OCCUPATIONAL DISEASES OF THE CHEST, EXCLUDING SILICOSIS.[1]

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EVEN if one excludes silicosis, industrial lung disease is still a tremendous subject and far too large even to attempt to cover in one short lecture. I propose, therefore to limit my remarks to two conditions which have come within my personal experience more frequently than other industrial lung diseases—namely, asbestosis and coal-workers' pneumonoconiosis. Also I shall deal with these from the point of view of the thoracic physician, and not that of the industrial hygienist.

The uses of asbestos have been known for over two thousand years, one of the earliest references to it being by Herodotus in 450 B.C.; he described how the Romans mined it in the Italian Alps and Urals and used it for enshrouding their corpses before cremation. The name "asbestos" is derived from the Greek meaning "inconsumable", and Plutarch referred to the wicks of the lamps of the Vestal Virgins as "asbesta". The dust associated with the handling of asbestos created difficulties in those days as at present, and Pliny in A.D. 50 referred to the use of respirators to avoid the inhalation of dust.

The Australian production of asbestos is not large; it amounted to 2500 tons in 1954, of which all but 450 tons came from Western Australia. This by no means satisfies the requirements of industry, and large quantities are imported, mainly from South Africa. It arrives in sacks as the crushed rock, and men employed in emptying these have been exposed to a high concentration of the dust. Asbestos fibres of extreme fineness can be obtained, spun into yarn and woven into cloth which has a great variety of uses. In addition to being woven, it can be ground and mixed with other materials to make insulating slabs, boarding, brake and clutch linings, electrodes for welding, acid-resisting filtering cloths, packings, jointings and wrappings for steam pipes. The dust given off in the process is mostly less than 5μ in diameter and 10μ in length. As with other dust diseases of the lungs, the occurrence of asbestosis depends upon the concentration of dust to which the worker is exposed and on the duration of exposure. There are, nevertheless, individual variations, as with silicosis, among workers apparently exposed to similar conditions.

Clinically the mode of presentation is with dyspnœa of gradually increasing intensity, often with a cough which is generally non-productive. There are usually no physical signs in the early stages of the disease, but later râles develop, with evidence of impaired respiratory function and cyanosis and clubbing of the fingers in advanced cases. As was mentioned previously, the cough is mostly non-productive, but examination of the scanty sputum produced will often reveal asbestos bodies. These highly characteristic structures are long and tapering, up to 75μ in length, and are a golden-yellow or brown colour. They tend to be segmented, and are of an elongated, dumb-bell shape. They have been shown to consist of a central core of asbestos, upon which a colloidal combination of tissue protein and silica has been adsorbed. This colloidal covering is soluble in strong acids and alkalis which leave the central asbestos fibre bare. Asbestos bodies obtained in a pure state by tryptic digestion of the lungs have been found to consist of approximately 70% organic matter and 30% inorganic matter. They may be found in sputum before there is any radiological evidence of asbestosis, and must be regarded as an indication of inhalation of asbestos dust, but not necessarily of clinical asbestosis.

Radiologically the appearances are fairly characteristic. Usually there is a diffuse haziness throughout the lower lung fields generally with obliteration of the normally sharp margins of the cardiac silhouette (Figure I[1]). Occasionally there are scattered, granular and punctate mottled shadows, but these have not the well-defined margins of the coarser silicotic nodulation.

Histologically, asbestosis produces diffuse fibrosis, starting mainly around the terminal bronchioles and atria as collar-like sheaths and extending peripherally as the disease progresses; it produces profound thickening of the alveolar walls, the interlobar septa and the pleura, and throughout the lung tissue are seen fibres of asbestos either singly or in clumps, as well as the characteristic asbestos bodies. Those parts of the lung not involved in the fibrotic process naturally become emphysematous.

Dyspnœa is the outstanding symptom, and reduction of respiratory capacity will be progressive unless the patient is removed from dust exposure at a very early stage of the disease. This is readily understood; because the primary condition is pulmonary fibrosis which becomes more marked with the passage of time even if the subject is removed from contact with the dust. Pleural thickening and emphysema are natural sequelæ and add to the patient's increasing respiratory disability. He becomes liable to chronic or recurrent bronchitis, and ultimately to the development of cor pulmonale.

From a study of 25 patients with asbestosis, Hugh Jones, John Read and Roger Williams (to be published) concluded that the primary disability was a diffusion defect, as would be expected from the thickening of the alveolar walls. They also found that those patients who had much fibrosis had diminished vital capacities, but no appreciable reduction in maximum breathing capacity unless bronchitis was present.

