ABSTRACTS

(This section of the JOURNAL *is published in collaboration with the two abstracting Journals,* Abstracts of World Medicine, *and* Abstracts of World Surgery, Obstetrics, and Gynaecology, *published by the British Medical Association. The abstracts are divided into the following sections : toxicology ; industrial physiology ; industrial lung disease ; industrial skin diseases ; accidents and orthopaedic surgery ; industrial ophthalmology ; environment ; general. Not all sections will necessarily be represented in any one issue)*

INDUSTRIAL TOXICOLOGY

Blood Cholinesterase Levels in Workers Exposed to Organo-phosphorus Insecticides. BARNES, J. M., and DAVIES, D. R. (1951). *Brit. med. J.*, **2**, 816.

By the method described by Callaway, Davies, and Rutland (see below) the erythrocyte and plasma cholinesterase levels were determined in 130 specimens of blood from 80 men and women exposed to organophosphorus insecticides in field or factory. In 12 individuals abnormal variations were found which were probably attributable to absorption of the insecticide. Reduction of the erythrocyte cholinesterase content was found in 3 cases, and of that of the plasma in 5. Of 14 workers whose blood was analysed on more than 1 occasion, abnormally great individual variations were found in 8 ; of these, 4 had been removed from contact with the insecticides after the first examination and the level of the enzyme had risen in the interval, whereas in the other 4 contact continued during the interval and the fall in the enzyme level indicated some absorption. All departures from normal were slight, and no serious case of poisoning was encountered. *A. Schott.*

Blood Cholinesterase Levels and Range of Personal Variation in a Healthy Adult Population. CALLAWAY, S., DAVIES, D. R., and RUTLAND, J. P. (1951). *Brit. med. J.*, **2**, 812.

Certain compounds used as insecticides or proposed for chemical warfare may produce a significant reduction in the erythrocyte or plasma cholinesterase content before symptoms of systemic poisoning occur. Estimation of the level of these enzymes may therefore yield important information regarding the absorption of such compounds and also during the recovery from intoxication. The present paper deals with the normal levels, as determined in 247 healthy adults—100 men aged 18 to 30 years from the armed Services, 81 civilian men aged 18 to 60 years, and 66 women aged 27 to 74 years. The method is described in detail, and the unit of enzyme activity used is defined. No statistically significant difference was found between the mean values for plasma enzyme activity in the 3 groups, but the mean value for erythrocyte enzyme content was significantly higher in the Service group. The limits of variation for plasma enzyme activity in the whole series were between 57% and 143% of the mean value and for erythrocyte enzyme activity between 78% and 122% of the mean in the Service group and between 68% and 131% in the civilian groups. There were no sex, age, or seasonal differences in either value.

The individual variations in level were investigated in 10 soldiers by examining blood samples on 8 separate occasions. From this it is concluded that where it is possible to make only a single estimation of blood cholinesterase levels in an individual exposed to such compounds, while it can be ascertained whether the level is above or below the lower limit of normal for the whole population (which is well above that at which symptoms of systemic poisoning are likely to occur), a level found to be within the normal range for the whole population may be well below the normal range for that subject, since individual variations are much smaller than those of the whole population. Successive estimations in the same subject will yield more information regarding the possible absorption of such toxic compounds.

A. Schott.

Vapor Toxicity of Trichloroethylene Determined by Experiments on Laboratory Animals. ADAMS, E. M., SPENCER, H. C., ROWE, V. K., MCCOLLISTER, D. D., and IRISH, D. D. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 469.

In this paper is described experimental work undertaken as part of a comparative study of the vapour toxicity of four chlorinated hydrocarbons.

