BRITISH JOURNAL OF INDUSTRIAL MEDICINE

VOLUME TWELVE
1955

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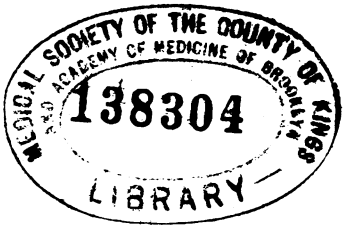

LONDON
BRITISH MEDICAL ASSOCIATION
TAVISTOCK SQUARE, W.C.1

Printed in England by Hazell, Watson & Viney, Ltd., London and Aylesbury—3040–1955

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disagreed with the histological interpretation. But a book of this type is not likely to be used so much for its conclusions as for its references. For the latter it is invaluable, not only because of the number cited but also because of their catholicity. It is a pleasure to read a book in which full attention is paid to data coming from all countries. It is refreshing also to find an author who lays so much stress on the doctor's responsibility to apply his conclusions in the field of prophylaxis.

RICHARD DOLL

A Practical Manual of Diseases of the Chest. By Maurice Davidson. (Pp. x + 647; 255 figures. 84*s*.) Oxford University Press (London: Geoffrey Cumberlege). 1954.

A reviewer, with specialist knowledge in a limited field, is in a somewhat difficult position when reviewing a chapter on his speciality in a large textbook. It is impossible for textbooks, however frequently revised, to be completely up to date at the time of publication, and the problem is simply, "How out of date is good enough?", and, for a well known and widely respected textbook, it seems reasonable to expect it to be not more than two or three years behind the times.

Unfortunately, by this standard, the chapter on "The Pneumonconioses" must be severely criticized. The histogram (Fig. I) of the number of references related to date of publication tells the story quantitatively, and when the quality of the four most recent references are examined, three are found to refer to experimental animal pathology with very doubtful human applications, while the other refers only to the social problem of pneumoconiosis.

Although many of the old references have great value, it is impossible to neglect nearly all the work of the last 10 years without seriously misleading the reader. The reader should be told something of the work of Gough and Heppleston in differentiating the pathology of coalworkers' pneumoconiosis from that of silicosis. Similarly, the modern British radiological classification, which has been accepted as the international classification, should surely at least be mentioned. The author refers to the differences of opinion amongst doctors in interpreting *x*-ray films showing pneumoconiosis but makes no reference to the work of Fletcher in measuring this error and in reducing it by means of standard films. The medico-legal section too has an odd flavour. It must

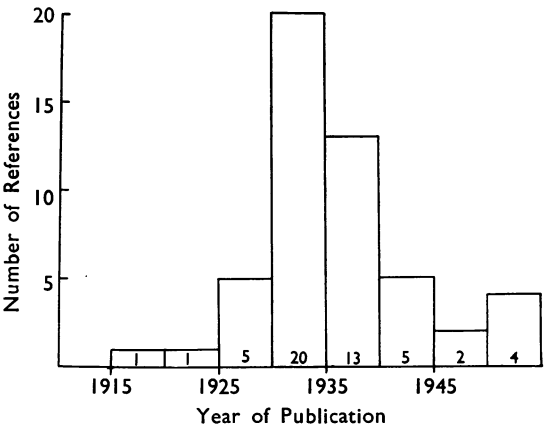

FIG. 1

antedate the National Insurance (Industrial Injuries) Act of 1946. The general effect is rather like a textbook on tuberculosis omitting the section on antibiotics.

However, a relative of mine once, to my horror, threw an expensive guide book to Scotland into the fire because it gave insufficient attention to the war memorial in Galashiels, so I would like to stress the probability of the excellence of the rest of the book, while advising readers to steer clear of the chapter on the pneumoconioses.

A. L. COCHRANE

Association of Industrial Medical Officers

In 1956 the Association of Industrial Medical Officers will celebrate its 21st anniversary by holding a meeting in London from September 24 to 29. The hosts will be the London Group of the Association, and the Scientific sessions will be held at the London School of Hygiene, which was the venue of the first meeting in September, 1935.

ABSTRACTS

(This section of the JOURNAL *is published in collaboration with the abstracting Journal,* Abstracts of World Medicine, *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

Acute Carbon Monoxide Poisoning—an Analysis of One Hundred and Five Cases. MEIGS, J. W., and HUGHES, J. P. W. (1952). *Arch. industr. Hyg. occup. Med.*, **6**, 344.

