calcified plaques but are usually quite small and visible only by electron microscopy.

Animal experimentation. The pleura of most experimental animals will show granulomatous response when asbestos is injected, but similar reactions are produced by other mineral dusts, so that specificity is wanting (175). The cellular reactions to asbestos, however, closely resemble those that occur in the parenchyma.

Influential factors. Many authors suggest that some sensitivity reaction is involved in at least plaque formation, and others [including the IARC Advisory Committee (274)] take pains to leave the door open for the cooperation of some other factor not yet recognized, but no details are given. The importance of age has been repeatedly demonstrated, particularly in agricultural workers not exposed to any industrial source of asbestos, but this may simply mean that the individuals have to live long enough for the very slow process to produce recognizable results. The question as why only a certain proportion of the population apparently exposed to very low doses develop the disease remains here, as with other responses to asbestos, entirely open. To ascribe the phenomenon to variations in susceptibility is simply to dress ignorance in a transparent cloak.

9

Prevalence, Diagnosis, and Course of the Asbestoses

With the nature of parenchymal and pleural asbestosis established, this chapter will turn to practical questions that confront the medical practitioner who has to deal with the spectrum of asbestotic disease that may be encountered in his patients. Parenchymal and pleural effects can appear in almost any combination and will be considered together. As in preceding chapters, the account given here combines the experience gained in the course of extensive studies and practice at Mount Sinai with that reported by others (153,215,333,531,562,677,801,803).

PREVALENCE

The medical practitioner needs to have an estimate of how often he is likely to see cases of asbestosis in his practice, or what the epidemiologist calls the prevalence in a specified population group. The medical literature is not devoid of data; numerous reports appear of the number of cases seen, the frequency with which asbestosis is found at autopsy, mortality figures, etc. But many of these data have limited usefulness because they do not indicate the size of the exposed population from which the cases were drawn, or what is called the population at risk, so that the reader cannot try applying the reported experience to the group of patients that he is likely to see. One trouble is that the disease that is seen today had its origin in the past, 20 or more years ago, and too little is known about the number of people who were employed in various occupations at that time. This is particularly true

Published by AsbestosClaims.Law
asbestosclaims.law/library/25477/1

where employment rates were changing or where there was a large turnover in employees, as occurred, for example, in World War II years, and in the slump that followed. We are on still shakier ground when we talk about those who were exposed (at risk). A few years ago persons working in a clerical capacity at a shipyard would not have been regarded as being at risk, but we now know that they may be, and in some cases obviously have been. We still have to find out if persons using consumer products containing asbestos are often at any significant degree of risk; we still have to acertain whether the burden of asbestos fibers found in the lungs at random autopsies has any bearing on health.

The data that we really need for talking realistically about probable prevalence are fragmented and subject to various kinds of bias. Nevertheless, a review of the available data, conservatively treated, can give the practitioner at least a "feel" for the situation that he will encounter, and for those occupations or acticities that are likely to expose people to significant intake of asbestos fibers. In reviewing the data he will surely come to appreciate both the difficulty and the importance of finding out, by careful and persistent inquiry into a patient's residential as well as occupational history, the extent to which he may have been exposed. From the data perhaps, and certainly after a little experience with cases, the practitioner will realize that a further difficulty confronts us—the nature of the disease resulting from exposure to asbestos that we now see is changing. The decrease in tuberculous infection changed the character of the expressed disease in the first half of the century; we are now witnessing some rather unexpected effects of asbestos dust control. Parenchymal asbestosis, as it used to occur from relatively heavy exposure, could develop in ten to twenty years initial exposure and lead to death in the following decade, and calcified pleural plaques were simply part of the generalized asbestotic process. We are now finding pleural plaques in persons exposed to much lower levels of dust, twenty to forty years after first exposure, with miniinal accompanying parenchymal change (48,471). If pleural plaques were the only process to be permitted development by increased longevity, we would probably not be greatly concerned; plaques, after all, do not of themselves impose much disability. But, as we shall see in later chapters (Chapters 10–12), two other disease processes of much greater clinical significance have, so to speak, been liberated by the reduction of fibrotic development—mesothelioma and carcinoma. It is somewhat chastening to find that when we developed the control of industrial dust that was demanded by regulations, albeit slowly and somewhat reluctantly, it turned out not to be good enough; that instead of dying at a fairly early age from

the effects of pulmonary fibrosis, a wider segment of the population runs an increased risk of somewhat later, but still premature, death from other diseases invoked by asbestos dust. However, the asbestoses are still with us, partly as the result of past excessive exposure; and they will continue to be with us for some time, since they are still developing in persons exposed to what was, until recently, regarded as a fairly safe standard—5mppcf (534). (For current standards, see Chapter 19.)

We will now review the information that we have on the prevalence of the asbestoses in various population groups, but once again remind the reader that the overall impression of prevalence is more important than individual sets of data, most of which have distinct limitations.

Miners and Millers

The data on this occupational group are not extensive. In 1943 Siegal *et al.* reported that 23 out of 221 men working in talc mining and milling in the St. Lawrence County of New York State showed a marked degree of pulmonary fibrosis in most radiographs (681). They did not examine men who had left work for ill health or other reasons, nor did they take account of borderline radiographic findings, so that their figures are on the low side.

In 1971 McDonald *et al.* reported on 2,457 deaths that had occurred in 10,421 persons who had been employed at some time before 1967 in the chrysotile mines and mills of Quebec Province (499). Table 9–1A gives

TABLE 9–1A

Equivalent Average Death Rates Per 1,000 Persons for Respiratory Diseases in Chrysotile Mines and Mills in Quebec Province (499)[a]

Disease category	Dust index[b]						
	<10	10–99	100–199	200–399	400–799	800+	All
Pneumonia and bronchitis	3.8	4.7	4.2	6.6	5.6	6.9	4.9
Pneumoconiosis	1.5	2.0	0	3.7	3.9	22.6	3.2
Other respiratory diseases	4.2	8.6	15.3	6.3	5.9	12.1	7.5
All respiratory diseases	9.5	15.3	19.5	16.7	15.3	41.6	15.6

[a] With kind permission of J. C. McDonald and American Medical Association, publishers of *Archives of Environmental Health*.

[b] Dust index is obtained by adding together the products of the time spent on each job and the estimated average concentration of dust in mppcf.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25477/2

the equivalent average death rate* per 1000 persons for respiratory diseases in relation to the cumulative exposure. The latter is expressed as a "dust index," obtained by adding together the products of the time spent on each job and the estimated average concentration of dust on that job in mppcf. These data were interpreted by the investigators to indicate that, while the chrysotile miners enjoyed the usual superiority of a work force over the general population as regards mortality rates in general, this advantage was lost by the men with the highest cumulative dust exposure, who showed a mortality rate 20% above that of the rest, and that two-thirds of the excess mortality in this group was probably due to pulmonary fibrosis, shown on the death certificate either as fibrosis or in the guise of various respiratory or cardiovascular diseases. In a later paper they express data in a more familar form of age-corrected death rates per 1000 men (Table 9–1B), from which it

TABLE 9–1B

Age Corrected Death Rates Per 1,000 Men Born 1891–1929: Deaths to December 1969 (500)[a]

	Dust exposure level (mppcf -yr)					
Cause	<10	10	100	200	400	800+
All causes	365	355	354	313	323	395
Respiratory cancers[b]	10.3	13.1	13.4	15.5	21.4	32.1
Abdominal cancers	18.0	13.6	18.7	11.6	26.3	28.7
Pneumoconiosis	1.6	1.5	0.8	4.9	4.9	23.6

[a] *With kind permission of J. C. McDonald and American Medical Association, publishers of Archives of Environmental Health.*

[b] Includes malignant pleural mesothelioma.

appears that not only the death rates from pneumoconiosis, but also those for respiratory and abdominal cancers rise in the groups with higher dust exposure levels (500).

