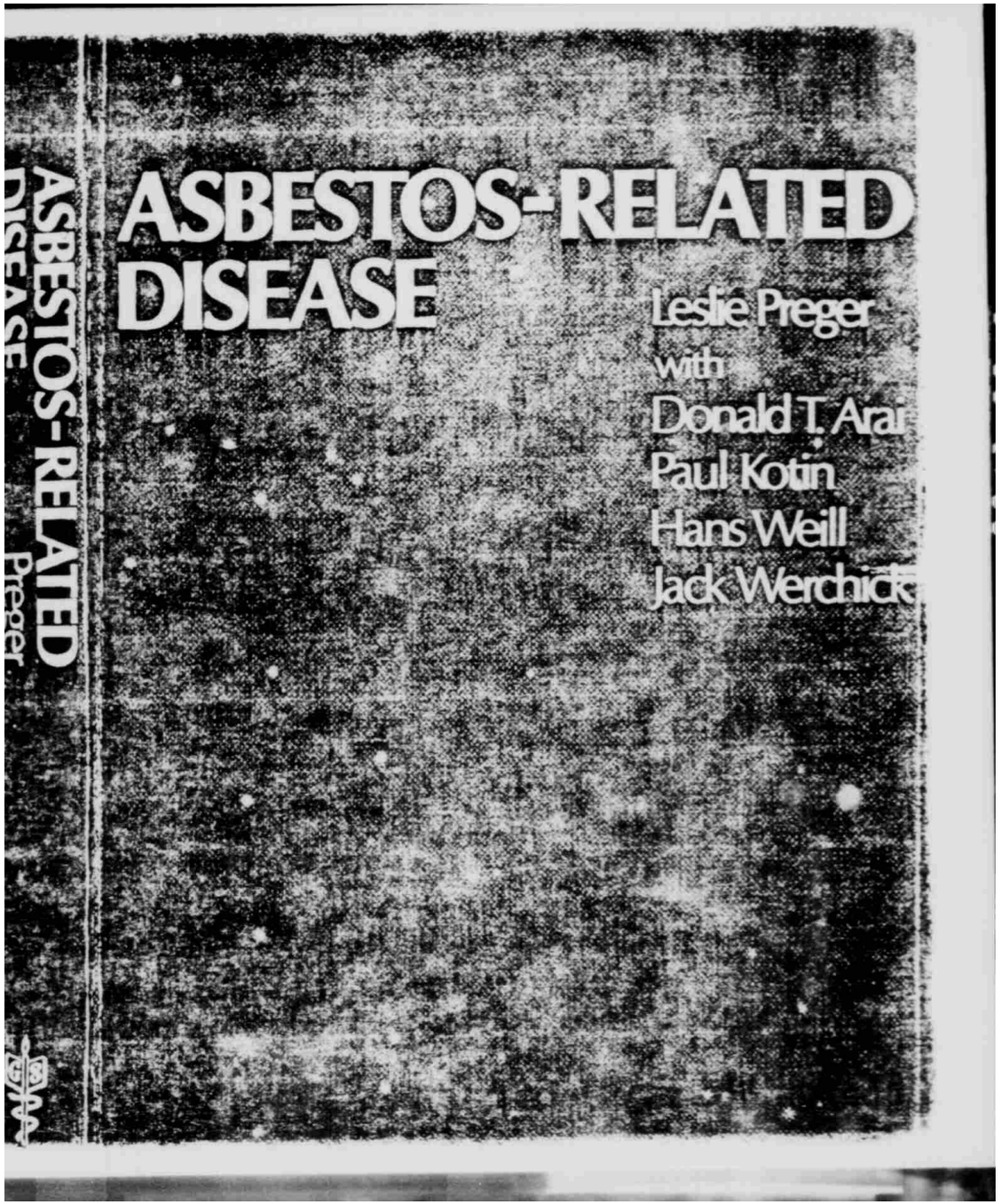

ASBESTOS-RELATED DISEASE
Leslie Preger
with
Donald T. Arai
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Hans Weill
Jack Werchick
ASBESTOS-RELATED DISEASE
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2

ORGAN INVOLVEMENT IN ASBESTOS-RELATED DISEASE: SECTION 1 PLEURAL PLAQUES

Leslie Preger

Plaques of the parietal pleura are associated with exposure to all types of asbestos fibers. The plaques are probably not harmful in themselves but are an objective sign of prior exposure. Their number usually increases with dose and elapsed time after exposure and so are more common in older patients; but there is no definite relation with total dust exposure.[1] A certain minimal exposure will ensure development of radiographically identifiable plaques, usually after an interval of more than 20 years; in some patients, plaques may appear after a shorter interval, this usually but not invariably implies a heavier exposure.

LOCATION

The parietal pleural plaques are multiple and lie against rigid areas such as the inner surface of the lower ribs, costovertebral angles, and central tendinous portion of the diaphragm. They may, if large, extend into the intercostal space and even penetrate the diaphragm by direct extension into the abdomen (see

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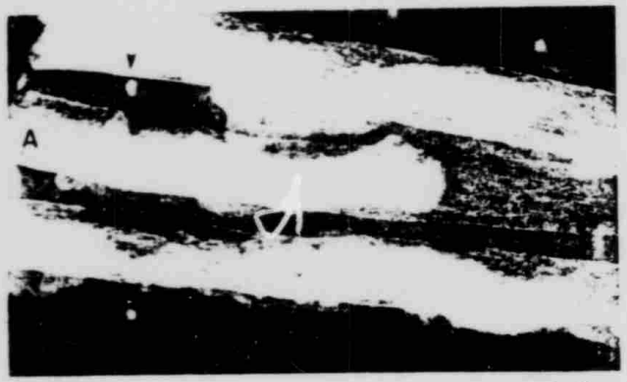


Fig. 2-1. *Autopsy specimen of rib. X-rayed in two planes (A and B). The plaques originate against the inner surface of the rib and extend with growth onto intercostal muscle. (B) Calcification is present in parts of pleura lying against inner surface of rib and in the intercostal spaces. The ease with which plaques may be stripped from ribs, despite their consistency akin to that of cartilage, is seen in B.*

Fig. 2-1). Sometimes they are seen posterior to the upper sternum, arising in the mediastinal pleura reflected over the aorta. The mediastinal pleura along the cardiac border is another less common site. In the case of the parietal pleura adjacent the aorta and left ventricle, both these segments may be construed as being against rigid areas, analogous to the locations near ribs and the tendinous portion of the diaphragm. Involvement of diaphragmatic and mediastinal pleura is usually a late event and not seen in absence of peripheral plaques. The plaques do not develop adjacent to the costal cartilages or against the ribs in the costophrenic angles or apical areas. Distribution is bilateral; occasionally the left side is slightly more involved, and the posterolateral aspects are nearly always the most involved.

GROSS APPEARANCE

The plaques are raised and are gray or yellowish-white. The surface may be flat or uneven, with a mammillated appearance. The edges may be abrupt, overhanging, or gently sloping. Their consistency is very hard, quite surprising to a radiologist accustomed to seeing them as "soft tissue" shadows on an x-ray. The thickness varies up to 1 cm. Their size and shape is variable, from tiny spots to many square centimeters, covering up four or five ribs and their



intercostal spaces. Their shapes may be punctate, oval, or irregular. When involvement has extended over an intercostal space, the earlier formed part lying against a rib is usually thicker than that over the intercostal space. They strip easily from the chest wall, as seen in Figure 2-1*B*.

MICROSCOPIC APPEARANCE

The plaques are extramesothelial structures in that they develop in connective tissue external to the mesothelial cells which usually remain as a surface covering for the plaque.[2] They are not associated with adhesions, but occasionally fibrinlike material may overlie the mesothelial cells.[1] The substance of the plaque consists of wavy interweaving bands of collagen fibers that lie parallel to the plaque surface. In early lesions, in their deeper growing part, there are some vessels and plump, nucleated fibroblasts and occasionally a slight infiltrate of lymphocytes and plasma cells. Older lesions are avascular, the fibrocytic nuclei are few, and the inflammatory cells are absent. The covering mesothelial cells may be difficult to identify. Since growth occurs from the deeper part of the plaque, it is there that the lesion is much more vascular and has nucleated fibroblasts and a cellular infiltrate.

