. In vesicular erythoderma about 80% of cures have been recorded and in the U.S.A. 90% success has been claimed in the treatment of agranulocytosis. Similar benefit has been observed in other forms of arsenic poisoning. In arsenical jaundice, however, the results have been poor and no improvement has been observed in aplastic anaemia. In poisoning from arseniuretted hydrogen it has been thought that BAL caused aggravation. As mercury has an affinity for thiol groups in the tissues, it was thought that BAL might also be valuable in cases of poisoning by this metal. Good results have so far been described in cases of acute mercury poisoning, but not as yet in chronic cases. Some cases of corrosive sublimate poisoning with renal involvement have responded well to BAL, while a patient with neuritis and ataxia improved quickly when BAL was given, and elimination of mercury by the kidneys was accelerated. In lead poisoning in experimental animals it has seemed that BAL did harm. In human subjects the quantity of lead in the blood diminished when BAL was used, but soon rose again. Some cases of saturnism with colic are said to have been improved by giving BAL together with acid remedies. The effects in poisoning by gold, antimony, bismuth and other metals are also discussed. By reason of its strong reducing power, BAL is itself toxic. Nausea, headache, arthralgia and myalgia occur when too much is given.

While it is to be hoped that in time the indications for the use of BAL in occupational metallic poisoning may be better understood, notably in respect of lead and mercury, at present its effects are not easy to foresee and, in view of its toxicity, the author urges that the treatment of such cases with BAL should be confined to hospitals and special clinics. Even more emphatically does the author oppose the prophylactic use of BAL in industry and for the same reasons—the difficulty and uncertainty of dosage and the danger of untoward effects.

G. C. Pethar.


The author reports what he states to be the second case of diazomethane poisoning on record. Diazomethane is a very unstable, odourless, yellowish gas used chemically as a methyllating agent. (Because of its high degree of chemical activity it is generally employed dissolved in ether or benzene.) The patient was a 28-year-old research chemist, engaged in an investigation in which diazomethane was involved. He accidentally inhaled the gases produced in his experiment, before which he had eaten some rather greasy food, which might have some significance on account of the fat solubility of the chemical. He soon developed a dry, persistent cough. The next day he also complained of fatigue and soreness behind the sternum, while his temperature had risen to 102.4° F. (39.1° C.). Two days after the exposure there were signs suggestive of inflammatory changes in the lungs. The temperature remained elevated and cyanosis developed. On the fourth day the blood pressure started to rise and reached 180/90 mm. Hg after 12 hours. The pulse rate increased to 176, while the respiration rate was 44. Death occurred 100 hours after exposure. Terminal manifestations included
mental confusion, muscular twitching, and a small hæmatemesis. Apart from an initial leucocytosis of 11,300 (polymorphonuclears 83%) which rose to 23,600, laboratory examinations showed the presence of albuminuria with casts, and perhaps some degree of hyperglycæmia. At necropsy the findings were: acute ulcerative tracheo-bronchitis, bronchiolitis, and bronchopneumonia; toxic changes in heart, kidney, and liver; acute oesophagitis, gastritis, and duodenitis; and microscopical foci of necrosis in the brain with thrombosis in small blood vessels. The author considers the changes in the respiratory tract to be due to direct contact with the inhaled gas, while those in the upper alimentary tract were possibly caused by the diazomethane-contaminated sandwich. He points out that the patient never complained of dysphagia.

R. Salm.

INDUSTRIAL PHYSIOLOGY


There was a close correlation between haemoglobin concentration and pulse rate during work on a bicycle ergometer, indicating a correlation between total blood volume and stroke volume of the heart during physical exercise. The cardiac volume, radiologically determined, diminished in some people only slightly during work until the heart rate reached 150 per minute, to decrease more rapidly at higher rates. In others, the heart volume diminished in proportion to the increase in heart rate. Left ventricular and aortic electrokymography showed that the systolic emptying of the left ventricle greatly increased during exercise, in spite of unchanged or slightly diminished heart volume. No direct correlation was found between heart volume and stroke volume in the same subject. It is realized that this finding is in contradiction to Starling's "law of the heart".

