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CHAPTER 12

ASBESTOSIS

Asbestos has been used for at least 2,000 years. Pliny[415] A.D. 23-79 in his *Naturalis historia* described its properties and spoke of the uses of asbestos fabric. "A linen has now been invented that is incombustible. It is called 'live' linen and I have seen napkins made of it and burnt more brilliantly clean by the fire than they could be by being washed in water. The linen is used for making shrouds for royalty which keep the ashes of the corpse separate from the rest of the pyre. The plant grows in the deserts and sunscorched regions of India where no rain falls, the haunts of deadly snakes, and it is habituated to living in burning heat." But the material was rare: "When any of it is found it rivals the prices of exceptionally fine pearls. The Greek name for it is *asbestinon* . . . this kind of linen holds the highest rank in the whole of the world."

Asbestos is mined by blasting and quarrying. Between 2 and 10 per cent of the mined rock is fibre. The crude asbestos of commerce contains 5–20 per cent of rock, dust and fine fibre which is removed in opening and spinning. The long-fibre yarn (chrysotile fibre-length ¾-inch or more, amosite up to 6 inches) is used for textiles, packing gaskets, felted paper and for goods made from asbestos cement where maximum strength is desirable. Asbestos of medium fibre-length goes into paper and board, filter pads, brake blocks, brake linings and asbestos cement. The short-fibre (fibre length about ⅛ to ¼ inch) material is useful only for paint, plaster and asbestos cement. Asbestos is used extensively as a mineral filler in plastics.

The dangerous nature of asbestos dust was recognized only recently. In this country the first case of asbestosis to be described was that of a man who died in Charing Cross Hospital in 1900; the second case was reported 25 years later.[416] By 1928 there were still no more than ten cases on record.[417] The recognition that a pneumoconiosis was caused by the inhalation of asbestos dust followed a few years later, after several other cases had been described in England[418] and a heavy incidence had been observed in some German factories.[419, 420] About this time Hunter[421] saw 30 cases of asbestosis at the London Hospital in 5 years. Wyers[422] has shown

that a sharp decline in the incidence of asbestosis has occurred since the adoption of proper methods of dust control in the factories, and the character of the disease has altered; it is now manifested in a more chronic form.

Composition of asbestos dust

Chrysotile, $3MgO.2SiO_2.2H_2O$, is the main asbestos of commerce but crocidolite and amosite are also used. The native fibrous asbestos is embedded in other non-fibrous or micro-fibrous minerals such as serpentine and talc. Another fibrous mineral, the non-siliceous

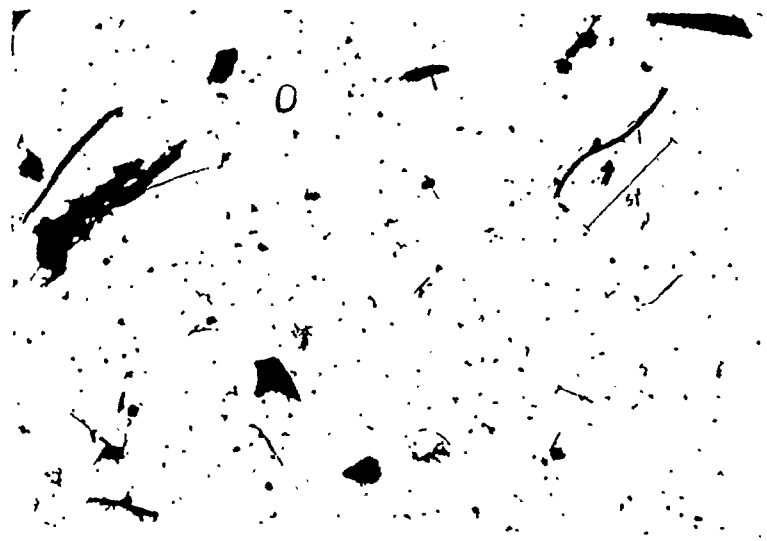

Fig. 12.1. Electron micrograph of a dust sample taken by a Thermal Precipitator from the air of an asbestos works.

Asbestos fibres are present but there are also many particles of other minerals and amorphous soot particles.

Micrograph by Safety in Mines Research Establishment ; Crown Copyright

brucite, $MgO.H_2O$, sometimes occurs with the asbestos. Brucite is of no value as a textile fibre because its fibres are insufficiently flexible.

