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Asbestosis Following Brief Exposure in Cigarette Filter Manufacture[1]

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Abstract. Severe asbestosis was found on lung biopsy in a 47-year-old woolen mill worker, who, 16 years before, was exposed to asbestos dust for a period of only 9 months. He made cigarette filters which consisted of a mixture of Cape Blue asbestos and acetate. The presenting symptom of dyspnea on exertion and classic radiographic changes first became evident 13 years later. Characteristic pathophysiologic findings of restrictive insufficiency and diffusing impairment were demonstrated. Pulmonary insufficiency progressed over a 2-year period to total disability.

The myriad products containing asbestos are reviewed and the dangers of exposure in secondary industries using these products are emphasized.

Key Words
Asbestosis
Cigarette filters-asbestosis

The rapidly escalating world consumption of asbestos, a useful but dangerous 'magic mineral' [1], presents one of today's most important occupational health hazards [2, 3]. Early descriptions of asbestosis related the disease to prolonged and intense exposure, mainly in mining and asbestos manufacturing industries [4, 5]. Today, asbestos is a common ingredient of innumerable building, paint, automotive, textile, filtration and other products (table I). Inadequate labeling, careless handling and unsupervised use of such materials may represent greater occupational hazards than exposure in the asbestos industry itself and, indeed, it may lead to worrisome environmental contamination [6]. The following case illustrates the dangers of even very brief exposure, the apparently 'benign' occupations where asbestos may be a serious hazard, and the importance of detailed occupational history-taking.

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Table I. Common materials containing asbestos

Textiles
Fireproof curtains, insulation, conveyor belts, safety clothing, textiles for ladies dresses and coats, pot holders, ironing board covers, draperies and rugs, motion picture screens, filters for gas maks, mail bags, prison cell padding, aeroplane fittings, stove and lamp wicks, spark plugs, fire hose, yarn, tape and rope, filters for processing fruit juice, beer, acids and medicine, cigarette filters.
Paper and felt
Roofing, piano padding, stove lining, heaters, filing cabinets, military helmets, mufflers and hoods, carpets, cartridges, boiler jackets, radiator covers.
Mill board
Acoustic ceiling, plaster board, wall board, electric switch boxes, safes, table pads, stove mats, ovens dry kilns.
Friction material and gaskets
Automotive brake linings and clutch facings, engine gaskets, seals, power shovels, hoists, other industrial machines, cloth packing.
Cement and building materials
Shingles, siding, interior and exterior walls, clapboard, insulation board, casings, pipe, roofing and siding tar, cements and coating, plaster, stucco.
Floor tiles
Vinyl-asbestos tiles, asphalt tiles.
Sprays and fireproofing
Structural steel, lagging, automobile undercoating.
Miscellaneous
Paints, ceramics, pottery, sculpture clay, sealants, adhesives, road building, putties, caulking, crack fillers, artificial snow, welding rods, fibrated greases, magnesite floors, battery boxes.

Case Report

G. S., a 47-year-old, white woolen mill worker, was admitted in August 1968, complaining of increasing dyspnea for 2 years. Slight cough and sputum disappeared after he stopped smoking 6 months previously. The past history was negative for respiratory disease. During a single, brief hospitalization following an auto accident in 1965, a chest roentgenogram was reported 'negative for fracture, effusion or pneumothorax'.

Initially, he stated that he had worked continuously in woolen mills for 26 years. However, on close questioning following the discovery of asbestos bodies in the sputum,

Table II. Serial lung function studies (ambient air)

	Determined		Predicted
	8/68	9/69	
I. Mechanics and lung volumes			
Maximal breathing capacity, l/min	98	69	118
Vital capacity, l	2.1	1.9	3.8
Timed vital capacity, % 1 sec	68	73	>75
Residual volume, l	1.1	1.1	1.2
Total lung capacity, l	3.2	3.0	5.0
II. Alveolar gas, arterial blood, rest			
Alveolar O_2 pressure (P_AO_2) mm Hg		100	104
Arterial O_2 pressure (P_aO_2) mm Hg		82	95
A-a O_2 difference, mm Hg		18	10–20
Arterial CO_2 pressure (P_aCO_2), mm Hg		38	40
Diffusing capacity, ml/min/mm Hg:			
Steady state, CO		8.3	>15
Single beath, CO	16.0	15.4	28
Fraction CO removed, %		33.4	>50
Absolute shunt, % of cardiac output		10	<4

