WV-0221#4

PLAINTIFF'S EXHIBIT

UC-2178

UNION CARBIDE

INTERNAL CORRESPONDENCE

SILICONES & URETHANE INTERMEDIATES P. O. BOX 8361, SOUTH CHARLESTON, WEST VIRGINIA 25303

To (Name) H. W. Himes, M.D.
Division R. W. Holland, M.D.
Location F. M. Plechaty, R.N.

Date June 9, 1983

Originating Dept. Safety & Health

Subject Asbestos-related Health Information

Copy to
Mr. R. E. Crum
Mr. C. E. Fry
Mr. E. B. Harris
Mr. T. E. Lawrence
H. C. Lewinsohn, M.D. (letter only)
Mr. L. E. McGlure
Mr. J. B. Mickey
Mr. D. G. Moshier
Mr. M. C. Polniaszek
S/H Managers

The attached information provided by Dr. Lewinsohn should be of interest to you. Fran Plechaty is requested to pass along a copy to Dr. Boone.

Very truly yours,

J. S. Cornell

JSC:ke
Ext. 3406

Attachment

UNION CARBIDE HEALTH SAFETY AND ENVIRONMENTAL AFFAIRS

HILTON C. LEWINSOHN, MB.BCH., F.C.C.P.
Assistant Corporate Medical Director
P2590
(203) 794-5214

June 7, 1983

TO: Dr. S. Austin
Dr. B. Ballantyne
Mr. J. Cornell
Dr. T. G. Fortney
Dr. W. F. Gorham
Mr. V. H. Johnkoski
Dr. A. A. Lang
Dr. T. A. Lincoln

RECEIVED BY
JUN 9 1983
J. B. CORNELL

The attached "abstracts" are from papers presented May 27-29, 1983 at the Thirteenth Annual Symposium on Chest Disease of the The Fleischner Society.

They may be of interest to you.

Hilton

OLD RIDGEBURY ROAD, DANBURY, CT 06817

ASBESTOS RELATED DISEASE:EPIDEMIOLOGIC & CLINICAL ASPECTS

Edward A. Gaensler

There are about 4 million persons alive today who have had heavy exposure to asbestos and an equal number with significant but lesser exposure (1). Among the several asbestos related disorders asbestosis, a term that should be reserved for pulmonary fibrosis (2), will remain an important but diminishing concern among the heavily exposed. The 3 pleural manifestations, hyaline plaques, benign asbestos effusion and mesothelioma, because of the lesser required exposure and long latent period, very likely will become increasing problems in all of the 8 million exposed persons. Concerning bronchogenic carcinoma, it is thought that 2 per cent of the 135,000 cases reported each year are related to asbestos exposure. Indeed, estimates of the percentage of all cancers caused by the single substance asbestos have varied from 1 to 18 per cent (1).

The radiologist's contribution in sorting out the asbestos related problems can be greatly enhanced by 1.) a strict adherence to accepted terminology and definitions, 2.) a working knowledge and use of the International Labor Organization Radiologic Classification of the Pneumoconioses, 3.)an effort to assemble old films for comparison, 4.) a detailed acquaintance with exposure history and with past history and 5.) an acquaintance with relevant epidemiology.

Asbestos: Fibrous hydrated silicates:Serpentine (chrysotile): white, curly, filamentous (90% of industrial use) and amphiboles, mainly Crocidolite (blue) and Amosite (brown). Others, including Anthophyllite and Tremolite mainly contaminants of talc.

Occupations at risk: (Incomplete list)

Process	Products (more than 3,000)	Occupations (Ex)
1. Production		
Mining, Milling	Asbestos Fiber	Mining,Crushing Transport, etc.
2. Primary use		
Spray Insulation	Fiber mixed with oil	Insulators, Construction
3. Manufacturing		
Textile	Cloth,Belts,Padding	Spinning,Card.
Cement Products	Roofing,Pipes,Gutters	Blending,cut.
"Paper" products	Felt,Electrical paper	Paper makers
Friction material	Gaskets,Clutch,Brake	Mixers,Blenders
Insulation	Pipe,Boilers,Bulkhead	Slurry,Chemical workers

Process	Products	Occupations
4. Application		
New Construction	Boards,Tile,Siding	Carpenters, Laggers,Heat.
Repair,Demolition		
Shipbuilding	Insulation:Pipes,hull	Laggers,Pipe-coverers
"Repair, Refits	"	Direct & In-direct (all)
Automotive	Undercoating,brake,etc.	Service,body shop

Epidemiology:

a.) Disease is dose-related.Exposure may have been brief and massive or lower dose for many years.
b.) "Twenty-year rule": Regardless of severity of exposure, signs and symptoms today are never seen until 10 or 15 years later and usually not until 20 years later.
c.) Asbestos manifestations correlate best with years since first exposure rather than number of years exposed. Manifestations progress despite removal from exposure.

I. Asbestosis:

This term should be reserved for a pneumoconiosis consisting of a diffuse chronic pulmonary interstitial pneumonia and fibrosis due to respirable asbestos fibers. The term should not be used for other asbestos-related manifestations or disease (2).

Asbestosis is demonstrably dose related, requires a considerable dose and therefore is not seen with out-of-plant neighborhood exposure or in relatives.

Cigarette-smoking is not a co-factor; and COPD occurs in asbestos workers with the same frequency and severity as in controls matched for age, sex and smoking history (3).

Clinical Diagnosis is based on history of exposure at least 15 and more usually 20 years earlier and at least 4 of the following 6 signs and symptoms:

1.) X-Ray: Linear-irregular markings (usually of lower lung fields) of ILO severity 1/1 or greater
2.) Dyspnea of Fletcher grade 2 or greater
3.) End inspiratory crackles (cellophane or velcro rales in 2 or more areas)
4.) Definite finger clubbing (usually late manifestation)
5.) Vital Capacity less than 80% of predicted
6.) Single breath diffusing capacity less than 80% of pred.