A. Schweitzer.

INDUSTRIAL LUNG DISEASES


Out of 110 coal miners suffering from silicosis 3 were found to have cardiac lesions unconnected with their lung condition, 12 systemic hypertension, and 9 silico-tuberculosis. The remaining 86 provided the material for this paper, in which is stressed the value of a clinical history and clinical examination as an aid to the diagnosis and prognosis of cardiac disease secondary to silicosis. As evidence of cardiac damage from silicosis the author considers that the four main signs in order of decreasing importance are: (1) right-sided bruit de galop, (2) filling of the jugular veins while the subject is seated, (3) a tender enlarged liver, (4) accentuation or duplication of second cardiac sound heard between the pulmonary and tricuspid areas. Among the symptoms and signs indicative of severe alteration of cardiac function the author notes dyspnoea, obstinate headache, a pulse rate greater than 100, cyanosis, and marked wasting.

H. E. Harding.


The author considers briefly the methods available for assessing lung function. Methods giving the ventilatory and respiratory components of breathing are held to be insufficient for this purpose if used singly. The author therefore tried to overcome this difficulty by estimating the so-called ventilation or respiratory equivalents of oxygen and carbon dioxide, which are simply the differences between the percentages of the two gases in the inspired and expired air. It will be recalled that the ratio of these equivalents is what used to be called the "false respiratory quotient." From estimations on normal subjects it was found that the value for the O₂ equivalent should normally always exceed 3-4 and for the CO₂ equivalent 2-9, while to ensure that hyperventilation has not occurred the true respiratory quotient should lie between 0-7 and 1. Both ventilation equivalents were estimated by the author simultaneously and continuously by means of the Zeiss interferometer—a very accurate method—and later with the Rochester continuous analyzer, which is less accurate. Determinations were made during rest, exercise, and recovery in a large number of normal and abnormal subjects, including cases of pneumoconiosis with and without dyspnoea. The exercises employed were the bicycle test with a load of 2,000 kg. per 10 minutes (or in severely disabled patients, 900 kg. per 10 minutes) and the climbing test of stepping up 20 cm. 20 times per minute. The author prefers the treadmill method, but did not have a treadmill available. He prefers the bicycle test to the climbing test.

Comparison of the average values obtained at rest in normal subjects and subjects with varying degrees of dyspnoea (and many kinds of disease) showed that only in cases of very severe dyspnoea did the equivalents fall markedly below the normal values. The ventilation equivalents of normal subjects during exercise and recovery all fell substantially within the normal range, but in the case, for example, of patients who were dyspnoeic when walking on level ground, the O₂ equivalent was always less than 3-4 and the CO₂ equivalent less than 2-9. It would appear that in silicosis and in diseases of the lungs and circulation in general there was poor correlation of ventilation equivalents with x-ray findings, but good correlation with the symptomatic state.

M. W. Goldblatt.


Abnormalities were found in the radiographs of the lungs of 10 out of 50 to 60 men attending a chest clinic between 1941 and 1949 whose work involved exposure to sulphurous fumes from coke fires—boiler-
stokers, blacksmiths, plateers, and men employed in coke-oven and gas-works. All 10 patients complained of respiratory symptoms, the chief of which were productive cough, dyspnoea on exertion, and irritation of the nose and throat. The severity and duration of their symptoms bore no direct relation to the duration of exposure to the fumes or to the extent of the x-ray abnormality. Radiography showed irregular patchy fibrosis involving both lungs, but usually one much more than the other. In most cases the mid- and lower zones showed nodulation occasionally becoming confluent and producing patchy consolidation. Emphysema was usually present. The fine fraction of a sample coke-flue dust obtained by a coke-oven worker contained 34% total silica, 2:1% free silica, and 17.5% iron compounds (expressed as Fe₂O₃).

The authors conclude that the cases in this series are examples of an occupational pulmonary disease, and suggest that protracted exposure to sulphur dioxide causes localized chronic inflammation in the lungs, with accumulation of siliceous dust in the affected areas.

[Four radiographs are reproduced, but too poorly to be useful. A good deal of confirmatory evidence will be necessary before this type of industrial exposure can be taken as producing a new form of pneumoconiosis. Sulphur dioxide has been claimed by previous authors to play a part in industrial lung diseases and has been used in animal experiments (often in combination with oxides of nitrogen) as an adjuvant or accelerator in studying the effects of particulate matter introduced into the lungs.]

