

Ventilatory Failure Due to Asbestos Pleurisy

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Seven patients are described who had a distinctive syndrome of chest wall restriction caused by asbestos-induced pleural fibrosis. All had severe dyspnea and predominant pleural disease on radiographic examination, with pulmonary function findings of reduced vital capacity, total lung capacity (measured in five patients), and maximal voluntary ventilation. Five patients had ventilatory failure with carbon dioxide retention; four of these have died and one is close to death. Examination of the thoracic organs in five patients showed minimal or no parenchymal fibrosis in three and less severe involvement of the parenchyma than of the pleura in the remaining two. Neoplasms were suspected in three patients because of extension of the pleural fibrosis into the lung. Two of these patients had pleural uptake of 67-gallium citrate attributable to the inflammatory reaction. With the increasing duration since onset of exposure in the nine million workers who have been exposed to asbestos, as well as in other exposed persons, it is expected that additional cases of ventilatory failure caused by asbestos-induced pleural fibrosis will be encountered.

It has not been recognized that ventilatory failure, cor pulmonale, and death may result from extensive pleural fibrosis caused by exposure to asbestos, even though little or no parenchymal disease is present. Benign pleural effusions, pleural fibrosis presenting as localized plaques or diffuse thickening, and pleural calcification are well-known complications of asbestos exposure. The recognition of these lesions has been emphasized, but their functional impact has received little attention and has generally been described as minor.

We herein describe five patients who met the following criteria for ventilatory failure due to asbestos-induced pleural fibrosis: (1) exposure to asbestos in the absence of other pleuropulmonary disease; (2) severe dyspnea (grade 3 on the British Medical Research Council three-point scale [1]); (3) vital capacity and total lung capacity (measured in four patients) of less than 50 percent of predicted; (4) maximal voluntary ventilation of less than 80 percent of predicted; (5) carbon dioxide retention (P_{aCO_2} of 52 torr or more); (6) absence of airway obstruction: ratio of one-second forced expiratory volume to forced vital capacity of more than 0.75; (7) predominant pleural disease evident radiographically (all five patients) and on examination of the thoracic organs at surgery (three patients) or autopsy (one patient).

In addition to these five patients with carbon dioxide retention, we have encountered others with a similar but less severe pattern of functional impairment. We report two representative cases without carbon dioxide retention to illustrate the range of physiologic findings.

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TABLE I Clinical Findings in Seven Patients with Asbestos Pleural Fibrosis

Patient	Asbestos Exposure	Lung Tissue	Significant Pulmonary Fibrosis*	Observed Pleural Effusion	Pleural Uptake of Gallium	Outcome
1	Insulation	Necropsy	No	NA	NA	Cardiorespiratory failure, death
2	Insulation	Bilateral thoracotomy	No	No	Negative	Cardiorespiratory failure, improvement after first decortication, death
3	Insulation	Thoracotomy	Yes	No	Positive	Cardiorespiratory failure, suspected neoplasm, death
4	Aboard ship	Thoracotomy	No	Bilateral	Negative	Cardiorespiratory failure, suspected neoplasm
5	Shipyard	None	No	Recurrent	NA	Cardiorespiratory failure, death
6	Insulation	Thoracotomy	Yes	Yes	Positive	Cardiorespiratory insufficiency, suspected neoplasm
7	Insulation	None	No	Yes	NA	Dyspnea on exertion

* Defined by radiography and by gross findings at autopsy (one patient) or surgery (four patients).
NA = information not available.

Three of the total of seven patients described were suspected of having neoplasms of the pleura or lung because of extension of pleural fibrosis into the lung.

The seven cases reported herein, together with others recently described [2], exhibit a distinctive picture of restrictive ventilatory impairment with entrapment of the lung due to asbestos-induced pleural fibrosis. This is to be differentiated from the better-known restrictive impairment due to asbestotic interstitial fibrosis. Asbestos pleural fibrosis is another cause of chest bellows dysfunction that has been reported with post-traumatic or postempyematosus fibrothorax. With many patients from the huge pool of asbestos-exposed workers (estimated to number more than nine million) [3] now presenting with asbestos-induced disease, resulting ventilatory insufficiency will be increasingly common. It is important, for legal and financial considerations, to recognize asbestos exposure as the cause and, for clinical considerations, to separate pleural from parenchymal fibrosis as the mechanism for ventilatory failure, because management and the role of surgery differ.