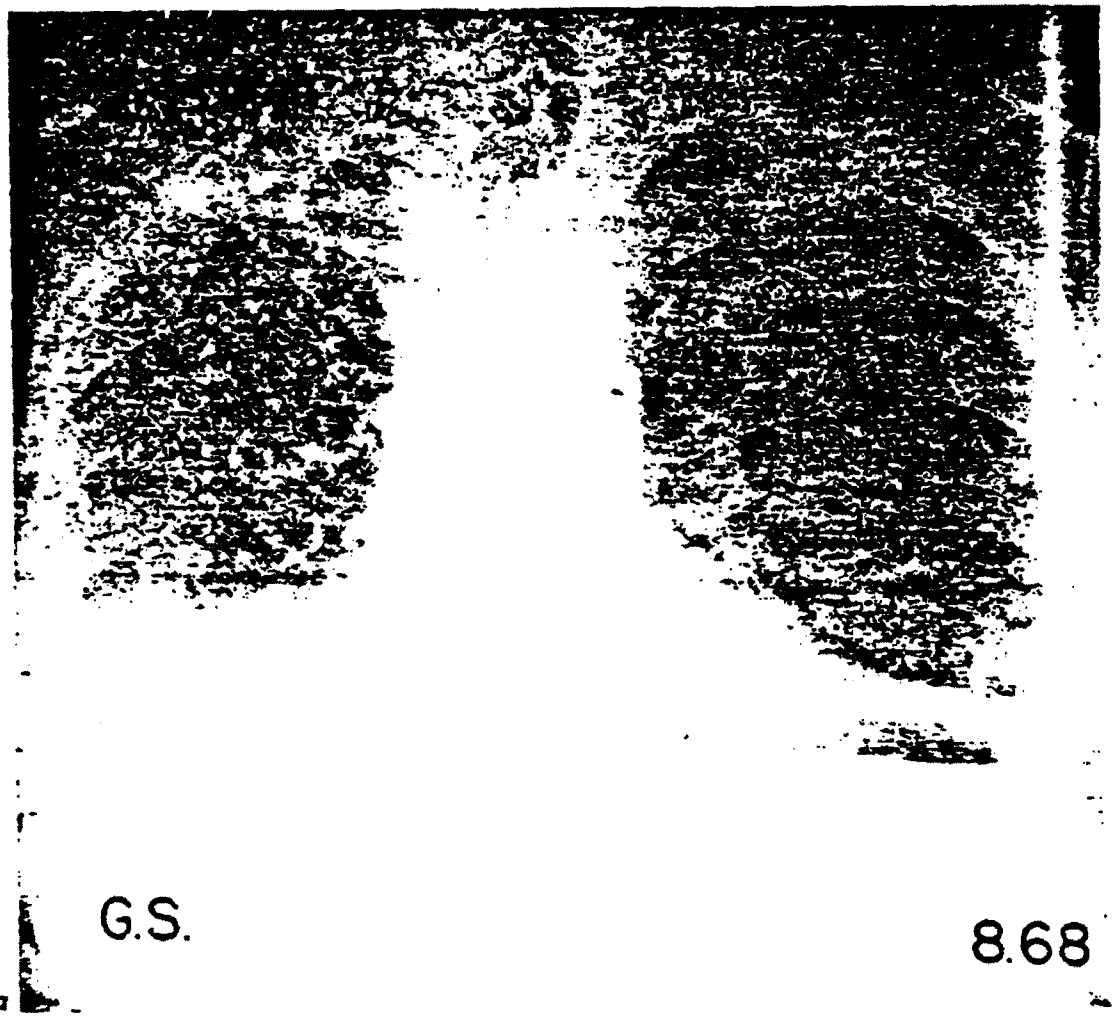
he recalled that, in 1953, he had worked for 9 months in a factory which made cigarette filters containing asbestos. For the next 14 years, chest roentgenograms, taken at 3-year intervals in the woolen mills, were reported as normal. From 1965 onwards films taken at 6-monthly intervals showed elevation of the diaphragm with progressive loss of lung volume.