In a cohort of 544 Quebec miners and millers, with 20 or more years in 1961 after first exposure, Nicholson reported the observed as compared with expected deaths through 1973 shown in Table 9–2 (549). Of 132 deaths from all causes over the period 1961–1973, 18 (13.6%) were attributed to asbestosis, 20 (115.2%) to lung cancer, and 1 to

* The *equivalent average death rate* is described by Hill as the arithmetic mean of the rates at quinquennial groups of ages. The resulting figure is equivalent to calculating a standardized death rate at ages under 65 based on a population equally distributed over 13 age groups (332).

mesothelioma. Deaths from other causes were not significantly different from the expected.

Meurman and his colleagues recently reported that 35 out of 248 deaths in anthophyllite miners in Finland had asbestosis as a primary cause or as a cofactor (514). [A]

Factory Workers

In 1969 Vigliani reported that in Italy 586 persons had been compensated for asbestosis and associated diseases in the period 1964–1966, and gave the distribution by trade (758). Unfortunately, he did not indicate how many workers, past and present, this group had been drawn from. The data are given Table 9–3.

In reviewing the causes of death up to mid-1960, among 1495 persons who had been employed at some time during the period 1938–1939 in a company manufacturing asbestos products in Ohio, Mancuso and Coulter found 186 deaths as compared with an expected 145 (475). The excess of observed over expected deaths was largely accounted for by two classes of disease: 58 as against 27 for neoplasms and 23 as against zero for asbestosis. The facts that this was a young population, that there was an unrecorded turnover and intermittency in employment, and that those employees with jobs involving low exposures are included in the total, increase the significance of the observed excesses for these two diseases.

Horai and his colleagues published the results of a survey in 1955–1959 of 328 employees in a Japanese plant using asbestos in the manufacture of insulation materials and textiles (351). The distribution of radiographic indications of fibrosis by length of service is given in Table 9–4.

The data reported by Nakamura for 1955–1957 showed a greater frequency (536). Among 1,100 employees of asbestos factories in Osaka and Nara, the prevalence of fibrosis was 21% in those employed between 5 and 10 years, 54% between 10 and 15 years, 70% between 15 and 20 years, and 100% for those employed over 20 years!

Asbestos deposits were not worked in Bulgaria until 1944, when an asbestos cement plant was opened (483). A 1960 study of 115 men, only 13 of whom had worked for more than 10 years, showed a surprisingly high prevalence of asbestosis (44%) in mine, mill, and factory workers. At about the same time in Germany, Bohlig *et al.* found radiologically detectable asbestosis in 44.5% of those with 20 or more years of exposure, and in 28% of those with 10–15 years (97).

TABLE 9–2

Expected[a] and Observed Number of Deaths among 544 Quebec Miners and Millers January–November 1961–December 31, 1973, Twenty or More Years after Onset of First Exposure to Asbestos (549)

Period:	1961 (part)–1965			1966–1969			1970–1973			Total 1961 (part)–1973		
Number of men:	544			506			457			544		
Person-years of observation:	2,303			1,926			1,735			5,962		
	Exp.	Obs.	Ratio	Exp.	Obs.	Ratio	Exp.	Obs.	Ratio	Exp.	Obs.	Ratio
Total deaths, all causes	32.64	38	1.16	36.69	49	1.34	43.52	45	1.03	112.85	132	1.17
Cancer, all sites	6.95	9	1.29	8.31	11	1.32	10.29	13	1.26	25.55	33	1.29
Lung	1.89	5	2.65	2.45	7	2.86	3.38	8	2.52	7.52	20	2.66
Pleural mesothelioma	n.a.[b]	1	—	n.a.	—	—	n.a.	—	—	n.a.	1	—
Peritoneal mesothelioma	n.a.	—	—	n.a.	—	—	n.a.	—	—	n.a.	—	—
Stomach, esophagus	1.03	—	—	1.09	—	—	3.21	1	0.83	3.33	1	0.30
Colon, rectum	0.96	2	2.08	1.17	1	0.85	1.44	2	1.39	3.57	5	1.40
Other	3.07	1	0.33	3.60	3	0.83	4.46	2	0.45	11.13	6	0.54
All respiratory disease	2.11	6	2.84	2.47	10	4.05	3.23	9	2.79	7.81	25	3.20
Asbestosis	n.a.	4	—	n.a.	6	—	n.a.	8	—	n.a.	18	—
All other causes	23.58	23	0.98	25.91	28	1.08	30.00	23	0.77	79.49	74	0.93

[a] Expected deaths are based upon Canada white, male, age-specific mortality data of the Dominion Bureau of Statistics, Ottawa, Canada.
[b] n.a., Specific rates for these causes of death are not available. They are, however, extremely rare in the general population.

TABLE 9–3

Number of Persons Compensated for Asbestosis and Related Diseases in Italy, 1964–1966 (758)[a]

Mining	39
Asbestos–cement and refractory material	342
Textiles and friction material	57
Dockyards and shipbuilding	101
Building (including insulation)	22
Plastics and rubber	18
Others	7
	586

[a] With kind permission of E.C. Vighani and the publishers of *Med. Lavoro*.

TABLE 9–4

Results of a Survey in a Japanese Asbestos Plant 1955–1959 (351)[a]

Length of employment	No. of employees	Normal	Grade I	Grade II	Grade III	Total abnormal
Under 5 yr	178	178(100.0)	—	—	—	0
5–10 yr	64	54 (84.4)	10 (15.6)	—	—	10 (15.6)
10–20 yr	77	27 (35.1)	41 (53.2)	9 (11.7)	—	50 (64.9)
Over 20 yr	9	0	3 (33.3)	5 (55.6)	1 (11.1)	9(100.0)
Total	328	259 (79.0)	54 (16.5)	14 (4.3)	1 (0.3)	69 (21.0)

[a] Percentages in parentheses. With kind permission of Z. Horai.

In 1971, Navratil and Trippé presented their findings on employees of a large factory manfacturing asbestos products over a period of 40 years who had been systematically examined over the previous 15 years (541). In 800 employees with more than 10 years of experience they found 42 (5.3%) with pleural calcifications, 23 of whom also had radiological evidence of parenchymal fibrosis. In 155 persons over the age of 20, living in the vicinity of the factory but never employed there, they found pleural calcifications in 9(5.8%); 5 of the 9 were blood relatives of employees. In 114 relatives of those employees who had pleural calcifications there were 4 (3.5%) who also had calcifications. (We will take up the matter of prevalence in relatives and neighbors a little later.)

In a cohort of 611 New Jersey asbestos factory employees, who in 1959 had passed 20 years or more after initial exposure, Nicholson reported the observed as compared with expected deaths through 1973 shown in Table 9–5 (549). Of 213 deaths from all causes over the period 1959–1973, 23 (10.8%) were attributed to asbestosis, 30 (14.1%) to lung

TABLE 9–5

Expected[a] and Observed Number of Deaths among 611 New Jersey Asbestos Factory Employees January 1, 1959–December 31, 1973, Twenty or More Years after Onset of First Exposure to Asbestos (549)

Period	1959–1963			1964–1968			1969–1973			Total 1959–1973		
Number of men	611			562			480			611		
Person-years of observation	2,955			2,646			2,195			7,796		
	Exp	Obs	Ratio	Exp	Obs	Ratio	Exp	Obs	Ratio	Exp	Obs	Ratio
Total deaths, all causes	39.87	49	1.32	52.06	82	1.58	59.60	82	1.38	151.53	213	1.41
Cancer, all sites	7.60	19	2.50	10.52	24	2.28	12.75	34	2.67	30.87	77	2.49
Lung	2.31	6	2.60	3.48	10	2.87	4.37	14	3.20	10.16	30	2.95
Pleural mesothelioma	n.a.[b]	1	—	n.a.	3	—	n.a.	6	—	n.a.	10	—
Peritoneal mesothelioma	n.a.	1	—	n.a.	3	—	n.a.	4	—	n.a.	8	—
Stomach, esophagus	0.72	3	4.17	0.87	—	—	0.90	—	—	2.49	3	1.20
Colon, rectum	0.92	4	4.35	1.28	2	1.56	1.56	2	1.28	3.76	8	2.13
Other	3.65	4	1.10	4.89	6	1.23	5.92	8	1.35	14.46	18	1.24
All respiratory disease	2.32	9	3.88	3.34	10	2.99	4.17	10	2.40	9.83	29	2.95
Asbestosis	n.a.	8	—	n.a.	7	—	n.a.	8	—	n.a.	23	—
All other causes	29.95	21	0.70	38.20	48	1.26	42.68	38	0.89	110.83	107	0.97

[a] Expected deaths are based upon United States white, male, age-specific mortality data of the National Center for Health Statistics.
[b] n.a., Specific rates for these causes of death are not available. They are, however, extremely rare in the general population.

cancer, and 18 (8.5%) to mesothelioma. Deaths from other causes except cancer of the colon and rectum were not significantly different from the expected.