A detailed analysis is presented of the records of 105 cases of acute poisoning with carbon monoxide admitted to the Grace–New Haven Community Hospital during the 29-year period 1920–48. The degree of poisoning was mild in 46 cases, moderate in 22, and severe in 37, 7 cases being fatal. No fewer than 98 of the patients showed signs of mental abnormality on admission. Of these, 74 were comatose or semi-comatose, the remainder disorientated, excited, depressed, or apprehensive. Lesions of the central nervous system occurred without apparent pattern, but the presence of incontinence (21 cases) was closely correlated with severity. The colour of the skin was more often cyanotic or flushed (71 cases) than pink or cherry-red (27 cases). Abnormal chest signs, mainly rales or rhonchi, were noted in 52 cases, being usually transitory and noted in different parts of the chest on successive days. Skin lesions developed in 31 patients, commonly an erythema resembling that of a burn. Some patients showed injection of the pharynx or suffusion of the conjunctivae. The heart rhythm was irregular in 18 cases. The findings on abdominal examination were recorded in 100 cases, in 23 of which the liver edge was palpable. Excessive sweating during the first 24 hours and occasionally recurring up to the 28th day was noted in 30 cases, localized pain and soreness in 20, localized oedema in 13, and a tendency to bleed in 11 cases. In 9 cases a remission of symptoms occurred between the 1st and the 4th days, followed by relapse between the 2nd and the 18th days. Of these patients, 4 died and 2 of those who recovered has serious permanent personality changes.

Clinically, these 105 cases could be divided into two groups : (1) In 68 cases poisoning was mild or moderate in degree and the clinical picture was of a rapid pulse, increase in blood pressure, irregular cardiac rhythm, suffusion of mucous membranes, localized oedema, tremor, headache, vomiting, increase in erythrocyte count, and slight glycosuria—signs suggesting disturbed function of the autonomic nervous sytem. (2) In 37 cases poisoning was more severe and the patients showed signs of injury to the central nervous system with associated stimulation of the pituitary-adrenal mechanism, changes in the colour of the skin, skin lesions, chest signs, tachypnoea (30 or more per minute), sweating, enlargement of the liver, localized oedema, tendency to bleed, rise of temperature to 102° F. (38·9° C.) or more, leucocytosis (18,000 or more cells per mm., with a decrease in the lymphocyte and eosinophil counts), albuminuria, and abnormalities in the urinary sediment. The presence of these signs together with those of Group 1 is of serious prognostic significance, all being present in the 7 fatal cases in this series.

M. A. Dobbin Crawford

Inhalation of Sulfuric Acid Mist by Human Subjects. AMDUR, M. O., SILVERMAN, L., and DRINKER, P. (1952). *Arch. industr. Hyg. occup. Med.*, **6**, 305.

Experiments were performed at Harvard University School of Public Health in which, for periods of 5 to 15 minutes, healthy adult volunteers inhaled sulphuric acid mist in concentrations varying from 0·35 mg. to 5·0 mg. per c. metre. Concentrations below the maximum allowable level of 1 mg. per c. metre were undetectable by odour, taste, or irritating effect, whereas a concentration of 3 mg. per c. metre was noted by all subjects, and 5 mg. per c. metre was objectionable, a deep breath causing cough. At all concentrations, however, pneumotachograph records showed that there was a quickening of respiration, breathing becoming shallower and expiration relatively prolonged. With a concentration of 0·4 mg. per c. metre the tidal volume in 5 subjects fell from about 900 ml. to 650 ml. after only 2 minutes' inhalation, and then remained constant until the end of the period of exposure. It then rapidly rose to about 1,090 ml. before returning to its initial level. At higher concentrations the type of response was the same, but it was more rapid and more marked and the return to normal was slower. Exposure to 0·5 mg. per c. metre for 5 minutes caused a marked reduction in minute volume in all cases, but the response was more varied in character since it was now no longer entirely reflex.

The mist particle size was found to average 1 μ and the average retention of inhaled acid was 77% for concentrations between 0·4 mg. and 1 mg. per c. metre.

Retention of the acid was found to decrease as the rate of respiration increased, and it is suggested that the more rapid, shallow breathing observed may be the result of a protective reflex mechanism.

Details are given of the experimental procedure and of the pneumotachograph tracings.

M. A. Dobbin Crawford

Tetraethyl Lead Poisoning. Report of a Non-fatal Case. WALKER, G., and BOYD, P. R. (1952). *Lancet*, **2**, 467.

The authors report in detail a typical case of tetraethyl lead (TEL) poisoning in a foreman who, for 3 years, had been employed in removing the TEL-containing sludge from storage tanks which had contained leaded petrol. The investigations carried out when the patient was admitted to hospital are described, special reference being made to the radiological demonstration of lead in the liver and to changes in the electroencephalogram. The routes of absorption of the TEL, the clinical picture, diagnosis, and treatment are discussed, and short notes on 5 other cases are given.