On physical examination there was marked finger clubbing. Chest expansion was diminished and the diaphragm was high with limited excursion. Fine, crackling 'cellophane' râles were heard over the bases. There was no evidence of heart disease, and the examination was otherwise normal.

The chest roentgenogram at this time showed 'small lungs' (fig. 1). The diaphragm was elevated and, on full inspiration, it reached only the 8th ribs posteriorly. There was a dense linear and reticular parenchymal infiltrate, more marked in the lower zones. Characteristically, the outlines of the heart and diaphragm were ill defined, and there was marked bilateral pleural thickening.

Laboratory studies, including hematocrit, sedimentation rate, serum enzymes, latex fixation, LE preparations, liver and renal function tests, as well as EKG, were within normal limits. Skin tests for tuberculosis and fungi were negative as were sputum cultures for aerobes, anaerobes, acid-fast bacilli and fungi. Asbestos bodies were readily demonstrated in the sputum.

Lung function studies (table II) were performed by methods previously described [7]. There was a marked restrictive impairment with vital, timed vital and total lung capacities reduced to 50% of predicted. Residual volume, FEV_1/FVC and the mixing index were in the normal range. Resting ventilation and the ventilation equivalent for oxygen were



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moderately increased. The alveolar-arterial PO_2 difference at rest was at the upper limit of normal with an arterial PO_2 of 82 mm Hg. The patient was unable to exercise. Diffusing capacities for carbon monoxide by steady state, single breath and fractional uptake techniques were all reduced; and the "absolute shunt", measured while breathing pure O_2, was increased.

One year later his chest roentgenogram was unchanged (fig. 2) and physiologic studies showed increasing restriction.

Lung biopsy in August 1968 revealed a greatly thickened pleura and a solid, rubbery lung. Sections showed advanced pleural and interstitial fibrosis (fig. 3). Clusters of siderophages filled some alveoli and a few asbestos bodies were seen in these spaces and in the adventitia of bronchioles (fig. 4).

Discussion

Asbestos has been used for more than a thousand years. Its outstanding qualities of incombustibility, indestructibility and flexibility, as well as its

Fig. 1. The postero-anterior chest roentgenogram (A) shows small lungs with only 8 posterior ribs visible. There is a dense lineal infiltrate which is more marked in the lower zones. The outlines of the heart and diaphragm are ill-defined and there is diffuse pleural thickening. The lateral film (B) emphasizes the high diaphragm, basal infiltration and perhaps a pleural plaque anteriorly.

adsorptive properties have led to its use in an ever-widening range of products (table 1). This is reflected by the rising world consumption, from 300 tons in 1870, to 4 million tons in 1968 [1].

Uses of asbestos. A meaningful occupational history requires some knowledge of likely asbestos-containing products, because workers may be una-