The manufacturing processes entail the breaking-up of massive asbestos into fibres. In this process the non-fibrous material is shattered to give a fine dust having an average particle size much smaller than that of the asbestos fibres. The non-fibrous dust, consequently, settles more slowly than the asbestos and the atmosphere of an asbestos mill with modern ventilating equipment contains far

more non-fibrous than fibrous particles. Most samples of airborne dust collected from industrial atmospheres show few asbestos fibres in the size range which is thought to be of pathological import. In the electron micrograph of a sample taken from the air of an asbestos mill by a thermal precipitator, shown in Fig. 12.1, fibrous asbestos particles, non-fibrous mineral particles and the amorphous sooty particles found in the atmosphere of any industrial town can be distinguished.

Hurlbut and Williams gave the following analyses for dust collected from the rafters of the different departments of an asbestos textile mill:

Dust Produced in the Manufacture of Asbestos Textiles

	Preparation	Carding	Spinning
	Per cent	Per cent	Per cent
Talc	6	9	5
Carbonate	30	27	21
Magnetite	28	21	27
Serpentine	14	29	27
Asbestos	15	8	8
Others	7	6	12

Pathology of asbestosis

Asbestosis is a chronic disease. The lesions take some years to develop. In some circumstances silicosis may develop extremely rapidly and death may occur within two or three years of the first exposure, but such rapid development never occurs with asbestosis.

Superficially the lungs of a person who has died from asbestosis resemble the lungs of a victim of silicosis. They are hard and incompressible and they adhere to the pleura. X-ray pictures of the chest of an asbestotic patient show the lung fields to be interspersed with very fine, shadowy, round spots. They are mostly undefined but occasionally are sharp and they are denser in the middle and lower regions of the lung. The shadows on the X-ray films correspond to regions in which abnormal connective tissue growth has occurred. This growth of diffuse connective tissue is greater in the lower part of the lung.[423]

The fibrosis due to asbestos is much more diffuse than that due to silica.[421] Whilst in silicosis there are numerous almost isolated foci, in asbestosis there is a fine network throughout the lungs. Hunter describes the radiographs as having the appearance of ground glass. The degree of shadowing present in advanced asbestosis

would have little significance if seen in silicosis.[421] However, Wyers[422] states that the more chronic form of the disease shows up in radiographs as shadows which are coarser and more granular in appearance.

The most obvious symptoms shown by a patient are those of dyspnoea. Tuberculosis may be present but asbestosis is not accompanied by a predisposition to tuberculosis, as is silicosis.[125] Quite often there are no tubercle bacilli in the lungs. Asbestos fibres have occasionally been found in the urine of patients with asbestosis.[426] Half the group of workers with asbestosis which Wyers[422] studied had clubbed fingers.

According to Knox and Beattie[427, 428] who examined lungs from 25 deceased persons who had been exposed to asbestos dust, the severity of asbestotic lesions is related to the time elapsing between the first exposure to the dust and death rather than to the mineral content of the lungs.

A visual examination of a sectioned diseased lung reveals tissue which is interspersed with sharply outlined, air-free, greyish-black areas. When examined microscopically, the lung sections are seen to contain many asbestos fibres which lie in the alveoli and air ducts and in the lymphatics and connective tissue. There are few fibres in the bronchial lymph glands. An asbestos fibre may penetrate the walls of one alveolus into a neighbouring alveolus. The most important position of protective tissue response in the case of asbestos fibres is the terminal portion of the respiratory bronchiole whereas with minute silica particles it is the peribronchial and perivascular lymph nodes.[318] Emphysema is always observed in sections from the lungs of a person who has died with asbestosis.

Strobe[429] found hardly any phagocytes connected to fibres in the alveoli but there was considerable phagocytosis of the disintegration products of the fibres. Gloyne[430], however, frequently observed the ingestion of fibres and bodies by macrophages and regarded this as a normal method for the removal of fibres from the lung. Sundius and Bygden[431] could find only hornblende in the lungs of persons who had died from asbestosis although the persons had handled materials which contained mostly serpentine.

A recent paper on the pathology of asbestosis is that of Lynch.[624]

Asbestosis bodies

The characteristic feature of the lung sections from an asbestosis case is the presence of large numbers of peculiarly shaped structures. There may be 8,000 of these asbestosis bodies visible in a section of lung tissue 1 cm.^{2} in area.

Asbestosis bodies form rapidly in the lungs. They have been

found in the lungs of a patient exposed to the dust for only 2 months and they may appear in the sputum within 5 months of exposure. Asbestosis bodies in the sputum or in the lung do not necessarily indicate a condition of pulmonary asbestosis. Indeed it has been observed[433] that most workers in asbestos factories have asbestosis bodies in the sputum. Asbestosis bodies were found in the lungs of one man who had lived near to an asbestos factory for many years but had never been inside it!