A patient recently studied was a man, aged 47 years, with a history of exposure to asbestos dust from 1927 until 1956, when he left the industry because of increasing dyspnœa. He had worked mainly as a lagger and asbestos mattress maker with various firms, with a break of four years during which he served with the Royal Australian Air Force. He was dyspnœic on mild exertion, and had a plethoric facies and definite clubbing of the fingers. Medium crepitations were heard over the lower lobes of both lungs, and an X-ray examination of his chest showed the appearance seen in Figure II. It is interesting that the film taken three years ago (1957) was passed as normal, and it was seen from a comparison of these skiagrams that the fibrosis has mainly developed since he left the absestos industry. The respiratory function tests were carried out by Dr. John Colebatch, with the following results. The vital capacity was 3·8 litres (100%), the maximal breathing capacity was 82 litres per minute (73%), and the diffusion capacity for carbon monoxide was 10 (55% of normal).

This shows that, though there is slight airway impairment, the main defect is in the matter of diffusion across the alveolo-capillary membrane.

Last October a group of six men suffering from asbestosis was examined. They had all worked for a period of approximately ten years in what was known as the "asbestos gang", whose work involved emptying bags of imported asbestos into a crushing machine. All complained of dyspnœa and cough of varying degrees of severity. Some had no abnormal physical signs, and in others crepitations were heard at the lung bases. The X-ray appearances were characteristic, and although the men were removed from contact with asbestos dust when the disease was first discovered to be present in them in 1956, there has been progressive fibrosis. There was a fairly good correlation between the severity of symptoms and the results of limited respiratory function tests carried out, but the correlation between these and the X-ray appearances was not close.

Asbestosis is not only a crippling disease in itself, but renders the subject more susceptible to two serious diseases. The studies of Wood and Gloyne and of Middleton and McLoughlin indicate that there is an increased susceptibility to tuberculosis in patients with asbestosis, although this has been questioned by other workers. Studies by Doll, and by Lynch *et alii*, both in 1955, indicate quite definitely that carcinoma of the lung has a higher incidence in asbestosis sufferers than in the general population. This high incidence of bronchial carcinoma has

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 30, 1959.

[1] For Figures I-VI see art-paper supplement.

en confirmed by others and is widely accepted. Doll's alysis has shown that the average risk among men ployed in asbestos for 20 years or more has been ten nes that experienced by the general population.

The second topic I wish to present is coal-workers' neumonoconiosis, and again in the time available only rtain aspects can be dealt with. The coal deposits of w South Wales are the most important and extensively orked in Australia, and are situated in the vicinity of ewcastle, Bulli and Lithgow, being referred to respecvely as the northern, southern and western coalfields. pproximately 13,250 men are employed in these mines; d the extent of the pneumonoconiosis problem can be nged by the fact that at present 870 men are receiving mpensation for this condition (420 total and 450 partial). great deal of work has been done on this subject by the 'al' National School of Medicine at Cardiff, and our no(ge of the pathology of the disease is largely based the observations of Professor J. Gough. In our own ate, pioneering work was done in this field by the late arles Badham and carried on by Dr. W. E. George and bers.

It was once thought that miners who developed pneumononiosis did so because of the inhalation of silica mixed ith coal dust, and that the coal dust itself was inert. R. James has shown that the dust to which the miners South Wales are exposed rarely contains more than 1% ee silica; moreover, in 1000 cases of coal-workers' neumonoconiosis studied by James, 19 subjects had orked as trimmers loading coal into the holds of ships d had never been underground; seven of these had nfluent massive fibrosis. This accords with Gough's 1940) findings, and disposes of the theory that the conition is due to highly siliceous dust from rock strata djacent to the coal seams. Moreover, the pathology is ifferent from that of silicosis.

The development of pneumonoconiosis depends on two roups of factors, one relating to the dust itself and the her to the individual; given identical dust factors with gard to particle size and atmospheric concentration, dividuals vary in the degree and extent of the pneumoconiosis that develops. These factors are difficult to sses but it appears that the efficiency of the nasal filter nd presence of other pulmonary disease, such as ron itis, are important. The claims of metabolic and enetic factors, physiological peculiarities of the lung and s lymphatic system and physiological response to limatic factors and others have been invoked, but none ppears to have been substantiated.