PATIENTS AND METHODS

A prospective study was begun when Patient 2 had the same relation between asbestos pleural fibrosis and ventilatory failure that we had noted previously in Patient 1. All patients were hospitalized at the Mount Sinai Medical Center in New York City.

Conventional pulmonary function tests were performed by standard techniques [4]. Predicted spirometric values were those of Morris, et al [5], as confirmed by our laboratory in an investigation of 229 normal nonsmoking adults [6]. For maximal voluntary ventilation, values were those of Baldwin et al [7], and for total lung capacity were those of Goldman and Becklake [8]. The single-breath diffusing capacity for carbon monoxide was not corrected for venous carbon monoxide or anatomic dead space. Prediction equations were those of Bates et al [9], as well as those derived by our laboratory from a survey of 511 subjects randomly selected from

the general population [10]. For the single-breath diffusing capacity for carbon monoxide/alveolar volume, alveolar volume was the single-breath helium-dilutional total lung capacity; predicted values were those from our survey [10]. Static pulmonary compliance was measured in three patients by the technique of Mead and Whittenberger [11] using an esophageal balloon pressure sensor.

Spirometric values, maximal voluntary ventilation, and arterial gas values were obtained in all patients. Patient 1 was studied before other techniques were available, Patient 5 could not tolerate the body plethysmograph, and Patients 4 and 5 were unable to hold their breath long enough to allow determination of the single-breath diffusing capacity for carbon monoxide.

RESULTS AND SELECTED CASE REPORTS

Clinical findings are summarized in Table I. All seven patients had progressive, severe shortness of breath. Additional symptoms were pleuritic pain or chest tightness and nonproductive cough.

Pleural effusions were noted during the course in four of the seven patients described. These were recurrent or bilateral in two patients. Pleural thickening described at thoracotomy was predominantly parietal. When decortication was performed (in four patients), the pleural was described by the surgeon as a thick "rind," often 2 cm in thickness. This is illustrated in Figure 1 from Patient 3 and Figure 2 from Patient 6.

In all patients except one (Patient 5), the pleural fibrosis was bilateral. This exceptional patient, who had dense fibrosis and calcification of the lower half of the left pleura on radiographic examination, died of carbon dioxide retention.

The extensive pleural disease with the development of mass-like shadows on roentgenographic examination suggested mesothelioma or lung cancer in three of the seven patients. These were evaluated by scanning with 67-gallium citrate, which revealed uptake of the isotope in two patients who were shown to have proliferative pleuritis but no malignancy at thoracotomy. Positive

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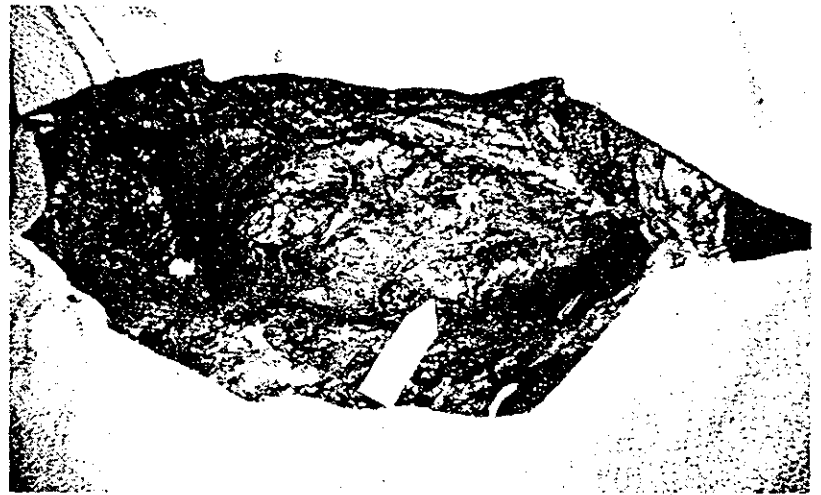
1. Surgical view of the right thorax in Patient 3. The chest wall is exposed; the thickened parietal pleura is held by a clamp. The pleural space and visceral pleura are visible.