As was mentioned above, a large proportion of the cases that make up these statistics had their origin in past years when dust levels generally were much higher than they are today. We would expect that with improvement in working conditions the number of new cases would be less and that a longer time would elapse before the disease reaches the stage of being radiologically detectable. In view of what was said above, we would expect this to be true particularly for parenchymal fibrosis, but to be less apparent for pleural calcifications.

In 1966 Smither offered evidence supporting one of these expectations (700):

> One means of observing the change in the incidence of asbestosis is to compare the average length of exposure before the onset of certifiable disability. In the early 1930's Hunter's (1955) cases averaged seven years. In the 1940's Wyers' (1949) cases averaged 10.4 years. In the 1950's the cases reported had an average of 14.5 years. My present series of cases, all from the same factory and certified since 1960, shows an average exposure of 17.5 years.

This does not mean, however, that the number of new cases is falling. In their 1973 paper (701), Smither and Lewinsohn report 53 new cases diagnosed from textile mills in the eight year period 1955-1963, and 65 in the six year period 1963–1969. Patients may be coming later in life, but they are coming in increasing numbers. How long this legacy of the past will last is difficult to say.

Construction and Engineering Workers

Asbestos was increasingly used in the manufacture of building materials and in the insulation of pipes and ducts until comparatively recent years, when other materials have been gradually substituted in many instances (see Chapter 3). Persons cutting asbestos materials, wrapping asbestos blankets, spraying asbestos slurries, and engaged in kindred operations were directly exposed to the dust, as were others working in the vicinity. Persons employed in removing old insulation will run the risk of exposure for many years to come.

Langlands and co-workers published in 1970 a detailed table of relevant health conditions among 252 men working as insulators in Belfast. They related various clinical findings to the radiographic appearances as in Tables 9–6 and 9–7 (439). It should be noted that 20% of the workers had less than 10 years of employment, and 60% less than 20 years. The frequency of abnormalities can be confidently expected to

Published by AsbestosClaims.Law
asbestosclaims.law/library/25477/5

TABLE 9–6

Abnormal Radiographs in Insulators in Belfast, 1970 Related to Years of Work (439)*

Years as insulator	No.	Pleural abnormality only	Parenchymal ± pleural abnormality	Total abnormal
0– 9	55	5	2	7 (13%)
10–19	103	12	16	28 (29%)
20–29	75	10	29	39 (52%)
Over 30	19	1	15	16 (85%)

* With kind permission of J. Langlands and the publishers of the *British Journal of Industrial Medicine*.

TABLE 9–7

Clinical Details Related to Radiographic Findings in Belfast Insulators Aged 40–59 Years (439)*

	Normal X-ray		Pleural abnormality only		Parenchymal ± pleural abnormality	
No.	65		15		44	
Rales	8	(12%)	1	(7%)	25	(57%)
Hyponychial angle ≥ 205°	0/16		1/14		4/42	
Mean right hyponychial angle	185°		185°		186°	
Mean left hyponychial angle	186°		186°		188°	
Dyspnea grade 2	20	(31%)	8	(53%)	14	(32%)
Dyspnea grade 3–5	13	(20%)	2	(13%)	12	(27%)
Dyspnea total	33	(51%)	10	(67%)	26	(59%)
Cough	43	(66%)	9	(60%)	33	(75%)
Sputum	44	(68%)	13	(87%)	35	(80%)
Wheeze	46	(71%)	9	(60%)	29	(66%)
Weather affects	20	(31%)	3	(20%)	10	(23%)
Cigarette smokers	45	(69%)	11	(73%)	35	(80%)
Over 25/day	12	(18%)	3	(20%)	13	(30%)
Asbestos warts on hands	41/62	(66%)	9/14	(64%)	36/42	(86%)
Asbestos bodies in sputum	17/51	(33%)	2	(13%)	15/38	(40%)
Limpet spraying	19	(29%)	3	(20%)	10	(23%)

* With kind permission of J. Langlands and the publishers of the *British Journal of Industrial Medicine*.

increase as the duration of employment and the time from initial exposure are extended. The high prevalence of respiratory symptoms in those without radiographic abnormality suggests that superadded infection and/or cigarette smoking may have been rather high in this population.

The prevalence of radiographic evidence of parenchymal asbestosis found in Mount Sinai studies of 1,117 insulation workers (out of a membership of 1,258 in the New York–New Jersey Local of the IAHFIAW in 1963), is given in Table 9–8. The marked increase in prevalence with time since first exposure, in this series strongly correlated with duration of exposure, is very evident.

TABLE 9–8

Grade of Asbestosis in Relation to Time from First Exposure in 1,117 Insulation Workers

Time from first exposure	No.	Normal (%)	Abnormal (%)	Grade of asbestosis 1	2	3
40+ yrs	121	5.8	94.2	35	51	28
30–39 yrs	194	12.9	87.1	102	49	18
20–29 yrs	77	27.2	72.8	35	17	4
10–19 yrs	379	55.9	44.1	158	9	0
0– 9 yrs	346	89.6	10.4	36	0	0
Total	1,117	51.5	48.5	366	126	50

In a cohort of 412 New York–New Jersey insulation workers, who in 1959 had passed 20 or more years after initial exposure, Nicholson reported the observed as compared with expected deaths through 1973 as shown in Table 9–9 (549). Of 258 deaths over the period 1959–1973, 31 (12.0%) were attributed to asbestosis, 60 (23.3%) to lung cancer, and 29 (11.2%) to mesothelioma. Deaths from gastrointestinal cancer were higher than expected, but deaths from all other causes were somewhat reduced by the above competing risks.

Sheers and Templeton, in their 10% sampling of employees of the Devonport (England) dockyards in 1968, found marked differences in the prevalence of both parenchymal and pleural changes with type of occupation as indicated by Figs. 9–1 and 9–2 taken from their report (677). The preponderance of pleural changes in this group is illustrative of the changing type of response now being seen in those with low levels but long periods of exposure. Three years later Mackenzie reported a similar prevalence had been found at other naval dockyards (470). Edge

TABLE 9–9

Expected[a] and Observed Number of Deaths among 412 New York–New Jersey Insulation Workmen January 1, 1959–December 31, 1973, Twenty or More Years after Onset of First Exposure to Asbestos (549)

Period	1959–1963			1964–1968			1969–1973			Total 1959–1973		
Number of men	412			316			222			412		
Person-years of observation	1,823			1,341			919			4,083		
	Exp	Obs	Ratio	Exp	Obs	Ratio	Exp	Obs	Ratio	Exp	Obs	Ratio
Total deaths, all causes	52.79	96	1.81	47.66	94	1.97	40.40	68	1.68	140.85	258	1.83
Cancer, all sites	9.28	44	4.74	8.79	48	5.46	7.79	39	5.00	25.86	131	5.06
Lung	2.36	18	7.62	2.51	25	9.96	2.36	17	7.20	7.23	60	8.29
Pleural mesothelioma	n.a.[b]	2	—	n.a.	2	—	n.a.	4	—	n.a.	8	—
Peritoneal mesothelioma	n.a.	3	—	n.a.	10	—	n.a.	8	—	n.a.	21	—
Stomach, esophagus	1.01	5	4.95	0.80	3	3.75	0.58	2	3.45	2.39	10	4.18
Colon, rectum	1.28	8	6.25	1.20	4	3.33	1.04	1	0.96	3.52	13	3.69
Other	4.63	8	1.72	4.28	4	0.93	3.81	7	1.83	12.72	19	1.49
All respiratory disease	3.34	11	3.29	3.39	16	4.72	3.09	8	2.58	9.82	38	3.87
Asbestosis	n.a.	9	—	n.a.	15	—	n.a.	15	—	n.a.	31	—
All other causes	40.17	41	1.02	35.48	30	0.85	29.52	21	0.71	105.17	89	0.85

[a] Expected deaths are based upon United States white, male, age-specific mortality data of the National Center for Health Statistics.

