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Prevalence and Incidence of Benign Asbestos Pleural Effusion in a Working Population

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• **Benign asbestos effusion was defined by (1) exposure to asbestos, (2) confirmation by roentgenograms or thoracenteses, (3) no other disease related to pleural effusion, and (4) no malignant tumor within three years. There were 34 benign effusions among 1,135 exposed workers compared with no otherwise unexplained effusions among 717 control subjects. Prevalence was dose related with 7.0%, 3.7%, and 0.2% effusions with severe (III), indirect (II), and peripheral (I) exposure, respectively. The latency period was shorter than for other asbestos-related disorders. Benign effusion was the most common asbestos-related abnormality during the first 20 years after exposure. Incidence studies showed 9.2 effusions per 1,000 person-years for level III exposure, 3.9 for level II, and 0.7 for level I. Most effusions were small; 28.6% recurred, and 66% were asymptomatic. There was one mesothelioma six years after effusion. Asbestos exposure should be carefully searched for in patients with "idiopathic" pleural effusion.**

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AMONG the several asbestos-related pleural manifestations, benign effusion is the most recent to come to our attention.[1] Though still considered a rare complication of asbestos exposure, the clinical and histological features of such effusions are now well described.[2,3] However, these case reports do not permit conclusions concerning frequency in asbestos-exposed populations. We studied serial roentgenograms and medical records of 1,135 employees in the asbestos industry and compared these with the records of 717 control subjects to determine (1) the prevalence and incidence of asbestos effusions, (2) the duration and amount of exposure associated with effusion, (3) the relationship between effusion and diffuse pleural thickening, and (4) the frequency of subsequent mesothelioma.

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MATERIALS AND METHODS

Definition

Benign asbestos effusion was defined by four criteria, as follows: (1) direct or indirect exposure to asbestos, (2) an effusion confirmed by a transient pleural change in serial chest films or by thoracentesis, (3) lack of evidence for any other disease related to pleural effusion, and (4) no malignant tumor detected within three years after the effusion.

Study Populations

Two groups were selected: a "survey group" of working exposed persons seen by us at yearly intervals at industrial sites and a "control group" of male employees from a large university.

The survey group consisted of 1,135 employees, including 45 women, from six plants:

1. Shipyard A (new-ship construction): 93 directly exposed pipecoverers and 74 indirectly exposed shipfitters first seen in 1965. Asbestos had been used regularly since 1930.

2. Shipyard B (submarine new construction and refitting): 317 employees, including directly exposed pipecoverers and sweepers and indirectly exposed welders, lead bonders, and others seen annually since 1976. Asbestos had been used from 1952 to 1975.

3. Fireproofing product manufacturing: 144 employees, some heavily exposed since the early 1930s, seen annually for the last ten years.

4. Mill A (specialty paper): 90 employees heavily exposed to crocidolite in the manufacture of filter paper during a special project between 1952 and 1956 and 126 employees with slight exposure, seen since 1971.

5. Mill B (specialty paper): 211 employees manufacturing filter paper and gaskets, who had slight but strictly monitored exposure since 1968, seen annually for the past six years.

6. Mill C (specialty paper): 80 employees exposed to "bonded asbestos" used for electrical insulation since 1930, seen for the last three years.

The control group consisted of faculty and employees of a large university. Serial films, some dating back to 1940, were available because of a law requiring roentgenograms of school employees before employment and every three years thereafter. We located an active "three-year recall list," which excluded persons who had been exposed to beryllium or asbestos at the university. We coded all films of the 717 male employees on this list according to methods used during our industrial surveys.

For comparison of clinical features and follow-up data, we referred to a group of 178 asbestos exposed clinical patients (Table 1). These included 26 with benign pleural effusion, 12 of whom have been described in detail.[1]

Methods of Investigation

The survey group answered a respiratory questionnaire, had a physical examination, screening pulmonary function studies consisting of force vital capacity (FVC) and its time derivatives, including a forced expired volume in 1 s, and also a single-breath diffusing capacity (Dsb).[4] Occupational histories, obtained by specially trained physicians, included a listing of all past jobs and exposures, present job description, year first exposed, and total years exposed. Dust exposure had been

monitored with variable consistency and duration, while instrumentation, manner of counting, and sampling sites had also varied over the years. Therefore, for this study we assigned employees to one of three exposure levels that were based in part on job description, in part on dust or fiber counts, and in part on personal observations. Generally, employees with peripheral exposure, such as administrators, clerks, and secretaries, were considered to have level I exposure; indirectly exposed employees, such as certain electricians, welders, mechanics, shipfitters, or machine operators, who worked in the plant were placed in the level II group; and the level III group consisted of directly exposed pipecoverers, asbestos mixers, or sweepers.

