ASBESTOSIS*

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Asbestosis has been brought to the forefront of industrial medicine in this country by a number of recent publications.

Smears were made from the cut lung surface of cases from the autopsy service in Cape Town. A relatively high percentage of cases showed the presence of asbestosis bodies in the smears.[1] From this finding it has been suggested that the air is becoming increasingly polluted by eventually 'indestructible and dangerous' asbestos fibres.[2]

Mesothelioma of the pleura has been considered to be a rare tumour, but over 100 cases have been diagnosed in South Africa. Almost all of these cases have had an association with the North-Western Cape or the asbestos coming from there.

THE INCIDENCE OF ASBESTOSIS

Asbestos of different types is used, unprocessed or as yarn, cloth, felt, tape, paper, millboard, in cement and in other different compounds. The *unprocessed material* is used in the chemical industry, in filters or for caulking; the yarn, in the manufacture of brake linings, clutch facings and gaskets; *the cloth,* in fireproof clothing, tapestry, blankets, theatre curtains, and oven linings; the felt, in acoustic and sound installations; *the tape,* in armature winding, conveyor belts and oil wicks; *the paper,* in linings for stoves and heaters; and the *millboard* for insulation. The *asbestos cement* is used in the manufacture of roofing, tiles, shingles, wallboards, in water pipes; and the various *compounds of asbestos* in the manufacture of battery boxes, in stucco paints, plastic appliances and in the ceramic industry.

It is difficult to obtain definite or comparable figures of the incidence of asbestosis in these different industries as diagnostic criteria vary from one country to another and in different parts of some countries. Such figures as are given vary from about 15 - 35% of employees examined. Such incidence figures are given in relation to the number of people examined at a certain time, and are often not related to the population at risk, which is indeed difficult to assess. Many reports only record the number of cases detected.

Because there were not accurate figures of the incidence of asbestosis and some of its possible complications, the Pneumoconiosis Research Unit of the CSIR approached the Asbestos Industry, the National Cancer Association, the Department of Mines, and the Department of Health, in order to carry out a survey of the asbestos areas. Financial assistance and moral support were most willingly given.

The results of the survey are shown in Table I, which shows that a certain percentage of the people living in the vicinity of the asbestos mines or mills may have asbestosis, and an explanation for this must be sought.

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TABLE I. RADIOLOGICAL CHANGES CONSISTENT WITH ASBESTOSIS

	Males		*Females*	
	Exposed	*Not exposed*	*Exposed*	*Not exposed*
Whites	1	2	0	0
Bantu	37	27	2	1
Coloured	24	5	9	2

Figures expressed in percentages.

The diagnosis of the cases found during the survey was made mainly on a radiological examination, where even without any evidence of disability, the appearance of the film showed features consistent with the diffuse fibrosis of asbestosis or when calcified pleural plaques were seen.

Many of the methods which are used in the milling or processing of asbestos preclude the use of water. In milling, for example, the asbestos fibre is separated from the grit by hot air. Although mining and milling conditions have been improved in the last few years, there was a period when the effluent from the mills *did* pollute the air in which people not employed in the industry lived or worked. In addition, some years ago the dangers of the inhalation of asbestos dust were not fully realized, even by research workers and industrial hygienists. The great binding property of asbestos grit was utilized in the making of roads and in certain places to make school playgrounds.

Cases of asbestosis have also been found in factories not using asbestos in any process, but situated close to factories which were. In such cases, every effort was made by the experienced people to exclude any known exposure to asbestos dust.

Professor Thomson and his co-workers[1] have shown that in 500 consecutive autopsies in subjects of 15 years or over, in a number of cases asbestos bodies were found in smears made from the bases of the lungs. It is suggested that asbestos dust is liberated into the urban air as a result of the increased use of asbestos materials. It is admitted, however, that the occupational histories, when available, were meagre, and it is commonly accepted that in order to assess the industrial exposure of a person to a hazard, the examiner must have full knowledge of the different processes involved.

One of the results of the survey mentioned confirmed that asbestos bodies could be found in the sputa of people with abnormal and with normal radiological films. This shows once more that the presence of asbestos bodies is indicative only of exposure to asbestos dust, and also poses the question of the relationship of the asbestos body to the interstitial fibrosis.

PATHOGENESIS OF ASBESTOSIS

Recently Beattie and Knox,[3] examining ashed lung tissue from cases of asbestosis, have shown that a smaller percentage of fibres over 15 μ in length is found in the severe degree of asbestosis than in cases of minimal or moderate asbestosis, and in those cases with asbestos

exposure but no evidence of asbestosis.