In carefully controlled experiments rats, guinea-pigs, rabbits, and monkeys were subjected to a single exposure to trichlorethylene, or repeated 7-hour exposures on 5 days weekly for periods up to 6 months. In rats the effect of a single exposure was to depress the central nervous system only ; full anaesthesia was induced at 4,800 parts per million (p.p.m.), but not at 3,000 p.p.m. All deaths occurred during the exposure or very shortly afterwards. Survivors recovered rapidly and completely. With repeated exposures a concentration of 3,000 p.p.m. produced no histological changes in the body tissues, but after the first week there were signs of functional disturbance during the times of exposure—hyperexcitability, restlessness, salivation, marked scratch reflexes, all of which subsided completely when the rats had been removed from the vapour chamber and had been fed. Repeated exposure to 400 p.p.m. caused no adverse effects in a monkey, but in guinea-pigs and rabbits it caused a slight increase in liver weight ; 200 p.p.m. produced no adverse effects in rats and rabbits, but a slight retardation of growth in guinea-pigs ; 100 p.p.m. caused no adverse effects in any animals. These results

support the accepted figure of 200 p.p.m. for the maximum permissible atmospheric concentration in industry.

It is suggested that the chronic effects experienced by workers exposed to trichlorethylene—fatigue, headache, gastro-intestinal malaise, and anorexia—represent primarily functional disturbances of the nervous system, and that the autonomic nervous tissues may be involved.

Tables and graphs give the mortality figures, growth curves, average final body weights, and organ weights of the animals exposed. *M. A. Dobbin Crawford.*

Vapor Toxicity of Ethylene Dichloride Determined by Experiments on Laboratory Animals. SPENCER, H. C., ROWE, V. K., ADAMS, E. M., MCCOLLISTER, D. D., and IRISH, D. D. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 482.

In this paper is recorded another part (see above) of a comparative study of the vapour toxicity of four of the chlorinated hydrocarbons.

The effects of single exposures and of repeated 7-hourly exposures to ethylene dichloride vapour at various concentrations were tested by carefully controlled experiments upon animals. With single exposures in rats three toxic effects were observed : (1) depression of the central nervous system, causing " drunkenness " at 12,000 parts per million (p.p.m.) and deep anaesthesia at 22,000 p.p.m. ; (2) shock : at all concentrations many of the rats died quietly and rather suddenly a few hours after removal from the vapour chamber ; and (3) organic injury, chiefly renal, causing death within 2 to 7 days of exposure. Only at concentrations above 3,000 p.p.m. were the lungs damaged, showing congestion and oedema. The adrenal glands were congested, with multiple haemorrhages.

Rabbits exposed to ethylene dichloride vapour in a concentration of 400 p.p.m. for 7-hour periods daily for 5 days a week and continued up to 6 months showed no adverse effects. The same concentration produced severe intoxication in monkeys, rats, and guinea-pigs, with cloudy swelling and fatty degeneration of the liver and congestion with degeneration of the tubular epithelium of the kidneys. A concentration of 200 p.p.m. caused no adverse effects in these animals except for a slight retardation of growth in rats. Repeated exposure to 100 p.p.m. produced no adverse effects in any of the animals. The adequacy of the generally accepted figure of 100 d.p.m. as the maximum permissible concentration of this gas in industry is therefore confirmed.

Detailed results are shown in tables and graphs.

M. A. Dobbin Crawford.

The Chylomicron Count as an Indicator of Phosphorus Poisoning. A Study Utilizing Experimental Animals. FLEMING, R. B. L., and COLLINGS, G. H. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 567.

In experiments on rats receiving a fat-free diet the base chylomicron counts were not significantly affected by acute or chronic phosphorus poisoning. The normal rise in level of blood lipids after the taking of food, however, was both diminished and delayed. This effect was greatest during the first 2 to 2½ weeks of the administration, and thereafter a tolerance developed. This tolerance was lost within 10 days of the last phosphorus injection. Details of procedure and chylomicron counts are given. *M. A. Dobbin Crawford.*

Response of Rodents to Repeated Inhalation of Vapors of Tetraethyl Orthosilicate. POZZANI, U. C., and CARPENTER, C. P. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 465.

Tetryl Toxicity : a Summary of Ten Years' Experience. BERGMAN, B. B. (1952). *Arch. industr. Hyg. occup. Med.*, **5**, 10.

Preliminary Observations on Toxicity of Elemental Selenium. HALL, R. H., LASKIN, S., FRANK, P., MAYNARD, E. A., and HODGE, H. C. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 458.