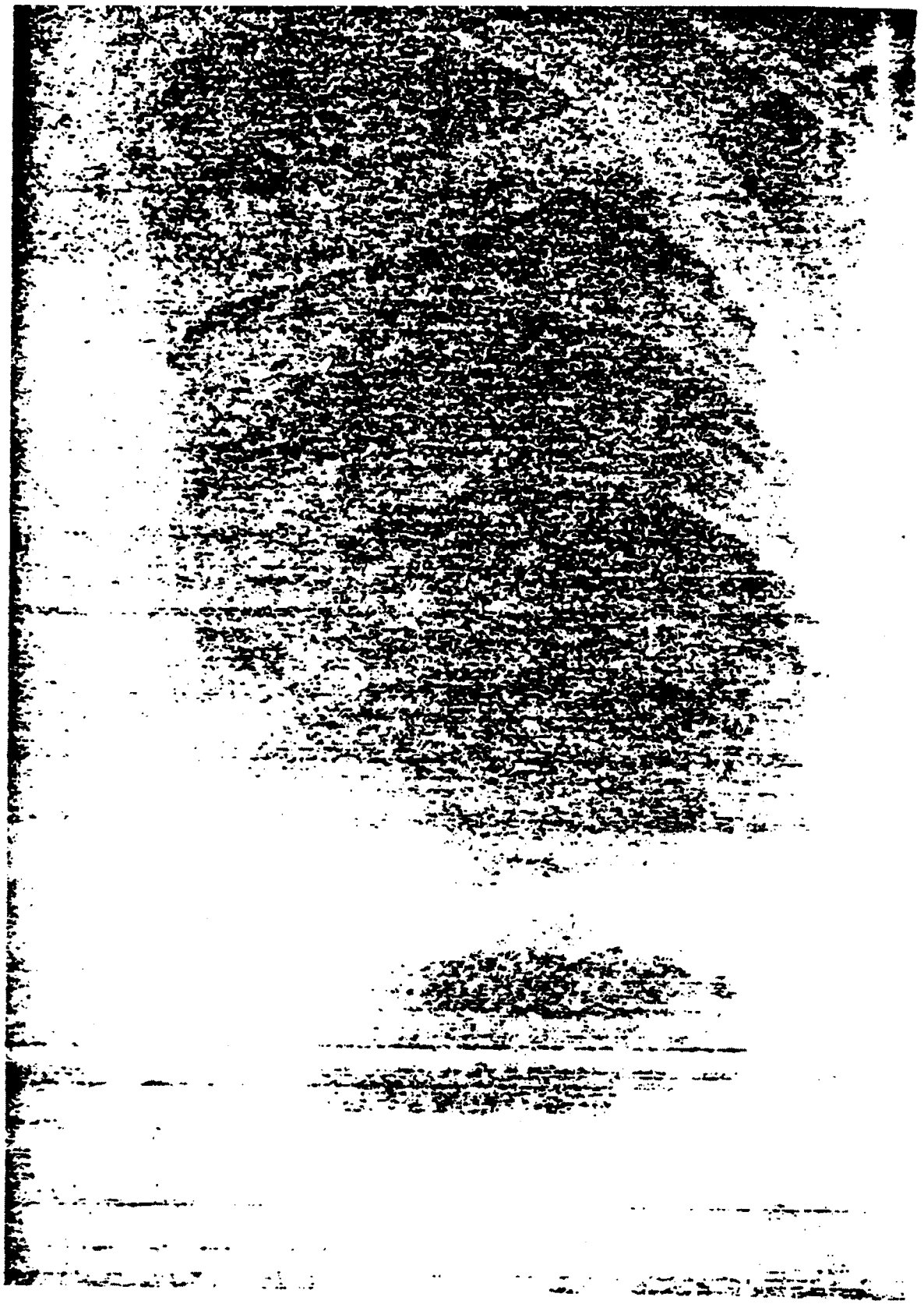

Fig. 2. A magnified view of the left lung, one year later, shows the diffuse, fine linear or reticular pattern of infiltration in more detail. Diffuse pleural thickening reduces the contrast between lung and soft tissues.