Asbestosis bodies are often seen in the lungs in clumps.[433] These formations may occur within the lung alveoli or they may be embedded in fibrous tissue. Stewart, Tattersall and Haddow[433] state that the clumps in the alveoli are almost certainly derived from clumps in the fibrotic areas since the alveoli which contain clumps are always adjacent to areas of advanced fibrosis. Clumps of asbestosis bodies have also been found in the sputum of patients suffering from asbestosis.[433, 434, 435] These formations in the sputum indicate that the lung tissue is disintegrating and they point definitely to a condition of asbestosis.

Originally, there was considerable controversy about the nature of asbestosis bodies. Some investigators believed that they were "essentially derived from, or associated with asbestos and were, in fact, portions of asbestos fibres in the process of alteration and absorption by hydrolysis, either by direct chemical action or by enzymes".[436] Others, such as Cooke,[417] believed that their "results completely negatived the theory that the bodies are asbestos or derivatives of asbestos". Cooke stated that "if organisms—staphylococci, for example—are incubated with a colloidal solution of asbestos they become silicated and resemble asbestosis bodies". He sought to prove that most of the bodies were actually "vegetable" in origin. A report on the discussion which followed a paper describing an early case of asbestosis[437] said: "A second interesting feature . . . was the presence of a body which some thought was an aspergillus. The authors of the papers held the view that it was a hyphomycete analogous to that found by Dr. H. H. Scott in batrachians, or that it belonged to the tuberculariaceae, a family of hyphomycetes described by Ehrenberg in 1818."

An outstanding investigation of the structure, formation, disintegration and chemistry of asbestosis bodies was made by Beger.[438] A lecturer in geology in the Technischen Hochschule of Hanover, Beger was asked to identify asbestos in the lungs of a foreman who had died from asbestosis. Beger had no knowledge of medicine, and stated: "I deferred all study of the literature on asbestosis and asbestos bodies, until such time as I should have completed my experimental investigations." In the lung sections which he

examined, Beger found asbestos fibres and asbestosis bodies. There were far more bodies than unchanged fibres. The fibres (Fig. 12.2) were straight, rigid, and extremely thin. Beger measured the length and thickness of the fibres in one field and gave the following values:

Length	(μ)	109	94	87	60	49	46	38	36	35	33	28	25	25
Thickness	(μ)	0·8	0·4	0·3	0·3	0·8	1·0	0·2	0·7	0·3	0·5	0·3	0·3	0·2

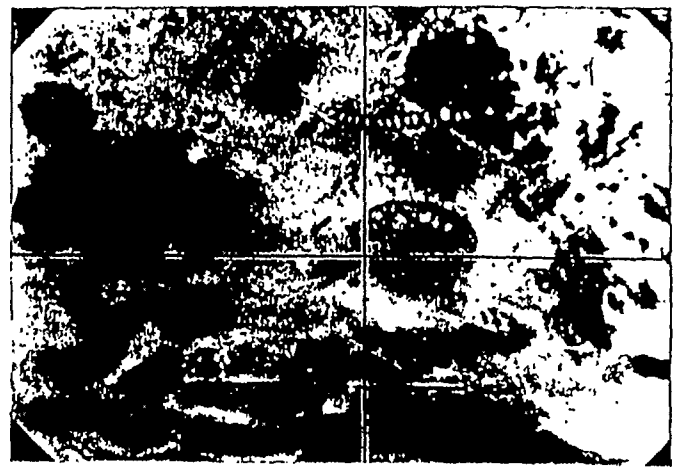

Fig. 12.2. An asbestos fibre in a section of a lung (lower half of photograph). A disintegrating asbestosis body is in the upper part of the photograph (×1,200). (Figs. 12.2 to 12.6 are reproduced from Beger, *Virchow's Archiv.*, 290, 282, 1933.)

There was no relation between the length and the thickness: sometimes long fibres were thin or short fibres thick. Very long needles, say 120 μ, were seldom seen, but long needles are easily broken in sectioning. By measuring the optical properties of the fibres (refractive indices, double diffraction, distribution of the optical vectors, etc.) and by considering other physical and chemical properties, the fibres were identified as chrysotile.

Beger described asbestosis bodies (Fig. 12.3) as golden yellow structures sometimes having the shape of a dumb-bell but more often in the form of a string of irregularly shaped discs, the "string of beads" form. At the ends are club-like protuberances. An average diameter of the head is given as 7·7 μ and of the shaft 3·5 μ. The length is from 10 to 100 times the thickness.