Calcification

Calcified pleural plaques contain more asbestos fibers than noncalcified plaques, and the fibers are more numerous in the calcified zone than in the fibrous zone of these plaques[3] (see Fig. 2-2). Whether this is a causal factor is



Fig. 2-2. *X-ray of excised portion of diaphragm to show calcified and noncalcified plaques of various shapes.*

not known. The incidence of calcification varies from area to area and may be caused by any type of asbestos fiber. Kiviluoto[4] found about a 40 percent incidence in persons working in or living near Finnish anthophyllite mines (see Fig. 2-3). Rom[5] found no pleural plaques, calcified or noncalcified, in a review of three independent radiological surveys of miners and their families in a Rhodesian asbestos-mining town. The older plaques may show dystrophic calcification in the more avascular central parts, and this is more common in older patients. Calcium deposits occur as fine granules that may coalesce into various shapes[6] (see Fig. 2-4).

The calcified zone is in the central or deeper zone of the plaques, with a thick layer of noncalcified fibrohyaline tissue lying between it and the mesothelium. The calcium crystals, mainly acicular apatite crystals but with some whitlockite nodules, are aligned along the length of the collagen fibers. Chemical analysis shows that the phosphorous calcium ratio in the calcified zones differs from natural calcium phosphates of the apatite or whitlockite type. Microscopic calcifications, however, which can be detected in the fibrous zone and which appear uncalcified on gross examination, have similar calcium phosphorous ratios to naturally occurring apatite and whitlockite.

The amount of calcification is not related to the thickness of a plaque.[8] Diaphragmatic plaques in particular, but also those in the subaxillae, may



Fig. 2-3. *A. Environmental asbestos plaques. A woman living in the neighborhood of Finnish anthophyllite mines who has peripheral pleural plaques and diaphragmatic calcified plaques. There was no occupational exposure. (Courtesy of Prof. R. Kiviluoto.) B. Environmental exposure. Calcified geographical plaques and diaphragmatic calcification in a patient living near Finnish anthophyllite mines but who was not occupationally exposed. (Courtesy of Prof. R. Kiviluoto.)*

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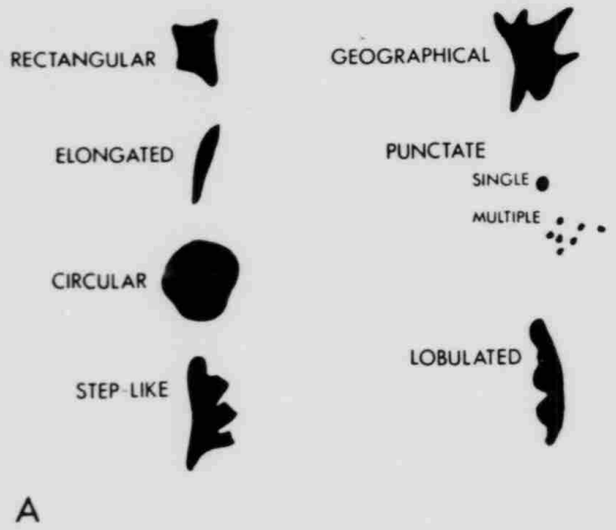
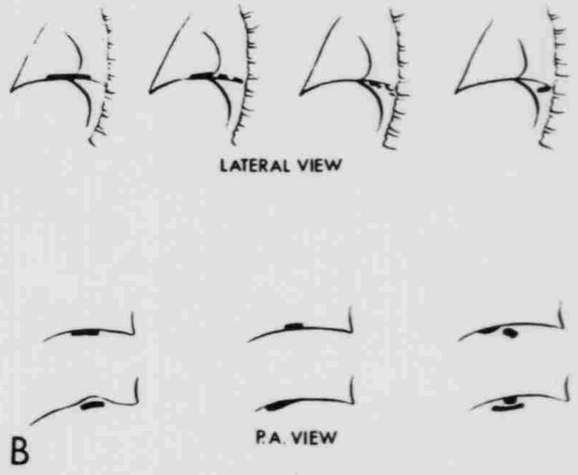


Fig. 2-4. (A and B) Diagrammatic representation of plaque shapes (see facing page).

attain large size and remain uncalcified (see Fig. 2-5). Calcification in diaphragmatic plaques is usually only linear in shape.

PRESENCE OF ASBESTOS PARTICLES IN PLEURAL PLAQUES

Earlier reports[8] did not reveal ferruginous bodies (coated asbestos fibers) in plaques. Later reports using ashed tissues[3,9] and electron diffraction techniques have demonstrated chrysotile fibers of very small size, length 0.1–2.0 μm and diameter 25–30 nm. They are only apparent when calcium deposits and collagen fibers have been removed by chemical techniques, the fibers being present 5 to 12 times more frequently in the calcified portion than in the fibrotic portion. Thus, asbestos may be difficult to detect in noncalcified fibrohyaline plaques, and such plaques, although commonly associated with asbestos exposure, are not specific to such exposure in that occasional plaques may be found at autopsies in which sophisticated techniques have not demonstrated



an asbestos fiber content and there is no known occupational environmental or leisure exposure.

PATHOGENESIS

The pathogenesis is unknown. Thomson[2] suggests that in an active patient with healthy lungs, inhaled asbestos fibers may move through lung parenchyma, breach the visceral pleura, and embed in the parietal pleura (see Fig. 2-6). The passage of asbestos fibers will be impeded if the fibers become coated with an iron–protein complex to become a ferruginous body (asbestos body), since these are wider and have blunter ends than uncoated asbestos fibers. Penetration of the pleura will be easier if adhesions are absent. Plaques are more common alongside the lower ribs and diaphragm because lung mechanics assure better ventilation of lower zones. Where upper zone plaques are present, a greater exposure or working or sleeping in the prone position may be a factor. When the fiber is held up against a rigid part (rib, tendinous portion of diaphragm, vertebra, aorta, left ventricle) plaque formation starts (see Fig. 2-7). The mesothelial cells do not contribute to development of the lesion but merely cover it, since development begins from the deeper part of the pleura; i.e., plaques are essentially extramesothelial. The fibrotic response is out of propor-

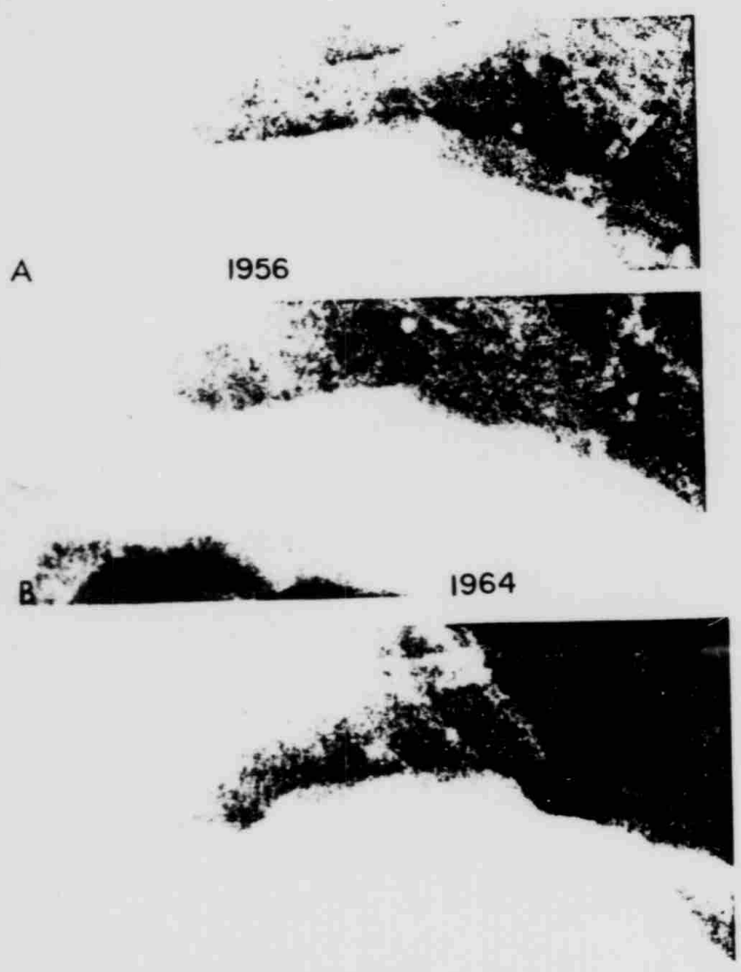


Fig. 2-5. Development of diaphragmatic plaque. PA view of left hemidiaphragm on [illegible] and 1972. In the earliest view, no abnormality is detected. A small plateau-shaped plaque is seen in 1964 and has become deeper in 1972. There was exposure to asbestos during work in World War II shipyards.