on [10]. For the single-breath monoxide/alveolar volume, single-breath helium-dilution techniques were those from our survey. Compliance was measured in three patients and Whittenberger [11] used a pressure sensor. Maximal voluntary ventilation was obtained in all patients. Patients used various techniques were available, including body plethysmograph, and patients held their breath long enough to measure single-breath diffusing capacity.

SELECTED CASE REPORTS

are summarized in Table I. All patients had massive, severe shortness of breath. Symptoms were pleuritic pain and a nonproductive cough. Symptoms were noted during the course of the disease described. These were noted in all patients. Pleural thickening was predominantly parietal. Surgery was performed (in four patients) and was described by the surgeon as a thickening. This is illustrated in Figure 2 from Patient 6. Except one (Patient 5), the patients had no pleural calcification of the lower lobe. Radiographic examination, died of



2. Top, the surgical field during thoracotomy in Patient 6. The parietal pleura is not well demonstrated, the visceral pleura is thickened but easily removed from the underlying lung. Bottom, the removed parietal pleura. Its right edge and thickness are well seen in the section.

pleural disease with the development of pulmonary hypertension on roentgenographic examination. No evidence of mesothelioma or lung cancer in the patients were evaluated by scanning, which revealed uptake of the isotope. The patients were shown to have pleural disease at thoracotomy.

TABLE II Pulmonary Function in Seven Patients with Asbestos Pleural Fibrosis

Patient	Date	VC (ml)	TLC* (ml)	FEV ₁ /FVC	MVV (liters per minute)	Lung Compliance (liters/cm H ₂ O)	D _L CO _{SB} (ml/torr per minute)	D _L CO/V _A	Paco ₂ (torr)	Pao ₂ (torr)
1	1965	1,420 (47)	...	0.86	76 (78)	...	...	...	52 → 64	Sat 81%
2	10/72	1,420 (33)	2,440 He (41)	0.97	60 (59)	...	...	...	41	70
	preop 8/75	1,820 (43)	3,170 (48)	0.70	58 (55)	..	9.2 (41 versus 36)†	3.5 (89)	39	85
	2/80	1,500 (37)	...	0.80	49 (51)	...	10.2 (51 versus 40)	4.1 (102)	44 → 70	55
3	1/75	2,200 (44)	3,760 (49)	0.84	...	...	9.9 (46 versus 31)	3.1 (76)	...	75
	8/78	1,030 (20)	2,810 (36)	0.98	68 (65)	0.03	...	...	48 → 70	63
4	10/77	1,370 (28)	3,650 (48)	0.96	74 (74)	...	...	...	51	77
	preop 7/79	1,190 (25)	3,460 (46)	0.92	...	...	...	...	66	56
5	4/78	1,720 (39)	...	0.89	50 (44)	...	...	...	>70	<45
6	2/78	1,940 (34)	4,080 (47)	0.87	79 (54)	0.05	17.1 (48 versus 55)	5.6 (137)	41	77
	preop 12/80	1,780 (32)	3,530 (43)	0.65	60 (42)	...	...	...	34	71
7	5/77	2,720 (63)	4,150 (66)	0.86	78 (70)	0.07	20.7 (85 versus 72)	5.9 (122)	34	88

Values in parentheses are percent predicted.

* Results are plethysmographic unless indicated (He = multiple breath helium dilution).

† The first value is from reference 10, the second from reference 11.

D_LCO/V_A = ratio of carbon monoxide diffusing capacity to alveolar volume; D_LCO_{SB} = single-breath diffusing capacity for carbon monoxide; FEV₁/FVC = ratio of one-second forced expiratory volume to forced vital capacity; MVV = maximal voluntary ventilation; TLC = total lung capacity; VC = vital capacity.

findings on gallium scanning have been described in benign asbestos pleural disease [12].