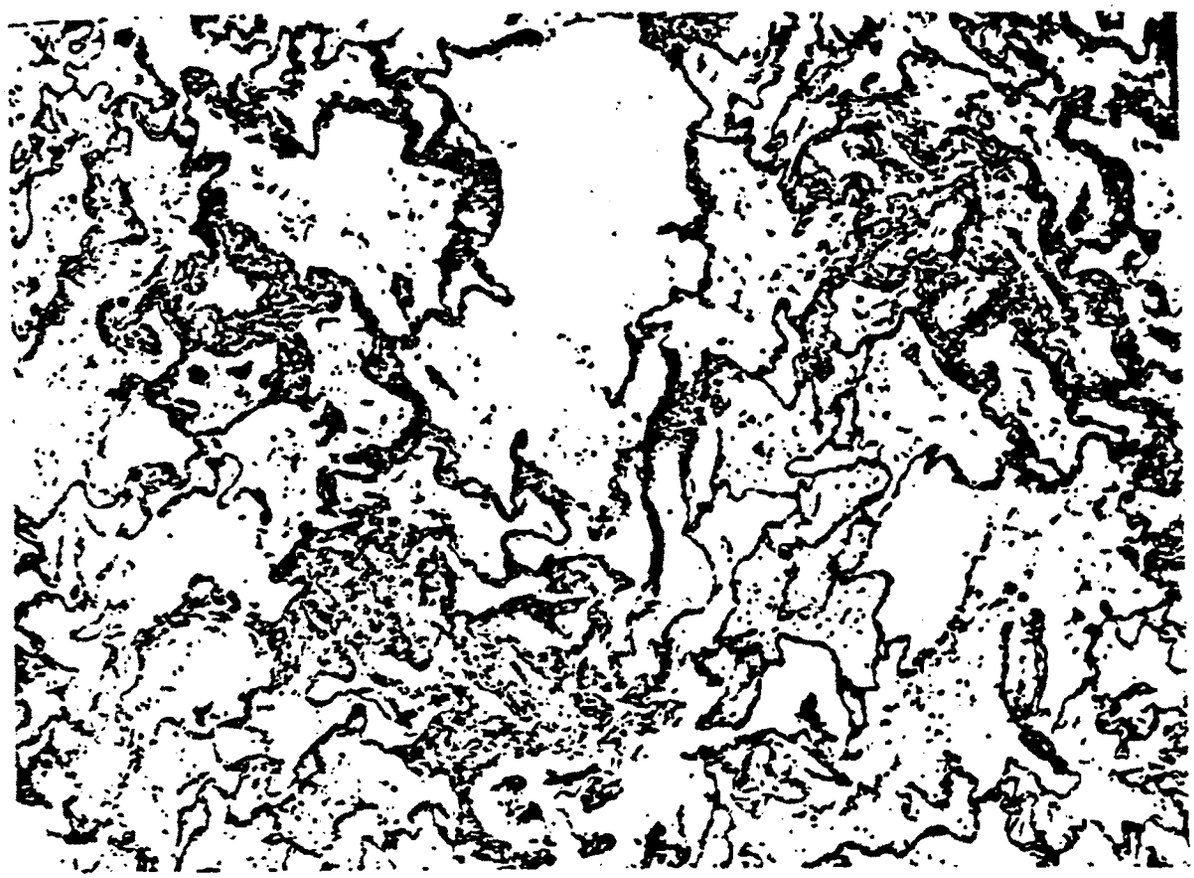

Fig. 3. In the lung biopsy from the lower lobe there is interstitial deposition of collagen in some alveolar walls and marked focal fibrosis. Several alveolar spaces contain nests of hemosiderin-laden macrophages and asbestos bodies; the latter are seen more clearly in figure 4. Hematoxylin and eosin 50 ×.

ware both of the constituents of the materials that they use and of the dangers of asbestos. More than 3,000 such products have been mentioned [1] some of the best known of which are listed in table I. The heaviest consumers are the cement, floor tile and insulation industries, with increasing adaptation in textiles, paper, felt, mill board, and friction materials. Generally recognized sources of exposure have been mainly in mining, processing, spinning, weaving, insulation and asbestos goods manufacturing. However, many products of this industry contain asbestos in friable form and their subsequent use, as in our case, may relate to crafts and trades where the hazard is quite unsuspected. If the mineral is left in exposed form then it may cause continued exposure of the end user [8]. Indeed, air pollution from these sources has been of increasing concern [6]. Finally, sources other than com-

