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Section of Public Health, Tuberculosis, Tropical Medicine and Industrial Medicine.[1]

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Vice-Presidents: Professor A. H. Baldwin, M.B., B.S., F.R.A.C.P., D.P.H., D.T.M. and H., New South Wales; G. Cole, D.S.O., M.B., B.S., D.P.H., Victoria; C. E. A. Cook, C.B.E., M.D., Ch.M., D.P.H., D.T.M. and H., Western Australia; A. Fryberg, M.B.E., M.B., B.S., D.P.H., D.T.M., Queensland; C. L. Park, M.D., B.Ch., D.P.H., Tasmania.

Honorary Secretary: D. Gordon, M.B., B.S., Queensland.

President's Address.

Dr. D. R. W. Cowan (Adelaide) took as the subject of his presidential address "The Role of the Voluntary Worker in the Control of Tuberculosis". He referred to the steps taken at the Perth congress to establish an Australian Tuberculosis Association, and he recalled the fact that in speaking to the subject on that occasion he had quoted a statement by William Osler that by "joining actively in the work of local and national anti-tuberculosis societies" medical practitioners could help in the most important and most hopeful campaign ever undertaken by the profession. Gratifying progress had been made with the Australian Tuberculosis Association; voluntary associations had been formed in four States. Queensland and Tasmania had so far done nothing. It was for this reason that Dr. Cowan had chosen his subject. It only required some ardent soul to give a lead and the rest would follow without much trouble. Perhaps an isolated individual could do little, but joined to others in an association, he might be able to accomplish much. Voluntary associations would not embarrass official efforts; the best results would be obtained by a combination of both. All that governments could do was to formulate plans, to provide money and facilities, and to give a lead. Without the help of the medical profession and the people a government could not make its plans effective. Though governments had done much, they had left much undone; they would go on leaving it undone unless urged on by the force of public opinion. Dr. Cowan referred to the report of Dr. M. J. Holmes in 1927 and said that nothing had come of it. Many recommendations had been made by the Tuberculosis Subcommittee of the National Health and Medical Research Council, but none of them had been implemented. A well

[1] The meetings held by the Section of Public Health, Tuberculosis, Tropical Medicine and Industrial Medicine with the Section of Pathology and with the Section of Radiology and Radiotherapy and the Section of Medicine have been recorded

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ance in the prevention and/or limitation of paralysis, and referred to the difficulties associated with a diagnosis of "non-paralytic poliomyelitis". Until it was possible to tell with precision which patients actually were suffering from poliomyelitis, research would be hampered, and at Fairfield patients admitted as suffering from "non-paralytic poliomyelitis" had been found to be suffering from mumps, infective mononucleosis and other conditions. He was convinced of the danger of moving patients over long distances and urged that if patients had to be moved, they should be transported to the nearest base hospital and not to an infectious diseases hospital, perhaps many miles away. Provision of an adequate supply of respirators was important, and, at Fairfield, some two dozen which had been built during the earlier epidemic had been available at the outset of the 1949 epidemic. However, many of them were child respirators and useless in the recent epidemic, when most respirator patients had been adult. Accordingly the policy of his hospital now was to build only adult respirators, which could be adapted for use for patients of any age. Dr. McLorinan could offer no explanation of the shift of the age group incidence in the Victorian epidemics.

Dr. C. E. Cook (Sydney) said that Dr. Cole's paper supported his own experience in the Western Australian epidemic in regard to "age shift" and the higher mortality among country patients, which he thought had perhaps resulted from their transport to Perth over long distances. Many such patients had been sent in ambulances past base hospitals with respirators and had arrived in Perth moribund. Dr. Cook asked had there been any investigation during the Victorian epidemic of the question of mortality among pregnant women, who had contracted poliomyelitis, to which attention had been directed by Snow. In the Western Australian epidemic, of twelve pregnant women who contracted poliomyelitis, six had survived and given birth to female infants, and six had died, five of them bearing male infants, the sex of the sixth child not being recorded.