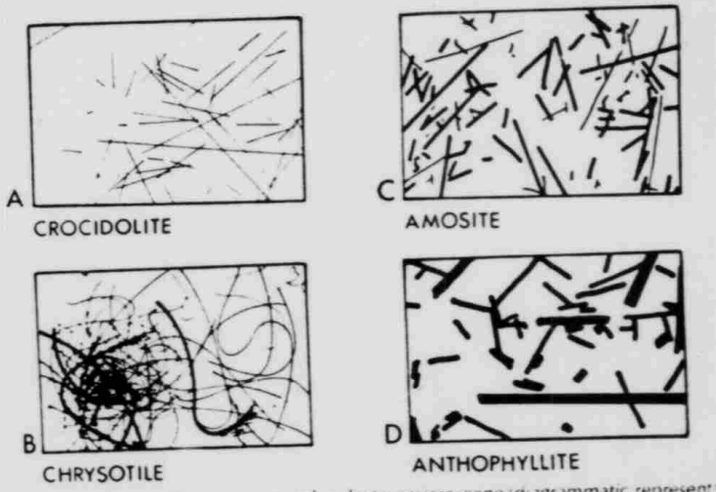


Fig. 2-6. Asbestos fibers as seen under electronmicroscope (diagrammatic representations). Fiber shape plays a part in lung penetrability. The amphiboles crocidolite, amosite, anthophyllite are rectilinear and penetrate lung more efficiently than do the curly chrysotile fibers. Chrysotile fibers tend to fragment in the lung after a period of storage, and the short fragments produced may then behave more like the amphiboles. Amphibole fiber diameter is in increasing order—crocidolite, amosite, anthophyllite; the wider diameter fibers are more likely to be deposited in the proximal airways.

tion to the few asbestos fibers found in the plaques, and this, together with the presence of lymphocytes and plasma cells in the early lesions, suggests a pleural sensitivity reaction for which there is some evidence (see below).

Other earlier hypotheses have stressed mechanical irritation or induction of minute hemorrhages by fibers and lymphatic rather than airway transport to the parietal pleura (see Fig. 2-8). Kiviluoto[4] has suggested that fibers project from the visceral pleura into parietal pleura to initiate an inflammatory response that subsequently organizes and fibroses. The absence of adhesions between the two pleural layers and the absence of surface inflammatory cells of parietal pleura (when present, they are deeply situated) make this explanation questionable. Meurman[8] thought that the sharp asbestos fibers might initiate subpleural hemorrhages that organize into plaques. The absence of adhesions again is not in keeping with this hypothesis. Instead of airway transport, earlier reports considered intracellular transport of asbestos particles within the lymphatics to the parietal pleura.[7,10] The hypothesis is based on the fact that plaques develop in areas of lymphatic drainage and on the possibility



Fig. 2-7. *Retrosternal plaques. Calcified and noncalcified plaques are seen behind the sternum (A and B). The calcified plaques are in the mediastinal parietal pleural reflection over the aorta. This is a rigid area. In A a circular mass between arrowheads simulates a lung mass. Involvement of mediastinal pleura is a late event and is not seen in absence of peripheral subaxillary plaques.*



Fig. 2-8A. *Pathogenesis of plaques. Plaques are thought to originate when asbestos fibers have penetrated lung and pleura and are held up against rigid parts. Lateral view of chest shows plaques lying against costovertebral areas.*

of lymph stasis: the diaphragmatic muscle propels the particles toward the inactive central tendinous portion, while the intercostal muscles may move particles toward the ribs. To reach these sites, particles would have to reach hila of lungs before the mediastinal pleura. Since hilar and mediastinal lymph nodes are unaffected in asbestosis, and since the mediastinal parietal pleura is less involved than the subaxillary pleura, this theory is also questionable. The lower parietal pleurae, both costal and mediastinal, can absorb particulate matter, however; the visceral pleura cannot.[11] This fact may explain the site of plaques. There is experimental evidence that, in mice,[12] asbestos fibers injected subcutaneously migrate to submesothelial tissues in the chest and abdomen and that in humans, asbestos fibers are found in many parts of the body, e.g. brain, kidney, and liver. In view of these various hypotheses, the pathogenesis is still considered sub judice.



Fig. 2-8B. *Parietal pleural plaques are seen better when they extend medially and lie against air-filled lungs. The large lateral subaxillary plaques merge with the chest wall shadow. Most of the calcifications appear at the periphery of plaques and some traverse plaques (horizontal arrow).*

IMMUNOLOGY OF PATIENTS WITH ASBESTOS PLAQUES

A sensitivity reaction is suggested by the disparity between the size of the plaque and the few asbestos fibers found in them.[2] The incidence of circulating rheumatoid factor, antinuclear antibodies, and the level of gamma globulin is greater in asbestos workers with plaques than in those without plaques and in the nonexposed population.[13,14] The relation of immunologic factors in asbestos-related pulmonary fibrosis is described in Chapter 1.



Fig. 2-8C. *Large parietal pleural plaques. Calcification is absent in these plaques, which extend medially in a lobular manner. Differentiation from a mesothelioma or metastatic pleural cancer would be difficult.*

EPIDEMIOLOGY

The prevalence of plaques has been assessed by necropsy examinations and x-ray surveys; the latter will show only a small portion of plaques noted at necropsy, the proportion varying with the sophistication of the x-ray techniques used. Exposure may be occupational, environmental, or leisure-related or may result from household (conjugal) contact. The incidence of plaques increases with age, i.e., with lapsed time since a minimal first exposure.[15] The

pleural plaque incidence is considered dose-related by some, but this correlation is more apparent with mild to moderate pulmonary fibrosis. A 20-year interval between first exposure and radiographic recognition of plaques is common.[16a,16b] Persons below the age of 30 years who have plaques have usually had an environmental exposure, e.g., living in an asbestos-mining area or near a shipyard. The effect of age on incidence of calcified pleural plaques is described by Rom and Palmer.[5] A period of 20 to 29 years after first exposure gave an incidence of 10 percent; 30 to 39 years, 35 percent; and over 40 years, 58 percent. Pulmonary parenchymal ferruginous bodies are present in 86 percent of patients with bilateral plaques;[8] but autopsy reports on patients dying in large cities quote similar prevalence of ferruginous bodies in the general population.

All types of asbestos fibers may produce pleural plaques. Anthophyllite mined in Finland, has been associated with a particularly high rate of 40 percent, but many of these were patients living near the mine and therefore subject to an environmental exposure at an early age (see Fig. 2-9).

Pleural plaques seen in radiographic surveys have been classified into three groups by Jones and Sheers[1]: (1) occupational groups, (2) environmental groups, and (3) nonexposed groups.

1. *Occupational exposure*

General asbestos workers in central Europe	5 percent incidence of plaques
New York asbestos insulators	38 percent incidence of plaques
United Kingdom shipyard workers	5 to 14 percent incidence of plaques
Quebec chrysotile mines and mills	0.5 to 9 percent incidence of plaques
Cypriot chrysotile mines and mills	6 percent incidence of plaques

The Quebec data varied from 0.5 to 9 percent incidence according to age, and the incidence at one mine was 13 times greater than that at an adjacent mine working chrysotile from the same geologic formation. The reason is unknown.[7] Pleural calcification in those exposed to talc and mica has been attributed to associated asbestos fibers.

2. *Environmental factors.* Pleural plaques may occur in those living near anthophyllite, crocidolite, amosite, and chrysotile mines although they are not occupationally exposed. Similarly, those living near factories using



Fig. 2-9. (A) Environmental exposure (town capitanal to Finnish anthophyllite). There are calcified diaphragmatic plaques, geographical peripheral plaques, and mediastinal pleural plaques. (B) An enlarged view of A showing the mediastinal calcific deposit better. Differentiation from visceral and parietal calcific deposits is possible. (Courtesy of Professor K. Kivisaari.)