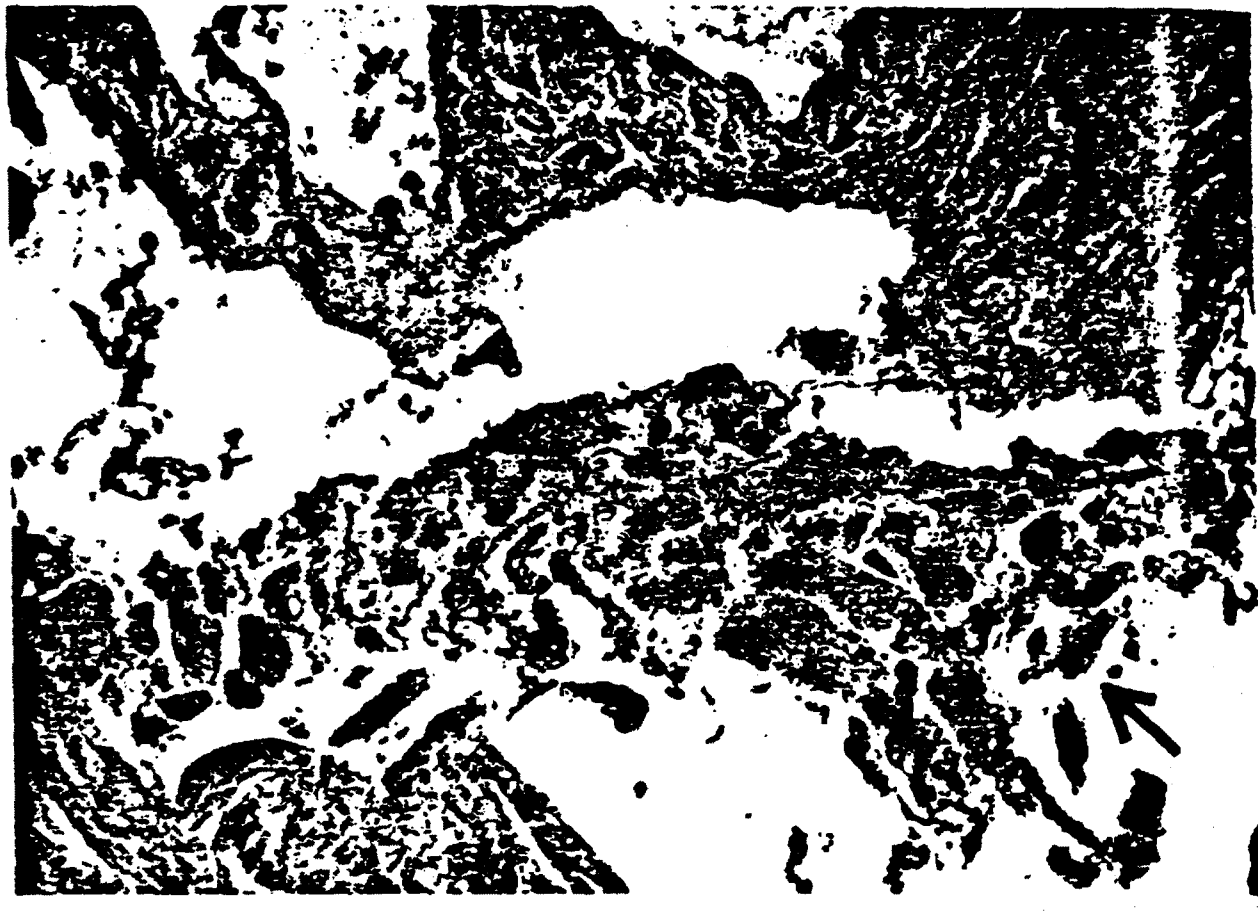

Fig. 4. Between the markedly thickened alveolar walls is a collection of macrophages which contain many golden-brown granules and some asbestos bodies. Several other partly fragmented asbestos bodies lie free in the alveolar space at the lower right. The arrow marks an asbestos fiber which is slightly coated by ferritin granules. Hematoxylin and eosin 500×.

mercial asbestos are important in producing pulmonary complications. Commercial talc, the annual consumption of which now exceeds that of asbestos [2], commonly contains asbestos amphiboles, including tremolite and crysotile, which may cause pulmonary fibrosis [9, 10].

Duration and type of exposure. The importance of dust concentration was first emphasized by MEREWETHER and PRICE [4] who showed that the prevalence of asbestosis was consistently higher in the dustiest occupations. For example, within 5–9 years, 44% of workers developed asbestosis with severe exposure while only 6.7% did so with slight exposure. One result of these observations was the introduction of safety measures in terms of dust concentration in 1931. 8 years later, SAYERS and DREESEN [5] gave the first useful information for calculating threshold limit values: they showed clear-

cut asbestosis among those exposed to concentrations exceeding 5 million particles per cubic foot (mppcf) with none at lower concentrations. More recent work has suggested that exposure even below this level is hazardous if it continues for more than 10 years [11, 12].

