Asbestos is a mineral that is used commercially in three main forms — chrysotile or white asbestos, crocidolite or blue asbestos, and amosite or brown asbestos. All three forms of asbestos were used at Acre Mill, Hebden Bridge. The consumption of asbestos has rocketed both nationally and internationally since the end of the First World War (Table 1).

|                                | 1920  | 1976  |
|--------------------------------|-------|-------|
| UK usage of asbestos (in tonnes)| 20,738| 143,300|
| World output of asbestos (in tonnes)| 205,000| 4,874,000|

Asbestos has many important and valuable uses in textiles, pipe and boiler lagging, cement products, brake linings, clutch facings, thermal insulation and in ships. Since 1970, the importation of blue asbestos into the UK has been discontinued on health grounds.

In 1939, the Cape Asbestos Company opened a factory named Acre Mill at Hebden Bridge near Halifax. This plant was engaged in the carding, doubling, weaving, and so on of white and blue asbestos, much of which was stored in permeable hessian sacks. Asbestos was used mainly in the manufacture of textile products but also in asbestos pipes and ropes, respirator filters and insulation material. The factory was closed in 1970. In the 31 years of its operation approximately 2,200 workers were employed. At first, labour was recruited mainly from the indigenous population of the Calder Valley near Halifax. At the end of the Second World War a considerable number of European Displaced Persons were employed. After 1956 there was an influx of refugee Hungarians, followed by a wave of Pakistani immigrants. So far, 262 workers, 12 per cent of the labour force at Hebden Bridge, have developed asbestosis of the thorax (lung or pleura), although there may be some difference of clinical opinion on some of
these patients. This compares with 1.05 per cent who have been certified as suffering from asbestosis found among all the workers in the same company's factories throughout the UK. Of the 262 workers who contracted asbestosis of the thorax, 77 have died of asbestosis or a complication of this disease. Of these, 44 (57 per cent) died of lung cancer or mesothelioma, 18 (24 per cent) of heart failure, cor pulmonale or pulmonary thromboembolism, 11 (14 per cent) of bronchopneumonia, and 4 (5 per cent) of asbestosis.

This dreadful human balance-sheet probably underestimates the true situation, as a number of European Displaced Persons and Hungarian political refugees who worked at Acre Mill have emigrated to North America and Australia, and many Pakistani workers from this factory have returned to the Indian sub-continent. They cannot be followed up and their subsequent health is therefore unknown.

Six members of one extended family contracted or died of this disease and, to date, over £2,000,000 have been paid by the company in compensation to the victims of asbestosis or their dependents from the Halifax area.

DEPARTMENTAL JOBS AT ACRE MILL AND PARA-OCCUPATIONAL ASBESTOSIS

One hundred consecutive workers (69 males and 31 females) who developed thoracic asbestosis were carefully analysed (Table 2). They were employed in a variety of different departments in the factory and, in the course of their employment, many were engaged in two or more departments.

| Nature of job                         | Number of employees |
|---------------------------------------|---------------------|
| Disintegrator, stripping and grinding | 2                   |
| Doubling, opening and carding         | 18                  |
| Spinning, weaving and winding         | 47                  |
| Pipe-making                           | 12                  |
| Bandsaw                               | 3                   |
| Rope-making                           | 7                   |
| Warehouseman                          | 13                  |
| Other workers                         | 11                  |

An important observation was that 11 'other workers' among 100 consecutive employees who developed thoracic asbestosis did not actually handle asbestos. These consisted of 2 gardeners, 3 office workers, 2 painters, 2 lorry drivers and 2 canteen workers employed at Acre Mill. None were actually engaged in any stage
of asbestos production. They were therefore only minimally exposed to asbestos dust. The duration of employment in the industry of this ‘para-occupational’ group that developed asbestosis was twice as long as that of the employees who developed asbestosis while working closely and continuously with asbestos fibre. There is clearly a real hazard of contracting asbestosis among ‘para-occupational’ workers not directly engaged in the handling of this mineral.