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Fig. 2-10. *Conjugal asbestos plaques. A woman with plaques that are better seen in the LAO view (B) than in the PA (A).*

asbestos may develop plaques. In the past, asbestos workers might bring home fibers in their hair, clothes, and footwear. Families of these workers occasionally develop household contact (conjugal) asbestosis[17] (see Fig. 2-10). For centuries, villages in southeast Turkey around Cermik have used chrysotile asbestos for painting walls and floors. The nature of this material was not realized until recently. The villagers have a high incidence, up to 14 percent, of plaques, and the condition was known in Turkey as Cermik disease; only recently has it been realized that the raw material dug up by these villagers was chrysotile ore with some talc included.[18]

3. *Nonexposed groups.* In virtually every patient with large calcified bilateral plaques typical of asbestos exposure, an appropriate history of exposure is obtainable. In some urban patients with small noncalcified plaques, a definite history of exposure is not obtainable. Are these plaques due to unusual susceptibility to the low levels of exposure in ordinary urban life or to some unelicited or forgotten exposure? A dentist the author has followed for some years may be in the latter group. His chest film showed small bilateral subaxillary plaques, and a history of asbestos exposure was not apparent until some years later, when he remembered that for some 35 years he had used asbestos string to line casting rings used in dental plate construction.[19]

CLINICAL MANIFESTATIONS

Hyaline or calcified pleural plaques may be present alone or in combination with visceral pleural fibrosis. They are most frequently found as an isolated finding and indicate asbestos exposure with all its significance to the clinician for the possible development of pulmonary fibrosis, intrathoracic (pleural and pulmonary malignancy) and extrathoracic (larynx, peritoneal, esophagus, colon, and possibly pancreas, kidney and breast) neoplasia, and splenic and hepatic capsular thickening. In the absence of other findings, the plaques do not produce symptoms. Lung function tests are not usually impaired if the worker does not smoke,[20,21] but some do show minor reductions in lung volumes.[22] Thus, although they themselves do not produce symptoms, the presence of plaques may be ominous; Fletcher,[23] in a survey of 408 shipyard workers with pleural plaques but with no pulmonary fibrosis, noted 3 workers with mesothelioma and 16 with bronchogenic carcinoma. Smoking history was not recorded. Edge[24] found a 2.5-fold increase in bronchial carcinoma in shipbuilders with plaques but no radiographic evidence of pulmonary fibrosis, as compared with controls from neighboring areas whose x-rays were strictly normal but who may have had some degree of asbestos exposure. The pleural plaques are not premalignant; however, Lewinsohn[25] reported a tubulopapillary mesothelioma with some undifferentiated cells arising by a narrow stalk from mesothelia cells covering a hyaline parietal pleural plaque. This case appears to be exceptional, if not unique.

When parietal pleural plaques are associated with diffuse visceral thickening (which may have resulted from a prior asbestos exudative effusion) and when there is lower lobe parenchymal fibrosis, the prognosis is different. About 20 percent of those with plaques have radiographically diagnosable pulmonary fibrosis. This condition has been termed "complicated hyalinosis," as opposed to simple hyalinosis, in which parietal pleural plaques are not accompanied by diffuse visceral pleural changes. Navratil studied a group of 30 present or former asbestos workers,[26] and his data are shown in Table 2-1. The difference between data for simple and complicated hyalinosis is statistically significant.

Table 2-1

	Simple Hyalinosis	Complicated Hyalinosis
Length of exposure	21.0 years	22.4 years
Elapsed time since onset of exposure	27.6 years	33.7 years
Mortality data	12%	40%

RADIOGRAPHIC APPEARANCE

Three problems confront the clinical radiologist:

1. Choice of optimal radiographic technique to demonstrate plaques;
2. The identification and differentiation of pleural plaques from other similar pleural shadows;
3. Pleural plaques may be associated with, and obscure, more serious asbestos-related thoracic disease.

Choice of Optimal Radiographic Techniques to Demonstrate Plaques

Film Size

Since pleural plaques have a peripheral location on a standard PA chest film, a 14 in. × 14 in. (355 mm × 355 mm) film, as used in the United Kingdom, or 14 in. × 17 in. (355 mm × 432 mm), as used in the United States, may not include the periphery of a large thorax. Although inconsequent, if films are inspected at the time of development prior to the patient leaving the department, the use of standard films in surveys or in rolls where film is examined when the patient is not available for exposure of a better centered or additional film may be a major disadvantage. Audsley[27] has therefore suggested a film size of 400 mm × 400 mm so that less than 2 percent of the population screened would not be fully covered by a single film. There is no published data on the efficacy of plaque detection by 100-mm films and photofluoroscopy compared with conventional size film.

Applied Kilovoltage

For many years, radiologists have used a high-kilovolt technique (110–140 kV) in preference to the older standard kilovolt technique (60–80 kV) for routine radiography (see Fig. 2-11). High-kilovolt techniques have a practical advantage in that there is a large latitude in exposure, so that variations in milliampere-seconds (mAs), which control film blackening or density, may be made over a relatively large range and an acceptable film still be obtained. This safety margin is at the expense of production of the film with less subject contrast (long-scale contrast), so that there is a long scale of shades of gray between the lightest and darkest parts of the film image. This is an advantage in separating adjacent and overlapping images, since the more radiographically dense image does not fully obscure the less dense. Noncalcified hyaline plaques adjacent to the ribs are therefore seen more easily with the high-kilovolt technique.

Conversely, low-kilovolt techniques produce an image with high subject contrast because there is large variation in the intensity of the transmitted x-ray beam through the various organs of the chest. This is based on the physical fact that low-kilovolt x-rays show more photoelectric type absorption in substances with high atomic numbers such as bone ($Z = 13.8$) as compared with muscle



Fig. 2-11. *High- and low-kilovolt techniques. In A (120 kV) hyaline plaques are seen along the lateral chest wall, but the calcified plaque projected over the right fourth rib anteriorly is poorly visible (arrow); it is seen better in B (80 kV). The minimal degenerative changes in the right lower lobe are seen better in A (120 kV). In C (120 kV), the calcified plaque overlying the left sixth rib anteriorly (see next page) is seen less well [illegible]*

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(Z = 7.4). Calcified pleural plaque will therefore be the more visible, since its Z number will approximate that of bone, whereas a noncalcified plaque will be less apparent, since its Z number will be similar to that of adjacent intercostal muscle. Many of the epidemiologic surveys have been made with mobile units carrying x-ray generators capable of generating a low kilovolt only, and although images so produced may show calcified plaques clearly, noncalcified plaques may be inapparent.

The information regarding the choice of technique may be tabulated as shown in Table 2-2 (altered from Bohlig and Gilson[28]).

General Radiographic Principles Affecting Image Quality

These principles are well known to all practicing radiologists and are succinctly described in modern texts on radiological physics.[29] Briefly, the important controlling factors are contrast and image quality. Contrast, as seen

Table 2-2

Lesion	High Kilovolt	Low Kilovolt
Pulmonary fibrosis	+	–
Hyaline plaque	+	–
Calcified plaque	–	–
Pleural effusion	+	–
Visceral pleural thickening	+	–
Mesothelioma and lung cancer	+	–

+ signifies preferred technique. Thus, because absorption of x-rays by calcium decreases with a rise in kilovolts, small areas of calcification may be inapparent on a high-kilovolt film, and two exposures, at high and low kilovolts, may need to be made for optimal detection of both types of plaques

on a film, depends on the contrast of the subject, film contrast (an inherent photographic property of the x-ray film used which may amplify subject contrast), fog, and scattered radiation. Image quality, the ability for point-for-point reproduction of parts of the subject x-rayed on the film, is governed by the degree of radiographic mottle, the ability of the apparatus (x-ray machine, grid, film, and screens) to define a sharp edge, and the resolving power, or the ability to record separate images of small objects near together.

Ultrasound Techniques

The use of ultrasound to identify pleural effusions, to select a suitable aspiration site,[30] and to monitor removal of fluid by use of special needle-transducer assemblies is now routine. Less utilized is ultrasonic detection of pleural plaques. Using A and B modes, plaques thicker than 2–3 mm are detectable whether calcified or not. Ease of detection increases with thickness and presence of calcification. Plaques thinner than 2 mm usually contain little or no calcium. The plaques appear as echo reflectant linear structures 2–5 cm from the skin surface. It is not possible to differentiate asbestos-related plaques from pleural thickening due to other causes. Plaques may be detected by ultrasound examination when not apparent on conventional radiographic examination. Viikeri[31] suggested that ultrasound be used for epidemiologic surveys, but despite the advantage of utilizing a nonionizing form of radiation, the technique is time-consuming.