[b] n.a., Specific rates for these causes of death are not available. They are, however, extremely rare in the general population.

Fig. 9–1. Types of abnormality related to exposure group in British dockyard workers (677). Group 1, continuous exposure: lagger, sprayer, mason, sailmaker–lagger, asbestos storeman, painter. Group 2, intermittent exposure: electrical fitter, riveter-caulker-driller, burner, welder, ship fitter, plumber-coppersmith. Group 3, intermittent exposure: shipwright, engine fitter. Group 4, varied or insignificant exposure: all other occupations including semiskilled and unskilled laborers. Reproduced with the kind permission of G. Sheers and the editor and publishers of the *British Medical Journal*.

also conducted a one-in-ten survey of 80,000 employees in shipbuilding in Barrow-in-Furness, England, in 1968 and found that 6% had pleural plaques, but only 0.5% had pulmonary fibrosis. In 100 consecutive autopsies on males in the local general hospital, he found that one-third had pleural plaques (207).

Fletcher in 1971 reviewed the films of 4,223 persons who had been routinely radiographed for "administrative" reasons at the clinic of a shipbuilding and engineering works at Barrow-in-Furness (248). He found evidence of pleural plaques in percentages varying from 33% in joiners to 1.2% in draftsman. Caulkers, burners, and drillers came second with 21%, and the laggers (in whom high prevalence would be expected) only a third with 17.5% Even in the largest class of 1,472 "unskilled, semi-skilled, and average of all trades" there was an incidence of 5.5%. Joiners on the roster of the carpenters' union, most of whom had worked in the shipyard at some time, showed a 35% prevalence of plaques, but only 7% had calcified plaques; the remainder showed only (radiologically) hyaline plaques. From what was said in the preceding chapter (Chapter 8) about radiological identification of plaques, it will be clear that the significance of reported prevalence

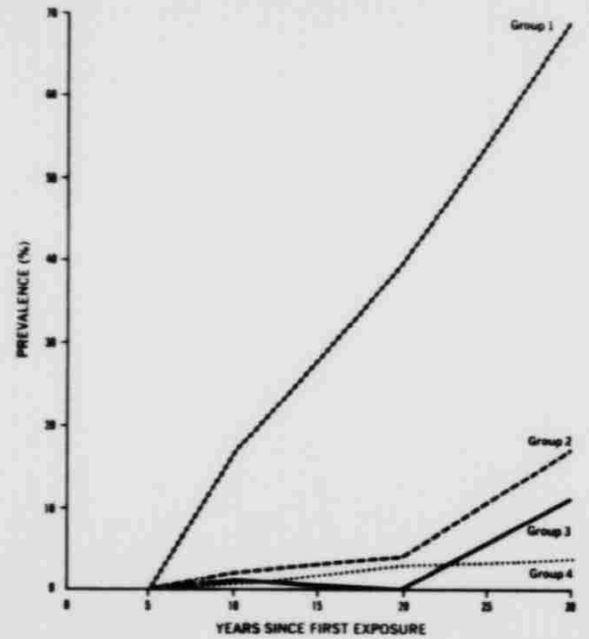


Fig. 9-2. Prevalence of abnormalities in exposure groups in British dockyard workers related to time since first exposure (1968). Groups as in Fig. 9-1 (677). Reproduced with the kind permission of G. Sheers and the editor and publishers of the *British Medical Journal*.

figures will depend upon what kind of plaques were counted and how thorough the examination was. In any case, the number of persons showing plaques radiologically is likely to be less than that of those who actually have plaques, and the apparent number of plaques in any one person markedly less than the actual number.

Table 9-10, quoted by Smither and Lewinsohn in 1973, compares the number of new cases of asbestosis diagnosed by four British pneumoconiosis panels for different periods and different occupations, but gives no indication of the relative number of persons engaged in the various trades (701). The increase with time of the number of cases of

TABLE 9-10

New Cases of Asbestosis Diagnosed by Four British Pneumoconiosis Panels (701)[a]

Principal occupation	No. of cases 1955–1963		No. of cases 1963–1969		Total	
Opening and disintegrating	41 (17%)		45 (10%)		86 (12%)	
Insulating						
Laggers	72 (29%)		134 (28%)		206 (29%)	
Sprayers	13 (6%)	42%	15 (3%)	50%	28 (4%)	49%
Mattress makers	5 (2%)		21 (4%)		26 (4%)	
Others	12 (5%)		71 (15%)		83 (12%)	
Weaving	16 (7%)		14 (3%)		30 (4%)	
Carding, spinning, etc.	37 (13%)		51 (11%)		88 (12%)	
Slab and pipe making	20 (8%)		19 (4%)		39 (5%)	
Brake lining	4 (2%)		10 (3%)		14 (2%)	
Miscellaneous	27 (11%)		91 (19%)		118 (16%)	
	247 (100%)		471 (100%)		718 (100%)	

[a] With kind permission of W. J. Smither and the International Agency for Research on Cancer

Published by AsbestosClaims.Law
asbestosclaims.law/library/25477/8

asbestosis is evident. In contrast, the number of workers estimated to be "at risk" rose only from 18,700 in 1958 to 19,600 in 1963.

Nonoccupational Groups

A study conducted by the Mount Sinai group in 1973–1975 of 490 family contacts of amosite asbestos workers employed over the period 1941–1954, revealed radiographic abnormalities in 194 (39.6%) (Table 9–11).

Parenchymal changes were found to be present in 25, pleural changes in 34 and both parenchymal and pleural changes in 22. Rales were significantly more frequent in those with radiographic changes (see Table 7–6 in Chapter 7).

As mentioned earlier in this chapter, Navrátil and Trippe found a 5.8% prevalence of pleural calcifications in blood relatives of employees of an asbestos manufacturing plant and a 3.5% prevalence in relatives of those workers who had calcifications (541).

The occurrence of pleural calcifications in persons living on asbestos-containing soils in central Europe, but without any opportunity for industrial contamination, was reviewed in Chapter 8, as was their prevalance in persons only vicariously exposed through living in the neighborhood of mines of mills, or engaged in incidental domestic handling of asbestos.

South African experience indicates the risk of neighborhood exposure. Webster, for example, states that a certain percentage of the people living in the vicinity of the asbestos mines and mills may have asbestosis, but gives no exact figures (778). Oosthuizen *et al.* reviewed the radiographs of 2,383 persons in the Northwestern Cape Province and the Northeastern Transvaal and found 166 cases of definite uncomplicated asbestosis (562). They stated that only some of the 2,383 had occupational exposure in which they worked with asbestos fibers; more specifically, of the 59 with pleural plaques, only 32 had had occupational opportunity for exposure. A similar experience was reported by Anspach in 1965 for the area of Dresden in East Germany: 429 cases of pleural calcification in which 54.5% had opportunities for industrial exposure, 35.9% for neighborhood contamination (47). Petrovic correlated the appearance of asbestosis in persons around the mines and mills of Serbia with the concentration of asbestos dust (574).