Chest roentgenograms at the time of study and all available previous films were read twice, first by a group of two to three chest physicians who knew the occupational history, and later by a chest radiologist who was unaware of the history. Films were coded according to the ILO U/C 1971 international classification of radiographs of the pneumoconioses.[11] This allowed for description of pleural disease, such as extent and thickness of plaques, calcifications, and diffuse thickening. Blunting of costophrenic angles was also recorded. A separate notation was made for "effusion" only if (1) there had been hospitalization with thoracentesis or (2) when effusions were obvious from transient roentgenographic changes or the sudden appearance of blunting of the lateral or posterior costophrenic angles with a miniscus typical of pleural effusion.

RESULTS

The survey and control groups were comparable in most respects. In the survey group, 96.0% were men, and for control subjects we selected only men. The mean age of the survey group at the first visit was 41.7 years and of the control group at the time of reading, 42.0 years. Although most of our control group were not professors but, rather, laboratory technicians, maintenance personnel, and grounds workers, the university environment admittedly is different from that of large shipyards or factories. Nevertheless, other than for an increased amount of trauma that may cause effusion, there seemed to be no important exposures except for asbestos, which have been related to pleural disease. Initial roentgenograms of the oldest asbestos workers dated back to 1935; for the university group, to 1940. The entire survey group was presumed to have had asbestos exposure, however slight; while among the control subjects, those who had been exposed to asbestos at the university were excluded. A few control subjects did have previous exposure, largely because of the many shipyards in this area. Indeed, 13 control subjects (1.8%) had typical pleural plaques, four with calcifications. Occupational histories of these 13 showed that eight had previously worked in shipyards or as Navy machinists or steamfitters. Diffuse pleural thickening was seen in two controls, a former shipyard worker and a former ship's engineer.

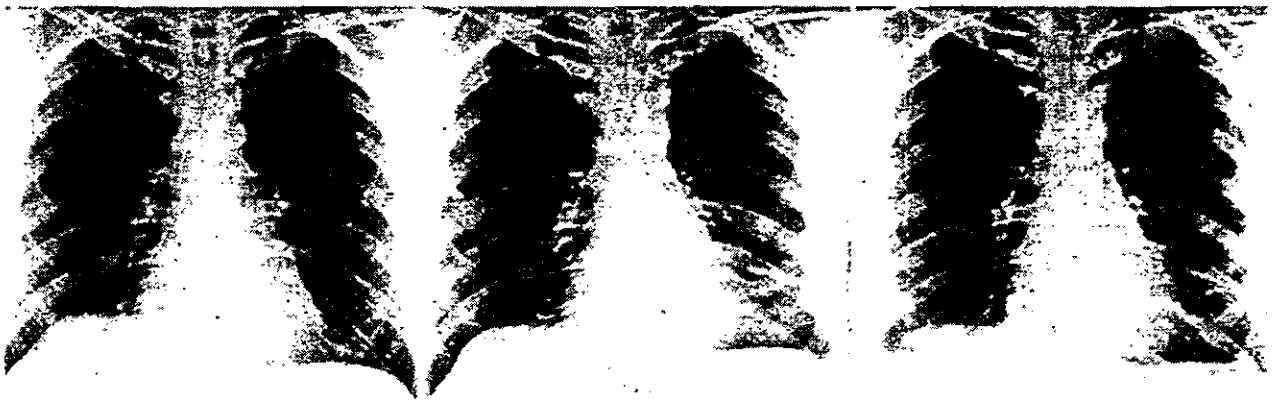
Table 1.—Pleural Effusions in Survey and Control Groups and Exposed Clinic Patients