Dust particles greater than 10μ in diameter seldom reach he lung alveoli, either settling out of the atmosphere or cing filtered off by the respiratory tract; however, articles less than 3μ can reach the alveolar spaces in rge numbers. Here they are taken up by phagocytes hich apparently originate in the alveolar walls, and then ither are wafted upwards and finally expectorated, or nter the lymphatic vessels and are transported to the mphoid tissue of the lungs and thence to the hilar mph nodes, from where they may travel to the scalene odes which are accessible to biopsy. Still other phagoytes with their carbon load gather in foci around the espiratory bronchioles, where they constitute the characristic lesion of coal-workers' pneumonoconiosis. These sions are stellate and intensely black, are scattered roughout the lung, and vary in diameter from microcopic dimensions to about 5 mm.; but they contain comaratively little fibrous tissue. *Gough*, by the use of whole-ung sections, has demonstrated these lesions beautifully, howing all gradations of simple coal-dust pneumononiosis.

Gough pointed out the essential difference between ilicosis and coal-workers' pneumonoconiosis, and described he characteristic focal emphysema of the latter. However, it remained for A. G. Heppleston, by painstaking erial section technique, to demonstrate that the proximal mit of this sheath of dust cells is regularly in the region of the terminal bronchioles, but distally extends for a variable distance up to the division of the final order of respiratory bronchiole, of which there may be three, depending upon the degree of dust accumulation. In the course of time the ensheathed bronchioles dilate, producing what is known as focal emphysema. Until Heppleston's work it was generally considered that focal emphysema was the result of obstruction of air passages by dust fibrosis. This histological condition of focal emphysema forms the basis on which it is possible to understand the dyspnœa of simple pneumonoconiosis. As respiratory bronchioles dilate, their cross-sectional area, and therefore their volume, increases in proportion to the squares of their radii. This is equivalent to an increase in the volume of functional dead space, and leads to deficient aeration of the distally placed segments of the airway.

To add to the coal-miners' disability, generalized vesicular emphysema not infrequently develops. This is distinct from focal emphysema, and is characterized by dilatation of alveolar ducts, atria and alveolar sacs. The cause of this condition in coal-miners is the same as that in the majority of non-miners who develop emphysema—namely, chronic bronchitis. Pemberton has shown by a careful comparative study that chronic bronchitis is approximately three times as common in coal-workers as in other industrial groups. In case it could be considered that smoking was a factor, he investigated this possibility and found that a greater proportion of the coal-workers were lifelong non-smokers or had given up smoking, than in the non-mining groups studied. There was also a considerably lower proportion of heavy cigarette smokers among the coal-workers. It would be surprising if the inhalation of dust-laden atmosphere over a long period did not cause such mechanical irritation as would, in the long run, lead to the development of chronic bronchitis.

Radiologically, simple pneumonoconiosis is characterized by fine opacities described and classified in the International System as category 1, 2 and 3. In some cases a condition is superimposed which has been termed "confluent massive fibrosis". This consists of well-marked collagenous fibrosis forming masses varying in size from 0·5 to 15 cm. in diameter. They occur most commonly in the apices of the upper and lower lobes—that is, in positions most favoured by tuberculosis. Although the ætiology of this condition has been disputed for some years, there is now fairly general agreement that it results from superimposed tuberculous infection. James in 1954 was able to find histological and bacteriological evidence of tuberculosis in 40% of the massive lesions present in 245 South Wales coal-workers. Since it is difficult to believe that the bacilli penetrate into existing massive lesions, one must postulate that the organisms reached their situations by being inhaled before or with the dust. These lesions sometimes cavitate, causing the production of intensely black sputum which has been likened to printers' ink. Confluent massive fibrosis has been divided into categories A, B and C on the radiological appearances of increasing severity.

In 1953 Caplan described another form of massive fibrosis, in which the round opacities were multiple and well defined, and distributed throughout both lung fields, but particularly at the periphery. These lesions occurred in coal-miners with a mild degree of simple pneumonoconiosis who also had rheumatoid arthritis, and were shown to be histologically distinguishable from confluent massive fibrosis. The condition has been given the name of "Caplan's syndrome".

In recent years it has become increasingly recognized that severe respiratory disability can exist without radiological evidence of pneumonoconiosis. There is a category 0 in coal-workers' pneumonoconiosis; that is, significant and even disabling disease can exist with an X-ray picture in which recognizable dust opacities are either absent or sparser than in the standard category 1 film. I have seen a number of such patients from the Cessnock mines, and the details of one patient who had a lobe removed because of a suspicious apical lesion will illustrate the point.