Summary of Prevalence

Where high levels of asbestos dust prevailed 10–20 years ago, a high incidence of asbestosis can be expected, even up to 100% of those exposed for more than 10 years, but more usually at the rate of 50% of

TABLE 9–11

X-Ray Abnormalities* (1973–1975) among 490 Family Members of Amosite Asbestos Workers Employed 1941–1954

Relative	Total examined	Pleural thickening only	Pleural calcification only	Pleural plaques only	More than one pleural change present	Irregular opacities only	Irregular opacities and some type of pleural change	Total abnormalities
Wives	114	19(16.7%)	3(2.6%)	4(3.5%)	4(3.5%)	15(13.2%)	16(14.0%)	61(53.5%)
Daughters	163	15(9.2%)	3(1.8%)	4(2.5%)	7(4.3%)	10(6.1%)	2(1.2%)	41(25.2%)
Sons	112	23(20.5%)	4(3.6%)	2(1.8%)	6(5.4%)	12(10.7%)	13(11.6%)	60(53.6%)
Siblings	57	4(7.0%)	0	0	0	6(10.5%)	5(8.8%)	15(26.3%)
Other	44	4(9.1%)	1(2.3%)	1(2.3%)	1(2.3%)	4(9.1%)	6(13.6%)	17(38.6%)
Total	490	65(13.3%)	11(2.2%)	11(2.2%)	18(3.7%)	47(9.6%)	42(8.6%)	194(39.6%)

* ILO U/C Classification.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25477/9

TABLE 9-12
Recent Statistics on Pleural Plaques and Asbestos Exposure (513)[a]

Authors	Population type	Country	Number of cases	Plaques		Asbestos exposure			Lung fibrosis		Mesothelioma (if mentioned)
				Number	(%)	Occupational	Environmental	None	Number of cases	Cases (%)	
Siegal *et al.*, 1943	Talc workers	United States	221		6.3	+				14	—
A. R. Smith, 1952	Asbestos workers	United States	261	0	0	+					—
Jacob and Bohlig, 1955	Asbestos workers	Germany	343		5	+					—
Frost *et al.*, 1956	Insulation workers	Denmark	31	11		+					—
Fehre, 1956	Steinholz moulders	Germany	35	7		+					—
Kiviluoto, 1960	Hospital cases from asbestos area	Finland		126			+			24	—
Müller, 1962	Asbestos workers	Germany	651		6.38	+					—
Anspach, 1962	X-ray mass survey	Germany		244		59	118	67			—
Hromek, 1962	X-ray mass survey	Czechoslovakia	9,871	273		–	–	+		0	—
Lawson, 1963	Asbestos workers	England		7		+			5		1
Garavaglia, 1963	Asbestosis	Italy	65	14	21.5	+				100	—
Neef, 1963	X-ray mass survey	Germany	111,069	33		4		29			—
Oosthuizen *et al.*, 1964	Asbestos workers	South Africa	2,383	59		+					—
Marsova, 1964	X-ray mass survey	Czechoslovakia	44,569	241		–	–	241		0	—
Cartier, 1965	Asbestos workers	Canada		126		+				14	—
Selikoff, 1965	Insulation workers	United States	1,117	150		+			"Many"		—
Meurman, 1966	Autopsy cases	Finland	438	172	39.3	–					—
Hourihane *et al.*, 1966	X-rays of hospital	England	27,500	36					7		2
Hourihane *et al.*, 1966	Autopsy cases	England		56	4.2				24		15
Kleinfeld *et al.*, 1966	Asbestos workers	United States	21	5		+					—
Raunio, 1966	X-ray mass survey	Finland	631,000	1,516		2	Many		1		—
Zolov *et al.*, 1967	X-ray mass survey	Bulgaria	3,429	175		43	132		7		—

[a] With kind permission of L. O. Meurman.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25477/10

those exposed for more than 20 years, with increasing frequency with longer employment.

The incidence to be expected falls as the exposure level decreases and the time to appearance of significant disease increases. But even at the level of 5 mppcf, some disease can be expected to appear after 20 years of exposure.

Familial and neighborhood cases can be expected at lower frequencies than those seen in persons occupationally exposed.

As the exposure level falls, the character of the disease tends to change from predominantly parenchymal to that of pleural involvement, which, although less productive of disability is less readily diagnosed, and may indicate that the population is liable also to the risk of mesothelioma.

Some pleural plaques may be found in persons with no ascertainable exposure to asbestos or talc, or to a less provocative mineral, mica. This may be particularly true in areas where asbestiform minerals are common in the soil. In any one particular case, the cause may be conjectural.

Meurman has provided a useful table of the reported prevalence of pleural plaques and their relations to exposure which is reproduced as Table 9–12 (513).

DIAGNOSIS

In comparison with other diseases, medical science has not yet provided very much in the way of reliable and practical methods of arriving at a firm diagnosis of asbestosis in the absence of a reliable history of occupational or other established exposure. As the field of opportunities for exposure widens, the possibility of asbestosis and particularly that of the pleura increases, and the range in which diagnosis may be doubtful is at the same time increased. This is particularly true in the incipient stages of disease when further decrease in functional disability and shortening of life might be somewhat reduced by removal of the affected person from further exposure, and perhaps new skills learned that would be compatible with the patient's functional capabilities. The art of diagnosis, always a matter of weighing probabilities and of looking at the total evidence, is seldom subjected to greater challenge than in the lesser stages of asbestosis (532,534,602). Even the demonstration of fibers or of asbestos bodies in lung biopsy, itself not a procedure to be undertaken lightly, lends supporting rather than conclusive evidence.

The interstitial pulmonary fibrosis produced by asbestos and termed parenchymal asbestosis, described in Chapter 7, is essentially similar to that seen in some persons with no history of exposure, to which the terms Hamman–Rich syndrome or "idiopathic" diffuse interstitial fibrosis are sometimes applied (285,753). This unhappy dependence on a history of exposure for the making of a diagnosis is not uncommon in occupational medicine, as witness the state of diagnostic procedures in so-called coal workers' pneumoconiosis, byssinosis, and beryllosis. It is only late in the disease that the indications add up to a virtual certainty, and by then very little can be done for the patient or for his resultant socioeconomic problems.

The relative evidence can be conveniently considered in four categories: exposure history, functional impairment, radiographic changes, and pathological indications. Many of the items have been considered in the two preceding chapters (Chapters 7 and 8), and will be reviewed here only as they pertain to diagnosis.

Exposure History

As indicated above, a history of exposure is a most important element in the diagnostic evidence. Unfortunately, it may be quite difficult to acquire. Persons currently employed in the mining, milling, manufacture, or utilization of asbestos constitute only a part of the population at risk. Furthermore, the dustiness of their workplace 20 years ago, when the disease process was initiated, was probably much greater than it would be today. Persons exposed for only short periods many years ago, as for example in wartime temporary employment, may not think the experience worth mentioning, or they may actually have forgotten about it. Even short periods a couple of decades ago, when controls were not so stringent, may have been quite sufficient for pathogenesis to begin. Such short exposures are easily overlooked, but they cannot be lightly dismissed. The possibility of short exposure needs to be borne particularly in mind where the presenting picture is one of pleural change with little in the way of parenchymal involvement. (We will see later in Chapters 10 and 11 that these short exposures are even more critical in the induction of mesothelioma. In any case of interstitial pulmonary fibrosis of undetermined origin, of pleural calcification, or of alveolar capillary block syndrome, no effort should be spared to elicit possible exposure to asbestos. A correct diagnosis is not an academic matter; the patient's entitlement to compensation or care may be jeopardized, and he has little else to look forward to. Appropriate

clinical care, moreover, depends on the diagnosis: steroids, often used in cases of Hamman–Rich syndrome, are contraindicated in asbestosis, and persons with asbestosis should be kept under surveillance for the possible development of neoplastic changes.

The possibility of incidential or vicarious exposure must also be remembered. Mention was made in discussing the etiology of parenchymal asbestosis (Chapter 7) of the possibility of neighborhood or family exposure and of the appearance of disease in persons working around but not in the actual handling of asbestos, as in shipyards or building operations. This is certainly important in the construction industry (with approximately four million employees in the United States alone), in the shipbuilding and ship repairing industry, in power plants, in brake repair and maintenance operations, and in railroading as well as in various other major industries. One cannot neglect the fact either that there are over 3,000 asbestos consumer products on the market that may permit exposure in the course of their manufacture, use, or disposal. We have as yet only partial estimates of how important this last category of exposure may be.