Running down the centre of the asbestosis body, a fibre or a modified fibre may sometimes be seen. Usually the fibre is still

straight but it has reduced rigidity and is less brittle. Occasionally the fibre is curved or even coiled. The coating of the fibre is a gel-like substance.

Electron micrographs[439] of asbestosis bodies collected from the sputum also reveal a central asbestos fibre which is coated with a gel. When the gel is removed by micromanipulation, the fibre appears to have transparent edges suggesting that a constituent of the fibre has dissolved. No fibres shorter than 10 μ were found, suggesting that short fibres dissolve completely.

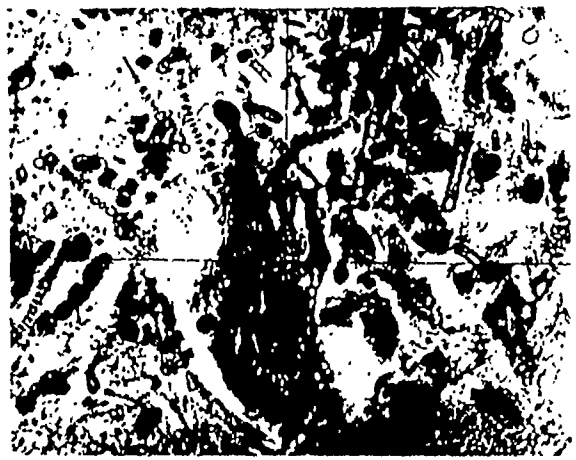

Fig. 12.3. Asbestosis bodies (×800).

Chemical composition of asbestosis bodies

Microchemical tests on the bodies show that the gel coating is a protein and that the granules therein are ferric compounds. The gel is not removed by hypochlorite and this reagent has been used[440] to separate asbestosis bodies from lung tissue. Sundius and Bygden[431] gave the following analysis of an asbestosis body:

Organic matter .	31% (S, 0·43%)
Iron oxide . .	40%
P_2O_5 . . .	6·9%
Water . . .	16%
"Asbestos" needle .	4·6%

An elementary analysis established that the organic matter was mainly protein. Koppenhoffer,[441] however, thought that the sheath was not composed of protein.

Formation and disintegration of asbestosis bodies

Beger suggests that the bodies are formed by the deposition of protein from the tissue fluid on the asbestos needles until the fibres become completely ensheathed. He succeeded in forming structures which were similar in appearance to asbestosis bodies by coagulating egg albumin on acid-washed asbestos fibres. The gel generally takes the form of beads because of surface tension effects. If they come into contact with one another, the beads can coalesce. The commencement of the disintegration of the asbestos fibre in the asbestosis body is shown initially by cleavage lines in the cylindrical

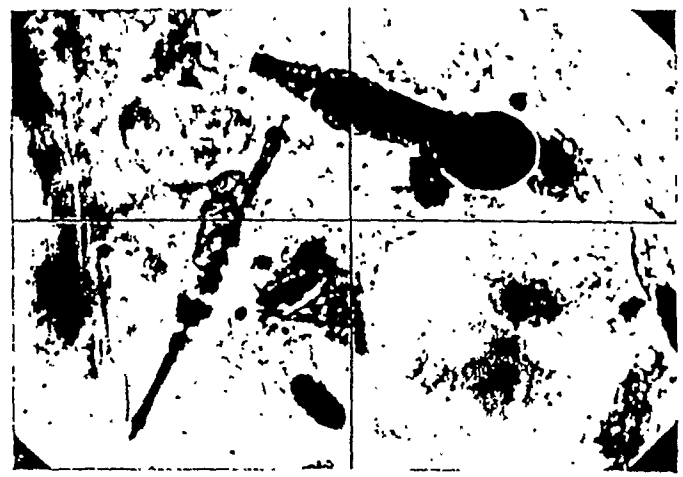

Fig. 12.4. The first stage in the disintegration of an asbestosis body is shown by cleavage lines in the cylindrical portion of the sheath (×1,270).

portion of the sheath at right angles to the length of the fibre. The heads then show cleavage. Finally the asbestosis body disintegrates completely; afterwards nothing remains but a series of granules from which the shape of the original body can be surmised. These stages are illustrated in Figs. 12.4, 12.5, 12.6.