Dr. Cole, in reply, said that in Victoria no action had been taken regarding food supplies, but the Consultative Council on Poliomyelitis had recommended the boiling of milk, and that recommendation had been interpreted by milk interests as an attack on the industry. It was not considered that ice cream was likely to be a vector, provided it reached the consumer after pasteurization and packaging in a factory, but ice cream dispensed from metal containers in retail shops could be contaminated. The closure of schools had always been opposed in Victoria, unless some special reason existed, and, in the 1949 epidemic, the Education Department had been asked to exercise clemency in those cases in which parents had kept their children from school, and not to admit transferred children to schools until those children had resided for not less than two weeks in the new area. The closing of kindergartens did not interfere with education and parents had been advised to keep young children at home. All schools had been urged to abandon competitive sports on account of the risk of precipitating paralysis in children, who might be in the incubation period of poliomyelitis and in whom exhaustion might have disastrous results. Dr. Cole said that he had no information regarding poliomyelitis in pregnant women.

Dr. McLorinan said that in the Victorian epidemic, of 45 women over twenty years of age who had been admitted to Fairfield with poliomyelitis, 14 were pregnant and three had died, but no record had been kept of the sex of their children.

Occupational Factors in Pulmonary Dust Disease.

Dr. G. C. Smith (Sydney) read a paper entitled "Occupational Factors in Pulmonary Dust Disease". He stated the definition of pneumonokoniosis adopted at the Third International Conference of Experts on Pneumonokoniosis held at Sydney early in 1950, and outlined its scope. Dr. Smith stressed the need for a complete and accurate occupational history in the diagnosis of pulmonary dust disease. He said that the potential capacity of a dust to cause disease depended chiefly on its composition and nature, its particle size, and the intensity of exposure. In addition to dusts containing high percentages of free silica, others from which free silica was absent (asbestos and certain metals) were capable of producing severe pulmonary damage. In most dusts, asbestos being the exception, the harmful particles were those less than about 5-0μ in size. Intensity of exposure, which varied for different dusts, depended on the atmospheric concentrations of dust and on the duration of exposure. Dr. Smith gave a brief account of a case in which silicosis developed rapidly. With regard to the main occupations and dusts associated with a risk of pneumonokoniosis, Dr. Smith mentioned diseases caused by silica, coal and asbestos, and also the radiographically evident abnormalities of certain basalt workers and biograph operators, and the pulmonary effects of talc, graphite, cement, zinc, cadmium, beryllium, manganese, iron, aluminium, synthetic abrasives, cotton, bagasse and wheat. He also drew attention to the occurrence of pulmonary carcinoma in workers exposed to chromates, arsenic and asbestos. Dr. Smith then set out the following nine measures to be applied in the suppression and control of dust and in the protection of workers; he said that they did not vary with the industry or with the type of dust. (i) The abolition of processes creating harmful dust. If that was not possible, the total enclosure of such processes in mechanically ventilated structures, or their segregation from other operations in the factory. Isolation could be applied either geographically or by time—the carrying out of a dusty process when no other workers were in its vicinity. (ii) The use of a harmless material in place of one that was dangerous. (iii) The removal of dust at its point of origin by local mechanical exhaust ventilation. (iv) The use of water or other wetting agents to prevent dust from certain processes from becoming air-borne. (v) Electrostatic precipitation. (vi) The provision of adequate general ventilation to dilute contaminated air. (vii) The maintenance of a high standard of plant cleanliness and housekeeping. (viii) Personal respiratory protection of the worker. Respirators should be worn only when other methods of protection were not practicable, or for jobs of short duration. They should be well-fitting, comfortable and efficient, and should be kept clean. (ix) Preemployment and periodical medical examinations, including chest radiography, of workers exposed to a dust hazard.