H. E. Harding.

ENVIROMENT


In the vicinity of a factory producing beryllium (Be) and its compounds, “cases of chronic pulmonary granulomatosis similar to that seen in beryllium workers were found among people not working in the factory. Of these patients 5 lived up to \( \frac{1}{3} \) mile (0.4 km.), 3 between \( \frac{1}{3} \) and \( \frac{1}{2} \) mile (0.4 to 0.8 km.), 2 between \( \frac{1}{4} \) and \( \frac{1}{2} \) mile (0.8 to 1.2 km.) and 1 between \( \frac{1}{4} \) and 2 miles (2.4 to 3.2 km.) from the plant. This last case was in the wife of a beryllium worker who handled her husband’s working clothes every day and probably inhaled particles from the clothes, but, apart from this case, atmospheric contamination arising from the factory appeared likely. The dusts, fumes, and mists from the factory discharged from a 185-foot (56.5 m.) stack and from a number of short roof stacks about 33 ft. (10 m.) above street level. The atmospheric concentration of beryllium downwind from the factory, measured spectrographically, ranged from 0.2 \( \mu \)g. per cm. at \( \frac{1}{3} \) mile to 0.03 \( \mu \)g. per cm. 5 miles (8 km.) from the stack. When the manufacture of beryllium-copper alloy was going on the concentration of Be in the atmosphere at 400 feet (122 m.) downwind from the factory was more than doubled—0.7 \( \mu \)g. as compared with 0.3 \( \mu \)g. per cm. when normal operations were proceeding. Continuous atmospheric sampling at fixed points 350 to 750 feet (107 to 228 m.) from the factory during 10 weeks of normal operation showed wide variations of Be concentration during any given day, but daily averages over the 10 weeks agreed closely (0.05 to 0.15 \( \mu \)g. per cm.).

Since concentrations of less than 1 \( \mu \)g. per cm. are not held to be toxic, the authors sought to estimate the probable concentrations existing some years previously. Since the processes remained unchanged and, while production units had been added during the last 7 years, no important ones had been discontinued, any changes in atmospheric concentration could be attributed to quantitative changes in production and to the manner of handling exhaust air. The former was estimated never to have exceeded twice that at the time of the present study. From the curves of the theoretical fall-off in relative concentration downwind from the 33-ft. stacks and from the 185-ft. stack, and from meteorological considerations, the authors concluded that the discharge from the 33-ft. level was the more significant as a possible cause of the cases detected. Up to \( \frac{1}{3} \) mile the Be concentration would be almost entirely due to the 33-ft.-level discharge; from \( \frac{1}{3} \) mile to \( \frac{1}{2} \) mile and from \( \frac{1}{2} \) mile to \( \frac{1}{3} \) mile the effect of discharge at both levels would be markedly felt. Since 1944 effluent gases had been collected by local exhaust and a dust separator connected to the tail stack. Thus by determination of the Be exhausted from each operation estimates could be made of the former delivery of Be into the atmosphere. From 1940 to 1944, during the Be-Cu operation, 12 g. Be a day was delivered at roof level (33 ft.) ; from 1944 to 1945 this was reduced to 2 g. a day; and from 1945 to 1948 to none at roof level and 2 g. a day from the tall stack. Similar estimates were made for many other plant operations.

During the 7 years’ operation of the factory it is estimated that the average beryllium concentration in the atmosphere \( \frac{1}{3} \) mile away ranged from 0.01 to 0.1 \( \mu \)g. per cm. The cases detected seem to have resulted from exposure during the first 5 years or so, when the concentration at \( \frac{1}{3} \) mile was probably about 0.1 \( \mu \)g. Be per cm. It is recommended that the average daily atmospheric concentration of beryllium in the neighbourhood of a plant should not exceed 0.01 \( \mu \)g. per cm. The principal compound to which the patients had been exposed was beryllium oxide (BeO). Curiously enough, the incidence of berylliosis among workers in the factory itself has been low in spite of high atmospheric contact: the authors suggest that particle size may play a part in determining this difference in incidence. It was also estimated that the laundering at home of working clothes (shaking, washing and scrubbing, shaking, and folding) may lead to a daily inhalation of 17 \( \mu \)g. of beryllium.