The breakdown of asbestos fibres by the formation and disintegration of asbestosis bodies in the lung is emphasized by differential counts of fibres made on the lungs of 27 deceased persons who had been employed in the asbestos industry.[428] The figures shown in

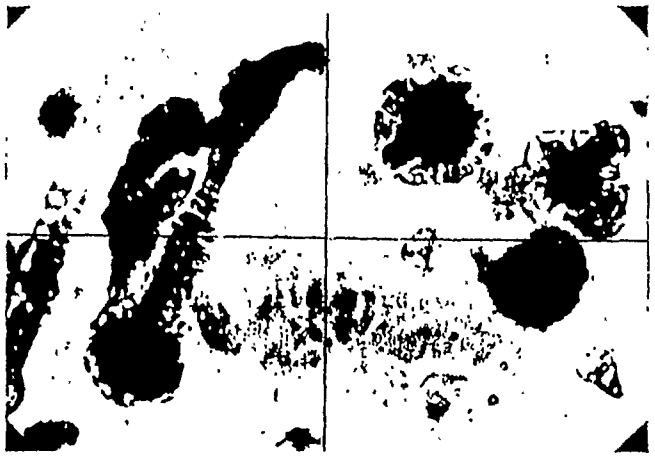

Fig. 12.5. The disintegration of an asbestosis body is later shown by cleavage of the "heads" (× 1300).

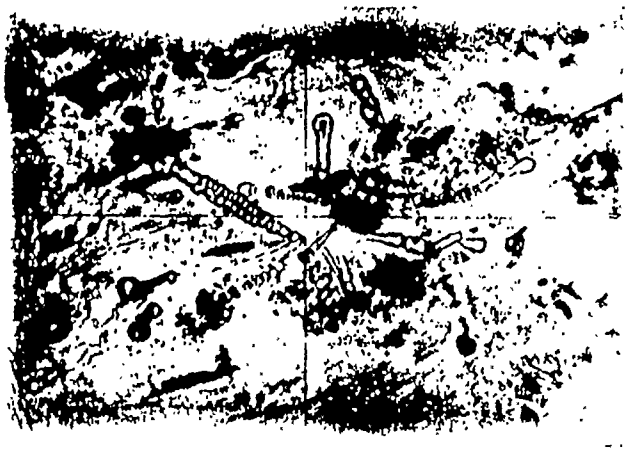

Fig. 12.6. An asbestosis body finally disintegrates into a series of granules (× 650).

Table 12.1 indicate that, when the time interval between the last exposure to the dust and death was more than 8 years, there were usually no fibres in the lung longer than 25 μ. Fibres in this size range were abundant in the lungs of persons who survived for a

shorter time after the last exposure to the dust. Gloyne[130, 442] also believed that there were fewer and smaller fibres and asbestosis bodies in the lungs of long-standing cases of asbestosis than in the lungs of workers exposed to the dust for a shorter time.

TABLE 12.1

Particle-size Distributions Expressed as Percentages of Count in the 16–25-μ range

Case No.	Sex	Total exposure time, Yr.	Survival time, Yr.	Asbestosis, − +	Mean particle counts					
					Less than 5 μ	5–15 μ	16–25 μ	26–35 μ	36–45 μ	Over 45 μ
3	M	26	0	++	364	193	100	36	21	0
23	M	28	0	+	458	217	100	42	0	0
26	M	20	0	+	510	290	100	51	15	6
9	M	11	0	−	612	389	100	62	18	11
17	M	12	0	+	394	281	100	79	9	2
25	M	8	0	−	627	329	100	38	17	21
12	F	32	1	−	501	257	100	48	0	17
2	M	27	2	+++	689	387	100	33	0	9
5	M	28	2	+	433	225	100	50	17	8
21	F	33	2	−	573	381	100	14	2	6
4	F	14	2	+	627	296	100	62	20	5
19	F	8	2	+	522	359	100	41	8	7
6	M	22	3	+	421	325	100	51	25	17
10	M	9	3	−	510	322	100	50	20	0
14	M	27	4	+	517	281	100	46	18	2
22	M	7	6	−	486	369	100	31	12	0
1	M	23	7	+++	610	487	100	0	0	0
27	M	21	8	+++	786	521	100	22	2	0
20	M	25	8	+++	829	644	100	11	4	0
8	F	27	8	+++	898	591	100	4	1	1
13	M	27	8	+	496	321	100	0	0	0
7	F	6	9	+	611	418	100	0	2	0
28	M	2	11	−	587	292	100	0	0	0
11	M	22	14	+++	720	599	100	0	0	0
24	M	19	14	+++	1,006	707	100	0	0	0
15	M	14	17	−	529	386	100	2	0	0
16	M	10	21	+	647	411	100	9	0	0

(From Knox, J. F., and Beattie, J., *Arch. industr. Hyg.*, 10, 30, 1954.)