Dr. W. E. George (Sydney) said that in investigating cases of pneumonokoniosis, it was important to obtain a detailed record of former employment, as there was a tendency for a man changing from an industry with a silica risk to another non-siliceous industry, to suppress information of his earlier occupation. Unless, too, a careful occupational history was taken, ridiculous errors might arise. He quoted the case in which a radiological report had been received from a large Sydney hospital of "typical pneumonokoniosis", the patient being a bank manager. Speaking of coal miners, Dr. George said that intermittently they were exposed to a high concentration of silica dust, particularly when shaft-sinking, roof-brushing or driving in sandstone, and, as coal raisers, they did not realize the dangers of such dusts. It was at times difficult to estimate the number of years during which a coal miner had been exposed to dust risk, as coal miners worked intermittently at their occupation. Probably a third of the number of years a coal miner said he had worked in the industry could be deducted before an estimate of the actual years of employment was made. Experience had shown that unless there was free silica in the dust inhaled it was unusual for tuberculous complications to develop. Many of the industrial dusts were not of great importance because only small numbers of employees were at risk. In factories the dust problem could be dealt with better than in coal mines, but it was a good working rule to regard all dusts as harmful. At the recent Pneumonokoniosis Conference in Sydney the possibility that free silica might reactivate tubercle bacilli of low virulence had been mentioned, and it was suggested that "if C. might have its virulence raised if subsequent to administration free silica dust was inhaled

DR. W. COTTER HARTEY (Sydney) said that he was interested in Dr. George's last statement, and referred to a patient, an insurance inspector in his late forties with pneumonokoniosis, who twenty years earlier had spent five years in the mines at Kalgoorlie. At times it was difficult to find tubercle bacilli in some fibrotic cases, and he recalled one case regarded as pure silicosis with "a snow-storm X-ray" picture in which two years later tubercle bacilli had been found with no demonstrable changes in the radiological picture. Commenting on the case of the two brothers (biograph operators) quoted by Dr. Smith, Dr. Harvey said that he had examined them both; one brother had emphysema and was dyspnoic and disabled, whereas the other brother with equal evidence of pneumonokoniosis had no emphysema and was fit to continue with his work—an illustration of the evil effects of the development of emphysema in such cases.

DR. ALAN PENINGTON (Melbourne) said that two questions were of importance. One was whether a predisposing infection was necessary before pneumonokoniosis developed. The other was whether removal from exposure prevented progress of the disease. In his own experience it did not.

DR. D. GORDON (Brisbane) said that at times it was difficult to obtain a good industrial history and the industrial hygienist could help the clinician considerably because dangerous pits were known and foundries had been classified in relation to dust risk. There was a tendency for the clinician to associate disease with occupation and to assume, for example, that a painter suffered from lead poisoning, or that a sewer digger was exposed to a dust hazard, whereas, in fact, it might be only certain sections of an industry which were dangerous. Again, an industry carried on in different localities might be dangerous in one and not in another, as exemplified by the fact that sewer constructors in Sydney were working in sandstone, whereas in Brisbane they worked in a schist which was usually damp and offered no hazard.

In further illustration of the difficulty sometimes encountered when an attempt was made to trace the past industrial history of a patient, Dr. Gordon referred to a man with silicosis who worked in an atmosphere contaminated with chlorine; it was at first puzzling as the man denied any possibility of exposure to silica dust, but eventually it was found that at an earlier period he had worked for thirteen years in the abrasive soap industry.

Dr. Gordon said that bagassosis occurred in Queensland, and the main risk lay in the handling of dry bagasse in bulk and, although it was the custom in the cane mills to burn waste in the furnaces, the resultant ash containing about 25% of silica, the exposure of boiler cleaners was limited and intermittent, and they were not subject to any great risk. One type of patient who caused worry was the elderly man, not infrequently a grinder in a factory, who on his first X-ray examination showed almost massive fibrosis, but in whom no tubercle bacilli could be detected. It was not desirable to put such a man off work, but there was always the possibility that he might be harbouring bacilli and be a risk to fellow workers.