Beattie[4] considers that the 'asbestosis body' will eventually break down, but that usually this is a slow process and that fragmentation can occur without the development of fibrosis. When, however, the break-down is rapid, owing to pulmonary infection or congestion, fibrosis occurs. As the fragmentation of the body will also include the fracture of the central fibre, an explanation is offered by the smaller numbers of long asbestos fibres in severe degrees of fibrosis.

Another explanation must be considered and that is that the most fibrogenic particles of asbestos are not the long fibres but rather the smaller particles. This would suggest that the fibrosis of asbestosis is caused by the fine particles and not by the asbestos body.

These particles, being silicates, may produce the type of fibrosis found in the lungs of nepheline workers,[5] which is indeed similar to that of asbestosis. But this concept will require a great deal of investigation.

It is probable that accurate records of service, correlated with the composition of dust retained in the lungs, will be of help, particularly in those cases with known exposure and no radiological evidence of asbestosis.

PATHOLOGY OF ASBESTOSIS

The first change which is found in the lungs owing to the inhalation of asbestos is an aggregation of dust-laden phagocytes in the region of the respiratory bronchioles and below the pleura. The dust which reaches the interstitial tissue causes an initial interstitial fibrosis in those regions. The fibrosis extends to the adjacent alveoli and associated alveolar ducts.

The fibrosis involves more and more of the alveolar ducts and alveoli supplied by the respiratory bronchiole and eventually there is no normal alveolar tissue between adjacent respiratory bronchiolar segments. Thus a histological picture of widespread diffuse fibrosis is produced.

Although it has been stated that asbestos affects mainly the lower zones of the lungs, the upper lobes may be affected without much fibrosis of the lower lobes.

270 cases have been examined at autopsy and 38 (12%) showed the presence of areas of massive fibrosis, but this was not necessarily related to the severity of the interstitial fibrosis.

Active tuberculosis was found in 44 (24·4%) out of the 204 cases examined by Dr. J. C. Wagner[6] and in 21 out of a series of 90 cases examined by the Unit since his departure. In this latter group more cases of active tuberculosis were found in the moderate and severe degrees of interstitial fibrosis.

The evidence of active tuberculosis in relationship to asbestosis in one of the areas surveyed, is given in Table II. The relationship of tuberculosis to the peculiar bilateral calcified pleural and diaphragmatic plaques of asbestosis has not been established and their pathogenesis remains obscure. In association with asbestosis they have been reported by Kiviluoto[7] and this has been confirmed not only by Wagner[6] but also in this series. In 3 cases the plaques were fibrous in nature and were found on the parietal pleural surface and on the diaphragm. It is of interest that similar calcified plaques have been reported as occurring in talc miners by Siegal and his co-workers[8] and in people working with mica by Smith.[9] Both of these substances, like asbestos, are fibrous silicates.

TABLE II. THE RELATION OF ASBESTOSIS TO ACTIVE TUBERCULOSIS

	Active TB	*No active TB*	*Total*
Asbestosis	37 (17%)	175	212
No asbestosis	115 (7%)	1,577	1,692
Total	152	1,752	1,904

In addition to the presence of asbestotic plaques, 50% of the cases described here showed the presence of a chronic non-specific pleurisy which in some cases produced marked thickening of the pleura.

MALIGNANCY IN ASBESTOSIS

There can no longer be any doubt that there is an increase in the incidence of malignant disease of the lung in cases of asbestosis. In this series 20% of the cases showed the presence of malignant neoplasia of the lungs and of these 16 cases of malignancy described here, 7 were bronchogenic carcinomata, 4 multicentric bronchiolar carcinomata and 5 were mesotheliomata of the pleura.

TABLE III. MALIGNANCY IN ASBESTOSIS EXPRESSED BY DIFFERENT AUTHORS

Author	*Pulmonary malignancy**
Stoll, Bass & Angrist[10]	13·2
	7·5
	14·8
Bonser, Faulds & Stewart[11]	M 26·1
	F 8·7
Merewether[12]	13·2
Jacob & Bohlig[13]	12—17
Wagner[6]	18

*Expressed in percentage.