[This most interesting and lucid article serves to emphasize the importance of occupation as a factor in the differential diagnosis.] *A. Thelwall Jones*

Chronic Toxicity of Ammonia Fumes by Inhalation. WEATHERBY, J. H. (1952). *Proc. Soc. exp. Biol. (N.Y.)*, **81**, 300.

Exposure of male guinea-pigs to an atmospheric concentration of 170 p.p.m. of ammonia for periods of 6 hours per day, 5 days per week, for as long as 12 weeks gave no significant evidence of chronic intoxication. However, such exposure for 18 weeks resulted in relatively mild though definite changes in spleens, kidneys, suprarenal glands, and livers, with severity of the changes being most prominent in spleens and least in livers. Hearts, lungs, stomachs, and small intestines showed no consistent changes suggestive of chronic intoxication.—[Author's summary.]

INDUSTRIAL LUNG DISEASE

Criteria for the Evaluation of the Health Hazard in Dusty Occupations. PARMEGGIANI, L. (1953). *Med. d. Lavoro*, **44**, 65.

In this paper from the University of Milan the author discusses the criteria available for the evaluation of the risk of silicosis in dusty trades. Enquiries based on the present health of groups of employees give information on past exposure, but are of little value in estimating whether a risk is still present. Reliance must therefore be placed on physico-chemical methods of examination of dust ; analysis of the materials from which dust is produced is not sufficient, as the dust may differ widely in composition from the material itself.

The relative value and uses of 15 methods of dust sampling are given. It is concluded that thermal and electrostatic precipitators give the best results in numerical counting of particles, the former particularly for particles of less than 5 μ. in diameter. For the quantitative determination of free silica by chemical methods, the Soxhlet filter paper and the salicylic-acid filter are recommended. Petrographic analysis is only of value where the particles are larger than 8 μ. in diameter ; with small particles the *x*-ray diffraction method gives good results. The quantitative determination of free silica in any sample of industrial dust may be undertaken by the combination of *x*-ray diffraction analysis with chemical methods, preferably by fusion of the sample with potassium bisulphate and subsequent treatment with phosphoric and hydrofluoric acids.

The risk of contracting silicosis depends on many different factors which are summarized as follows : (1) objective factors, that is, nature of the material, or presence of free silica ; (2) environmental factors ; (3) the effectiveness of any methods of prevention in use such as local ventilation or respirators ; (4) human factors, both collective and individual. The estimation of the silicosis risk in a working process must be based on the study of all these factors and must therefore be undertaken by experts in this field. Examples are quoted of various attempts which have been made to establish a maximum permissible dust concentration, but it is concluded that in the present state of our knowledge it is not possible to do so. Investigations are being continued by the U.S. Public Health Service, the Silikoseforschungsinstitut, Bochum, and the Clinica del Lavoro, Milan, and it is hoped to establish figures of maximum allowable dust concentrations for particular industries.

L. G. Norman

Pneumoconiosis due to Cement Dust. DOERR, W. (1952). *Virchows Arch.*, **322**, 397.

The author reports what he believes to be the first recorded case of severe pneumoconiosis attributable to the inhalation of cement dust—in a man of 47 who had worked for 25 years (1921–46) in the same Portland cement works near Heidelberg in very dusty occupations. There was no other history of exposure to dust. The condition was first detected in 1939 as the result of a routine *x*-ray examination, when nodular shadows in the lung fields were observed, but the man was fit and continued at work. In 1946 a radiograph showed massive shadows in the lung fields, and he became progressively more disabled and died in 1951. At necropsy massive fibrosis was found in both lungs, which contained numerous collagenous nodules interconnected by a fibrous reticulum. There was thickening of the pleura, and fibrosis in the regional lymph nodes. The massive fibrosis was collagenous, with hyaline connective tissue : it resembled silicotic fibrosis, but was of a somewhat looser texture and densely impregnated with cement dust. The blood vessels in the fibrotic areas showed endarteritic changes. There were deposits of cement dust in the liver and spleen. There was no evidence of tuberculosis.

The author then describes investigations carried out in an attempt to determine the aetiology of the fibrosis. Incinerated and acid-treated sections of the lung showed profuse dust deposits, the dust particles being, in general,

somewhat elongated, measuring up to 20 μ in length. Dust from the lungs and from the factory was examined mineralogically and by *x*-ray diffraction. The lung ash contained 50% total silica, which is a greater proportion than is commonly found in silicotic lungs. There was only 4% of calcium, compared with 40% in the parent dust, and the author considers that the calcium of the cement dust must have been dissolved away during life leaving a silicate residue behind. *X*-ray diffraction showed small quantities of quartz in the lung, but not in the parent cement dust, and also tracts of clay and mica. The author discusses the question of whether the fibrosis should be attributed to the predominant silicates so that the condition should be regarded as a silicatosis, or whether the disease is a " mixed-dust silicosis ". The presence of quartz in the lung might be accounted for by the presence of free silica in the parent material despite its apparent absence on analysis, or dust in earlier years might have contained quartz ; but the author dismisses these possibilities and concludes that the quartz must have arisen from the " weathering " of silicates in the lung in the course of time. He concludes that the condition must be regarded as a silicatosis (chiefly because of the character of the fibrosis) and that its development in this single case must be attributed to individual susceptibility on the part of the subject, coupled with excessive exposure over many years.

