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EXHIBIT 164

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AUSTRALIAN BLUE ASBESTOS LIMITED

TELEGRAPHIC ADDRESS
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PRIVATE

Managing Director,
Australian Blue Asbestos Limited,
SYDNEY, N.S.W.

P.626.
24th June, 1960.

Dear Sir,

ASBESTOSIS. We enclose for your information two copies of an extract taken from the British Medical Journal. It is quite an interesting article and reveals several facts not known to us before. There is quite a lengthy clinical description of the case also in this issue of the Journal.

Yours faithfully,

Manager.

GAA/HS.

EXTRACT taken from the British Medical Journal of 30th April, 1960 headed "Complications of Asbestosis".

ASBESTOSIS REVIEWED.

Professor J.McMichael: Dr.Hugh-Jones, would you sum it up?

Dr. Hugh-Jones: The case is of great interest, as it demonstrates so many of the problems associated with asbestosis which we should review. Asbestos itself is chiefly a double silicate of magnesium and iron. It occurs in a number of mineralogical forms, one of the most valuable being chrysolite. Asbestosis is a pneumonoconiosis arising from the inhalation of asbestos dust during the manufacture of asbestos goods, such as asbestos sheeting, fireproof clothing, brake-linings, lagging for boilers and pipes, etc.

One way of thinking of the pneumoconioses is in relation to the chemical toxicity of the different dusts. At one extreme are those, such as iron, which are entirely non-toxic and simply produce a characteristic radiological appearance of the lungs with no change in bodily function. Then come others, such as coal, which do this but also have a pathogenic action by altering the reaction of the lungs to tubercle. Next there are dusts, such as silica, which produce a local reaction in the lungs and pulmonary fibrosis. Finally there are others, such as beryllium, of such toxicity that they can be regarded as general tissue poisons, which produce widespread effects in the body as well as local fibrosis in the lungs. Asbestos is very toxic and produces not only pulmonary fibrosis but reactions in the pleura and even in other organs. Dr.Heard has excellently demonstrated the pathology of asbestosis in our patient, but by way of summary here is a picture (Fig.12) of a whole lung section of another patient who died from asbestosis. There is an interstitial fibrosis, as in the present case, gross pleural thickening and some patchy bronchiectasis. Besides these three features, which are common in asbestosis, there may be associated tuberculosis, as there was in this second case, or, even more important, associated lung cancer, as in our patient. Sometimes the interstitial fibrosis gives rise to the formation of bullae, or occasionally even to a widespread "honeycomb lung" - i.e., that which occurs with other rare forms of pulmonary interstitial fibrosis, such as the xanthomatoses.

RADIOLOGICAL APPEARANCES.

These are typically those of a fine lace-like reticular pattern, particularly affecting the lower lung fields and causing a "shaggy" border to the heart, as in our patient. Besides this, cystic changes may be seen and very often the pleural thickening. Although the X-ray picture is typical and almost pathognomonic in a few advanced cases, there are many patients who have undoubted asbestosis (as judged by a history of exposure and the clinical features of clubbing, rales in the lungs, and typical changes in lung function), who do not have specific changes in their chest radiograph. That is extremely important in relation to the diagnosis, as we shall see later, because in general the diagnosis of pneumonoconiosis depends on an appropriate industrial history and a characteristic X-ray appearance. There is a characteristic X-ray appearance in asbestosis, but unfortunately from the medico-legal standpoint not all patients with asbestosis have the characteristic radiograph. Finally, the radiological appearances of asbestosis cannot be classified in the same categories as those accepted internationally for the other pneumoconioses.

CLINICAL FEATURES AND COURSE.

Gross clubbing of the fingers and persistent rales in the chest are the characteristic clinical signs of the condition. Exertional dyspnoea is the main symptom as in other pneumoconioses. Patients suffering from asbestosis may die either from the complication of tuberculosis (more common in past years), or more particularly from that of carcinoma of the lung. Those not killed by chest infection or cancer may finally get cor pulmonale.

The disease usually makes its appearance rather suddenly, over the course of a few months, often long after the initial exposure to the asbestos inhalation. It has been suggested that the asbestos lies dormant as an asbestos body, which has to "ripen" over many years before it can break down and liberate its toxic contents. In this respect asbestos is again like beryllium, which may also lie dormant in the body and then suddenly produce its effects concurrently with some other infection.

Carcinoma of the lung is a serious and well recognised complication in asbestosis. Its frequency in asbestosis is difficult to determine, for it is now a common condition in the general population. Moreover, exposure to asbestos may have occurred many years before the cancer develops. Nevertheless, the paper by Doll makes it fairly clear that a patient with asbestosis has a risk about 10 times that of the general population of getting carcinoma of the lung. Another hazard is mesothelioma of the pleura. This rather rare tumour may draw attention to the fact that a patient has worked in asbestos dust. Finally, in women who work with asbestos there is a high incidence of cancer of the ovary.