M. W. Goldblatt.


In this important report the results are analysed of a survey of nurses’ health in an urban hospital, “in the hope that the facts revealed will lead to the more effective care of nurses’ health and greater efficiency in the nursing services”: The extent of sickness among 300 nurses.
during the years 1943-5 was assessed by the number completely free from sickness, the average amount of sickness per head, and the proportion sick for more than 50 days in the year. The proportion sick for more than 50 days per annum was 5% for trained nurses, 10% for student nurses, and 12% for assistants and auxiliaries. The commonest cause of illness was respiratory infection; the next most important was skin sepsis, followed by infective diarrhea, infectious fevers, and tuberculosis. The author quotes figures given by other workers showing higher sickness rates in nurses than in other students, but has no such figures for comparison with his own.

In 23 out of 39 nurses tuberculin negative on first examination, the reaction became positive during the survey. Tuberculosis developed in 7 of these 23; one of the 7 had a pleural effusion only, and the other 6 had a primary focus or complex only; three required sanatorium treatment, but no "serious bronchogenic or haematogenous lesions" developed within one to two years of the initial illness. Eleven cases of tuberculosis developed among the 264 initially positive; in 7 of the 11 disease was latent or detected by routine radiography and in 4 it was clinically active; five nurses required sanatorium treatment for periods varying from 6 to 18 months. There were no deaths in either group.

The number leaving nursing before training is completed and in the years immediately after qualification is a grave problem, and the author found that ill health plays a large part in wastage; 17% of those leaving the hospital and 25% of those leaving the profession did so for this reason. He reviews the principles concerned in the reasonable care of the health of hospital nurses, and suggests a number of practices which could be carried out readily in most hospitals. M. Daniels.


Investigations were carried out at a steel-rolling mill to assess the dependence of the productivity of the workers on their physical fatigue. The former was estimated by the time requested by individual workers for the various parts of the procedure—this is called the rhythm of the work; the latter was assessed by the temperature and pulse rate of the worker. The selected group of 12 workers were of approximately the same age and had the same industrial experience; 500 observations were made on each individual worker under investigation.

It was found that the 12 workers could be divided into four groups with reference to productivity as measured by the time requirement for the various parts of the steel-rolling process. The group of workers with the longest time requirement showed also the highest degree of fatigue. This was particularly noticeable in the afternoon. Further analysis of the various parts of the procedure showed that it was the setting of the steel product in the narrow right-angled hole of the rolling table which gave rise to such great individual differences. It is suggested that the right-angular shape of the apertures should be replaced by conical ones, and that 20 minutes rest is required for every 10 minutes of work.

These suggestions, it is expected, will bring about the reduction of the individual time requirement, improve the so-called work rhythm, and reduce fatigue in the workers irrespective of their individual skill. E. W. Collis.

GENERAL


In addition to innocuous colouring matters of vegetable and animal origin, coal-tar dyes may be used for colouring foodstuffs. Owing to their high colouring power, only small amounts are required. Repeated ingestion of these compounds over prolonged periods may lead to development of malignant neoplasms. Various chemical compounds ingested at the same time may increase the deleterious effect of these dyes. A petition has been presented to the Austrian Ministry of Health for the prohibition of the use of coal-tar dyes in colouring butter and cheese. Margarine is not mentioned. The author is of the opinion that the import from abroad of coal-tar dyes for the colouring of foodstuffs should be prohibited. C. den Hartog (Excerpta Medica).


This paper is based on 750 cases of head injury presenting medico-legal problems. Of these patients 685 did not subsequently suffer from fits. The remaining 65 (8.6%) had fits afterwards. This latter group has been subdivided into 2 subgroups. The first subgroup of 13 patients sustained focal brain damage. None of these patients had fits before the injury. The second subgroup consisted of 52 patients who alleged head injury of at least concussional severity. No patient in this second subgroup had had a skull fracture. Such patients usually recover completely within 30 days and many return to normal in 48 hours. Those who are only briefly dazed or have a brief period of traumatic amnesia have even less basis for persisting brain-tissue changes or symptoms.

Convulsive attacks never take place as an immediate result of concussion. This has been confirmed by animal experiments and by observations on pugilists. Single seizures may occur soon after recovery of consciousness, but no case of this was seen in the present series. The 52 cases of alleged head injury were further subdivided. The author found that in 28 cases (54%) there was malingering or fraud with reference to the character of the injury and its effects. In 18 cases a seizure had caused the head injury. Five patients had an organic nervous disease which accounted for the fits in the absence of head injury. The author's general conclusion is that in a healthy person an injury limited to concussion never causes recurring seizures at a later date. A more severe focal lesion may occasionally do so. Gilbert Forbes.