INDUSTRIAL LUNG DISEASE

Coal Workers' Pneumoconiosis. Pathological and Etiological Considerations. HEPPLESTON, A. G. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 270.

In order to compare the general pathological features of pneumoconiosis as it occurs in coal-workers in other areas with those of the disease as it occurs in South Wales, the author studied material from the U.S.A. (50 coal-workers, 19 employed in anthracite mines and 27 employed in bituminous-coal mines), Scotland (8 cases from steam coal mines), England (2 cases), Germany, and Russia (1 case each). As controls, lung tissues from 15 individuals with classical silicosis (mainly sandblasters and pottery workers) and from 25 town dwellers without occupational exposure to dust were examined.

As in South Wales coal-workers' lungs 2 main types of pathological change were observed in the lungs of coal-workers examined : massive collagenous fibrosis, which was found in 14 of the cases, and simple pneumoconiosis characterized by " coal macules ", which were present in all cases. The massive fibrosis was histologically similar to that found in Welsh cases, with coarse, irregular, hyaline, collagenous fibrosis, lymphocytic infiltration, endarteritis, and colliquative necrosis causing cavitation. Some cavities had a lining suggestive of chronic tuberculosis, and the author suggests that cavitation may be of two kinds, infective and ischaemic. In all the massive lesions there was an eosinophilic necrosis suggestive of tuberculosis, especially in the neighbourhood of the tuberculous granulation tissue which was found in some cases.

The macular lesions are described at greater length, and 4 stages of development are distinguished : (1) An early stage of dust collection around the divisions of the respiratory bronchioles in the interstitial tissue and extending into the surrounding alveoli, with a delicate network of reticulin. (2) Formative lesions develop, similar to the early lesions but extending more widely into the surrounding alveoli, and there is early trans-

formation of the reticulin into fine collagen. (3) Retrogressive lesions are found which are shrunken and compact, with bundles of fine collagenous fibres. These lesions have a stellate outline and there is emphysema of the surrounding and enclosed alveoli, which have attenuated walls and altered elastic fibres. (4) The progressive extension of the focal lesions leads to confluent lesions, so that the focal origin is largely obscured. In such cases there is extension of dust and fibrosis subpleurally, in the septa, and also along the bronchoarterial tree. The essentially focal nature of the lesion of the coal macule is stressed. Since there is no histological evidence of infection in the coal macules these must be attributed to the action of the dust alone. The massive lesions bear no resemblance to the macules and cannot therefore be due to their coalescence by collapse, so that they must be due to some factor other than the dust. The obvious factor is tuberculous infection. Of massive lesions in South Wales, 30% prove positive on culture for the tubercle bacillus, and the histology of the remainder is so similar that it may reasonably be supposed that in these cases the tubercle bacilli have been overcome, leaving a healed lesion ; moreover, the site at which massive fibrosis occurs is also the site of predilection for tuberculosis. However, it remains possible that other infections may be involved. The vascular changes are considered to be secondary to the fibrosis. It is pointed out that the large lesions are often surrounded by larger, hard nodules which have the same histology as the massive fibrosis and are probably also of infective origin. The distinction between this type of nodule and the coal macule has not been clearly made by previous workers.

The author then discusses whether any particular fraction of the dust is responsible for the macular lesion. In the past the silica fraction of the dust has been generally incriminated, even though it forms a very small proportion of the total dust, but if this were so the histology of the lesions should bear some morphological resemblance to that of silicotic nodules. The obvious differences from silicotic nodules are stressed : in particular, the disproportion between the amount of dust and the amount of fibrosis and the entirely different arrangement of the fibrosis. It is concluded that the two lesions have little in common, and it is pointed out that whereas in animals silica produces definite nodulation, coal dust produces no material fibrosis. The author therefore concludes that the silica is not responsible and suggests that the coal is not just a diluent, but is the active cause of the lesion, the lung being simply overloaded so that it can only respond by developing sufficient fibrosis to immobilize the dust. It is further pointed out that inhalation of rock dust by coal miners cannot be incriminated, since coal-trimmers exposed only to commercial coal dust in the holds of ships develop the same lesions. It is also stated that town-dwellers may have similar early lesions. The development of collagen in the retrogressive lesions cannot be taken as evidence of the action of silica, for a similar change may occur in other tissues (as in old uterine fibroids). Figures for the prevalence of pneumoconiosis in hard-coal workers in the U.S.A. are shown to depend more on total dust exposure than on the free silica content of the dust. Chemical analyses of lungs are unlikely to throw much light on this question because of the very poor association between silica content and severity of silicosis even in workers exposed to a pure silica risk. The focal emphysema is attributed to the force of inspiration acting on a focus of consolidation and causing mechanical stress in the surrounding lung. It is considered to be the cause of disability in simple pneumoconiosis. The final conclusion is that the word " anthracosilicosis " should be dropped and the words " coal-workers pneumoconiosis " substituted. *C. M. Fletcher.*