The formation of asbestosis bodies has been described as a defence mechanism of the body for the detoxication of asbestos fibres.[440, 443] The bodies appear to be innocuous. Asbestosis bodies were recovered from a human lung, then they were injected intratracheally into guinea-pigs' lungs. No fibrosis was produced although the bodies remained in the lungs for a year or more. It was possible to produce fibrosis, however, by injecting asbestos fibres.

Experimental asbestosis

Asbestos dust has been administered to animals by inhalation and by intratracheal, intravenous, intraperitoneal and subcutaneous injection. The maintenance of an atmosphere containing a suitable concentration of asbestos dust is not easy. The inhalation chamber used in the Saranac laboratory was a cubical room, length of side 8 ft., in which asbestos dust was thrown into the air by a paddle slowly rotating in a hopper. Since the fibres tend to form balls in the hopper, wire brushes were attached both to the paddle blades and to the floor of the hopper. Animals were kept in this room for periods of 2 to 5 years.

Stewart[444] caused guinea-pigs to inhale asbestos dust. After 12 weeks, he found numerous asbestosis bodies in the alveoli and some in the alveolar walls. Some of the bodies were "beaded", in the process of degeneration, and they all gave an intense Prussian blue reaction, showing the presence of iron in the outer coating. Scattered, giant phagocytes in the alveoli contained asbestosis bodies. No fibrosis in the lungs or thickening of the alveolar walls was observed; the experiments lasted 20 weeks.

Large-scale animal studies were made during some 25 years in Gardner's laboratory at Saranac Lake. Vorwald, Durkan and Pratt[440] reviewed and summarized this work. When injected intravenously into rabbits, finely ground asbestos (chrysotile, amosite, crocidolite, anthophyllite and tremolite) produced only the reaction of an inert foreign body. There was no fibrosis of the liver, spleen or other organs as is observed when quartz is injected intravenously. When chrysotile (0·25 g. or less) was injected intravenously 5 of 6 animals died; no reason for the deaths could be found.

Guinea-pigs inhaled asbestos for periods up to 33 months. The dust collected about the respiratory bronchioles, where fibrosis developed; the peripheral alveoli were not involved. A segregation of the fibrous and non-fibrous particles was observed, only the latter being transported to the lymph nodes. Asbestosis bodies were formed in 2 months. A cellular reaction was first observed about one year after the first exposure to the dust and a delicate fibrosis appeared after a further 4 months. The fibrosis progressed thereafter and, as the collagen fibres contracted, alveoli were partially closed and distorted. The appearance of the tissue resembled that of an adenoma; similar structures have been described in the lungs of guinea-pigs which had inhaled silicon carbide.[445]

When administered to guinea-pigs by intratracheal injection, chrysotile produced fibrosis. Two months after the dust was administered, there was well-marked cellular proliferation and focal

fibrosis about the respiratory bronchioles; asbestosis bodies were not observed. After six months, asbestosis bodies were abundant in the lungs but, perhaps due to the contraction of the fibrous tissue, the reaction was less extensive. Later, the fibrous tissue, still localized around the terminal bronchioles, became hyalinized until, after 12 months, there were well-developed peribronchial and intrabronchial adenomatoid areas of fibrosis.

Species differences.—Although Vorwald and his associates[440] obtained a definite fibrotic response to inhaled asbestos fibres in guinea-pigs, no fibrosis could be produced in rabbits. As there were no fibres or asbestosis bodies in the lungs at autopsy, it was argued that the upper respiratory tract in these animals is an efficient filtration mechanism, adequate to exclude fibres from the lung.

Well-marked peribronchiolar fibrosis has been observed in rats following the inhalation of long fibre asbestos dust but hardly any asbestosis bodies were present; in mice numerous asbestosis bodies were observed but no fibrosis. Some cellular connective tissue forms a sheath around bronchioles in the lungs of cats following the inhalation of asbestos fibres, but typical asbestosis bodies are not produced. The location of the lesions is similar to that found in guinea-pigs but the lesions develop much more slowly.

Fibre length.—Vorwald and his associates[440] compared the fibrogenic activity of asbestos dust of short fibre length (3 μ) with that of samples containing much longer fibres (20 50 μ). The dust was administered to guinea-pigs both by inhalation and by intratracheal injection.