CLINICAL SYMPTOMS (Table 3)
The average interval between initial exposure to asbestos and the development of symptoms was 17.6 years, but there were wide variations in time scale. In one worker, symptoms developed in as little as two years after first exposure, while in a number of others symptoms did not start until they had been working at Acre Mill for 30 years. It will be seen that in a number of patients, two or even three different symptoms presented simultaneously.

| Table 3. First symptoms of one hundred consecutive asbestosis patients |
|---------------------------------------------------------------|
| Dyspnoea                                                      | 78 |
| Chest tightness or pain                                      | 39 |
| Cough                                                        | 36 |
| Lassitude                                                    | 25 |
| Haemoptysis                                                  | 2  |

CLINICAL SIGNS
The most frequent sign was bilateral basal crepitations. In three patients, despite a history of occupational exposure and definite radiological and physiological evidence of parenchymal disease, crepitations were not detectable even at periodic examinations up to two years after a definitive diagnosis. Hence, it appears that clinical signs may very occasionally follow as well as precede radiological evidence of parenchymal asbestosis. It should also be noted that crepitations heard repeatedly over a period of months may diminish and almost disappear with the slow development of a ‘shell’ of pleural asbestosis. A crepitation can be defined as a crisp note heard at the height of inspiration, which is not cleared by coughing and can always be heard at the same site when the patient is in the same posture. If the crepitations are caused by asbestosis, there may be additional evidence, such as a history of occupational exposure, clubbing of the finger nails, chest X-ray abnormalities and a reduction in gas transfer on lung function testing. Unilateral crepitations were occasionally detected, and when they were, they were accompanied by radiological parenchymal disease on the same side.
ASBESTOSIS, LUNG FIBROSIS, AND HL-A ANTIGENS

One of the most fascinating features many investigators in occupational health have noted is the extraordinary variability in the severity of pulmonary and pleural disease among asbestos workers who are exposed for equivalent periods of time to approximately similar amounts of asbestos dust. Lung damage can, apparently, vary from trivial disturbances of lung function, with minor radiological pleural damage, to gross physical incapacity with major impairment of respiratory function and extensive chest X-ray shadows.

It has been suggested that host factors may determine the individual response to asbestos dust. Turner-Warwick and Parkes (1970) noted an increased frequency of antinuclear antibody and rheumatoid factor in subjects with asbestosis. Later, Merchant and his co-investigators (1975) found that W27 was three times more common among the asbestos workers they investigated than among normal controls. Of the 10 patients with W27, 6 had moderate to severe asbestosis. However, they found that the distribution of ANA and rheumatoid factor did not seem to be associated with W27 or any other antigen. These results were not confirmed by Evans and his colleagues (1977), who found no significant difference in the prevalence of antigens in 37 patients with asbestosis, 37 matched controls with equivalent asbestos exposure, or a control group who had not been exposed to asbestos. In fact, they observed that asbestos workers without pulmonary fibrosis had an unexpectedly high frequency of HL-A BW5 and suggested that this antigen might protect against the development of pulmonary fibrosis. At present, therefore, one must conclude that the individual pleural and lung reactions to asbestos dust are variable and the precise role of HL-A remains elusive.

RADIOLOGICAL CHANGES IN ASBESTOSIS

There are many well-documented radiological patterns described in pulmonary asbestosis (Solomon, 1970). These are most commonly fibrosis and parenchymal shadowing in the lower portions of the lung fields (Sheers and Templeton, 1968; Anton, 1968; Soutar et al., 1974; Mackenzie and Harries, 1968; Harries, 1976). Pleural thickening was regarded as being caused by asbestosis if it was radiologically greater than 3 mm in thickness, over 5 cm in length, and was located on the axillary or diaphragmatic lung surfaces (Fig. 1). It was occasionally accompanied by calcification on the diaphragm, heart and lungs. In some patients pleural thickening was unilateral and did not advance, but in other cases there was progression over the years into bilateral pleural fibrosis without further occupational exposure having taken place. The definition of pleural asbestosis was strictly reserved for asbestos workers who had the degree of pleural thickening mentioned above and had never been exposed to trauma, pulmonary tuberculosis, or chest infection. Pleural changes, which are often difficult to recognise in the
postero-anterior position can frequently be more readily identified in oblique views taken at 45 deg. or retrosternally in the lateral chest X-ray.