A Statistical Study of Pneumoconiosis in the Cement Industry. PARMEGGIANI, L. (1951). *Rass. Med. industr.*, **20**, 400.

***X*-ray Diffraction Study of Sputum in Silicosis.** MEYER, F., and SOLOMON, S. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 443.

The presence of silica was shown in the sputa of persons with silicosis by *x*-ray diffraction analysis. Positive *x*-ray diffraction identification of silica was obtained in the sputa of all 4 persons with severe silicosis investigated. No evidence of silica was found in the sputa of 10 control patients. While the presence of silica has been previously demonstrated by *x*-ray diffraction in lung tissue obtained at autopsy, the present study appears to be the first report on the successful application of this method to sputum obtained during life.

The method as used in this study was not quantitative. Considerable work will be required to determine whether *x*-ray diffraction of sputum has a place in the clinical diagnosis and investigation of silicosis.—(Authors' summary.)

Rönnskär Disease. (" Rönnskärssjukan ") LUNDGREN, K. D., RICHTNÉR, N. G., and SJÖSTRAND, T. (1951). *Nord. Med.*, **46**, 1556.

Since the ore-smelting works at Rönnskär, Sweden, were opened in 1929 there have been complaints of lassitude and shortness of breath on exertion from the men working there. In 1945 the Swedish State Hygiene Institute initiated a thorough investigation into the problem, the findings of which are now presented. This involved the medical examination of some 1,500 workmen employed in the plant and 500 control subjects, together with technical inquiries. The prevalence of a chronic rhino-pharyngo-tracheo-bronchitis with atrophic and hyperplastic changes in the respiratory mucosa was confirmed ; thick mucus is secreted and there may be emphysema. Rönnskär ore has a high concentration of arsenic, and it was possible to associate the occurrence of the condition among the employees at the works with the concentration of arsenic trioxide and sulphur dioxide, both of which are liberated with the fumes during the roasting and smelting processes. *W. G. Harding.*

Effect of Aluminium Dust on Susceptibility to Lobar Pneumonia—Animal Experiments. VINTINNER, F. J. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 217.

The experiments reported here are similar to those described above, except that pure metallic aluminium dust was used in high concentrations. The results were also similar to those in the coal dust experiment, in that there was a lower mortality and infection rate in the dust-exposed animals in the mucin experiments but no appreciable difference in the broth experiments. Indeed, in the latter there was a slightly higher mortality in the rats exposed to dust than in the controls, when the exposures were continued over a long period.

C. M. Fletcher.

ENVIRONMENT

Lobar Deposition and Retention of Inhaled Insoluble Particulates. STOKINGER, H. E., STEADMAN, L. T., WILSON, H. B., SYLVESTER, G. E., DZIUBA, S., and LABELLE, C. W. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 346.

The authors were unable to find in the literature any quantitative information on the retention of dust in the lungs after appreciable lengths of time, although a number of studies have been made on its deposition in various parts of the respiratory tract. They therefore carried out an investigation of the retention of particles of uranium oxide in the lungs of rats.