Guinea-pigs kept in an atmosphere containing long chrysotile fibres developed lesions consisting of cellular connective tissue about the terminal bronchioles after 8 months. The adjacent parenchyma was affected after 16 months. Lesions were large enough to be seen macroscopically after 20 months.

By contrast, when the guinea-pigs inhaled chrysotile fibres which were mainly shorter than 3 μ they showed but little reaction to the dust; only scattered phagocytes and an occasional minute asbestosis body were observed. Cellular accumulations about terminal bronchioles were visible microscopically after 2 years, but no abnormality was sufficiently gross to be detected with a hand lens. There was a slight increase in reticular tissue, but no true fibrosis in the tracheobronchial nodes after 30 months.

Chrysotile fibres in the size range 20 50 μ are evidently pathogenic. The conclusion that particles less than 3 μ long are relatively harmless must be drawn with some reservations because of the experimental difficulties encountered in the tests. Vorwald showed that strictly comparable dusty atmospheres containing asbestos of different

fibre lengths could not be produced. The average values (Table 12.2), which are taken from the detailed control analyses of the atmospheres, emphasize the degree of variation to be expected in experiments of this type, even when the most careful attention is given to technique.

The deductions made from the results of inhalation experiments are supported by those obtained by intratracheal injection. Long fibre (20–50 μ) chrysotile will produce the cellular reactions in the lungs of guinea-pigs which were described earlier; there are foci of cellular proliferation after 1 month and well-developed areas of fibrosis after 12 months. When the administered dust particles were below 3 μ in size, they evoked an initial proliferative reaction

TABLE 12.2

Differential Counts (percentage of total count)

	Non-fibrous particles			Fibrous particles			Analysis	
	< 3 μ	3–10 μ	> 10 μ	> 10 μ	< 10 μ	Clumps	Chrysotile (per cent)	Serpentine (per cent)
Long fibre	65·4	1·1	0	25·8	1·0	1·0	60	20
Short fibre (ball milled)	90·6	4·8	0	0·8	0·6	3·2	15	60

and the dust became localized about the bronchioles but after 12 months there was no considerable fibrosis. There were only a few microscopic patches where the alveolar wall was thickened, and some adenomatoid change in air spaces adjacent to thickened bronchi. No asbestosis bodies were observed. It has been suggested[427] that particles which are smaller than the critical size are removed by macrophages whilst those above this size do not reach the distal air spaces.

King and others[446] obtained rather different results when asbestos of graded fibre length was transferred to the lungs of rabbits by intratracheal injection. The long fibres (15 μ) produced a nodular reticulinosis, comparable with experimental silicosis, but the short fibres (2·5 μ) also produced a reticulinosis.

Effects of different types of asbestos and other fibrous particles.—Vorwald and others[440] found that inhaled serpentine, of particle size less than 3 μ, when injected intratracheally induced simple phagocytosis in the lungs of guinea-pigs but that it had produced no fibrosis 1 year after the experiment commenced.

When administered by intratracheal injection (dose 50 mg.) the fibrous minerals chrysotile (Thetford and Arizona), amosite, crocidolite (Bolivia and S. Africa) and tremolite all produced fibrosis about the bronchioles. Brucite, $Mg(OH)_2$, produced a similar reaction. In all cases asbestosis bodies were formed but often they were not observed until after the fibrosis was well established. Sundius and Bygden[431] found that "asbestosis" bodies formed around rutile needles.

Anthophyllite, another fibrous mineral of the amphibole group, produced no fibrosis. After 1 month scattered intrabronchiolar dust foci were observed but after 8 months there was little evidence of dust. Lymphocytic infiltration of the bronchiolar walls, giant cells, and a few atypical asbestosis bodies were seen after a month. No explanation of the inert character of this asbestos has been offered.

Glass wool was also inert. Giant cells collected the particles, which could be seen as fine spicules in the cytoplasm, and formed clumps in the peripheral air spaces. No fibrosis was visible after a year.

Progress of lesions after discontinuance of dust exposure.—Guinea-pigs were exposed to an atmosphere containing asbestos dust for periods of 6 or 9 months, then kept in a normal atmosphere for periods up to 3 years. The initial cellular reaction to the dust was replaced by thin strands of fibrous tissue 8 to 11 months after exposure ceased. As the fibres matured, they contracted so that the amount of scar tissue decreased with time. Experimental asbestosis does not, then, progress after the animal is removed from the dust-laden atmosphere.