Table 4 attempts to correlate the different types of radiological damage caused by asbestos dust with the duration of exposure to the offending mineral and with the age of the patient. Although 37 per cent of these cases sustained pleural damage exclusively, pleural asbestosis alone is not recognised as certifiable by the Pneumoconiosis Medical Boards. Radiological pleural thickening alone appears to represent an early and mainly benign form of thoracic asbestosis. Generally, workers with this type of pleural reaction and damage due to handling asbestos are either asymptomatic or suffer minor disability. It would, therefore, seem reasonable to include pleural thickening or fibrosis within the definition of certifiable thoracic asbestosis when it gives rise to definite disability on the part of asbestos workers. As a broad generalisation, the longer the duration of direct exposure of the worker to the asbestos dust, the more likely was the finding of pulmonary rather than pleural radiological asbestosis. The commonest type of

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Table 4

| Radiological changes in thoracic asbestosis | Duration of exposure (years) | Mean age |
|--------------------------------------------|-----------------------------|----------|
| Pleural                                     | 37                          | 51       |
| Pulmonary                                   | 22                          | 57       |
| Mixed pleural and pulmonary                | 41                          | 53       |

abnormal radiological chest X-ray changes noticed (41 per cent) was a mixed pleural and pulmonary asbestosis. Perhaps less familiar is the very occasional development of bullous cyst formation in asbestos workers. Two examples, one of which was confirmed at postmortem, were found among the 262 cases of asbestosis.

THE NORMAL CHEST X-RAY IN ASBESTOSIS

Williams and Hugh-Jones (1960) described asbestosis as occurring with a normal chest X-ray in asbestos workers with a reduced gas transfer factor. This finding has been corroborated by Kreeel (1976, 1977) in whole body computer tomography EMI scanning and also by Stanley (1976) in regional pulmonary ventilation studies. Four such cases of asbestosis with normal chest X-rays were found at Acre Mill and all were confirmed histologically at biopsy. Thus, the inescapable conclusion is that asbestos workers with lung tissue evidence of asbestosis, abnormal breathing tests, and suffering from breathlessness and fatigue, may be found to have a normal chest X-ray.

ASBESTOSIS, BRONCHITIS AND BRONCHIECTASIS

A number of workers with asbestosis complained of either a dry or a slight productive cough with white or grey sputum. These symptoms were worse in winter and were exacerbated by intercurrent infection. All these patients with asbestosis demonstrated a reduction in FEV₁ sec. and FEV₁ sec./VC that was partially improved by an antispasmodic drug. Initially, these patients were regarded as bronchitics whose condition was caused by cigarette smoking. However, nine workers suffering from asbestosis who had never smoked tobacco or had chest infections previously gave a history of several years of morning cough with occasional sputum. On pulmonary examination, all had abnormal adventitious sounds and a reduced FEV₁ second. There is therefore some evidence for concluding that parenchymal asbestosis can cause bronchitis or obstructive airways disease. This view is also shared by Rodriguez-Roisin (personal communication) who found a decrease in flow volume of 50 per cent among 40 cases of asbestosis.
Among this series of cases of asbestosis there were four who gave a history of trivial streaky haemoptysis noticed on two or three occasions during their working life. Initially, these patients with radiological evidence of linear streaking at the lung bases were regarded as being cases of infective bronchiectasis, and bronchograms were performed. These showed evidence of bronchiectasis. None of these patients had a history in childhood or adult life of measles, whooping cough, pneumonia or chest infection. In the light of this evidence and Leathart’s (1960) work in the same field, the possibility of pulmonary asbestosis causing bronchiectasis as well as asbestos bronchitis by lung fibrosis and scarring warrants further investigation.