Xerography

Xeroradiographic images have enhanced edges, and this technique permits greater latitude in the choice of exposure factors than does conventional filming. Edge enhancement should permit easier identification of plaques, but in practice, the increased radiation dose to the patient is a controlling factor.

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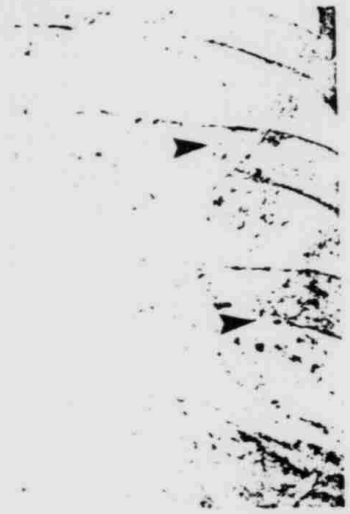


Fig. 2-12. *Xerogram of an insulating worker showing noncalcified pleural plaques.*

and conventional film oblique views are preferable. The plaques outlined in Figure 2-12 were easily apparent on conventional films; the xerograms were part of an examination to assess a malignancy in the opposite lung.*

Computed Tomography Techniques

Parietal and visceral pleural thickening, diffuse or plaquelike, calcified or not, causes an increase in soft tissue attenuation of x-ray beam and so is demonstrable by computed tomography (CT) techniques[32] (see Figs. 2-13 and 2-14). Diffuse and localized areas of thickening may be seen when inapparent in routine chest films and, if seen conventionally, may appear larger with CT. The inner (nearer lung) margins of plaques visualized by CT are denser than the



Fig. 2-13. *CT demonstration of pleural plaques. (A) Calcified paravertebral (horizontal arrows) and peripheral plaques (oblique arrows). (B) Calcified mediastinal (horizontal arrows), paravertebral, and peripheral plaques. (C) Calcified diaphragmatic (horizontal arrows) and paravertebral plaques; also seen is wedge-shaped area of fibrous tissue in right lung (oblique arrows) (see next page).*

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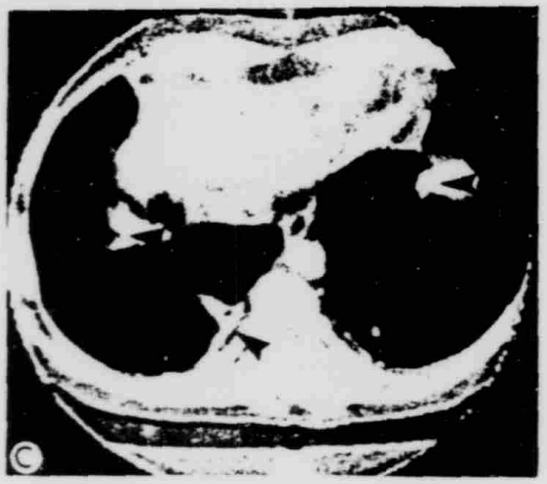


Fig. 2-13C

outer margins. Some changes are detectable only by this technique, e.g., extension of plaques along diaphragmatic crura below the level of the lungs, subpleural localized infiltrates that appear to be related to visceral pleural thickening as described by Solomon,[33] and bands, presumably fibrous, crossing the pleural cavity, especially at the cardiophrenic angle. This latter change is of extreme interest in that the absence of pleural adhesions at necropsy and thoracotomy had prompted the concept that parietal pleural plaques are noninflammatory in origin. Detection of these bands by CT should prompt a reassessment of the incidence of adhesions in asbestosis. Computed tomography is especially helpful in detection of plaques in the posterior mediastinum, adjacent to the heart, and those penetrating the diaphragm and lying adjacent to the liver and spleen (see Fig. 2-15). The latter may have been in part responsible for capsular fibrosis of liver and spleen described in asbestosis by pathologists in the past. The overall value of CT is shown by Kreel's[32] data; 11 of 13 patients, with exposures ranging from 1 to 40 years, have plaques demonstrable by CT, but only 8 of 13 were visible on chest films. Another value of CT lies in the detection of peripheral pulmonary malignancies that may be in part obscured by large plaques when using conventional techniques. Its value in the detection of pulmonary fibrosis, small nodular densities, and visceral pleural thickening is described in Chapter 2, Section 2.

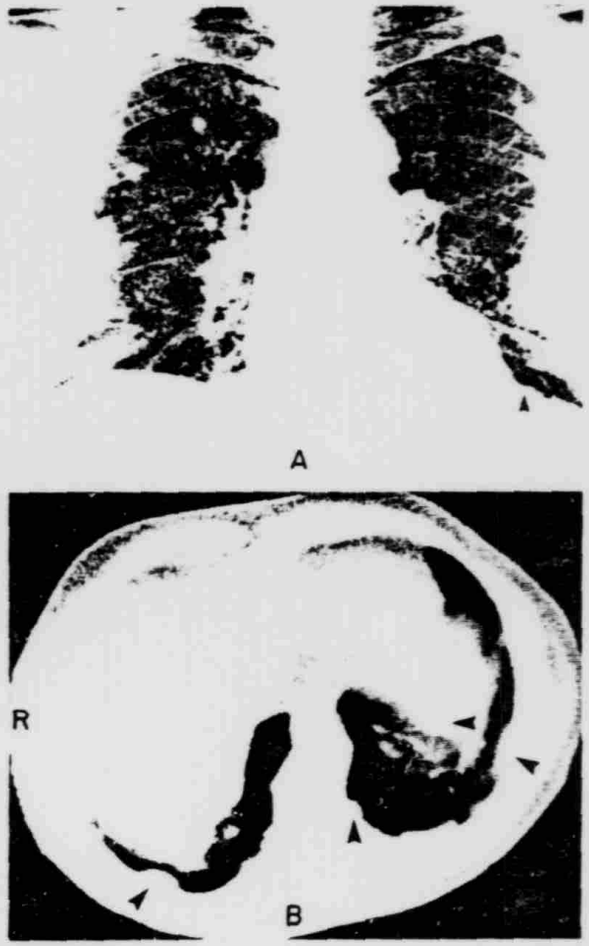


Fig. 2-14. *A: PA chest film of patient with an 18-month history of asbestos exposure while working in a shipyard in World War II. Large subaxillary plaques are seen. Small plaque on left diaphragm is seen poorly (arrow). B: CT section shows plaque on right diaphragm (horizontal arrow) as well as peripheral (oblique arrows) and paravertebral plaque (vertical arrow). C and D: CT sections show that peripheral plaques may be present anteriorly (horizontal arrows) in addition to more typical posterolateral location (vertical arrows).*

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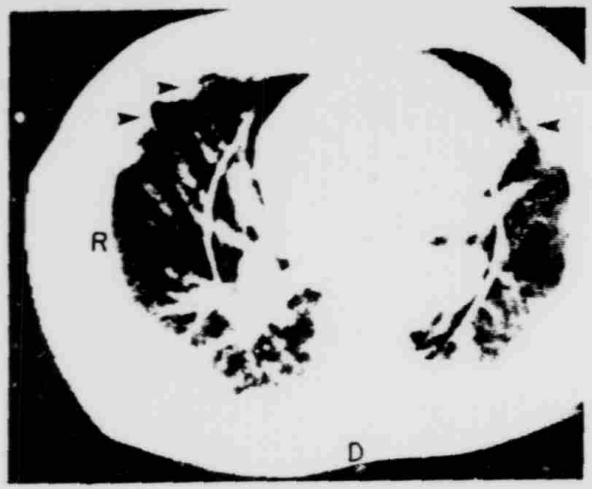


Fig. 2-14C,D



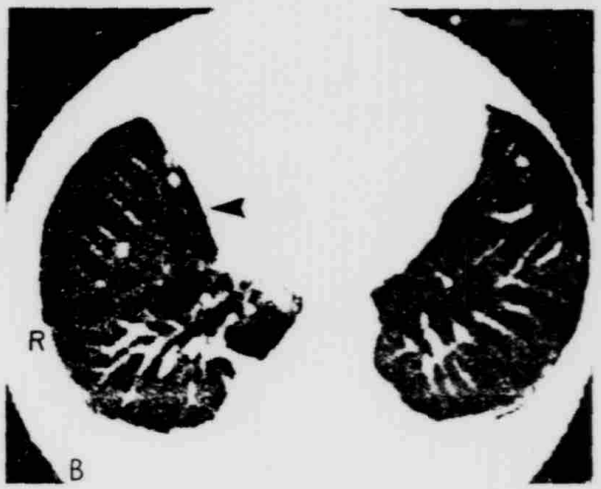


Fig. 2-15. Computed tomography. (A) Irregular border of left side of the mediastinum due to plaques (horizontal arrow); there is also fibrosis in the posterior part of the left lung (vertical arrow). In (B) there is flattening of the right side of mediastinum due to mediastinal pleural plaques.