Classification	Survey Group, n=1,135	Control Subjects, n=717	Exposed Clinic Patients, n=178
Total Pleural Effusions	54	7	45
Known disease related			
Mesothelioma	4	0	16
Lung cancer	2	0	2
Pneumonia or empyema	5	2	0
Chest surgery	2	2	1
Trauma	4	1	0
Spontaneous pneumothorax	2	0	0
Tuberculosis	0	1	0
Congestive heart failure	0	1	0
Benign asbestos effusion	35	0	26

Prevalence of All Effusions

Pleural effusions of all kinds were five times more common in the survey group (4.8%) than in the control subjects (1.0%), a highly significant difference ($P<.001$) (Table 1). This was in part because of a greater number of asbestos effusions and in part because of effusions related to mesothelioma and lung cancer in the survey group. Cancer-related effusions were even more common in our 178 asbestos-exposed clinic patients. There were 19 (10.7%) with effusions

Fig 1.—Chest roentgenogram of 55-year-old asymptomatic shipyard pipecoverer since 1969. Yearly films to January 1973 (left) were normal. During next 13 months asymptomatic left pleural effusion developed, resulting in diffuse pleural thickening in March 1974 (center). During next year asymptomatic right effusion with residua seen in March 1975 (right). No change occurred during next five years.



related to obvious disorders, 18 of which were pleural or pulmonary malignancy (Table 1).

Prevalence of Benign Asbestos Effusion

The overall occurrence of one or more asbestos effusions in the survey group was 3.1% (Fig 1 and 2), while there were no otherwise unexplained effusions in the control group (Table 1).

Dose Response

In the first four plants listed in Table 2, where there were many employees at each exposure level, there was an obvious relationship between exposure and prevalence of asbestos effusions. These prevalences ranged from 7.2% to 14.3% at level III and ranged from 0% to 4.3% at level I. In the fifth plant, mill B, where exposure had been recent, slight, and carefully controlled, there were no documented effusions. In mill C, there were few level III exposed employees left at the time of our first survey. For the entire survey group, the prevalences of asbestos effusion were 7.0%, 3.7%, and 0.2% at exposure levels III, II, and I, respectively.

There was also a relationship to occupation (Table 3). Asbestos effusions were most common among asbestos pipecoverers (7.3%) (Fig 1 and 2), less common in asbestos product and paper machine operators (5.3%), and least common among shipfitters, maintenance personnel, and welders. The fact that effusions were seen in level II exposures and in an office executive (Table 3), and have been seen in the wife of an employee,[16] suggests that the requisite exposure threshold may be low.

Latency

The latent period, that is, the interval between first exposure and clinical evidence of disease, is shown for several asbestos-related disorders in Fig 3. Only persons exposed at level II or III were included, because only for these was onset of exposure precisely known. There were no cases of asbestosis, pleural plaques, or calcifications during the first ten years after initial exposure. These three manifestations increased steadily over subsequent years. The latent period for benign asbestos effusion was shorter.