FUNCTIONAL EFFECTS.

There is a diminution of the inspiratory capacity which fits in with the "shrunken lung" demonstrated by Dr.Heard in this case. In contrast with other pneumoconioses there is often little disturbance to air flow, so that all the movement of the lungs there is is used to good advantage, and the maximum breathing capacity (M.B.C.) is often surprisingly well maintained. Thus if a patient suffering from asbestosis is assessed by the M.B.C., or forced expiratory volume (F.E.V.) test, he is unfairly assessed for compensation compared with a man suffering from silicosis or coal-workers' pneumoconiosis. The essential cause of the breathlessness in asbestosis is the difficulty of transference of oxygen across the altered alveolar membrance, more so than the reduced maximum breathing capacity, which is the cause in, say, coal pneumoconiosis. Carbon dioxide, being soluble, diffuses 20 times as rapidly as oxygen and is unaffected. Oxygen transfer is usually adequate at rest, so that the patients are rarely cyanosed, but when they start to exercise there is a rapid fall in oxygen saturation and gross hyperpnoea.

The diagnosis is made on a history of exposure, the presence of clubbing and rales, and usually by means of a radiograph. There is, however, evidence that the radiograph may not show changes as early as do the lung-function tests. Although the latter are not specific to asbestosis, they are only seen in other uncommon diseases which cause alveolar-capillary block and interstitial fibrosis (such as sarcoid, scleroderma, microlithiasis, and other rare conditions). These lung-function tests are useful for making the pathological diagnosis, for, if it can be shown that the patient has an interstitial fibrosis causing alveolar-capillary

block, has gross clubbing and rales, and a history of exposure to asbestos dust, it seems reasonable that he has asbestosis whether the radiograph is specific or no

ASBESTOS DUST

The asbestos bodies simply prove exposure to asbestos dust and of themselves do not mean asbestosis. Anyone may cough up asbestos bodies who has inhaled dust, without necessarily having the changes of asbestosis in the lungs. Vice versa, a patient may have definite and gross asbestosis without asbestos bodies necessarily being found in the sputum during life. Since, medico-legally, the diagnosis depends on a history of dust exposure together with the appropriate X-ray and other changes, the finding of asbestos bodies is of importance in proving exposure.

It seems probable that asbestos fibres, once inhaled, are coated with collage Over the course of years the collagen cover "ripens" by forming transverse cracks, and then the asbestos body can break down, liberating the toxin and causing the reactive fibrosis in the lungs. The nature of the toxin is uncertain. It is, however, presumably the same substance which is carcinogenic.

Asbestosis was first described in this country by M.Murray in 1907. After that, in 1930, Merewether and Price reported the dangers of asbestos dust in the lungs and made recommendations for dust suppression. Thereafter there was a great improvement. In the factory where our patient worked the utmost precautions are now taken to prevent asbestosis occurring. It is a disappearing disease. Nevertheless, asbestos dust is most toxic, and the amount needed to cause asbestosis is not known, so constant vigilance and new preventive methods a needed if this disease is to be abolished.

DISCUSSION.

Professor McMichael. There are many other facets to this patient, butbefore we go on to them are there any other points or queries which could be raised on the pathogenesis of asbestosis?

Dr.R.S.Williams. Ithink there is one point in support of the mechanical theory that is worth raising. If experimentally you give asbestos ground up into a fin dust, fibrosis does not occur, and it seems that asbestos fibres between 20 and 5 microns in length are necessary to produce the typical peribronchiolar fibrosis.

DISTRIBUTION OF FIBROSIS.

Professor E.G.L.Bywaters. What is the explanation of the distribution of fibrosis? Does it correspond to the distribution of asbestos in the lung, or is there some other explanation?

Dr.Heard. As a matter of fact, it doesn't seem to correspond. Although some of the best asbestos bodies in this case were in fibrosed areas, there were plenty surrounded by perfectly normal lung in other parts. Dr.Engelsmith, fro Sheffield, was here two days ago and he was saying that they have recently examined chemically a number of lungs from cases of asbestosis. In some there

was a lot of fibrosis and practically no asbestos. However, he did point out th
there was a possible error because the asbestos content was estimated as the
percentage of the dried weight and the dried weight of fibrosed lung is not
altogether comparable to the dried weight of the normal lung. It is quite a
problem, and, so far as I know, the peripheral distribution of the fibrosis has
been explained. It is not restricted to asbestosis, being seen in other forms
of honeycomb lung.