C. M. Fletcher

Printers' Asthma. FLOWER, P. B. S. (1952). *Lancet*, **2**, 755.

This is a report of 32 cases of asthma in printers who used a spray made of gum acacia and isopropyl alcohol. The literature is reviewed. Sensitivity to gum acacia was first described in 1941 by Bohner and others (*J. Allergy*, 1941, **12**, 290).

Following the discovery of the causal allergen in the original case on investigation at the London Hospital, 62 volunteers from the printing trade were examined. Of these, 31 suffered from asthma and a further 14 had early symptoms. Of the 32 men with asthma, 26 gave no past history or family history of allergy before exposure to the spray. The average duration of exposure before asthma developed was 4·8 years. It was not possible to correlate the period of exposure with the amount of spray to which men had been exposed, but all the 32 patients were of the opinion that the severity of their asthma varied with the amount of spray fluid inhaled. The onset was usually gradual, consisting at first of wheezing and cough at the end of a day of exposure to a heavy concentration. There was a tendency to become free from symptoms at the week-end. Occasionally the onset was quite abrupt. The time taken for symptoms to subside after cessation of exposure varied considerably.

It is suggested that the incidence of the condition is higher than may be apparent, as men were (for economic reasons) not always willing to volunteer for examination, and may be as high as 19% of all workers exposed, not including men with early symptoms. The gum acacia process is used only in colour printing, and the risk does not extend to those employed in black-and-white printing. Efficient ventilation will mitigate the hazard, but it can be eradicated only by the substitution of dextrose for gum acacia in the spray fluid, as suggested by Bohner.

L. W. Hale

INDUSTRIAL SKIN DISEASE

Interdigital Sinuses of Barbers' Hands. CURRIE, A. R., GIBSON, T., and GOODALL, A. L. (1953). *Brit. J. Surg.*, **41**, 278.

The authors review an occupational condition which is more prevalent than is usually realized, since the patient seldom seeks medical advice—namely the interdigital sinuses which occur on barbers' hands. At the Glasgow Royal Infirmary they examined the hands of 77 gentlemen's hairdressers, in 10 of whom some stage of interdigital sinus formation was present ; the hands of none of 61 ladies' hairdressers showed any similar lesion. Only 18 cases have previously been recorded, and clinical details of these and of the present authors' 11 cases are tabulated.

The condition is characterized by pits and sinuses on the webs of the fingers which occur most commonly between the fore and middle fingers or middle and ring fingers of the right (cutting) hand. These are due to penetration of the skin by the short, sharp hairs which accumulate between the barbers' fingers, followed by secondary infection and granuloma formation. The mechanism whereby the hairs penetrate to the corium and even to the subcutaneous tissue is discussed. The sinuses are all lined by squamous epithelium, and the microscopical appearances of the various types of sinus are described and beautifully illustrated. Brief mention is made of other hair-bearing lesions, including scarring pseudo-folliculitis of the negro beard, postanal pilonidal sinus, and similar sinuses which have been described in the perineum, axilla, umbilicus, sole of the foot, and suprapubic area, and a plea is made for a more accurate nomenclature.

Simple prophylatic measures would prevent this condition altogether. Once developed, it is an irritating disability which may require surgical excision of a deeply situated granuloma, sometimes followed by skin-grafting.

John Huston

Hop Dermatitis in Herefordshire. COOKSON, J. S., and LAWTON, A. (1953). *Brit. med. J.*, **2**, 376.

An investigation of the incidence of hop dermatitis among workers in the hop-yards in Herefordshire in 1952 revealed that one in every 30 pickers had a mild form of dermatitis. Severe dermatitis was, however, rare, and was associated with oedema of the face and hands and exposed parts. The incidence of this severe form was probably no higher than 1 in 3,000 pickers.

In some cases the dermatitis appeared to be due to a sensitizing ingredient of the hop-oil, which, however, was lost in the process of drying, thus accounting for the rarity of hop dermatitis among workers in dried hops. In some cases a positive reaction to hop resin was obtained.

John T. Ingram