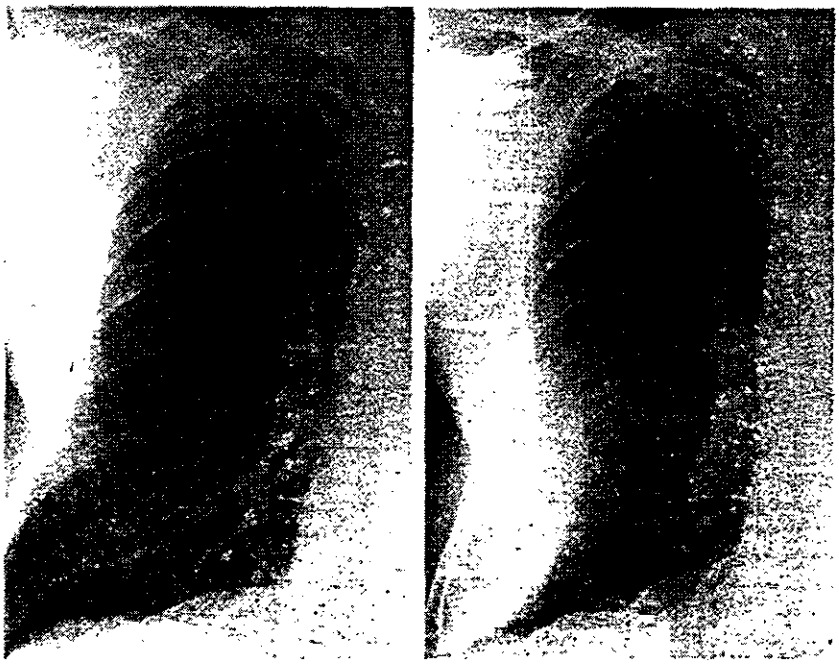


Fig 2.—Chest roentgenograms of 51-year-old shipyard pipecoverer had been normal for 18 years (left). Three months later, pleuritic chest pain developed on right side, as well as pleural-based density (right). Exploratory thoracotomy for "mesothelioma" showed encapsulated bloody effusion. Patient has remained well for three years.

Table 2.—Prevalence and Incidence of Asbestos Effusion

Industry and Exposure Level	No. of Employees	Asbestos Effusion, No. (%)	Observation, Person-Years	Rate per 1,000 Person-Years
Shipyard A				
III	93	6 (6.2)	572	10.5
II	74	1 (1.4)	494	2.0
Shipyard B				
III	126	9 (7.1)	1253	7.2
II	79	3 (3.8)	897	3.3
I	112	0 (0.0)	409	0.0
Asbestos products				
III	27	4 (14.8)	280	14.3
II	71	6 (8.5)	1010	5.9
I	46	1 (2.2)	230	4.3
Paper mill A				
III	70	4 (5.7)	322	12.4
II	20	0 (0.0)	79	0.0
I	126	0 (0.0)	289	0.0
Paper mill B				
III	9	0 (0.0)	50	0.0
II	15	0 (0.0)	88	0.0
I	187	0 (0.0)	416	0.0
Paper mill C				
III	4	0 (0.0)	23	0.0
II	36	1 (2.8)	230	4.4
I	40	0 (0.0)	76	0.0
Survey Group				
III	329	23 (7.0)	2,500	9.2
II	295	11 (3.7)	2,798	3.9
I	511	1 (0.2)	1,421	0.7
Total	1,135	35 (3.1)	6,719	5.2
Total Control Subjects	717	0 (0.0)	5,832	0.0

Table 3.—Job Descriptions of Employees*

Exposure Level	Job Title	No. of Employees	Effusion, No. (%)
III	Pipecoverers, asbestos	191	14 (7.3)
	Asbestos mixer	110	7 (6.4)
	Asbestos sweepers	28	2 (7.1)
II	Machine operators	114	6 (5.3)
	Shipfitters	41	1 (2.4)
	Maintenance	31	1 (3.2)
	Others	109	3† (2.8)
I	Machine operators‡	206	0 (0.0)
	Pipecoverers, fiberglass	71	0 (0.0)
	Office	59	1 (1.7)
	Others	175	0 (0.0)

*N=1,135.
†One each: welder, lead bonder, asbestos supply room worker.
‡Did not work with asbestos.