Functional Impairment

Meiklejohn, says Hutchinson, claimed that he could tell which of the girls in a factory had asbestosis by dancing with them (363). Our more prosaic generation seldom indulges in this approach to diagnostic evidence, and seeks perforce more quantifiable and perhaps more objective information. Even in fairly early stages of the disease, there are some indicators that can be sought once a suspicion of exposure has been aroused or in the course of a routine examination of exposed personnel. One that sometimes develops quite early is a reduction in the carbon monoxide diffusion capacity of the alveoli (243,535,602), which may be detectable before any complaint of dyspnea is offered. Changes in compliance are considered by some to provide an early suggestive sign (205,495). Reduced rate of lower lobe emptying in expiration, as revealed by an admixture of radioactive xenon, is advocated by others (711). At a somewhat later stage, dyspnea accompanied by reduced vital capacity and lung volume, and the presence of persistent, fine rales at the bases of the lungs constitute a very suggestive combination, even in the absence of demonstrable radiographic changes.

Later in the disease, clubbing of the fingers is often seen and cyanosis may be present. By this time, however, dyspnea is often marked and

radiographic appearances consistent with the diagnosis have usually appeared.

Radiographic Changes

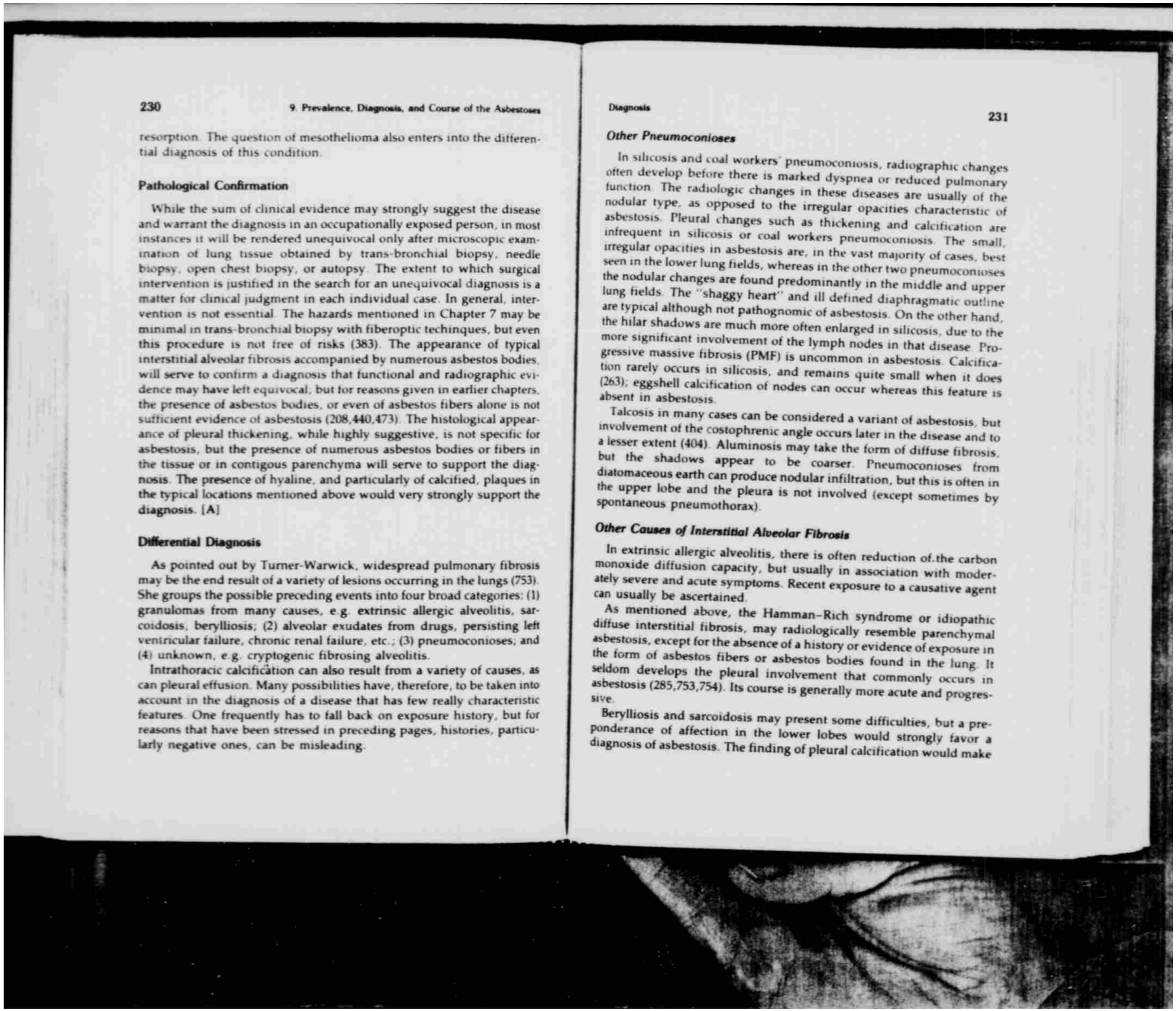
For systematic evaluation of chest radiographs in suspected cases of asbestosis, the UICC classification, developed for silicosis and coal workers' pneumoconiosis, has been modified to the form known as the ILO-U/C classification, and is particularly useful for the recording of pleural changes and irregular parenchymal infiltrates, as described in Chapter 7 (365).

Parenchymal changes provide few radiographic features that are characteristic of asbestosis, but some are highly suggestive particularly when they occur together (Plates 7–2,a–h). The irregular, reticular, and nodular changes are more commonly seen in the lower two-thirds of the lung fields and are generally more easily visible on the right side. The apices remain lucent and massive confluent shadows are uncommon. Hilar enlargement is not usually marked until pulmonary hypertension develops. A shaggy heart outline is not rare, interlobar septa may be visible, and shadows described as resembling Kerley "B" lines may appear. As lung volume diminishes, the apicocaudal diameter is diminished, the costophrenic angle is narrowed, and the lesser interlobar fissure descends.

Pleural thickening may lead to obliteration of the costophrenic, and sometimes of the cardiophrenic angles, and to tenting of the diaphragm, although the latter may be found without adhesions. The pleural fibrosis may appear as soft tissue between the lung periphery and the chest wall, and adhesions may distort the lung shadow. This is particularly true in the region of the lingula or mid-lung fields ("mid-lung tug"). The fibrosis may give some graded shadowing over the peripheral field where the rays become tangential to the thickened pleura.

Hyaline pleural plaques may appear as faint diffuse shadows that persist in repeated films when viewed *en face*, and may be seen a little more clearly when viewed end on. The presence of calcified pleural plaques, distinguished from intrapulmonary calcification, are practically pathognomonic of exposure to asbestos, talc, or (occasionally) mica, especially in the absence of past thoracic hemorrhage, trauma, or tuberculosis (Plates 8–1,a–o).

Asbestos exposure must be considered when pleural effusion, free or loculated, has come on quietly, particularly if it undergoes spontaneous



resorption. The question of mesothelioma also enters into the differential diagnosis of this condition.

Pathological Confirmation

While the sum of clinical evidence may strongly suggest the disease and warrant the diagnosis in an occupationally exposed person, in most instances it will be rendered unequivocal only after microscopic examination of lung tissue obtained by trans-bronchial biopsy, needle biopsy, open chest biopsy, or autopsy. The extent to which surgical intervention is justified in the search for an unequivocal diagnosis is a matter for clinical judgment in each individual case. In general, intervention is not essential. The hazards mentioned in Chapter 7 may be minimal in trans-bronchial biopsy with fiberoptic techinques, but even this procedure is not free of risks (383). The appearance of typical interstitial alveolar fibrosis accompanied by numerous asbestos bodies, will serve to confirm a diagnosis that functional and radiographic evidence may have left equivocal; but for reasons given in earlier chapters, the presence of asbestos bodies, or even of asbestos fibers alone is not sufficient evidence of asbestosis (208,440,473). The histological appearance of pleural thickening, while highly suggestive, is not specific for asbestosis, but the presence of numerous asbestos bodies or fibers in the tissue or in contigous parenchyma will serve to support the diagnosis. The presence of hyaline, and particularly of calcified, plaques in the typical locations mentioned above would very strongly support the diagnosis. [A]

Differential Diagnosis

As pointed out by Turner-Warwick, widespread pulmonary fibrosis may be the end result of a variety of lesions occurring in the lungs (753). She groups the possible preceding events into four broad categories: (1) granulomas from many causes, e.g. extrinsic allergic alveolitis, sarcoidosis, berylliosis; (2) alveolar exudates from drugs, persisting left ventricular failure, chronic renal failure, etc.; (3) pneumoconioses; and (4) unknown, e.g. cryptogenic fibrosing alveolitis.