In five of the seven patients (including four of the five with ventilatory failure), there was little or no radiographic evidence of interstitial fibrosis. On examination of the thoracic organs at thoracotomy (four patients) or autopsy (one patient), palpable fibrosis of the lungs was absent in three and less prominent than the pleural fibrosis in the other two.

Thoracotomy with decortication was undertaken in four patients. This was done to rule out a neoplasm in the three mentioned earlier and did not improve their clinical status, in part because the less involved side was surgically treated in two of them. In one patient (Patient 2), decortication of the more involved side was performed to improve respiratory function and resulted in a substantial increase in exercise tolerance and improvement in measured lung function. Decortication of the opposite side was recommended but refused by the patient until his condition was nearly terminal, at which time the procedure stabilized his condition and allowed

withdrawal of mechanical ventilation. In all cases, the lung expanded well after decortication.

Results of pulmonary function tests are summarized in Table II. Of the five patients who had respiratory failure with carbon dioxide retention (Patients 1 to 5), four have died and one is severely disabled. The vital capacity was less than 50 percent of predicted in all five patients, ranging as low as 20 percent (1.03 liters). Average was 33 percent of predicted. The maximal voluntary ventilation was reduced in all five (average 64, lowest value 44 percent of predicted) in the absence of airway obstruction. Indeed, the ratios of one-second forced expiratory volume to forced vital capacity were high (average 0.93, range 0.86 to 0.98), and flow rates such as maximal mid-expiratory flow were reduced to a lesser extent than the vital capacity, consistent with restrictive impairment. Full lung volumes were measured in three patients; total lung capacity was reduced (less than 50 percent of predicted) in all. The single-breath diffusing capacity for carbon monoxide (measured in two patients) was reduced; the ratio of single-

breath diffusing capacity for carbon monoxide to alveolar volume was normal.

Patient 6, who did not have carbon dioxide retention, had equally severe reductions in vital capacity, total lung capacity, and maximal voluntary ventilation. Although single-breath diffusing capacity for carbon monoxide was low, the ratio of carbon monoxide diffusing capacity to alveolar volume was increased. Patient 7 had a similar pattern but less severe reductions in forced vital capacity, total lung capacity, and maximal voluntary ventilation. In this patient, the ratios of one-second forced expiratory volume to forced vital capacity and single-breath diffusing capacity for carbon monoxide to alveolar volume were increased.

Pulmonary compliance was measured in three patients, with (Patient 3) and without (Patients 6 and 7) carbon dioxide retention; it was markedly reduced in all three.

Patient 1. Born 1912 and a nonsmoker, the patient had been an insulation worker from 1929 to 1946 and again from 1959 to 1961, with lesser exposure in intervening years. In 1963, routine chest radiography revealed bilateral infiltrates with extensive pleural thickening and pleuropericardial adhesions. Clubbing and rales were noted on physical examination. The vital capacity was slightly reduced (72 percent of predicted). Over the next two years, dyspnea and signs of right heart failure developed. On his first admission to the Mount Sinai Medical Center on November 12, 1965, the vital capacity was 1,425 ml (47.5 percent of predicted). Arterial oxygen saturation was 81 percent at rest and 68 percent on one minute of step-up exercise; P_{aCO_2} was 52 torr. The patient was readmitted in April and in October 1966, at which time P_{aCO_2} was 64 torr. The patient's condition continued to deteriorate; tracheostomy was performed, and the patient died on December 11. Postmortem examination revealed marked thickening of the parietal and lesser thickening of the visceral pleura. The lung parenchyma showed only minimal fibrosis. Right ventricular hypertrophy was observed.