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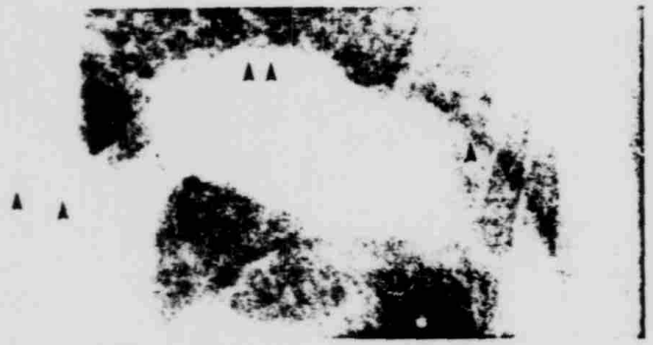


Fig. 2-16. *Diaphragmatic plaque in a lobulated diaphragm. Diaphragmatic plaques when noncalcified are best seen in full inspiration, which helps straighten out lobulated diaphragm. In this patient, the medial and lateral lobulations (arrows) do not have the plateau shape of the typical ventral diaphragmatic plaque (double arrows).*

Identification of Plaques and Their Differentiation from Similar Pleural and Chest Wall Shadows

Detection of Small Hyaline (Noncalcified) Plaques

Small, noncalcified hyaline plaques of mediastinal parietal pleura are not recognizable by conventional techniques. When adjacent to the diaphragm, parietal pleural plaques should be searched for assiduously in the middle one-third (the tendinous aponeurotic area); they are not found less than 2.5 cm from the costophrenic angles. A film exposed in full inspiration is needed, so that the raised plaque will stand out from the relatively flat, adjacent, uninvolved diaphragm (see Fig. 2-16). With poor inspiration and a domed diaphragm, the smaller plaques may be inapparent. The lobulations of diaphragmatic contour may obscure a plaque or be mistaken for one (see Fig. 2-17). Pleuro-diaphragmatic adhesions at the lower end of the major fissure may obscure adjacent plaques (see Fig. 2-18). The shape of the plaque varies from slight elevations, difficult to separate out from the diaphragmatic contour, to localized buttonlike or plateaulike masses when larger. The small diaphragmatic plaques are usually not distinguishable on lateral projections. Since they are more common posterior to the midcoronal plane of the diaphragm, oblique views are helpful.

Small lateral hyaline noncalcified pleural plaques should be searched for



Fig. 2-17. *PA view of right lung and lateral view. Muscle slips in frontal view may simulate diaphragmatic plaques; no plaques are evident in the lateral view.*

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A

Fig. 2-18. *Diaphragmatic linear calcified plaques may be obscured by adjacent nonaerated lung or fluid. Linear atelectasis in A obscures the fine diaphragmatic calcification, which becomes apparent when this area is reinflated (B) (arrow) (see facing page).*

on the lateral chest wall between the sixth and ninth ribs and on the posterolateral chest wall between the fifth and tenth ribs (see Fig. 2-19). The apices and sulci are not involved. Small noncalcified plaques are usually not discernible on the lateral view because of the overlap of the left and right posterior rib ends and vertebrae. If the lateral projection is not true but slightly oblique, small plaques may occasionally be seen, but in general this is more helpful in detecting small calcified plaques. Any overlap of soft tissue of the arm may obscure lateral plaques. This is especially important if oblique views are made, since the arm nearest the cassette may be difficult to move out of the way and still maintain good contact of the patient with the cassette holder. Oblique projections are of great value in showing a tangential view of small plaques not visible on routine PA projections; several films taken at different obliquities are sometimes of value. Fluoroscopy helps in choice of appropriate degree of obliquity.

The line diagram in Figure 2-20 shows that the radiographic image of a plaque whose long axis is parallel to the x-ray beam may be enlarged by the use of appropriate oblique projections. In doubtful cases, both oblique (left and



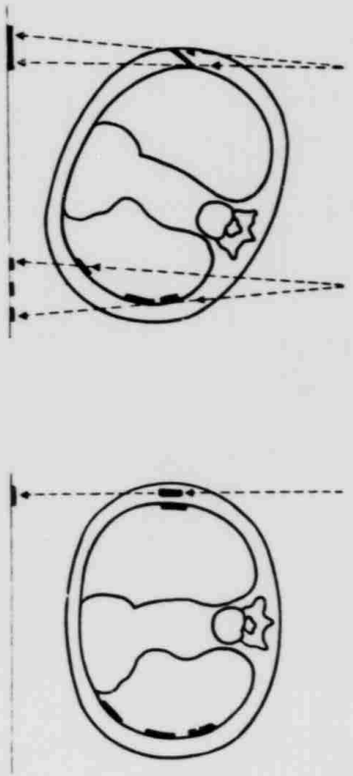
B

right) views should be used (Fig. 2-21). Views that are too steep an oblique will project the semispinalis and sacrospinalis muscles adjacent to the spine and will obscure the plaques, since they often are present adjacent to the spine.*

*The semispinalis muscle arises by a series of long muscle bundles from the backs of the tips of the first to tenth thoracic transverse processes and articular processes of third to seventh cervical vertebrae. It passes upwards to insert as three bundles. Semispinalis capitis inserts on the occipital bone between the superior and inferior nuchal lines close to the midline and immediately under the trapezius. Semispinalis cervicis inserts onto the spines of C2 to C5 vertebrae. Semispinalis thoracis inserts on the spines of C6 to C7 and T1 to T4 vertebrae. The sacrospinalis muscle arises from the sacral spines and lateral mass, the lumbar and lower thoracic spinous processes, and the posterior part of the crest of the ilium. It inserts as three columns—outer, intermediate, and medial—in a complicated manner from the mastoid process superiorly, all cervical vertebrae except C1, all thoracic and lumbar vertebrae, and all the ribs inferiorly.



Fig. 2-19. *A not-quite-true left lateral view of the chest shows large noncalcified plaques against left and right ribs adjacent to costovertebral angle. The left pleural gutter is obliterated (arrows); recurrent benign pleural effusions may follow asbestos exposure and, when resorbed, leave a blunted costovertebral angle.*



OBLIQUE PROJECTION

P.A. PROJECTION

Fig. 2-20. *Values of oblique projections. An oblique view will present more of a surface of a plaque "en face" to the incident beam. This is especially important in the detection of noncalcified plaques. (After H. Boblig.)*

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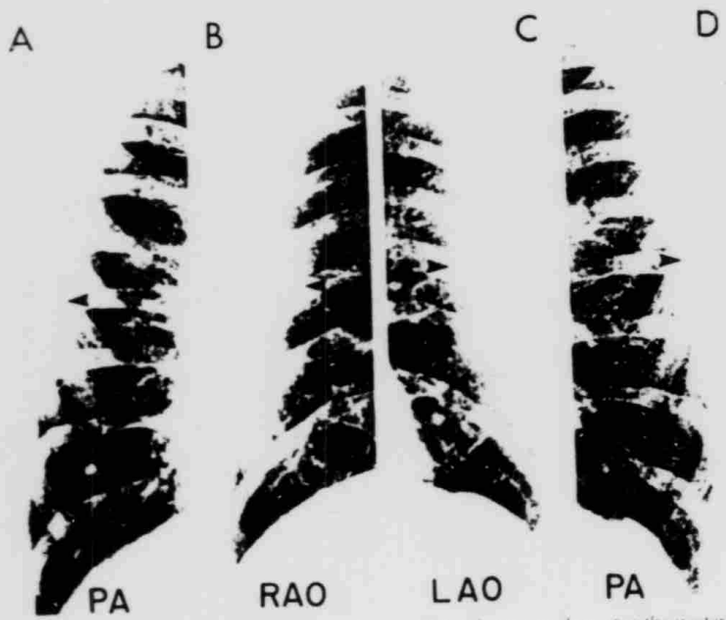


Fig. 2-21. *Value of oblique projections. Subaxillary plaques are largest in the posterolateral area between fifth and tenth ribs. Plaques are seen in A and D (PA views of right and left lung), those on left being barely visible. In the RAO (B) and LAO (C) views, the plaques become much more apparent.*

The plaques on oblique views may appear larger, and their shape will alter, usually from a flat or flangelike density to a more rounded one. Sometimes a coin lesion may be simulated; but plaques are usually less dense than a pulmonary coin lesion of similar size. Reference to Figure 2-21 will show that an oblique view may demonstrate several plaques, while a PA (or AP) view will show but one. In doubtful cases, instead of oblique PA films, oblique AP films may be helpful.

