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Asbestos-Associated Radiographic Changes Among Household Contacts of Amosite Asbestos Workers*

In growing numbers, toxic industrial agents are being detected in drinking water, urban air, and food sources. The halogenated hydrocarbons, [1,1,1-tri-chloro-2,2-bis (4-chlorophenyl) ethane] (DDT) and polychlorinated biphenyl (PCB) have spread into the general environment to such an extent that they can now be detected in the adipose tissue of many persons in the United States. Equally ubiquitous and widely used in industry is asbestos.

Asbestos pollution, a recently noted phenomenon, is associated with the rapid expansion of asbestos materials during and after World War II.[1] An extremely versatile mineral, the pervasiveness of its use is reflected in

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the Department of Health, Education and Welfare's estimate that 1.5 to 2.5 million full- or part-time workers are currently exposed to asbestos and that 8 to 11 million workers have been exposed since the beginning of World War II.[2]

Asbestos fibers may be freed and enter the general environment while being processed or used in manufacturing, construction or demolition. Air in urban, as compared with isolated rural areas may have 10 to 1,000 times the concentration of such fibers.[3] Using electron microscope examination and tissue ashing techniques, chrysotile asbestos can be identified in the lungs of virtually all urban U.S. residents.[4]

Another specific pollution problem (which, however, can be more easily controlled or eliminated) is contamination of the home environment. Wives experience exposure to chemicals and dusts when laundering contaminated work clothes. Other household residents may also be exposed to the gradual accumulation of industrial materials shed daily from a laborer's dirty shoes and work clothes and subsequently circulated and suspended in the air. The potential health risk of such exposures is only beginning to be recognized.

Second to occupational exposure, household contact is the most frequently identified source of asbestos exposure among reported mesothelioma cases.[5] Vianna and Polan[6] reviewed 52 cases of mesothelioma among women in New York State; ten were known to have had husbands or fathers who had worked in an asbestos trade. Compared to a matched control population, an estimated relative risk factor of ten was calculated for women with this type of exposure. Despite the reports of *mesothelioma* among persons having household contact with asbestos workers, few attempts have been made to assess the prevalence of *other* asbestos-related diseases.

CHARACTERISTICS OF ASBESTOS-ASSOCIATED DISEASE

Asbestosis (parenchymal fibrosis, pleural fibrosis, pleural calcification) and malignancy (lung cancer, pleural and peritoneal mesothelioma, gastrointestinal cancer) are well recognized hazards of occupational exposure to asbestos. Epidemiologic investigations of these illnesses in heavily exposed occupational groups have defined the important relationships that characterize asbestos-associated disease. Intensity and length of exposure as well as amount of time elapsed from time of exposure were already apparent in Merewether and Price's 1930 report on asbestos.[7] They reported that individuals working in the less dusty areas of asbestos textile mills were less likely to develop parenchymal fibrosis than those

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working in more dusty areas (ten percent versus 34 percent). Sixty-one percent of individuals with more than 15 years' employment had fibrosis as compared with 20 percent among those employed less than 15 years. None of the workers employed for less than five years in either area showed evidence of fibrosis. Subsequent reports confirmed and refined these observations for both clinical asbestosis and, later, mortality from asbestos-associated malignancies.[8]

Even after excessive dust exposure, sufficient time must be allowed for this chronic disease to become manifest. From the same perspective, adverse effects of brief or low-level exposures are even less likely to become apparent rapidly. Insufficient elapsed time from first exposure, when evaluating exposures, will lead to underestimates of risk. Because duration of employment and elapsed time from onset parallel each other and provide a composite effect, independent effects are often difficult to appreciate. Seidman et al.[9] analyzed lung cancer deaths among individuals who had worked in an amosite asbestos factory for varying periods between 1941 and 1954, and had had no subsequent or previous asbestos exposure. They found that a group with only one month of exposure began to show excessive lung cancer deaths after 30 years had elapsed from first exposure. For those with more than one year employment, latency was shortened to 15 years. As the duration and/or intensity of exposure lessens, the period of latency of a particular effect increases.

RADIOGRAPHIC APPEARANCE OF ASBESTOS DISEASE

The ILO U/C International Classification of Radiographs of 1971[10] coupled with the set of standard x-ray films that define the abnormality categories in the Classification, now provide a common language and definition of radiographic abnormalities that should help reduce inter-reader variability and provide more reliable comparisons among epidemiological studies.