Patient 2. Born 1916, this patient had been an insulation worker from 1939 to 1956. In 1963, he was found to have radiographic changes consistent with asbestos exposure; spirometric results were normal. He had smoked two packages of cigarettes per day beginning at age 14 but stopped smoking at this time. In 1972, he noted increasing shortness of breath and a 35-pound weight loss. Chest film (Figure 3, top) now showed massive bilateral pleural thickening, greater on the left than on the right. Pulmonary function tests revealed severe restrictive impairment (vital capacity 33 percent and total lung capacity 41 percent of predicted) with maximal voluntary ventilation 59 percent of predicted. Left pleurectomy was performed to improve pulmonary function. The pleura showed dense fibrosis, chronic inflammation, and areas of neovascularization and hemorrhage. Hyperplastic mesothelial cells, neutrophils, and fibrin on the pleural surface were noted histologically. Postoperatively, the patient had marked improvement in exercise tolerance. Previously unable to walk across the room, he was now doing household chores and shopping. The vital capacity increased 400 ml (31 per-

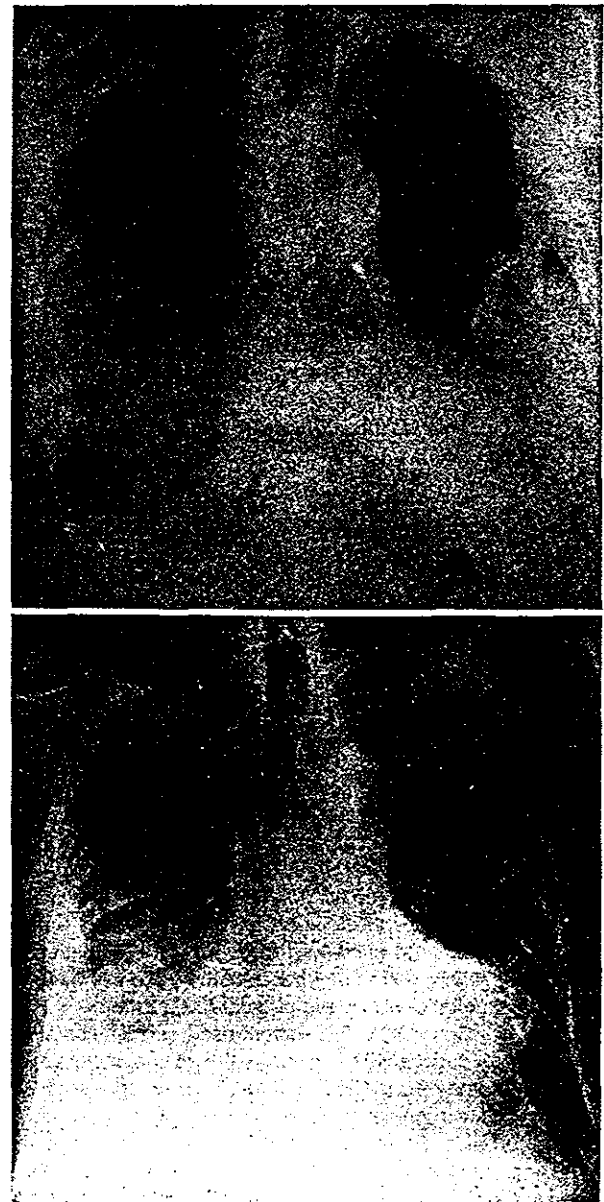


Figure 3. Top, Chest roentgenogram of Patient 2 in 1972 showing massive bilateral pleural thickening greater on the left and involving the lower two thirds of the thorax. Bottom, Patient 2 in 1980, eight years after left decortication showing extension of pleural thickening at the right base.

cent) compared with the preoperative value. This improvement was maintained until 1979, when he noted increasing dyspnea and limitation in activities. The pleural thickening on the right side had progressed, as seen in Figure 3, bottom. Decortication was advised but deferred by the patient. By August 1980, vital capacity had decreased 320 ml (18 percent) and maximal voluntary ventilation 9 liters per minute (16 percent) from their values in 1975. The patient required permanent mechanical ventilation through a tracheostomy.



Figure 4. Chest roentgenogram of Patient 6 showing bilateral pleural thickening greater on the left. On the right side, the short fissure is thickened and depressed, and there is a density medial to this that had not previously been noted.