A major problem in the identification of small hyaline plaques is their differentiation from the normal companion shadows of the chest wall. In the subaxillary areas of the chest wall the shadows of interest are those due to serratus anterior and external oblique muscles, rib companion shadows, overlapping ribs and extrapleural fat (see Figs. 2-22 and 2-23). Overlapping rib shadows may be traced out satisfactorily with a high-kilovolt technique and have clearly definable borders, which is often not the case with a plaque that usually has a poorly definable medial border unless it is very thick. Extrapleural fat in obese patients is usually symmetrical over many intercostal spaces. The companion shadow on the under surface of ribs is usually more marked in the upper ribs, and pleural plaques are more common nearer the lower ribs. The interdigitations of serratus anterior and external oblique muscles as they arise from the ribs are a problem. A line diagram (Fig. 2-22 A) shows that the serratus anterior slips have an isosceles triangle shape, while the external oblique slips may be triangular, V-shaped, or quadrilateral. Since the shadows from both sets of muscle slips are superimposed on a radiograph, the composite image may appear as a series of triangles, diamond shapes, or V or inverted V shapes. Differentiation from plaques may be difficult and is summarized in Table 2-3.

The line diagram of the anterior wall of the thorax shows the original of the external oblique muscles of the anterior abdominal wall from the outer surfaces of the lower eight ribs by slips, the upper of which interdigitate with the serratus anterior and the lower with the latissimus dorsi. The latissimus dorsi lies posteriorly and so is not shown in this diagram. The other muscle to be differentiated from pleural plaques is the serratus anterior, which originates by digitations from the outer surfaces of the upper eight ribs.*

Bilateral pleural thickening from prior inflammatory disease or hemothorax is usually not a problem in differential diagnosis because costophrenic sulci or costovertebral sulcus is usually obliterated. Pleuropericardial

*Another confusing shadow has been described as paralleling the medial surface of the upper 3 or 4 ribs along the lateral chest wall. It is more apparent on an oblique than PA view. It tapers inferiorly and may be up to 1 cm wide on the oblique view. It is caused by intercostal muscles, with subcostal muscles, endothoracic fascia, and fat contributing. (Sargent NE, Jacobson G, Gordonson JS. Pleural plaques. A signpost of asbestos dust inhalation. Semin Roentgenol 12(4):287–297, 1977.)

Fig. 2-22. *Diaphragmatic representation of origin of muscle slips of external oblique and serratus anterior (Access young judge). The triangular shape of these slips is seen in B.*

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Fig. 2-23. *Development of pleural plaques. In A (1949), the muscle slips of the serratus anterior are visible. In B (1967), noncalcified pleural plaques have obscured the muscle slips, and more anteriorly calcified plaques are seen. The value of an overpenetrated oblique film to show calcified plaques is seen in C (1967). In D (1972), the noncalcified plaques are larger, and the calcification is more extensive* (see facing page).

and pleurodiaphragmatic adhesions occasionally are problems. If a pleuropericardial adhesion is tentlike, differentiation is easy. If it is a localized area of thickening, as occurs as a sequel of transmural myocardial infarction, its differentiation from a plaque of the mediastinal parietal pleura may be difficult. Fortunately, such plaques are usually not isolated, and others are apparent in the subaxillary or diaphragmatic area. Similarly, pleural diaphragmatic adhesions, if tentlike, are identifiable, and even if such an appearance is absent on frontal view, it may be seen in lateral view to lie adjacent to the major fissure, while most plaques usually lie more posteriorly, at least in the lower zones. In



general, asbestos plaques are characterized by an absence of adhesions, and it is rare but not unknown to have an adhesion and a plaque coexist in the same area. The lack of distinctness of the medial part of the diaphragm seen in some patients with tiny pleural effusions or pectus excavatum is not a problem, since this area is not prone to plaque development. Plaques are most common in the approximate mid-third of the diaphragm in frontal view. Noncalcified callus seen soon after rib fractures, especially bilateral fractures, may be a radiographic problem in the absence of clinical information.

Assessment of Unilateral Plaques

Asbestos exposure produces bilateral plaques. Unilateral plaques are nearly always due to some other cause, e.g., pleural thickening subsequent to rib fractures or callus in a rib fracture simulating a plaque (see Fig. 2-24).

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Table 2-3

Muscle Insertion	Pleural Plaques
Disappear on oblique view	More obvious on oblique view
Usually uniform in shape and size in any individual	Not uniform in shape and size
More apparent in muscular patients and those with much adjacent fatty tissue	Muscularity may obscure small plaques
Medial borders sharp	Medial border indistinct
Extends over ribs 4 to 12, but last two ribs may not be apparent on PA view	Typically occur adjacent to sixth to ninth ribs of lateral chest wall and seventh to tenth ribs posterolaterally, but usually only a few are involved in early cases; in advanced disease, differentiation from muscle slips is easier

Modified from Fletcher and Edge.[7]

Unilateral asbestos-related plaques may occur, however, if there has been a pleural synthesis on one side preventing plaque development. Potential causes include hemothorax, empyema, tuberculous pleurisy, and thoracotomy. Preexisting unilateral chronic pulmonary parenchymal disease may prevent airway passage of asbestos fibers to initiate plaque development.

Detection of Small Calcified Plaques

Calcified pleural plaques occur in the same location as fibrohyaline plaques, i.e., parietal pleura adjacent to the lateral, posterolateral chest walls and, to a lesser extent, anteriorly, the mediastinal pleura, and the diaphragmatic area (see Fig. 2-23). The same projections as mentioned for detection of noncalcified plaques will therefore be useful. Because of the reduction of x-ray absorption with increasing kilovolts, a low-kilovolt technique is preferred. For mediastinal and diaphragmatic calcifications, an overpenetrated film frequently shows calcifications more clearly. While conventional tomography has a limited use in the detection of noncalcified plaques, it is helpful in the detection of small calcifications. The conventional lateral view is especially useful in identifying diaphragmatic calcifications. Although in North America and the United Kingdom posterolateral calcifications are more common, in the Finnish anthophyllite group, anterolateral calcifications predominate.



Fig. 2-24. *Unilateral pleural plaques are frequently due to old rib fractures, and their calcification is linear. This ex-asbestos worker has old fractures of the right tenth and left eleventh ribs (arrows). Plaques, both noncalcified and calcified, are present only on the left, are superior to the rib deformity, and are typically the shape of asbestos plaques. Unilateral plaques due to asbestos are an extreme rarity and usually an alternative cause should be considered.*

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Fig. 2-25. *Oblique projection, rib detail techniques to show calcified and noncalcified plaques.*

The shape of the calcifications differs according to location. Peripheral plaques (as opposed to mediastinal and diaphragmatic plaques) may exhibit the forms shown in Figure 2-4A. Usually in the geographic, rectangular, and circular forms the periphery is more dense on the x-rays. The steplike and lobulated forms that usually lie closely adjacent to the lateral chest wall on frontal views may appear homogeneously dense on the x-ray. The areas appearing most dense will alter with varying projections as thicker parts of the plaque become tangential to the beam.

Mediastinal plaques do not show such a varied radiographic appearance.

They are almost always linear shadows, continuous or interrupted, of varying thicknesses and length. They present in frontal radiographs as long plaques of calcification, 0.5–2 cm on either or both sides of the vertebral column. If both the anterior and posterior mediastinal pleural reflections are calcified, there will be a double or tramline shadow, about 1 cm apart. On the left side, differentiation from calcification of the descending aorta is needed. The mediastinal pleura may also be calcified where it lies against the heart. It is indistinguishable from localized periocardial calcifications or myocardial calcifications. In some asbestos surveys, in fact, it was considered as being pericardial in location. Like paravertebral mediastinal pleural plaques, it is linear. It parallels the heart border, being more common on the left side. It may lie adjacent to the left atrial appendage, near the area of opposite pulsations, i.e., just below the outflow tract of the right ventricle or in the area of the left ventricular wall. In the latter position, differentiation from pericardial calcification, calcification in a transmural myocardial infarct, or intracavitary myxoma may be difficult. The rare calcifications along the right heart border are adjacent to the right atrium. The preponderance along the left heart border compared with the right has been stated to result from the increased mechanical irritation of the asbestos fibers when adjacent to the pumping heart. Since more of the plaques have been reported adjacent to the left atrium than adjacent to the left ventricle, this concept may be incorrect.