Radiodiagnostic characteristics (parenchymal opacities, pleural thickening, pleural calcification, pleural effusion) have played an important role in the recognition of asbestos diseases since the 1930s. Interstitial fibrosis initially appears as an increase of fine reticular markings especially in the lower lung fields. Such opacities, which appear predominately as "irregular opacities" but can also appear as "rounded opacities" in the ILO Classification are not entirely unique to asbestosis. However, given a documented asbestos exposure and the absence of other conditions such as rheumatoid lung, sarcoidosis, scleroderma, radiation fibrosis, Hamman-Rich Syndrome and chronic aspiration pneumonia, which share a similar radiographic appearance but differ in other

clinical and past history parameters, such abnormalities can almost certainly be attributed to the asbestos exposure. If asbestos exposure is found concomitant with one of these other diseases, differential diagnosis may be difficult. Fig. 14-1 is an example of asbestosis. Often a minimal increase in opacities goes unnoticed unless standard normal films are used for comparison.

Pleural abnormalities as seen on the chest x-ray are more unique to asbestos exposure than are the increased reticular markings. Other possible causes of lateral chest wall pleural thickening or discrete pleural plaques on the diaphragm or chest wall, such as post-traumatic pneumothorax or hemothorax, post-thoracotomy changes from rib rubs, or empyema residua, are usually apparent from past medical history and physical examination. A more difficult differential diagnosis is that of early pleural mesothelioma. Rapid enlargement of the pleural thickening is indicative of malignant pleural mesothelioma. Similarly, metastatic malignant disease to the pleura presents a more dynamic course. Fig. 14-2 demonstrates bilateral pleural thickening in the typical scalloped pattern. Fig. 14-3 demonstrates pleural thickening and a diaphragmatic plaque.

Pleural calcification, such as that seen in Fig. 14-4, is often indicative of past asbestos exposure. As with pleural thickening, other explanations for its presence—old tuberculosis infection or trauma, for example—are readily apparent in the past medical history. Less commonly reported

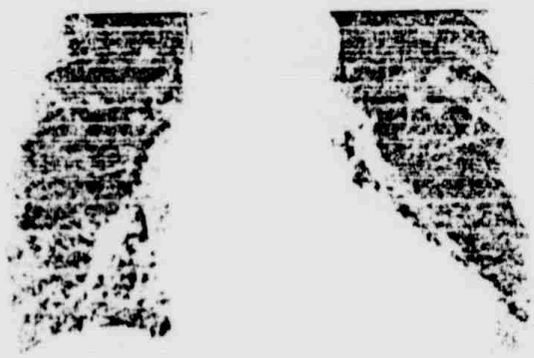


Fig. 14-1A. Sixty-seven year old wife of an amosite asbestos factory worker employed for two years (1942–1944). Never smoked cigarettes. ILO U/C Classification: irregular opacities s 1/1; bilateral chest wall pleural thickening, width b, extent 2; pleural calcification on right diaphragm and both chest walls. Grade 2.

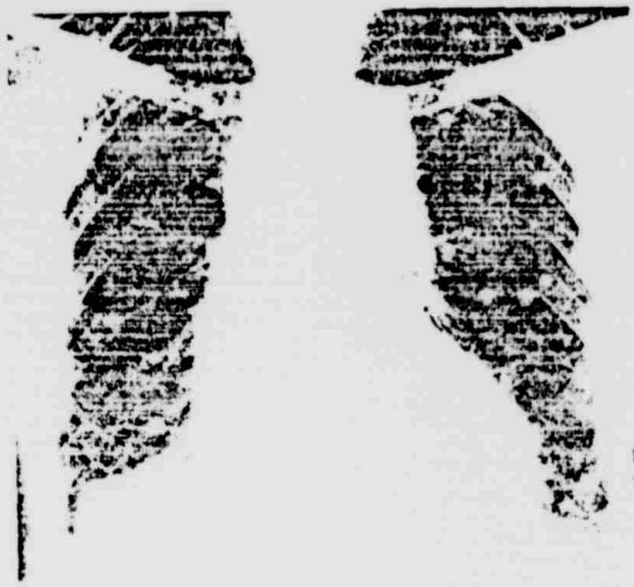


Fig. 14-1B. Sixty-seven year old wife of an amosite asbestos factory worker employed for 2½ years from 1943–1945. Never smoked cigarettes. ILO U/C Classification: irregular opacities s 2/2.

associations with calcification, such as mineral oil aspiration, must also be considered.