By spraying aqueous suspensions of sized particles 2 types of cloud were produced, 1 consisting of particles of U_3O_8 of a mass median diameter (M.M.D.) of 2·6μ, and the other of UO_2 of M.M.D. 0·45μ. [It is important to point out, although the authors do not do so, that these 2 oxides have densities of 10·9 and 7·3 respectively, so that the equivalent size of particles of unit density having the same terminal velocity (and therefore penetrating power) would be 7μ and 1·5μ respectively.] Rats were exposed to these clouds for periods varying from 6 to 54 hours, and killed after intervals ranging from 20 hours to 8 months. The amounts of dust retained in the whole left lung and in the various lobes of the right lung were estimated by a fluorophotometric method, and the following results were obtained: (1) After 6 hours' exposure to dust of M.M.D. 0·45μ, about 10 times as much was retained as after exposure to dust of M.M.D. 2·6μ for the same time and the same concentration, as measured with a gravimetric filter-paper sampler. (2) After 54 hours, exposure, 150 to 175% more dust was deposited in the right upper lobe of all rats, irrespective of particle size, than in other parts of the lung. (3) On comparing the dust content after 54 hours' with that after 6 hours' exposure, it appeared that dust was removed from this lobe about twice as fast as from all other lobes except the right median, from which no dust appeared to be removed at all. (4) Coarse dust appeared to be removed from the lungs more rapidly than fine, over periods of several months.

(The above observations were made on groups of 10 rats in each category, and the differences found were statistically significant.)

From the size distribution of particles in the 2 clouds, as studied by means of the cascade impactor, and from the observed difference in deposition of particles of different sizes, a "nasal filtration" cut-off point was calculated for the rat, "nasal filtration" being taken to include filtration by the whole upper respiratory tract, including the bronchi. This cut-off point was found to be 0·7μ which, the authors comment, compares with figures of 1·2μ found by Hatch and his co-workers. [Here again the authors ignore the effect of density; if this is allowed for, the cut-off point in the rat appears actually to be close to that in man.] Assuming a minute volume of 110 ml. per minute for the rat, the authors calculate the percentage retention for each size of particle and show that about 10% of the respirable fraction of both dusts was retained in the alveoli, although the percentage of the total was only 0·86 in the case of the coarse dust and 8·7 in the case of the fine. Anatomical studies of the rats' lungs suggested that the difference in deposition and retention of dust in different lobes could be explained on a physiological basis.

[This is an interesting and valuable piece of work but it is somewhat marred by the obscurity of its style, and by the failure to realize the fundamental importance of particle-density in lung retention studies.]

B. M. Wright.

Absorption and Excretion of Inhaled Fluorides. COLLINGS, G. H., FLEMING, R. B. L., and MAY, R. (1951). *Arch. industr. Hyg. occup. Med.*, **4**, 585.

This paper contains a brief description of an investigation into the absorption and excretion of inhaled fluorides under industrial conditions. Two human subjects (sex not stated) were exposed for 7 hours (being an 8-hour shift interrupted by four 15-minute intervals—three for decontamination and collection of urine, and one for lunch) to each of the following atmospheres: (1) an atmosphere containing hydrogen fluoride gas and silicon tetrafluoride gas at a mean concentration of 3·8 mg. per c. metre (maximum atmospheric concentration = 2 mg. per c. metre); and (2) (several weeks later) one containing rock phosphate dust (3·5% fluorine) at a concentration of 5·6 mg. fluoride per c. metre, 95% of the dust particles being 5μ or less in diameter. The total urine was collected at 2-hour intervals during the exposure and for the next 2 days. For the supply of 2-hourly specimens a large fluid intake, producing a relative diuresis, was necessary.

In terms of total air-borne fluorine, the exposure to dust was 1½ times greater than the exposure to gas. Both subjects felt the irritant effect of the gas, but quickly became acclimatized. The dust caused no subjective symptoms. The normal daily fluorine excretion by the kidneys—1·2 mg. or less—was increased on the day of exposure to gas to 8·1 mg., and on the day of exposure to dust to 8·9 mg. Within 24 hours of the beginning of the exposure the urinary fluorine concentration was almost normal again; and it is suggested that the very small carry-over to the following day would have little cumulative effect.

M. A. Dobbin Crawford.