Relation between asbestosis and tuberculosis.—When guinea-pigs which had inhaled asbestos dust for 26 months were transferred to a normal atmosphere and then infected with tubercle bacilli, the course of the tuberculosis was not appreciably altered. When the infection was coincident with the onset of the exposure to dust, the results were variable; in some animals the disease did not progress, but in most there was a temporary progression and subsequent healing. There is no evidence that asbestos dust significantly affects the course of a tuberculous infection.

Aetiology

Two theories have been used to explain the damage caused to the lung by asbestos. The one theory,[440, 431, 423] which is supported largely by the results of animal experiments, suggests that the damage is purely mechanical—that it is due to the fibrous nature of the dust. The following evidence supports this view. Only asbestos fibres which are more than 20 μ and less than 50 μ long have been found to cause fibrosis in the lungs of animals.[440] Moreover, fibrosis has been produced in the lungs of guinea-pigs by the intratracheal injection of brucite fibres, which contain less than 1 per cent SiO_2.

for the normal lungs were subtracted from those from the asbestosis cases, the extra mineral matter appeared to be only silica. He concluded: "One cannot avoid the conclusion that the asbestos has undergone decomposition in the lung, with deposition of SiO_2" and he suggested that the disease is "due to the chemical action of silica . . . liberated in situ from inhaled silicates capable of relatively easy decomposition within the lungs".

Asbestosis and cancer of the lung

Statistics have been published which suggest that the incidence of cancer of the lung is higher among asbestos workers than among the general population. Merewether stated in the Report of H.M. Chief Inspector of Factories,[448] that carcinoma of the lung was notified in 31 of 235 cases of asbestosis (13 per cent) but in 91 of 6,884 cases of silicosis (1·3 per cent), Gloyne[430] from personal examinations found carcinoma of the lung in 17 of 121 (14 per cent) autopsies on subjects with asbestosis but in 55 of 796 (6·9 per cent) autopsies on subjects with silicosis. Gloyne emphasized that it is not possible to get figures which are statistically significant because of the small number of asbestosis cases. The ratio of women to men workers in industries in which asbestos is handled is much higher than in the industries in which silica is used and amongst the general population. Lung cancer is more common amongst men than women. These facts give the figures an added significance. If only the male subjects who contracted asbestosis are considered, the incidence of carcinoma of the lung is 17 per cent in the group studied by Merewether and 20 per cent in that studied by Gloyne. A comparison of the incidence in different industrial groups of carcinoma of the lung associated with pneumoconiosis is made in the chart (Fig. 12.7).

Doll[449] studied the records of a group of 105 persons who had died after working in the asbestos industry. Of these, 18 had lung cancer and in 15 cases the cancer was associated with asbestosis. He compared the mortality in a group of 113 men who had worked for 20 years or more in the industry with that in a corresponding group of the general population. Thirty-nine deaths occurred in the one group whereas the expected number was 15·4. The excess was due to lung cancer, and other respiratory and cardiovascular diseases. He stated: "From the data it can be concluded that lung cancer was a specific industrial hazard of certain asbestos workers and that the average risk among men employed for 20 years or more has been of the order of 10 times that experienced by the general population."

He pointed out, however, that the workers examined had worked in the industry before the pathogenic nature of the dust was recog-

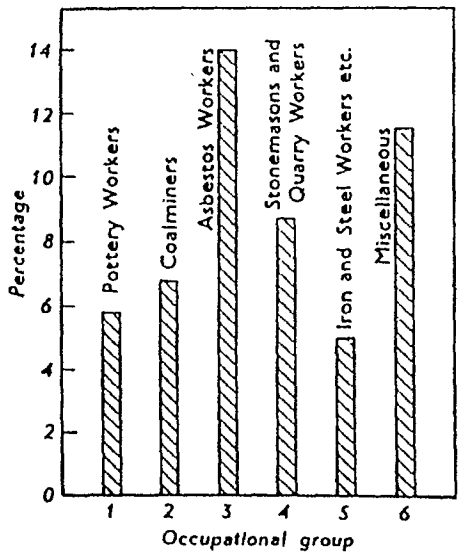

Fig. 12.7. Malignant neoplasms associated with pneumoconiosis. (After Gloyne, *Lancet*, 260, 810, 1951.)

nized and before the introduction of the Asbestos Industry Regulations, 1931, which controlled the conditions in asbestosis factories.

Whilst most investigators believe that a relationship is probable between asbestosis and carcinoma of the lung,[450, 637] others[153, 288] do not. Experiments with animals have shown negative or inconclusive results.[451, 452, 453, 454] See also Rombola.[636]

PNEUMOCONIOSIS

Industrial Diseases of the Lung caused by Dust

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