Calcifications along the diaphragm are restricted to the more rigid central aponeurotic area, as are the hyaline plaques. On lateral view, they are seen beginning at the posterior border of the heart and extending backwards. Since the deeper parts of the plaques calcify, and the outline of the diaphragm on the frontal view may be anterior or posterior to the plane of calcification, the calcifications may or may not parallel the diaphragm. In the lateral view, however, such parallelism usually exists. Low-kilovolt techniques in full inspiration are needed. The shapes are less varied than the subaxillary shadows, with the linear form predominating. Lower rib costal cartilage calcifications projected along or below the diaphragm on the frontal view may simulate a calcified plaque. Some examples are seen in Figure 2-26.

"Talc plaques" described in x-rays of talc workers appear as linear calcifications similar to the above. These calcified plaques were described in the early reports on talcosis. Talc is a hydrated magnesium silicate closely related chemically to several types of asbestos (chrysotile, tremolite, and anthophyllite), and talc deposits usually contain one or more of these minerals.[34,35] Plaques occurring after talc exposure are probably due to an associated asbestos content rather than to talc fibers, but current evidence is not

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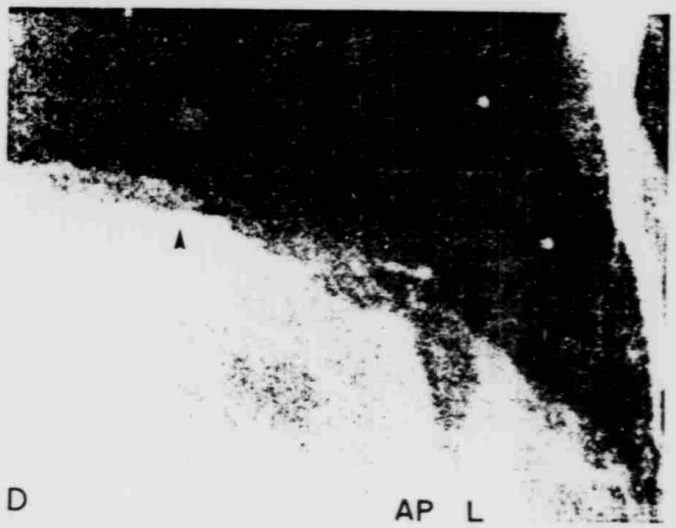


Fig. 2-26. Value of AP and PA projections. A and B are PA views of the right and left hemidiaphragm. In C and D which are the AP views, the calcification in medial part of the hemidiaphragm (arrows) become more apparent. For geometric reasons, [illegible] detection of posteriorly located diaphragmatic calcifications will be obtained by AP views. An overpenetrated view (D) is an additional aid.

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Fig. 2-27. *Anterior pleural plaques. Oblique view shows pleural plaques in unusually anterior locations. There is also linear diaphragmatic calcification (arrow).*

conclusive. Occasional case reports have described calcified pleural plaques in chronic mineral oil aspiration, scleroderma, and after irradiation.

Significance of Visceral Pleural Thickening After Asbestos Exposure

Radiography cannot distinguish between diffuse thickening of visceral and parietal pleura (interlobar fissures excepted) (see Fig. 2-27). Diffuse thickening of the visceral pleura is seen after asbestos exposure as a "dry" pleurisy[33] or as residual from a resolved asbestos effusion. Diffuse lateral chest wall shadowing and thickening of the interlobar fissures may result from many types of pleural exudates and transudates, and in an asbestos worker the contribution from asbestos exposure is always conjectural.

Pleural Plaques Associated with and Obscuring More Serious Asbestos-related Lung Disease

Masking of Small Peripheral Pulmonary Carcinoma by Large Parietal Pleural Plaques

There is a predominance of asbestos-related lung cancer in the lower lobes, the areas that usually contain the greatest proportion of ferruginous bodies and the greatest degree of pulmonary fibrosis. The difficulty in the early detection of bronchogenic carcinoma in severely fibrotic regions is discussed in Chapter 2, Section 2. Since parietal pleural plaques predominate adjacent to the sixth to tenth ribs and the diaphragm, and since many asbestos-related lung cancers are peripheral in location, especially the adenocarcinoma, the early radiographic detection of these tumors may be difficult. Stereoscopic and oblique projections should be used, but their value is limited if a large plaque and a small peripheral carcinoma are adjacent. Tomographic sections at close intervals should be made (Fig. 2-86). Oat cell and squamous cell carcinomas are also common in patients with asbestos exposure, but these tumors are usually more central and less likely to be masked by plaques. CT may prove very helpful. The axial images should be valuable in separating the adjacent shadows due to a plaque and those due to a peripheral carcinoma.

Asbestos Pleural Effusion Masking Parietal Pleural Plaques

A pleural effusion is a rare stigma of asbestos exposure (Chapter 2, Section 3) (see Fig. 2-28). It may be unilateral or bilateral and may resolve completely or be recurrent over many years. The fluid is an exudate that does not contain asbestos fibers. For this reason and also because such effusions are rarely associated with recognizable plaques, the cause is often difficult to ascertain. The easiest technique for identification of any accompanying plaques is a steep Trendelenburg* decubitus view which permits the exudate to move toward the lung apex and so uncover the pleura adjacent the lower ribs. Unlike the usual decubitus view for pleural effusion, the exposure should be made in both the inspiratory and expiratory phases. An elevated diaphragm in the expiratory phase may obscure some of the lower plaques, although the effusion is seen more easily in this phase.

*Friedrich Trendelenburg (1844–1924) is alleged to have performed the first successful pulmonary embolectomy." (Brunners Schweizer A.G. Hopten-Archiv.)

Fig. 2-28. *Pleural plaques and pleural effusion in 1973 (A and B, both lungs, upper zones) and in 1976 (C and D, same lungs, same zones). In 1973 an asbestos effusion had developed on the right (arrow), and on the left (b) were the pleural plaques (arrow) and a tiny effusion (arrowhead).*

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Association of Plaques in Asbestos-related Mesothelioma

The presence of pleural plaques is a potentially useful "marker" in the radiographic diagnosis of mesothelioma. Unfortunately, very few x-rays of patients with pleural mesothelioma show identifiable plaques. Pleural plaques are more evident in association with peritoneal mesotheliomata. These patients have usually had a heavy crocidolite exposure, and it is conjectured[36] that the type of fiber is of importance. It is acceptable that a mesothelioma of any size would envelop and obscure parietal pleural plaques. But why are they so rarely visible beyond the radiographic shadow of a mesothelioma or on the contralateral side? Postulating that some fibers predispose to plaque formation and others to mesothelioma induction would appear reasonable because anthophyllite asbestos production in Finland is associated with a high incidence of parietal pleural plaque formation but not mesothelioma.[37–39] Furthermore, anthophyllite gives rise to plaque production at an early stage of exposure, and these should have attained a large size and be reasonably recognizable should mesothelioma have developed in exposed workers. Chrysotile mining is known to be related to a low incidence of mesothelioma. Yet in two Quebec chrysotile mines, the one with the higher dust exposure had a high incidence of both mesothelioma and pleural plaque formation, quite unlike the situation in Finland, where plaque formation is common and mesotheliomata rare. South African N.W. Cape crocidolite exposure entails a high risk of mesothelioma induction and of pleural plaque formation, but the incidence of mesothelioma combination varies with the manganese content of the asbestos.[40] Furthermore, crocidolite and amosite ore often intermingle in the same rock formation, although pure amosite is a lesser cause of mesothelioma induction.[16a,16b] Excluding anthophyllite, which causes pleural plaques only (only one anthophyllite worker is known to have developed mesothelioma), the other three major fibers all cause both pleural plaques and mesotheliomata to varying degrees. It ensues that some unknown factor is responsible for the relatively few pleural plaques seen in asbestos workers who develop mesotheliomata.

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