CANCER AND ASBESTOSIS
A high incidence of cancer is found in patients suffering from pulmonary asbestosis. Martin (1970) of the Department of Health and Social Security and Buchanan (1965) declared that more than half the patients certified at death as having asbestosis have associated lung cancer. An editorial in the British Medical Journal (1976) reiterated this view and indicated that at least 60 per cent of those workers accepted by the Pneumoconiosis Medical Boards as being disabled by asbestosis have subsequently developed cancer of the lung. Even more striking was the finding of Selikoff et al. (1968) that the combination of asbestos exposure and cigarette smoking increased the risk of lung cancer 92-fold compared with non-smokers without occupational asbestos exposure. Newhouse and Berry (1976), Wagner (1960) and Elmes and Simpson (1971) have reported patients who had suffered from pleural asbestosis dying of mesothelioma. In this series of cases, at least three patients with pleural asbestosis subsequently developed mesothelioma and died. The presumed benign nature of pleural asbestosis therefore warrants fresh appraisal. There is no doubt that, as a generality, there is a quantitative relationship between the amount of asbestos retained in the lungs and the subsequent development of lung cancer or mesothelioma. However, it is facile to assume that mesothelioma and bronchial cancer can occur only in the presence of substantial quantities of asbestos in the lungs, with some associated interstitial pulmonary fibrosis.

In fact, a number of investigators (British Medical Journal, 1964; Hourihane, 1964; Wagner et al., 1960; Elmes and Wade, 1965; Selikoff et al., 1964) have indicated that mesothelioma may be associated with quite trivial asbestos deposition in the lungs.

The following two patients highlight two significant features of this problem. The first is the manner in which minimal exposure to asbestos can cause a mesothelioma. The second is that, as detected by optical microscopy only, a few asbestos bodies and very slight pulmonary fibrosis can be responsible for lung cancer in asbestos workers. Mr K. G., aged 35 years, was employed for only nine weeks stacking asbestos materials on a conveyor belt. A chest X-ray was taken
eight years later and showed extensive pleural and pulmonary damage. He died of a mesothelioma at 48 years of age.

Mr H. S., who undertook the stripping and lagging of pipes and boilers, died at 65 years of age of an undifferentiated oat-cell bronchial carcinoma. No asbestos bodies were found in most of the histological sections; in others, very few asbestos bodies were noted, and in only one section were six asbestos bodies observed, accompanied by a very slight degree of interstitial fibrosis. The Pneumoconiosis Medical Board accepted that the histological evidence was sufficient to confirm the diagnosis of mild asbestosis, which caused death by lung cancer. Radiologically, there was definite evidence of two plaques of calcification due to asbestos in the right diaphragm, in addition to a large bronchial carcinoma.

One of two assumptions is therefore possible in this second patient. Either we are dealing with a patient who inhaled marginally more asbestos than the ordinary urban resident and simply died of lung cancer, just as he might have ended his days with a coronary thrombosis; or it is possible, as Edge (1976) has suggested, that relatively small additional amounts of inhaled asbestos can in some instances cause lung cancer. To summarise, there is a quantitative relationship between the amount of retained asbestos fibre in the lungs of asbestos workers and bronchial carcinoma. However, it is cautiously suggested that occupational exposure to asbestos, even in the presence of minimal asbestosis, may cause lung cancer. The question to be posed at the end of the day is whether the biological reactions that give rise to asbestosis are identical with those producing cancer. Furthermore, cancer may develop in organs other than the lungs or pleura following exposure to asbestos. For example, four deaths due to cancer of the ovary were reported by Newhouse et al. (1972) in female workers who had 'severe and long exposure to asbestos dust'.

Again, cancer of the stomach, colon and rectum were found by Selikoff et al. (1965) to be three times as frequent as expected among asbestos laggers in the white male population of the USA. This same hazard of cancer of the gastrointestinal tract was found by Enterlin and Kendrich (1967) and Elmes and Simpson (1971) among a group of Belfast insulation workers. Libshitz et al. (1974) and Shettigara and Morgan (1975) in the USA have reported that the larynx is another site of cancer in employees working with asbestos.