Dr. Smith, in reply, said that the points raised by the speakers in reference to the importance of industrial histories emphasized what he had said in his paper. Referring to Dr. George's remarks, Dr. Smith said that it was very difficult to ascertain the actual degree of exposure of coal miners to silica, and there was no doubt that at times they were at risk, but examination of affected coal miners' lungs *post mortem* showed that they contained less silica than was found in classical cases of silicosis. In answer to Dr. Penington, Dr. Smith said that in many cases of silicosis there was a predisposing infective condition, but that it was not a factor essential to the development of the disease and, in his experience, removal from exposure to dust did not arrest progress of fibrotic changes. In conclusion, Dr. Smith said that recently a brick "dragger" carrying bricks from a barrow, and not entering the kiln, had been found to have advanced fibrotic changes with tubercle bacilli in his sputum, and it had not previously been thought that a hazard existed to those handling bricks in such circumstances.

The Diagnosis and Control of Smallpox.

DR. C. L. PARK (Hobart) discussed smallpox. He said that with the advent of aircraft the possibility of the introduction of smallpox into Australia was increased. As Australia was largely an unvaccinated country, the quick control of such a disease would depend on the isolation of patients at the earliest possible moment and on the prompt taking of precautionary measures. Smallpox remained endemic in certain eastern countries, with seasonal epidemic prevalence in the first quarter of the year. The causal agent was a virus, which was stable, and which retained its viability under unfavourable conditions of temperature. It was spread in two ways, directly from person to person, and indirectly through fomites. The incubation period until the development of the rash was fourteen days; the period was irregular in modified smallpox. The invasion period was characterized by an abrupt onset of toxæmic symptoms, which subsided on the appearance of a focal eruption. The eruption was at first macular; then papular, vesicular and pustular stages followed in regular sequence. The last stage was the drying stage when crusts formed; these left a pitted skin surface when the crust fell off. Buccal lesions appeared early, and the rash preferred the upper half of the body to the lower half. It was a rash of the face rather than of the arms and legs; of the distal ends of the limbs rather than the proximal ends; of the back of the trunk rather than the front; of the extensor surfaces rather than of the flexor surfaces. It shunned the most pronounced flexures such as the axillæ. The centrifugal distribution gave the key to the diagnosis, as Ricketts had shown in 1903. The laboratory diagnosis was made (i) by the detection of the virus in scrapings from lesions, and (ii) by the detection of variola antigen in vesicle fluid. The differential diagnosis had to be made from (i) measles in the early stage, (ii) chickenpox, by the distribution of the lesions (chickenpox was a rash of the body, smallpox was a rash of the face), and (iii) drug rashes. The clinical history and the distribution of the lesions would help. Formerly treatment had been by administration of the sulphonamides; these had been superseded by penicillin and streptomycin. With regard to prevention, Dr. Park said that successful vaccination and revaccination would give absolute protection. Administrative control required isolation of patients and vaccination and surveillance of every possible contact. Two complications might follow vaccination, post-vaccinal encephalitis and generalized vaccinia. Encephalitis was probably due to the presence of an unknown virus previously introduced into the body, but quiescent until activated by the vaccinia. It affected only a few countries, and the safest method of avoiding it was to carry out primary vaccination prior to school age. Generalized vaccinia was rare in adults, and nearly always affected children; it affected only those vaccinated for the first time.

DR. CYRIL COOK (Sydney) said that the subject of smallpox might at any time be of great importance to the medical profession in Australia. It was important to determine the values attaching to the work of Wilkinson, of Hong Kong, in relation to vaccination. He claimed that it gave protection for two years and then its effects rapidly waned, first in relation to toxæmia, then to the number of lesions and finally to maturation of lesions. Thus it was possible for a patient with smallpox, vaccinated more than two years previously, to die of toxæmia with no rash, or if contracting the disease at a later period, to have few lesions and yet die of toxæmia.

In Australia there were now a large number of vaccinated ex-service personnel and an outbreak of smallpox might present many diagnostic difficulties, because if infection occurred within the first decade after vaccination, the number of spots on the body might be of little help in diagnosis. Another question for consideration was the so-called "immune reaction". Until recently it had been understood that an early papular reaction following fre