Actually, as with other pneumoconioses, the amount of asbestos dust in the lungs is largely related to the *concentration of dust times the duration of exposure*, a factor often expressed in mppcf-years. This was recognized by MEREWETHER [13] almost 40 years ago when he reported in 1,512 workers incidence rates of 1.0, 5.6, 13.4, and 53.2% asbestosis after 5, 10, 20 and 20 + years respectively. He realized, however, that the inference from these figures, namely, that so long as the period of exposure did not exceed 5 years, the risk was negligible, was wholly untenable. He went on to say, 'the fact is that work in a dense concentration of asbestos dust of a comparatively short period will lead inevitably to the development of a profound fibrosis, provided the worker lives long enough for it to develop'. He estimated that the 'maturation period' for the trapped dust to cause extensive fibrosis was 7 years at least. He went on to describe a man who made asbestos mattresses for 4 years and died of asbestosis 8 years after leaving this work. Another, a mixer died 12 years after working with asbestos for only 2 years and 7 months. Since then, asbestosis generally has been described after a minimum of 4–5 years exposure though occasionally, shorter periods have been recorded. For example, a husband and wife developed signs and symptoms of severe asbestosis 20 years after building 2 bungalows from asbestos sheets and living in one of them, which remained unpainted, for 2 years [8]. Undoubtedly, many such cases are missed (1) because, by definition, the exposure was brief and often was not identified with the usual trade of the person, (2) considerable time, probably from 5 to 10 years must elapse between exposure and the development of fibrosis, and (3) clinical and radiologic evidence of interstitial fibrosis does not become evident until the disease is quite severe.

Occupational history taking Our patient illustrates the hazards existing in occupations not generally known to be related to asbestos. More and more such instances are being reported, for example, automobile undercoating, brake lining manufacturing, filter making, and floor tile laying. Increasingly, 'neighborhood cases' of asbestosis are being reported in workers who did not handle asbestos themselves but who worked in close proximity thereto. Electricians, steamfitters and general laborers working in holds of ships refitting are particularly prone to this type of exposure. We have recently described a bricklayer with asbestosis and asbestos pleural effusion, who

worked for only 4 years on highrise apartments. Laggers, working several floors above him had sprayed asbestos on steel beams. The laggers were protected by masks. but he never suspected the danger of the gray dust which continually floated from above and settled on his bricks [14].

Our patient highlights the great importance of detailed occupational history taking. He described himself as a woolen mill worker for 26 years. He recalled working in the filter factory only following repeated and detailed questioning. subsequent to a review of his chest roentgenograms and the finding of asbestos bodies in the sputum. Asbestos was not mentioned until he gave a detailed description of his work and of the materials which he handled: For 9 months he transferred raw asbestos by hand from bags to carding machines. The atmosphere was very dusty and he always wore a nose mask. This mask became clogged frequently and was cleaned by blowing the dust into the air outside the workroom. After 9 months, the filter manufacturing process was transferred to a tobacco company and he returned to the woolen mill. His chest roentgenogram was first reported abnormal 13 years later when he began to have mild dyspnea on exertion; and 15 years later there was advanced pleural and pulmonary fibrosis with marked physiologic disturbances. We know of other men who had worked in the same plant on the same project who have died of asbestosis.

The diagnosis of asbestosis depends upon the occupational history and on classic signs and symptoms. These include dyspnea. finger clubbing. fine dry râles with a close-to-the-ear sound, radiographs showing linear densities more marked in the lower lung fields, with ill-defined cardiac and diaphragmatic outline. often assiciated with pleural thickening, plaques, and pleural calcifications [15] and physiologic abnormalities of restriction and impaired respiratory gas exchange. A simple industrial history often does not reveal the problem. A careful description of the type of work, the tools and the materials used may uncover a totally unsuspected and unpredictable exposure.

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