**ASBESTOSIS CAUSED BY A PLACE OF WORK OR RESIDENCE**

A number of investigators have observed radiological abnormalities such as pleural calcification among farming communities in Bulgaria (Zolov et al., 1967) and Czechoslovakia (Hromek, 1962; Rous and Studeny, 1970). In the absence of an asbestos industry in the affected areas of these two countries, the cause of pleural calcification was assumed to be the presence of asbestos in the soil in the form of anthophyllite. Similar reports of radiological evidence of pleural calcification have
been noted in Finland among farmers and their wives by Kiviluoto (1960), who concluded that the cause was pollution of the atmosphere by an anthophyllite asbestos mine in the neighbourhood. Hourihane et al. (1966) in London documented six patients found at post mortem to have pleural plaques of calcification who had lived within a radius of half a mile of an asbestos plant. At Hebden Bridge, four residents have evidence of pleural thickening. None of these residents, or their families, have ever worked with asbestos. All four lived or worked within two miles of the Acre Mill factory for a period of more than 25 years (Fig. 2). None had a history of empyema, pulmonary tuberculosis, chest injury or any other cause that might have produced pleural thickening or calcification. It is therefore difficult to avoid the conclusion that these four residents developed some degree of pleural damage simply by living for over 25 years to windward of the factory that contained asbestos fibres. It must be
emphasised that hundreds of residents in this area have had X-rays that have shown no radiological abnormalities that could have been caused by asbestosis.

ASBESTOS TIPS
Old quarries are favoured sites for disposal of unusable industrial asbestos waste. In the past, there was a danger of the asbestos being partially weathered in winter and then carried by the wind during the dry months to adjacent homes and schools. This potentially serious hazard has been controlled by a code of practice agreed in 1972, in which it is laid down that each tipped layer of asbestos shall be covered by nine inches of 'consolidated earth', a condition that should ensure a high degree of safety in residential areas adjacent to tips.

There were four asbestos tips near Acre Mill. Tip No. 3 contained scores of tons of asbestos waste and it was located only 150 metres from a nearby primary school. Six 60 g soil samples were taken from a footpath running over this asbestos tip, and in every sample substantial amounts of crocidolite and/or amosite were discovered on or just below the soil surface. Clearly, this footpath was dangerous; it has now been closed. Similarly, considerable public concern was expressed about the safety of a residential area close to the Acre Mill factory where asbestos products had been manufactured from 1939 to 1970. There were two grazing fields adjacent to this factory and six years after the closure of the factory thirteen 60 g soil samples were taken from the perimeter and/or centre of each field. No asbestos fibres were found in eight sites; and in the remaining five sites a few short asbestos fibres were detected. In one area only, close to the Acre Mill factory, one long fibre and a few short asbestos fibres were detected by optical microscopy. It must be emphasised that all these investigations were conducted by optical microscopy. Neither X-ray diffraction nor electron microscopy was employed. In the light of recent work by many investigators (Timbrell, 1973; Nicholson and Pundsack, 1973) these findings must be interpreted with caution.

SAMPLING FOR ATMOSPHERIC ASBESTOS DUST CARRIED OUT NEAR AN ASBESTOS TIP
An attempt was made to assess the atmospheric hazard to school-children caused by Asbestos Tip No. 3 in the vicinity of a neighbouring primary school where dumping had ceased 15 years previously. These investigations were conducted in conjunction with the Department of Consumer Protection. Five sampling points were designated at varying distances between the asbestos tip and the school playground 150 metres away. The measuring instruments consisted of a pump and a filter assembly, and each pump was set to draw two litres of air per minute through the filter for three hour periods. In no instance was a single asbestos fibre detected on the filter membrane. Thus, after a 15-year interval, there was no detectable atmospheric hazard to the school-children from the asbestos dump revealed by optical microscopy.
Asbestos is an important, essential, yet dangerous mineral that is required in a highly industrial society. The tragic outbreak of asbestosis at Hebden Bridge devastated the health and even the lives of a considerable number of employees, and had a profoundly depressing effect on the morale of the entire community. The lessons of this study will not, I hope, be lost on all those concerned in the asbestos industry.

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