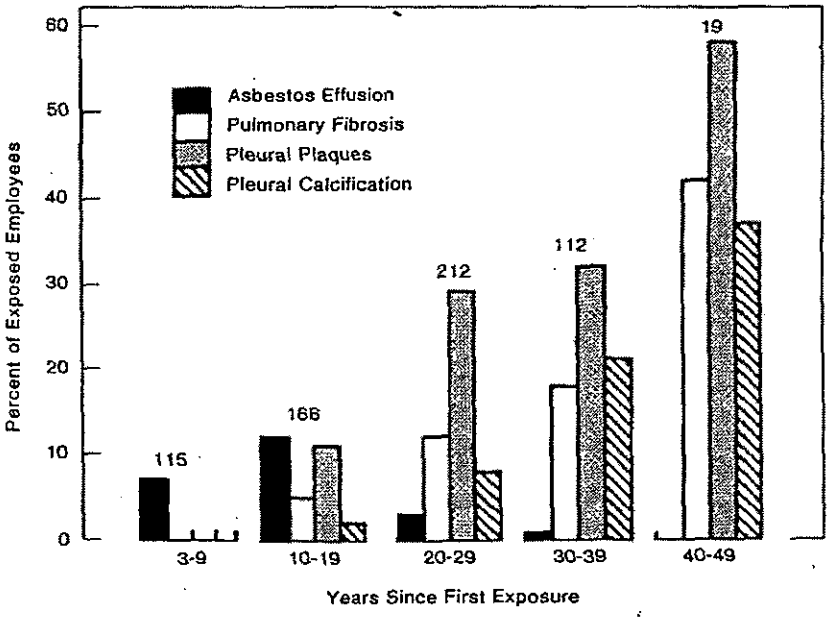


Fig 3.—Asbestos-related manifestations among employees exposed to asbestos at level II or III. Grouping is according to years since first exposure. Number of employees in each group indicated above columns. Benign asbestos effusion was observed earlier than other manifestations: it was the only asbestos-related disorder observed during first ten years after exposure and was most common during first 20 years.

It was the only manifestation seen within ten years, and it was the most common abnormality during the first 20 years (Fig 3). The possibility that latency is dose related could not be proved by these data. Although the latent period was shorter (13.3 years) for the 23 workers exposed at level III than for the 11 employees exposed at level II (15.2 years), this difference was not statistically significant.

Incidence

Serial chest roentgenograms were available for up to 45 years. Therefore, the number of new asbestos effusions per 1,000 person-years of observation could be calculated (Table 2). This analysis indicated an annual occurrence of nine asbestos effusions per 1,000 employees exposed at level III, four for those at level II, and fewer than one per 1,000 for those at level I.

Clinical Findings

The mean age at the time of the first benign asbestos effusion was 46.1 years, and two workers were only 28 years old. Eight also had chronic bronchitis, one had asthma, and four had had childhood pneumonia. Concurrent medical problems included hypertension in three, coronary artery disease in three, and diabetes in one. In none was there any evidence of tuberculosis at the time or subsequently. Two thirds reported no symptoms during their effusion, even when told of their condition (Fig 1) (Table 4). In contrast, more than one half (53.8%) of the 26 clinic patients with asbestos effusions had pleuritic pain.

Roentgenographic Features

Most asbestos effusions were small (Fig 1), and a few presented bilaterally (Table 4). Plaques were seen in one fifth, calcifications in only one, and moderate to severe asbestosis in less than 10%. All of these three manifestations were uncommon because they are usually late complications, while asbestos effusion often occurs relatively early (Fig 3). Follow-up films showed blunted costophrenic angles in virtually all cases, and residual diffuse pleural thickening was seen in one half (Table 4).

Follow-up and Prognosis

The mean follow-up after initial asbestos effusion was 9.7 years, with a range of three to 27 years. Recurrent benign effusions developed in ten persons (28.6%), sometimes on the same side, more often on the opposite side (Fig 1). Physical findings consisted of bilateral crackles in one third, and there were pleural friction rubs in two. The FVC was reduced in one half, and Dsb was reduced in two thirds. Few had evidence of airflow obstruction (Table 5).

In the survey group three persons have died, two from asbestosis, and one from a mesothelioma that developed six years after the first effusion. Mortality was greater among our 26 clinic patients with asbestos effusion. Five have died: two from infections, one from metastatic hypernephroma, and two from mesothelioma nine and 16 years after the first effusion.

COMMENT

Asbestos and other fibrous silicates are virtually unique among environmental hazards as a cause of pleural manifestations. Hyaline plaques were readily associated with asbestos clinically, because of the almost invariable history of exposure, however distant or brief, and epidemiologically, because of their exceedingly high preva-

Table 4.—Clinical and Roentgenographic Features of Benign Asbestos Effusion

Feature	No.	%
Presenting symptoms		
None	23	65.7
Pleuritic pain	6	17.1
Dyspnea	3	8.6
"Pneumonia"	2	5.7
Cold symptoms	1	2.9
Hemoptysis	0	0.0
Cigarette use		
Smokers	18	51.4
Ex-smokers	14	40.0
Nonsmokers	3	8.6
Roentgenographic		
Initial effusion		
Large, >500 mL	4	11.4
Bilateral	3	8.6
Signs of asbestos exposure		
Plaques	7	20.0
Calcifications	1	2.9
Parenchymal "fibrosis"		
Slight (s,t,u 1/0-1/2)*	15	42.9
Moderate to severe (s,t,u 2/1-3/4)*	3	8.6
Residual pleural findings		
Blunted angle	32	91.4
Diffuse thickening	19	54.3