In combination, these abnormalities—parenchymal opacities (irregular or rounded) and pleural thickening or calcification—make the radiographic diagnosis of asbestosis virtually certain.

OCCUPATIONAL DISEASES AMONG NON-OCCUPATIONALLY EXPOSED INDIVIDUALS

The occasional appearance of overt disease among residents of neighborhoods in close proximity to factories and individuals having household contacts with workers directly involved in handling toxic materials has been recognized for more than 40 years. Polychlorinated biphenyls, beryllium, lead and asbestos have been responsible for diseases seen among individuals whose only known contact with the offending agent has been indirectly—via a member of or visitor to the household who had handled the material in an industrial setting.[1,2,3,4,5] The most

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Fig. 14-2A. Forty-six year old son of an amosite asbestos worker employed for eight years from 1942 to 1950. Former cigarette smoker of 1½ packs per day. Employed as an engineer. ILO U.C Classification: irregular opacities s 1.0; bilateral chest wall pleural thickening, width C, extent 2, calcification on diaphragms Grade 1.

frequently described sources of home contamination are the chemicals and dust that adhere to shoes, hair and work clothes brought home for laundering. Some industries now provide change rooms and coveralls but many do not. Even when available, change rooms and showers frequently go unused because workers may not understand the potential long-term hazards and continue to inadvertently expose their unsuspecting families to the materials used and manufactured in their factories.

Case reports of overt disease among family contacts of workers have been frequently considered medical curiosities. The full extent of such risks is not known, for it is uncommon to inquire into the occupation and exposures of a patient's household contacts when investigating the symptoms of a disease. In all likelihood, most family exposure disease, especially with mild or sub-clinical manifestations which may only be contributory to a current health problem, goes unrecognized.

Because environmental exposures to asbestos are generally of low

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Fig. 14-2B. Thirty-one year old son of an amosite asbestos factory worker employed for six years (1948–1954). Two pack per day cigarette smoker. Employed in printing ink manufacturing plant. ILO U/C Classification: bilateral chest wall pleural thickening, width b, extent 2.



Fig. 14-3. Forty-six year old son of an amosite asbestos factory worker employed for six months in 1942–1943 (brother of individual in Figure 4). Never smoked cigarettes. Tire salesman for 32 years. ILO U/C Classification: irregular opacities p 1/1; bilateral pleural thickening extent 2; pleural plaques on both diaphragms.

intensity, effects would be expected to appear only after 20, 30 or more years. Unfortunately, because of this unusually long period of clinical latency, many of the consequences of today's exposures will not be detectable or quantifiable until well into the next century. On the other hand, health effects of environmental asbestos exposures that occurred in the 1940s would be expected to be seen now.

With the exception of pleural calcification, which was frequently reported as an incidental observation because of its striking appearance and novelty, few general surveys have provided data that can establish a general background prevalence of the x-ray abnormalities associated with asbestos exposure. Table 14-1 summarizes several large studies. In over 900,000 x-rays reviewed, pleural calcification was found in an average of



Fig. 14-4. Sixty-two year old wife of an amosite asbestos worker employed for two years, 1941–1943. One-half pack per day cigarette smoker. ILO U/C Classification: irregular opacities p 2/2; bilateral chest wall pleural thickening extent 2; bilateral chest wall and diaphragm calcifications Grade 3.

two of 1,000 x-rays. Pleural thickening and parenchymal fibrosis as described today, although accepted as uncommon, were not commonly reported; a broad base for statistical comparison is not, then, readily available.

Pleural calcification is noticeably more prevalent in surveys where non-occupational or environmental exposures to asbestos have been recognized. Table 14-2 summarizes several such environmentally exposed populations. The studies of Zolov and Bourilkov and Yazicioglu et al.[17,18] considered exposure from asbestos indigenous to the soil and rock outcroppings of the geographical areas examined. Kiviluoto[16] and Navratil[19] studied populations living in neighborhoods and districts with active asbestos mines and manufacturing facilities. Table 14-3 lists the spectrum seen in occupational groups. Comparing the three tables it is evident that when the prevalence of pleural calcification increases, asbestos exposure can be identified.