Intrathoracic calcification can also result from a variety of causes, as can pleural effusion. Many possibilities have, therefore, to be taken into account in the diagnosis of a disease that has few really characteristic features. One frequently has to fall back on exposure history, but for reasons that have been stressed in preceding pages, histories, particularly negative ones, can be misleading.

Other Pneumoconioses

In silicosis and coal workers' pneumoconiosis, radiographic changes often develop before there is marked dyspnea or reduced pulmonary function. The radiologic changes in these diseases are usually of the nodular type, as opposed to the irregular opacities characteristic of asbestosis. Pleural changes such as thickening and calcification are infrequent in silicosis or coal workers pneumoconiosis. The small, irregular opacities in asbestosis are, in the vast majority of cases, best seen in the lower lung fields, whereas in the other two pneumoconioses the nodular changes are found predominantly in the middle and upper lung fields. The "shaggy heart" and ill defined diaphragmatic outline are typical although not pathognomic of asbestosis. On the other hand, the hilar shadows are much more often enlarged in silicosis, due to the more significant involvement of the lymph nodes in that disease. Progressive massive fibrosis (PMF) is uncommon in asbestosis. Calcification rarely occurs in silicosis, and remains quite small when it does (263); eggshell calcification of nodes can occur whereas this feature is absent in asbestosis.

Talcosis in many cases can be considered a variant of asbestosis, but involvement of the costophrenic angle occurs later in the disease and to a lesser extent (404). Aluminosis may take the form of diffuse fibrosis, but the shadows appear to be coarser. Pneumoconioses from diatomaceous earth can produce nodular infiltration, but this is often in the upper lobe and the pleura is not involved (except sometimes by spontaneous pneumothorax).

Other Causes of Interstitial Alveolar Fibrosis

In extrinsic allergic alveolitis, there is often reduction of the carbon monoxide diffusion capacity, but usually in association with moderately severe and acute symptoms. Recent exposure to a causative agent can usually be ascertained.

As mentioned above, the Hamman–Rich syndrome or idiopathic diffuse interstitial fibrosis, may radiologically resemble parenchymal asbestosis, except for the absence of a history or evidence of exposure in the form of asbestos fibers or asbestos bodies found in the lung. It seldom develops the pleural involvement that commonly occurs in asbestosis (285,753,754). Its course is generally more acute and progressive.

Berylliosis and sarcoidosis may present some difficulties, but a preponderance of affection in the lower lobes would strongly favor a diagnosis of asbestosis. The finding of pleural calcification would make

Published by AsbestosClaims.Law
asbestosclaims.law/library/25477/13

it certain. Changes in compliance are less evident in these diseases (205).

Pulmonary Infections

Tuberculosis commonly involves the apical and subclavicular areas at an early stage. It may be confused with asbestosis of the upper lobe, but the latter is usually accompanied by fibrosis in the lower lobe or pleura. A risk of superadded tuberculous infection is not increased by the presence of asbestosis. With the decreasing frequency today of tuberculous infection, an upper lobe lesion has almost as good a chance of being asbestotic. Old calcified tuberculous lesions may occasionally resemble pleural plaques, but can be shown to lie at various depths in the lung substance. When located in the pleura, they are generally an aftermath of tuberculous empyema and are often much thicker than asbestotic calcifications.

Histoplasmosis is usually fairly well distributed over the lung field and radiographic shadows are larger and denser than the fine nodulation of asbestosis. Symptoms and radiographic indications are usually coincident.

The calcified lesions of trichinosis can be shown to lie chiefly in the intercostal muscles rather than in the parietal pleura. They do not take on the bizarre and confluent forms often seen with asbestotic plaques, nor are they commonly seen along the diaphragmatic contours.

The radiographic shadows of chronic bronchitis are principally along the lines of the bronchial tree and not in the form of a reticulation in the intervening spaces. There may, however, be some chance of confusion in the early stages of asbestosis. Productive cough is not a marked feature of asbestosis unless some bronchitis has supervened or the patient is a heavy smoker.

Other Causes of Pleural Calcification

The possibility of calcified tuberculous pleural lesions or of calcified hematoma remains will usually be revealed by the patient's medical history. Calcification secondary to pleurisy or tuberculous empyema is nearly always posterior or posterolateral when located in the lower or middle portions of the chest; it is very rarely diaphragmatic, mediastinal, or pericardial. Mica is the only mineral other than talc and asbestos reported to produce calcified pleural plaques. The rare occurrence in silicosis was mentioned above. [Floyd and Hepburn (249), Head (325), and Kristiansen (442) may be consulted for details on other causes.]

Other Causes of Pleural Effusion

Causes other than asbestos usually announce themselves by their own clinical patterns (e.g., tuberculosis or other infections, nephrotic syndrome, cardiac failure, neoplasms), including localized pain. Benign asbestotic effusions, on the other hand, add little in the way of symptoms, and frequently undergo spontaneous reabsorption, but may recur after reabsorption or after removal by tapping (215,455,471). The most important question is whether mesotheliomatous changes are present in the pleura. Cytological examination of the fluid may help to answer this question, but interpretation may be difficult (see Chapter 11).

Other Causes of Dyspnea

Asbestotic dyspnea characteristically comes on gradually, but it may be aggravated by respiratory infection. It is not accompanied by indications of other disease patterns, such as cardiac insufficiency, which could account for the symptom, at least until cor pulmonale supervenes late in the development. Chronci bronchitis and emphysema are not common in asbestosis unless it is correlated with cigarette smoking. In the absence of other discernable causes of dyspnea, and in the presence of a history of exposure to asbestos, changes of pulmonary function combined with dyspnea offer strong probability that the condition is one of asbestosis.

Other Pulmonary Affections

Asbestosis is not normally accompanied by marked or widespread emphysema. Carcinoma may occur as part of the tissue response to asbestos (Chapter 12), with or without indications of asbestosis. It may, of course, occur quite independently of exposure to asbestos. The effects and diagnostic features of carcinoma will be essentially similar in both instances, except for a predilection for the lower two thirds of the lung where asbestos is a significant cofactor in the causation.

Anginal pain may be mimicked in a person with asbestosis who also has some hypertension and is undertaking heavy manual labor.

CLINICAL COURSE

Progress of Disease

In most patients the degree of fibrosis progresses even after removal from further exposure. (See Table 7–9 for the later development of fibrosis after brief exposures.) Pulmonary function declines, radiological ap-

pearances intensify, and disability increases. This course of events is well reviewed by Leathart (444). In 14 cases that he followed for 2–9 years, there was a general downward trend amounting to an average fall of 19% in compliance, 16% in vital capacity, and 26% in diffusing capacity over the period of observation. There were a few individuals, however, whose lung function did not alter any more than would be expected from advancing age.

Experience at Mount Sinai is generally in accord with this (58). Bader *et al.* found that in 12 out of 13 persons who were re-examined after 10-years removal from industry, the vital capacity fell from 9–31%, more than could be expected on the basis of aging alone. The maximum breathing capacity also fell in 12 of the patients by 6–51%, with an average of 21% (58). (This contrasts with the normal to high maximum breathing capacity that these persons showed when first examined.) Arterial oxygen saturation at rest was reduced in only 3 of the 13, as was the saturation on exercise. Marked falls were observed, however, in one individual who died shortly afterward with cor pulmonale. The correlation between reduction in vital capacity and radiographic progression was good in six, fair in two, and poor in five of the patients. Those with poor correlation usually showed a fall in vital capacity without corresponding increase in radiographic signs. Further experience since 1965 confirms the observation, however, that some individuals seem to benefit by a reduction or cessation of disease progress after their removal from exposure.