He had upper gastrointestinal hemorrhage in January 1981. In April, right pleurectomy was performed. Pulmonary artery and right heart pressures were increased, although pulmonary capillary wedge pressure was normal. The parietal and visceral pleura were markedly thickened. The lung, which was described by the surgeon as "trapped," expanded well at the end of the procedure. Postoperatively, the patient's condition improved enough to allow him to perform the activities of daily living with a permanent tracheostomy but without mechanical ventilation. He died one year later at home.

Patient 6. Born 1935, this patient had been an insulation worker from 1955 to 1977. He had smoked two packages of cigarettes a day from 1953 until 1967, when chest radiography showed progressive bilateral pleural thickening, greater on the left side. From 1973 to 1977, the patient noted increasing shortness of breath. In 1977, a new density was seen in the right mid-lung field (Figure 4). The vital capacity was 34 percent and the maximal voluntary ventilation 54 percent of predicted. Transbronchial as well as percutaneous lung biopsy performed because of suspicion of bronchogenic carcinoma revealed pulmonary fibrosis and ferruginous bodies. Gallium scanning demonstrated increased activity in the right mid-lung field, corresponding to the abnormality seen on chest roentgenography. Thoracentesis showed a markedly thickened pleura. Protein concentration of the pleural fluid was increased. No abnormal cells were seen. Needle biopsy of the pleura revealed dense fibrosis with areas of inflammation and hemorrhage. Computed tomographic scanning revealed extensive pleural thickening on the left and pleural thickening on the right, continuous with a mass-like density intruding anteriorly into the lung (Figure 5). Because mesothelioma or lung cancer could not be ruled out, right thoracotomy was performed in March 1978 (Figure 2, top)

even though pleural involvement was greater on the left side. The parietal pleura was thick and rigid, described in the surgical report to be "like shoe leather" (Figure 2, bottom). The mass density seen on computed tomographic scanning proved to be a projection of visceral pleura into the middle lobe. The pleura was densely hyalinized, showing areas of neovascularization, chronic inflammation, and necrosis of collagen with neutrophils. An acute fibrinous exudate was seen on the surface. After parietal and visceral pleurectomy the lung expanded well. The patient still had dyspnea on minimal exertion when last seen 18 months after pleurectomy. The vital capacity was now 32 percent of predicted, similar to the previous value, but the maximal voluntary ventilation had decreased further to 42 percent of predicted. The left hemithorax showed progression of the pleural involvement, and decortication was advised but refused by the patient.

Comment: This patient resembles the first five. The degree of dyspnea, the pleural findings on radiography, computed tomographic scanning, and thoracotomy, and the reduction in pulmonary function were of similar severity. He differs only in the absence thus far of carbon dioxide retention.

Patient 7. Born 1937, this patient had been employed as an insulation worker from 1952 to 1969. He had smoked more than two packs of cigarettes per day from 1954 to 1975, at which time he presented to the Mount Sinai Medical Center with dyspnea on exertion of one year's duration. Six months previously, he had been hospitalized elsewhere for pleural effusions. Chest radiography revealed bilateral pleural thickening more extensive on the right, with calcification. Pulmonary function tests showed moderate restrictive impairment. Despite the reduction in total lung capacity, the single-breath diffusing capacity for carbon monoxide was well preserved. Arterial gases were normal except for a slightly

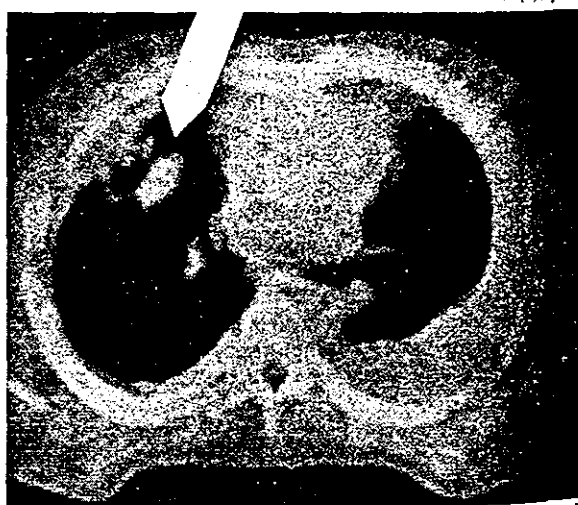


Figure 5. Computed tomographic scan of the thorax (Patient 6) showing bilateral diffuse pleural thickening more extensive on the left side but projecting into a mass-like density or pseudotumor (arrow) in the middle lobe.

increased alveolar-arterial gradient for P_{O_2} (18 torr). The patient declined further evaluation.