Radiographic abnormalities associated with asbestos exposure offer an opportunity to identify and evaluate past asbestos exposure in terms of one set of biologic effects, i.e., radiographic change. Unfortunately,

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Table 14-1
Prevalence of Radiographic Abnormalities Among Survey Populations Without Easily Recognizable Asbestos Exposures

Author	Country	Total Examined	Pleural calcification	Parenchymal fibrosis
Floyd & Hepburn (1959)	U.S.A.	115,000	28 (0.02%)	Not reported
Neef (1963)	East Germany	111,969	36 (0.03%)	Not reported
Matsova (1964)	Czechoslovakia	44,549	241 (0.54%)	None
Hourihane (1966)	Great Britain	27,500	36 (0.13%)	7 (0.03%)
Raunio (1966)	Finland	631,000	1,516 (0.24%)	1

* Includes some with environmental exposure

Table 14-2
Prevalence of Radiographic Abnormalities Among Survey Populations with Environmental and Neighborhood Asbestos Exposure

Author	Country	Total Examined	Pleural calcification	Parenchymal fibrosis
Kiviluoto (1968)	Finland	6,312	499 (7.9%)	Some
Zolov (1967)	Bulgaria	3,429	175 (5.1%)	7 (0.2%)
Yazicioglu (1976)	Turkey	5,989	288 (5%)	Not reported

Table 14-3
Prevalence of Radiographic Abnormalities Among Occupationally Exposed Workers in the United States

Author	Type of exposure	Number examined	Pleural calcification	Parenchymal fibrosis
Siegel (1943)	Talc workers	221	14 (6.3%)	31 (14%)
Selikoff (1965)	Insulation workers with >30 years exposure	315	137 (43%)	283 (90%)
	Insulation workers with <30 years exposure	802	13 (2%)	259 (32%)
Anderson (1978)	Insulation workers with >30 years exposure	113	37 (32%)	79 (70%)
	Insulation workers with <30 years exposure	539	24 (4%)	149 (20%)

radiological techniques are not sufficiently sensitive to detect asbestos-associated disease before a clinical latency period of 20, 30 or more years. Other means of determining earlier physiologic responses are needed.

RADIOGRAPHIC ABNORMALITIES AMONG HOUSEHOLD CONTACTS OF ASBESTOS FACTORY WORKERS

Navratil[9] studied factory workers, residents of a factory neighborhood, household contacts of the workers, and an unexposed control population in Czechoslovakia. Table 14-4 summarizes the results of that survey. Household contacts had a greater prevalence of pleural calcification than did the control. The population studied was small and only four

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Table 14-4
Prevalence of Pleural Calcifications in Populations with Different Sources of Exposure to Asbestos in Czechoslovakia

General non-exposed population	8,133	28 (0.34%)
Blood relatives of factory workers	114	4 (3.5%)
Neighborhood residents of asbestos factory	155	9 (5.8%)
Factory workers	755	42 (5.3%)

[a] Navratil et al. 1972

abnormal individuals were reported. Similar exposures occurred in the United States. Since the 1930s the number has grown sufficiently to warrant investigation of this potential public health issue.

For more than 20 years workers employed for varying periods of time from 1941 to 1954 in a factory producing amosite asbestos products have been under clinical observation by one of us (IJS.) The group first employed (1941–1945) is known to have suffered excessive deaths due to lung, gastrointestinal, and mesothelioma malignancies, as well as asbestosis.[5] Table 14-5 summarizes the mortality experience of that group. In

Table 14-5
Expected and Observed Deaths Among 933 Amosite Asbestos Factory Workers Employed 1941–1945, Observed to December 31, 1977

	Deaths 1941–1977		
	Expected[a]	*Observed*	*Ratio*
Total Deaths	368.62	594	1.61
Cancer all sites	73.35	195	2.66
Lung cancer	19.16	100	5.22
Pleural mesothelioma	a	8	—
Peritoneal mesothelioma	a	8	—
Gastrointestinal cancer	21.55	32	1.48
All other cancer	32.64	47	1.44
Asbestosis	a	30	—
Other non-infectious respiratory disease	8.47	19	2.24
All other causes	286.80	350	1.22

[a] Expected deaths based upon age-specific death rate data for New Jersey white males in corresponding years.

[b] Death rates not available, but these have been rare causes of death in the general population.