*ILO U/C classification.[15]

Table 5.—Follow-up Data Three or More Years After Asbestos Effusions

Feature	No.	%
Physical findings		
Clubbed fingers	4	11.4
Localized dullness	4	11.4
Bilateral fine crackles	12	34.3
Pleural friction rub	2	5.7
Pulmonary function*		
FVC<80% predicted	17	48.6
FVC, mean±SD	74.8±17.9	...
$FEV_1/FVC\%$ <70	13	37.1
$FEV_1/FVC\%$, mean±SD	72.0±13.7	...
Dsb <80% predicted	20	57.1
Dsb, mean±SD	69.2±20.7	...
Recurrent effusions, entire observation period	10	28.6

*FVC indicates forced vital capacity; FEV_1, forced expired volume in 1 s; Dsb, breath diffusing capacity.

lence among exposed workers. Mesothelioma, another pleural disease, was firmly related to asbestos by a single clinical study when this rare tumor appeared with great frequency in certain locations.[17] The significance of pleural effusion with respect to asbestos exposure was more difficult to evaluate, because effusion, unlike plaques and mesotheliomas, is a common complication of a large number of disorders.

The first suspicion, in 1962, of a relationship between asbestos exposure and effusion was based on histological findings: an insulation worker with bilateral recurrent effusions was found to have pulmonary fibrosis with asbestos bodies as well as hyaline plaques.[2] Over the ensuing nine years, additional observations of "exudative pleuritis" were described in ten exposed persons.[3-5] About that time, we saw an otherwise healthy patient with recurrent bilateral bloody effusions who eventually required decortication. Again, asbestos bodies and fibers were found in the lung and this time also in the pleura. This prompted a review of clinical and histological material of 11 other patients with asbestos exposure and effusion.[1] Since then, 27 additional cases of asbestos effusion have been reported from the United States,[6] South Africa,[7] Australia,[8] Hungary,[12] and France.[9-11,13] In most of the total 37 reported cases[2-13] and in our 12 previously reported,[1] there was physical evidence of asbestos exposure from biopsy specimens that showed pulmonary fibrosis,[1,2,6,7,11] asbestos bodies in the lung,[1-3,6,7,9,11] or from asbestos bodies in the sputum.[4,9,10] Asbestos bodies in the pleura were uncommon,[1,9,11,12] sometimes detected by x-ray diffraction or electron microscopy,[9,11] and they were not seen in the pleural fluid.

These case reports are uninformative concerning prevalence of asbestos effusion. Also, it has been pointed out that conclusions concerning the relationship of any two conditions must be made with caution, and that the more common they are, the greater the likelihood of error.[18] Clearly, both pleural effusion and asbestos exposure are extremely common. An analogy may be drawn to the long-suspected relationship between effusion and rheumatoid arthritis—another frequent condition. Here the answer came from comparison of rheumatoid patients with a control group with degenerative arthritis: pleural effusion was ten times more prevalent in the former.[19] This suggested our comparison of the survey group with a control group drawn from the general population. It showed that, over a similar observation period, effusions of any cause were five times more common in the exposed group and that there were 35 pleural effusions not related to other disease among the exposed but none in the control subjects (Table 1). Clearly, among these 35 effusions there may have been some that were related to other perplexing causes rather than to asbestos. However, the absence of such cases among the control subjects suggests that such problems must be relatively rare.

The prevalence of 3.1% asbestos effusions undoubtedly represents an underestimate. Sometimes the first available roentgenograms were obtained many years after initial exposure, and then an initial finding of a blunted costophrenic angle or of diffuse pleural thickening was not accepted as evidence of asbestos effusion. Also, lateral films were sometimes missing, and oblique films were rarely available. Therefore, residua of localized effusions might have been missed. Finally, some persons might have had an effusion that disappeared without residua during the interval between films. This raised the possibility of an underestimate of the prevalence of effusions in the control group because there were fewer films, usually every third year, while in the survey group films were obtained annually at least during recent years. However, most effusions, that is, 91.4% (Table 4), left a residual blunted angle so that such a finding is an important marker for past effusion. Among our 1,135 employees in the survey group, there were 92 who had blunted angles, 54 of which were recognized as evidence of effusions (Table 1), whereas in the control group there were eight persons with blunted angles, seven of which were counted as evidence of effusions (Table 1). Therefore, if the smaller number of films in the control group caused us to miss silent effusions without roentgenographic residua, there certainly were not many such cases.