It does seem probable that asbestos dust can produce malignant change at different levels of lung structure, namely bronchi, bronchioles and pleura. Although the bronchogenic carcinomata can usually be distinguished from the other two with accuracy, it is sometimes difficult to be certain whether a tumour is primarily of mesothelial origin or bronchiolar, as the mesothelioma can spread into the lung parenchyma and the multicentric bronchiolar tumour to the pleura.

The presence of hyaluronic acid in the mesothelioma has been used to differentiate the 2 types of tumours. For this the specimen should be preserved in a fixative containing formalin, alcohol and acetic acid or it could be done on a frozen section. Many specimens were preserved in formalin and in these the absence or presence of hyaluronic acid could not be established.

In South Africa there are different types of asbestos, of which the main ones mined and milled are crocidolite, amosite and chrysotile. Crocidolite is found mainly in the North-Western Cape. There are also deposits in the Northern Transvaal, as well as those of a closely related asbestos, known as Transvaal Blue and the big and only known source of amosite asbestos. All three of these are asbestos of which the main component is an iron silicate and they are associated with a banded ironstone.

Other deposits of blue asbestos are to be found in Western Australia and, according to Conn,[14] in the Labrador troughs of New Quebec in Canada. Prospecting in these latter beds has not yet started. In Canada, New-

foundland, Russia, Rhodesia and in the Eastern Transvaal there are deposits of a magnesium silicate asbestos known as chrysotile.

Wagner, Sleggs and Marchand[15] described a number of cases of mesothelioma of the pleura in people associated with the North-Western Cape asbestos fields, or with the asbestos mined there. Over 100 cases in which the histological features are consistent with those of a mesothelioma of the pleura are on record and in only 3 is there no connection with this area or the asbestos from it. It must be mentioned that in some cases the association with the North-Western Cape was very short indeed, being days or a few weeks.

Cases of mesothelioma of the pleura have recently been reported from Belfast, Southampton, Liverpool, Newcastle, Edinburgh and Portsmouth, and Wagner[16] considers that they may have been exposed to asbestos in the repair or building of ships. This has been suggested in the cases from Belfast.[17]

Keal[18] has reported a number of cases of peritoneal mesothelioma at the London Hospital where there may have been exposure to asbestos dust. Such mesotheliomata have also been reported by Wagner in his series of cases with some association in the North-Western Cape.

No case of mesothelioma has been found from the Northern Transvaal or from the chrysotile area of the Eastern Transvaal. However, 4 cases have been found in Canada in people exposed only to chrysotile asbestos.[19]

Deaths from lung cancer ICD 162 have been registered in Whites of the magisterial areas in which the amosite and chrysotile asbestos mines are situated.[20]

Intrapleural inoculation of animals with the different types of asbestos has produced a small number of tumours with histological and histochemical appearances similar to those of a mesothelioma. Also most of these have been found after inoculation of crocidolite, but one has been produced by chrysotile and another with silica.

There are, therefore, many unsolved problems in regard to the incidence and development of mesothelioma in asbestosis, which may be summarized as follows:

(*a*) No cases have yet been found from the Northern Transvaal, although crocidolite is to be found there.
(*b*) Cases have been found in people only exposed to chrysotile (Canada).
(*c*) Bronchogenic and bronchiolar carcinomata have found with exposure to all 3 types of asbestos.

In regard to the carcinogenic agent a further problem arises. If the hydrocarbons found in crocidolite and amosite asbestos are to be incriminated in the production of mesothelioma, why have there been no cases from the amosite area and why are there cases of bronchogenic carcinoma and mesothelioma found in people with only chrysotile exposure? Only trace amounts of the hydrocarbons have been found in some specimens of chrysotile.[21]

CONCLUSIONS

The pneumoconiosis produced by the inhalation of asbestos dust still presents a large number of problems, and the main ones appear to be:

(*a*) The particular property of the dust which causes fibrosis and how it does this.
(*b*) The length of exposure or dust load necessary to produce such fibrosis.
(*c*) The reason for the increased incidence of pulmonary malignancy in asbestosis.

It is a disease produced by the inhalation of dust and as such can be prevented. The managements of the asbestos industry have introduced many procedures to prevent aerial pollution in the last few years and the principals of these applied to manufacturing concerns using asbestos will reduce the contamination of the urban atmosphere. Under such conditions an ever-increasing atmospheric dust hazard, as has been suggested by some workers in South Africa, is not anticipated.

Reference to the survey carried out by the Pneumoconiosis Research Unit of the CSIR, and reproduction of the Tables, is made with the consent of the Director of the Unit.

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