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the course of periodic clinical evaluations of these workers, four pleural mesotheliomas were reported among their household contacts.[1]

In 1973, we began a clinical survey of the household contacts of these workers who could be identified as being resident in the workers' households during the period of their active asbestos employment. All identified individuals were invited to participate in a special clinical study. We have so far examined 776 of these household contacts. The examination included comprehensive occupational and residential histories, past and current medical histories, histories of current symptoms, smoking and respiratory questionnaires, physical examination, 14 in. × 17 in. postero-anterior and right and left oblique chest x-rays, and spirometry. Of the 776 examined, 685 had not had personal occupational exposure to asbestos and were household members at the time the factory workers were actively employed in the factory. The remaining 91 individuals were placed for analytical purposes in a separate category as outlined in Table 14-6.

For comparison purposes in the chest x-ray evaluation, a control population of urban New Jersey residents, living in the same community as the study population, was identified in the general medical clinic at the facility used to examine the workers and household members. Controls were matched for age and sex. Clinical records were reviewed, and none of the controls had recorded occupational or household contact with asbestos.

The chest x-rays of household contacts, factory workers and controls were interspersed and read without knowledge of exposure category. The ILO U/C Pneumoconioses Classification of 1971 was utilized by a panel of five experienced readers.

Table 14-6
Individuals Examined But Who Did Not Meet Study Criteria

Exclusion criteria		Number examined
Personal occupational exposure to asbestos		51
Entered household after active worker asbestos employment ceased		40
Total		91
* Direct asbestos trade	19	
Brake repair work	8	
Dry wall taper	1	
Shipyard trade	6	
Job includes work with asbestos	16	
Silica exposure	1	
	51	

Table 14-7 summarizes the overall results by exposure category. A film was classified as abnormal if one or more of the following abnormalities was recorded: irregular opacities of 1/0 or more; any lateral wall pleural thickening; any pleural calcification; pleural plaques on the diaphragm. It is interesting to note that of the children born shortly after the workers ceased active employment in the plant, but while the family still lived in the same house, six had abnormal x-rays. This would suggest that sufficient asbestos continued to be present in the home atmosphere to cause an increase of x-ray abnormality 30 years later.

As expected, individuals with personal occupational exposure to asbestos in addition to household exposure had the highest prevalence of abnormalities.

Table 14-8 summarizes the types of individual abnormalities seen among the 685 household contacts. Pleural abnormalities were predominant. No instances of pleural calcification were seen among the controls, while 8.5 percent of the study group had calcification present. In all abnormality categories, the household contacts had a statistically significantly higher rate of abnormality.

Given sufficient elapsed time from first exposure, it is apparent that for environmental and other low-level asbestos exposures, radiographic appearance of disease is predominantly pleural. In general, the presence of pleural change is of considerable significance and, when present, possible asbestos exposure should be investigated. The presence of the more "classical" appearance of asbestosis, with apparent parenchymal fibro-

Table 14-7
Prevalence of Radiographic Abnormalities Associated with Asbestos Exposure Among Household Members of Amosite Asbestos Workers

Exposure group	Total examined	One or more radiographic abnormalities present*	
New Jersey urban residents**	326	15 (5%)	$\chi^2 = 7.1$ $p < .01$
Entered household after active worker employment ceased†	40	6 (15%)	
Household resident during active worker employment†	685	240 (35%)	$\chi^2 = 6.8$ $p < .01$
Household resident and personal occupational asbestos exposure	51	23 (45%)	

* ILO U/C Pneumoconiosis Classification categories: irregular opacities 1/0 or greater; pleural thickening; pleural calcification; pleural plaques.
** No known direct occupational or household exposure to asbestos.
† No known direct occupational exposure to asbestos.

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Table 14-8
A Matched Comparison Group: Chest X-ray Abnormalities Among 685 Household Contacts of Amosite Asbestos Workers and 326 Individual Residents in Urban New Jersey

Group	Total examined	Pleural thickening present	Pleural calcification present	Pleural plaques present	Irregular opacities present
Household contacts of asbestos workers	685	146 (18.8%)	66 (8.8%)	61 (7.9%)	134 (16.6%)
Urban New Jersey residents	326	41 (12.2%)	0 (0.8%)	2 (0.6%)	4 (1.4%)

* ILO U/C Pneumoconioses Classification irregular opacities 1/0 or greater

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Table 14-9
Chest X-ray Abnormalities Among 685 Household Contacts of Amosite Asbestos Workers

Total examined	Number with only pleural abnormalities	Number with only irregular opacities	Number with both pleural and parenchymal abnormalities
685	126 (18%)	61 (9%)	53 (7.7%)

sis, is the exception rather than the rule after environmental exposures. In Table 14-9, the predominance of pleural abnormalities is further demonstrated. Pleural change was the only abnormality present in 18 percent of the cases. In nine percent, only irregular opacities were seen and in 7.7 percent both abnormalities were present. In over half of those with asbestos associated abnormalities, pleural change was the only evidence of exposure.