Two distinct types of pleural reactions are seen in the asbestos exposed: plaques and diffuse pleural thickening. A relationship between diffuse thickening and asbestos effusion has not been suggested previously. However, it has been recognized that diffuse thickening differs from plaques in most respects. The costophrenic angles are commonly involved, there is pleural symphysis, fibrosis is common, and there is usually loss of function.[20] Indeed, by 1970, diffuse thickness was so well recog-

nized that the International Classification for the Pneumoconioses allowed for a distinction from plaques.[15,21] Our longitudinal observations suggest that the sudden appearance of diffuse thickening frequently was caused by an effusion. Among our 35 workers with asbestos effusion, 54% had residua in the form of diffuse thickening (Fig 1). Furthermore, among the 1,135 employees, there were 44 with diffuse pleural thickening greater than 5 mm, and of these, almost one half had had a previous asbestos effusion. By contrast, there were 127 with typical plaques, but the development of the plaques never seemed to be related temporally to the effusion.

Mesothelioma, the other well-recognized asbestos-related pleural complication, generally is thought of as a rapidly growing and quickly fatal tumor. Therefore, we excluded from our count of benign asbestos effusions all persons who had a follow-up of less than three years. However, there has been a report of malignant mesothelioma of 17 years' duration,[22] and others have indicated that effusions sometimes occurred several years before the histological confirmation of a mesothelioma.[3,11] In our "survey group," one person was recognized to have a mesothelioma six years after his initial "benign" effusion, and in our clinic group, two had such a tumor nine and 16 years after initial effusion. There are several possible explanations: inasmuch as both a benign effusion and mesothelioma are relatively common in the asbestos exposed, it may be that the two disorders occurred in the same patient in nonrelated fashion. It is also possible that the pleural drift of asbestos fibers caused mechanical irritation resulting first in effusion and eventually in mesothelioma. Finally, it is possible that the earlier effusion was the first manifestation of the tumor. Mesothelioma grows along interstitial planes and is detected roentgenographically only late, and, therefore, its "doubling time" cannot be determined. It well may be that in some patients this lesion initially grows rather slowly. The spontaneous cessation of pleural exudation, the disappearance of most roentgenographic residua, and the usually long-term stable course thereafter suggest that most of our "benign asbestos effusions" were indeed benign. However, our follow-up was not long enough to determine the incidence of mesothelioma after asbestos effusion.

Historically, idiopathic pleural effusion was generally attributed to tuberculosis, and, among younger persons, two thirds eventually showed development of active disease.[23] Even now some textbooks and reviews on pulmonary disease deal at length with the concept of idiopathic effusion presumably due to tuberculosis and make no mention of benign asbestos effusion. Our study suggests that in the general population, effusions without immediately apparent cause have become extremely rare, while in the asbestos exposed, they are relatively frequent (Table 1). In our ambulatory consultation practice, asbestos exposure has become the most common cause of pleural effusion.[1] Mesothelioma presents the principal differential diagnostic problem to benign asbestos effusion. Our experiences and those of others suggest that benign effusion often occurs relatively soon after initial exposure, sometimes within ten years (Fig 1) and in two thirds within 20 years[1,4,7] (Fig 2 and 3), while mesothelioma usually is seen more than 20 and often 30 to 40 years later. Pleuritic pain is the most frequent symptom in both conditions though benign effusion goes entirely unnoticed by more than one half of patients (Table 4). Roentgenographic signs of other asbestos-related disease such as plaques or pulmonary fibrosis (asbestosis) may be absent both in benign asbestos effusion and mesothelioma, and the presence of plaques or asbestosis is not helpful in differentiating between the two.

It has been estimated that there are somewhere between 2 million and 6 million persons with significant asbestos exposure in the United States.[24] A prevalence of 3.1% benign effusions in the asbestos exposed should alert physicians to the importance of this disorder in the differential diagnosis of "idiopathic" effusion.

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