Treatment

In the absence of complications, the functional impairment is to be attributed almost entirely to the parenchymal involvement. Pleural changes, except perhaps for extensive thickening and recurrent effusion, add little to the disability. No specific treatment has been found to affect the course of the disease. Neither aluminum nor poly-2-vinylpyridine-1-oxide (PVPNO) (176) restrict the disease, and the former has been rejected by the American Medical Association (162,454). Some improvement has occasionally been reported after the administration of steroids (529), but there is a decided risk of lowering the immunity to superadded infection.

Infection occurring in the disabled lung is fraught with more than ordinary significance and early attention is essential. Prophylactic antibacterial therapy, however, is not necessary as it is with chronic bronchitis; the patient usually has time to consult his physician after the infection starts.

Embarrassment of Pulmonary Circulation

Fairly early in the disease, even when radiographic findings are minimal, the second pulmonary heart sound may be accentuated and the pulmonary arterial pressure raised. Later in the disease, the developing interstitial fibrosis frequently increases the embarrassment to the point that the right heart first dilates and then begins to fail. The resulting enlargement is not readily visible in the chest radiograph. The sequence of events that usually follows this development must be anticipated by the physician. It is difficult to say what proportion of deaths stem directly from this sequela, since immediate and ultimate causes of death are not always clearly distinguished on death certificates, and the terminal event may be a superadded respiratory infection tipping the patient's precariously balanced scales. Cor pulmonale may very well be present in cases whose death is attributable to some other event. Kleinfeld *et al.* reported that 19 out of 91 deaths in a cohort of talc workers were due to cor pulmonale, but there were also 25 deaths listed from other types of cardiac failure (409). Wagner is quoted (421) as finding in South Africa that 12% of 200 cases of asbestosis had this development. Jacob reported that of 730 deaths among workers in the asbestos industry of Saxony, 32 were due to cor pulmonale, with the following relationship to fibrosis: grade 0–I, 2/166; grade I, 6/298; grade II, 13/208; grade III, 11/58 (371). In general, it is clear that the frequency increased with the degree of fibrosis, but some instances occurred where there was little to be found radiographically.

Respiratory Infection

Tuberculosis

In the earlier years of the asbestos industry, tuberculosis was a common disease in the community that could affect asbestos workers just like anyone else. Scarpa is quoted (23) as reporting on 20 persons working with asbestos who died from tuberculosis over the period 1894–1906 in Turin, but we do not know the size of the working population from which these cases were drawn. Vigliani found that the disease accounted for 8% of 307 deaths in persons compensated for asbestosis in four Italian provinces over the period 1943–1969 (758). On the basis of experience in the Soviet Union, Dvizhkov considered in 1962 that tuberculous pneumonia appeared to be more common in the presence of asbestosis and that the lowering of resistance when asbestosis is present was evidence by the tuberculous endobronchitis that they have seen (206). Butkin and Shelkovina in 1963 analyzed the

prevalence of tuberculosis among asbestos workers in Asbestovsk and found 207 cases, but the size of the population is not given and we do not know the prevalence in the nonexposed population (124). In 1970, Ivanovskaya reported that of 441 patients from the asbestos industry in Sverdlovsk (45% inpatients, 55% outpatients), 100 suffered from "asbestotuberculosis" (369). Again we do not know the size of the population at risk, but it is clear that tuberculosis is still a menace in those parts.

In Britain and in the United States the general incidence of tuberculosis has markedly declined, and this is reflected in the falling rate to be seen in asbestos workers. Smither compared the 31.3% found at postmortem by Wyers in 1931–1949, with the 5.6% in his series in the same factory over the period 1957–1964 (700). In 98 deaths of insulation workers in Belfast, Elmes and Simpson found 3 out of 10 from tuberculosis in 1940–1944 and 3 out of 7 in 1945–1949; none after that through 1966 (219). In the United States, tuberculosis is seldom seen now as a complication of asbestosis.

Other Infections

Asbestos workers, of course, share, with the community at large, the risk of various respiratory infections. To the extent that bronchial deformation produced in the course of asbestosis results in the pocketing of secretions, the liability of pulmonary effects is increased (369,371,774). As the fibrosis advances, there is progressively less normal lung to deal with the threat of infection and to maintain adequate gas exchange. Consequently, bronchopneumonia is a common terminal event with pulmonary edema finally making the continuance of life impossible.

Rheumatoid and Other Immunologic Factors

In 1965 Pernis *et al.* found a definite increase in the frequency and titers of rheumatoid factors, mainly in that which reacts with human γ-globulins, in persons with asbestosis of grades 2 and 3 (573). Turner-Warwick and Parkes drew attention in 1970 to reports of the rare occurrence of necrobiotic nodules in the lungs of asbestos workers (754). They found antinuclear factors in 28% of 80 patients with asbestosis, and rheumatoid factors in 27%—at least a fourfold increase over their incidence in a random population. Here, also, there was a suggestion of correlation with the progressiveness of the disease. The conclusion was strengthened by a later comparison of these findings with two groups of persons exposed to asbestos but not showing evidence of disease (751). Lange *et al.* have recently reported similar observations

(430). Immunoglobulins are found to be higher in persons with parenchymal asbestosis or who have been exposed for a long time (382,432). There is a depletion of T lymphocytes in these people (382,429).

Four cases have been reported of Caplan's syndrome in patients with asbestosis; the overall significance of this uncommon finding is not known (488). Two or three cases of coexistent lupus erythematosus are known to us (799), one or two of periarteritis nodosum, one of Wagoner's granulomatosis, and at least one case of sarcoidosis superimposed on asbestosis (691).

To bring some order into this incomplete picture, and to attempt some explanation of why some individuals are quickly and markedly affected by asbestos while others continue with little or no involvement, Turner-Warwick and her associates have undertaken a systematic survey of three groups of asbestos workers in Britain. The survey is still proceeding, but a very useful review of the findings to 1973 was presented at the Lyon conference (752). The summary of the report was as follows:

> (a) Non-organ-specific autoantibodies, especially ANA (antinuclear antibody), are found with increased frequency in those asbestos workers with abnormal physical signs suggesting asbestosis, who have been heavily exposed to asbestos dust for a long time.
>
> (b) In contrast, the amounts of autoantibodies are only slightly increased in those with heavy exposure but without lung disease.
>
> (c) In the PMP [Pneumoconiosis Medical Panel] series [one of the three groups] there is an almost linear relationship between the percentage of positive-ANA and duration from the time of first exposure, with a plateau at 25–30 years.
>
> (d) A trend is shown in all three surveys of a slightly higher than expected prevalence of ANA in workers without clinical signs, and this may suggest that there is an early stimulus to ANA production in some asbestos workers.
>
> (e) Lymphocyte sensitization to nuclear antigens was not found in asbestosis.
>
> (f) No evidence of lung-specific antibodies has been obtained.
>
> (g) Challenge of lymphocytes by asbestos fiber results mainly in cytotoxicity.
>
> (h) Occasional intraavleolar macrophages appear to contain immunoglobulins, mainly IgM and complement. Whether this occurrence relates to an immunological response to asbestos fiber or to coincidental microbial infection has not been ascertained.

In 1975 Merchant added: "In a study of the HL-A system in 56 selected asbestos workers referred to the Pneumoconiosis Medical Panel with definite or suspected asbestosis, the W27 antigen had definite radiographic evidence of asbestosis compared to 13 out of 46 without the W27 antigen. These observations, if confirmed, suggest that the W27 antigen may provide a useful marker of an enhanced susceptibility to the tissue-damaging effects of asbestos dust" (505). Matej and Lange report similar findings (486).

Published by AsbestosClaims.Law
asbestosclaims.law/library/25477/16