Comment: This patient has a pattern of ventilatory impairment similar to, but less severe than, that in the other six.

COMMENTS

Hyaline and calcific plaques, diffuse thickening, and obliteration of the costophrenic angles are recognized pleural consequences of exposure to asbestos. They are specifically coded by the International Labor Office roentgenographic classification [13]. Pleural findings on radiographic examination are more common in certain cohorts than is interstitial fibrosis. Minor reductions in lung function have been reported in asbestos workers with these pleural abnormalities [14-16]. Diffuse pleural thickening was associated with ventilatory impairment on submaximal exercise testing [17].

Pleural fibrosis so severe as to result in ventilatory failure has not been reported. Wright et al [2] described six cases with marked pleural thickening that resulted in entrapped lungs. These patients had grade 1 dyspnea on the British Medical Research Council scale (walking stairs or hurrying on level ground) and had no evidence for respiratory failure as did the first five patients described herein. The pattern of physiologic impairment was otherwise quite similar. The vital capacity was decreased, although much less so than in our first five patients (mean 69 versus 33 percent of predicted), but the ratio of one-second forced expiratory volume to forced vital capacity was high. The authors emphasized that although single-breath diffusing capacity for carbon monoxide was decreased, the ratio of carbon monoxide diffusing capacity to alveolar volume was high. Sterling and Herbert [18] reported six cases similar to those of Wright et al.

The dyspnea, which was so prominent a symptom in all our patients, and the carbon dioxide retention, which supervened in five, can be explained on the basis of increased work of breathing and dead space ventilation secondary to decreased lung volumes and chest wall compliance.

Relative preservation of gas transfer despite restriction in lung volumes (high ratio of carbon monoxide diffusing capacity to alveolar volume) fits the pattern of "entrapped lung" that may be seen in musculoskeletal disorders (for example, kyphoscoliosis) or pleural disease (such as, fibrothorax resulting from old empyema or asbestos-induced pleurisy). The reduction in maximal voluntary ventilation, present in all seven of our patients, is consistent with impairment of the chest wall. Reduction in maximal voluntary ventilation is characteristic of kyphoscoliosis [19]; its absence has been stressed in asbestotic interstitial fibrosis [20].

Recently, Britton [21] demonstrated a significant decrease in vital capacity in asbestos workers that correlated with increasing pleural abnormality. When subjects with different degrees of pleural involvement were analyzed by holding constant the degree of interstitial fibrosis (profusion rating), the vital capacity decreased with increasing pleural involvement in each category of interstitial fibrosis. The investigator concluded that pleural involvement alone can result in disability.

Measurement of pulmonary compliance does not separate pleural from interstitial fibrosis. The usefulness of esophageal pressure recordings in the presence of gross pleural disease is questionable [2]. Pulmonary compliance was markedly reduced both in our series (three patients) and in others' [2,18].

In the seven patients we describe, four had one or more known episodes of pleural effusion. Benign pleural effusions due to asbestos inhalation have been described by several investigators [22-30], although a few of these cases resulted in mesothelioma after a period of time [23,30]. The latent period for benign pleural effusions is shorter than for all other complications of asbestos exposure. It is the only manifestation seen within 10 years of onset of exposure and the most common abnormality within the first 20 years. Epler et al [30] reviewed serial roentgenograms of 1,135 workers and noted 34 cases with effusion. Incidence was related to intensity of exposure and was 9.2 cases per 1,000 person-years for heavily exposed workers. Prevalence was 7 percent in this group. Fewer than 10 percent of the workers with effusions had significant parenchymal fibrosis. In follow-up study, virtually all subjects with effusion showed blunted costophrenic angles and 54 percent showed diffuse pleural thickening. Diffuse pleural thickening was generally a sequela of acute effusion. By contrast, in the more numerous workers with pleural plaques, there was no relation to effusion.