The actual extent and intensity of each household resident's asbestos exposure is unavailable. To help approximate a relative exposure dose index for each household contact, utilization was made of the employment history of the factory worker in whose household the contact resided. The resident factory worker for each household is referred to as the "index worker."

The importance of sufficient elapsed time from onset is demonstrated in Table 14-10. The length of index worker employment for both groups was roughly the same. Thirty-eight percent of the group with exposure beginning prior to 1945 had abnormalities, compared to 27 percent for those whose first exposure was after 1945.

Table 14-10
Prevalence of Chest X-ray Abnormalities in Household Contacts by Year of Onset of Exposure of Index Workers

Year of first exposure	Total examined	Number with abnormalities
Before 1945	403	152 (38%)
1945 and after	244	67 (27%)

Prevalence of x-ray abnormalities significantly greater among those exposed before 1945 compared to those first exposed after 1945.

$$\chi^2 = 7.1 \quad p < .01$$

* Thirty-four individuals with more than ten years of exposure are excluded from this table. The distribution of duration of exposure of individuals in each of the groups was then identical.

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Table 14-11
Prevalence of X-ray Abnormalities in Household Contacts by Duration of Employment of Index Amosite Asbestos Workers

Duration of employment	Number examined	Pleural abnormality present	Irregular opacities present	One or more abnormalities present
Less than 1 year	314	46 (15%)	46 (15%)	81 (26%)
1–5 years	288	90 (31%)	46 (16%)	110 (38%)
5–10 years	46	26 (57%)	14 (30%)	29 (63%)
10+ years	35	16 (46%)	8 (23%)	19 (54%)

Prevalence of abnormal chest x-rays is significantly greater among those with index workers with longer duration of employment.

$$\chi^2 = 34.6 \quad df = 3 \quad p < .001$$

Similarly, the importance of duration of exposure, in this case the length of the index workers' employment, is clearly seen in Table 14-11. Abnormalities were significantly more prevalent among those associated with index workers with a longer duration of employment. The most striking increment of abnormality prevalence is seen in pleural changes, which were three times as prevalent among those with ten years of exposure than among those with less than one year. These tables demonstrate the time dynamics for the appearance of radiographic abnormalities after this type of environmental exposure. Serial observations of this cohort will provide additional information concerning the interaction of length of exposure and lapsed time from onset of exposure. Tables 14-12, 13, and 14 give the distribution of parenchymal and pleural abnormalities recorded in ILO U/C Classification categories. In most cases, the abnormalities seen were in the lesser categories. Only two individuals had advanced parenchymal fibrosis of a category 2/2. Nineteen individuals had extensive pleural thickening and 10 had extensive pleural calcifica-

Table 14-12
ILO U/C Pneumonoconioses Classification of Irregular Opacities Among 685 Household Contacts of Amosite Asbestos Workers and 326 New Jersey Urban Residents

Total examined	0/0	0/1	1/0	1/1	1/2	2/2
685	406 (59%)	165 (24%)	47 (7%)	61 (9%)	4 (0.6%)	2 (0.3%)
326	288 (88%)	27 (8%)	8 (3%)	3 (1%)	0 (0.0%)	0 (0.0%)

Table 14-13
ILO U/C Pneumoconioses Classification of Pleural Thickening Seen Among 685 Household Contacts of Amosite Asbestos Workers

Number examined	Extent 1			Extent 2		
	Width					
	a	b	c	a	b	c
685	61 (9%)	40 (6%)	8 (1%)	4 (0.6%)	11 (1.6%)	4 (0.6%)
Total	109 (16%)			19 (3%)		

* Extent 1 = Total length of pleural thickening does not exceed one-half of projection of one lateral chest wall.
Extent 2 = Total length of pleural thickening exceeds one-half of the projection of one lateral chest wall.
Width a = Up to 5 mm thick at widest part of any shadow.
b = 5 mm to 10 mm thick at widest part of any shadow.
c = over 10 mm thick at widest part of any shadow.

tion. Figures 14-1–4 are examples of radiographs demonstrating the types of abnormalities seen.