The patient was a man, aged 54 years, who had been a miner for 40 years. He had first noticed dyspnœa three years before he was referred to me on June 19, 1958. This had reached the stage at which he said he found it difficult to walk out of the pit, and even the exertion of bathing and dressing caused breathlessness. He had only a slight cough, and the sputum he produced was white and frothy. His chest skiagram in 1956 had been passed as clear, but a current one showed an opacity at the apex of the right lung (Figure V). Tuberculosis was considered the most likely diagnosis; but repeated sputum tests gave negative results, and because of the possibility of carcinoma thoracotomy was advised. Clinically and radiologically the patient appeared to have marked emphysema, and this was confirmed by respiratory function test carried out by Dr. Colebatch. His vital capacity was normal (3·48 litres), but the maximum breathing capacity was considerably reduced, being 36·8 litres per minute (36% of the predicted value). The total lung capacity was 126% of predicted level and the residual volume 161%, giving a residual volume to total lung capacity ratio of 52. He was subjected to surgery, and although a less extensive operation would have been preferred, a right upper and middle lobectomy had to be performed because of technical difficulties. The lesion proved to be tuberculous; but the interest in the case is the fact that examination of the lung sections showed a marked degree of pneumonoconiosis, despite the absence of dust opacities in the skiagram (Figure VI).

The lack of correlation between the radiographic appearances of pneumonoconiosis and the degree of respiratory disability is also exemplified by studying the respiratory function tests of the patients whose skiagrams I used to demonstrate the radiological categories of simple pneumonoconiosis and confluent massive fibrosis. Reference to Table I will show that a man in category 3 can have considerably less respiratory disability than one in category 2; that virtually no respiratory disability may exist with category A, confluent massive fibrosis, and a category C man may have appreciably better respiratory function than one in category B. As long ago as 1948 Fletcher described the case of a coal-worker who was the local champion for the 75 yards sprint, and whose chest skiagram showed progressive massive fibrosis.

TABLE I.

Results of Respiratory Function Tests in Patients with Coal-Workers' Pneumonoconiosis.

Category.	Age. (Years.)	Years in Mine.	Vital Capacity.		Maximal Breathing Capacity.		R.V. : T.L.C.*
			Litres per Minute.	Percentage of Normal.	Litres per Minute.	Percentage of Normal.	
1	53	39	4·0	106	86	77	32
1	50	23	2·8	83	49	53	50
3	59	32	4·6	116	85	81	35
A	63	45	3·8	130	94	106	28
B	70	32	1·8	60	30	24	55
C	60	30	4·1	127	74	73	33

* Ratio of residual volume to total lung capacity.

Conclusions.

From the foregoing, as well as from other published work which time prevents me from including in this paper, the following conclusions must be drawn.

1. The respiratory disability of coal-workers is primarily an airway disease and is due to (*a*) focal emphysema and (*b*) chronic bronchitis and generalized vesicular emphysema.

2. There is no correlation between respiratory disability and the X-ray appearances in the chest.

3. In the matter of determining compensation, the X-ray findings in the chest may be misleading and unjust in two directions: (*a*) a worker with radiologically evident pneumonoconiosis may have no respiratory disability; (*b*) a worker with severe disability due to his occupation may have no radiological evidence of pneumonoconiosis.

4. Tests of respiratory function such as that of the maximal breathing capacity and timed vital capacity, in conjunction with clinical evidence of respiratory disability, provide a better indication of the state of the worker's pulmonary health than an X-ray examination, and should supplement the latter in the regular assessment of the miner.

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HYDATID DISEASE IN A CHILDREN'S HOSPITAL.

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SINCE 1938, 72 patients with hydatid disease have been admitted to the Royal Children's Hospital, Melbourne. Eleven of these were seen for the first time in 1957. The continued prevalence of hydatid disease provided the stimulus for a review of the paediatric aspects of the condition. Information obtained from other centres in Australia indicates that the problem is by no means purely local, because over 1000 patients with hydatid disease have been admitted to public hospitals in the capital cities of Australia during the past twenty years. A further 86 patients with pulmonary hydatid disease have been treated by Dr. S. C. Fitzpatrick of Hamilton, Victoria, since 1920.

Clinical Material.

Sixty-seven of the patients were Victorian, three were referred from Tasmania and two came from southern New South Wales. Thirty-five of the Victorian patients were living in Melbourne at the time of their admission to hospital. Some had previously lived, or spent holiday