Review of cases in the literature as well as of the patients presented herein suggests that the intense pleural inflammation secondary to asbestos, which often manifests itself as an effusion, eventuates in the extensive pleural fibrosis we observed. The effusions are exudative and often bilateral and/or recurrent. Since they may be small and asymptomatic [30], they may go undetected and their relation to consequent pleural thickening remain unrecognized.

Herbert and Sterling [31] described granulation tissue organizing a pleural exudate. Dense fibrosis extended into the interlobular septa. The series of 12 patients with asbestos effusions described by Gaensler and Kaplan [23] included four who underwent decortication (one patient had bilateral procedures) "to establish the diagnosis, to restore pulmonary function, and

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to prevent recurrence." Many findings at thoracotomy were similar to those in our patients. The pleura was vascular and thickened (up to 2 cm). There were areas of fibrinous exudation, vascularization, and proliferative mesothelium. The lung was partially atelectatic but inflated readily after decortication. Physiologic studies when no effusion or the least residual was present revealed restrictive changes in three patients. In one of these, the maximal voluntary ventilation was also reduced.

Navratil and Dobias [32] described seven patients in Czechoslovakia with "pleural hyalinosis complicata" whose courses were marked by acute inflammation with effusion. This was followed by diffuse pleural thickening and restrictive impairment. Unfortunately, no lung function or arterial gas data were reported.

Some degree of interstitial fibrosis is likely in all patients with pleural changes induced by asbestos, since the offending fibers are thought to reach the pleura by way of the parenchyma and lymphatics. Nevertheless, in five of the seven patients we describe, there was little radiographic evidence of interstitial fibrosis. Even when the thoracic organs were examined, in three of these five patients, palpable fibrosis of the lungs was absent and the lungs expanded well after decortication. We cannot state whether *microscopic* evidence of interstitial fibrosis was present in these three patients. Biopsy specimens of grossly normal lung were not obtained because surgery was undertaken to improve severely limited lung function and/or to rule out neoplasia.

Two patients had radiographic evidence of interstitial fibrosis and are indicated as having "significant pulmonary fibrosis" in Table I. Their interstitial fibrosis was less prominent at thoracotomy than was their extensive pleural involvement. In these two, and in the other five

patients, the physiologic pattern was more consistent with entrapped lung rather than with interstitial pulmonary disease.

Progressive weight loss, bloody pleural effusions, and extensive pleural thickening with mass-like shadows on radiographic examination strongly suggested mesothelioma or bronchogenic carcinoma in three patients. Differentiation could not be established by gallium scanning. Of the malignant mesotheliomas scanned at this institution, 12 percent failed to take up 67-gallium citrate [12]; however, uptake was demonstrated in two of our three patients who had extensive, nonmalignant pleural disease. Because closed pleural biopsy was believed to be unreliable, the diagnosis in these three patients was established by careful examination of the large amount of tissue removed at decortication. The need for diagnosis impelled surgical intervention on the less encased side in two of the three patients.

Similar "pseudotumors" secondary to asbestos have been reported by Hillerdal and Hemmingsson [33]. These were localized fibrotic thickenings of the visceral pleura and underlying lung parenchyma. Based on our experience in Patient 6 and Hillerdal's experience in three cases, computed tomographic scanning is valuable in recognizing pseudotumors but may not obviate the need to establish a histologic diagnosis. This can be accomplished by thoracotomy, as in our cases, or by thoracoscopy [34].

When pleurectomy was performed (in four of our patients), the underlying lung was noted to expand well. Since the functional impairment is due to entrapment of lung by fibrocalcific pleura, pleurectomy may improve ventilatory function. Our experience (one patient) with this procedure for this purpose rather than for diagnosis is insufficient to allow any generalization.

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