Dyspnea is the most commonly reported symptom among occupationally exposed asbestos workers. Only 4.7 percent of the family cohort complained of dyspnea. However, those with abnormal x-rays had a significantly higher prevalence of this symptom than those with normal x-rays (7 percent versus 3 percent). Dyspnea was the only symptom statistically associated with abnormal chest x-rays.

Table 14-15 summarizes the clinical abnormalities found and their distribution in relation to x-ray findings. (Chest x-rays were interpreted after the physical examination had been completed.) Although in-

Table 14-14
ILO U/C Classification of Pleural Calcification* Seen Among 685 Household Contacts of Amosite Asbestos Workers

Total examined	Grade 1	Grade 2	Grade 3
685	27 (4%)	17 (3%)	10 (2%)

* Grade 1 = one or more areas of pleural calcification, sum of greatest diameters does not exceed about 2 cm.
Grade 2 = one or more areas of pleural calcification, sum of greatest diameters about 2–10 cm.
Grade 3 = one or more areas of pleural calcification, sum of greatest diameters exceeds 10 cm.

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Table 14-15
Distribution of Symptoms and Physical Findings Among 685 Household Contacts of Amosite Asbestos Workers by Chest X-ray Findings

X-ray status	Total examined	Rales[a]	Dyspnea[a]	Cyanosis[a]	Clubbing[a]
Normal	445	14 (3.1%)	15 (3.4%)	0 (0.0%)	1 (0.2%)
Abnormal	240	21 (8.8%)	17 (7.1%)	4 (1.7%)	4 (1.7%)
Total	685	35 (5.1%)	32 (4.7%)	4 (0.6%)	5 (0.7%)

A statistically higher prevalence of abnormal symptoms and physical findings was seen among individuals with abnormal chest x-rays than among those with normal chest x-rays.

[a] $\chi^2 = 10.4$ $p < .01$
[a] $\chi^2 = 4.8$ $p < .05$
[a] $\chi^2 = 7.5$ $p < .01$
[a] $\chi^2 = 4.5$ $p < .05$

frequently seen, rales, cyanosis and clubbing were statistically more prevalent among those with abnormal x-rays.

DISCUSSION

The widespread use of asbestos and subsequent release and accumulation of fibers in non-work environments did not occur on a large scale until the mid-1940s. Highly exposed occupational groups were the first to be known to have increased risk of asbestos associated illnesses (asbestosis, pleural and peritoneal mesothelioma, lung cancer). Occupational exposures in the 1940s, 1950s and 1960s have generally been described as excessive. This is certainly true for the occupational groups studied from which clinical latency periods of 15 to 30 years were established.

Appearance of disease has been shown to depend upon duration and intensity of exposure. The greater the exposure, the sooner the disease could be expected to appear and, conversely, the shorter and less intense the exposure, the longer the time before appearance of disease could be demonstrated. Asbestos exposure in the 1940s, which might be expected to have had a clinical latency period up to 40 or more years, only now and in the next ten years may be detected. Household contacts of workers would then constitute a large population whose latency periods would be expected to be longer than those of the workers themselves, due to exposures greater than those of the general population but less than those of the index workers.

Using one measure of asbestos effect, the chest x-ray, we have seen in one well-defined group of 685 household contacts of amosite factory workers that x-ray abnormalities characteristically seen after occupational asbestos exposure are common. As in occupational groups, an increased duration of exposure was associated with a higher prevalence of abnormalities. As other investigators of environmental exposure asbestos disease have also reported, pleural abnormalities predominated.

We do not yet know the full extent of the disease risk that will be experienced by the other household contacts. Prospective observation of this young cohort (75 percent under age 50) and other similar groups will determine whether an unusual malignancy risk exists in addition to the demonstrated x-ray abnormalities. Continued observation will also help assess whether the presence or absence of pleural disease is of prognostic value in assessing future health risk.

Clinicians and radiologists need to become aware of the possible significance of pleural disease unaccompanied by evidence of parenchymal fibrosis. The presence of such abnormalities should be recorded and reported to the patient. Possible history of exposure to asbestos should be sought. Among household contacts we examined, only a few were aware that they had previously had an abnormal x-ray, although on review abnormalities were often present in past films, when these were available. None had been queried regarding prior exposure to asbestos.

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