A clinical diagnosis is made when 4 of 6 of the above manifestations are present. "Permanent and total disability"

usually is associated with at least 2/2 radiographic profusion, an FVC of less than 50% and D_LCO of less than 40%. Lung biopsy should be required only in persons with a good exposure history but with atypical clinical or radiographic findings, or, on the contrary, when there are suggestive clinical findings but the exposure history is brief or unconvincing. The recently fashionable transbronchial biopsy may result in recovery of asbestos bodies but it is not a suitable technique for evaluating the presence or severity of interstitial pneumonia and fibrosis.

II. Parietal Pleural Hyaline Plaques:

The most recent I.L.O. classification distinguishes between diffuse pleural thickening and plaques or circumscribed pleural lesions (4). The latter, hyaline plaques, are the most common and also the most benign asbestos-related disorders.

Neither the early descriptions of the pathology of asbestosis nor the first clinical surveys in the 1930's called attention to parietal pleural lesions because at that time exposure was often severe and workers became disabled or died of asbestosis before plaques could form (5). Radiographic pleural calcifications of occupational origin were first noted in talc miners probably due to tremolite. Jacob and Bohlig (6) in their study of Dresden asbestos workers first mentioned calcifications as one of several roentgenographic features of asbestos exposure, and soon thereafter pleural plaques became established as markers of both occupational and non-occupational endemic asbestos exposure. More recent epidemiologic studies have led to the following generalizations concerning pleural plaques: (1) They are seen only following exposure to fibrous silicates. (2) Unlike asbestosis, they may result from casual, peripheral or neighborhood exposure. (3) They are rarely seen in less than 15 to 20 years after initial exposure. (4) Among asbestos workers first exposed 30 to 40 years ago, the prevalence may be as high as 35 to 60 per cent (5). (5) Only a small percentage of those present are seen radiographically. (6) They are an almost invariable finding at autopsy. (7) Plaques and calcifications occur on the parietal pleura and rarely cause pleural symphysis. The mechanism of their development is not known but probably they are due to mechanical rather than chemical irritation. (8) In the absence of asbestosis they are not harmful in that 1.) they are not associated with functional loss, 2.) they are not associated with symptoms and 3.) they do not predispose to other asbestos-related disease (CA, Fibrosis, Mesothelioma) compared to similarly exposed persons who do not have plaques. (9) Asbestos-related plaques and calcifications must be recognized and

propery labeled as such by radiologists. This usually requires an occupational history and review of earlier films. Proper recognition of such lesions will avoid much anxiety, unnecessary surgery and unwarranted litigation.

III. Benign Asbestos Pleural Effusion:

Benign asbestos effusion was not described as an entity until 1971 and the several series of cases reported since then have been summarized recently (7). Our definition is based on 4 criteria: 1) direct or indirect exposure to asbestos, 2) an effusion confirmed by a transient pleural change in serial chest films, by thoracentesis or surgery, 3) lack of evidence for any other disease related to pleural effusions, and 4) no malignant tumor detected within 3 years after the effusion.

COLLECTED FLUID

Such effusions may result from slight or peripheral exposure much as all the other pleural manifestations. However, the 20-year-rule does not apply in that they may occur earlier than the other asbestos-related disorders. Indeed benign effusion is the most common asbestos-related manifestation during the first 20 years after initial exposure (7).

A recent epidemiologic study of 1,135 exposed workers followed for 3 to 40 years revealed 35 cases (5.2%) of otherwise unexplained effusions compared to none in an unexposed control group of 717 persons observed over a similar period (7). Effusions generally were small, one-fourth recurred and two-thirds were asymptomatic. Incidence was related to exposure: there were 9.2 effusions per 1,000 person-years for individuals working directly with asbestos, 3.9 effusions for those working in proximity to asbestos use by others, and 0.7 for administrative and office personnel who worked in shipyards or asbestos plants but probably had insignificant exposure.

IV. Mesothelioma:

Malignant mesothelial tumors have been described for 100 years and, notwithstanding recent interest, they have remained rare with incidences from 0.8 to 2.1 per million per year. Approximately 20 per cent are primarily abdominal. A relationship to asbestos exposure was first described by Wagner et al (8) in 1960 and this is now generally accepted. The actual percentage of the some 1,000 mesotheliomas reported in this country each year that result from asbestos exposure is not clear. Large cancer centers have reported infrequent asbestos exposure, 13 to 27%, but occupational histories before 1960 clearly were deficient. Other series reporting 100% exposure have come from near shipyards

or asbestos plants. Other types of exposure include long fiber Zeolites used for stucco and buildings. Sugar cane work, radiation and thorotrast cholangiography, as well as tuberculous empyema all have been associated with mesothelioma. McDonald and McDonald (9) have well summarized present knowledge by saying that the proportion of mesotheliomas due to occupational exposure is increasing but is certainly less than 100%, unlikely less than 50%, and probably accounts for two-thirds of all cases. They and others also have shown a gradient in mesothelioma-inducing potential from crocidolite down to chrysotile and anthophyllite.

Mesothelioma, much like the other pleural manifestations of asbestos exposure, may result from brief, slight or peripheral exposure - a finding of great concern. The latent period from initial exposure is usually very long, often 30 years or more and very rarely less than 20 years. Among asbestos workers the death rate from mesothelioma appears to be proportional to the third or fourth power of time from first exposure and is unrelated to smoking habits. The histologic diagnosis has remained difficult because the tumor arises from pleuripotential mesothelial cells and in consequence may present with widely varying histologic features. Three types are generally recognized: mesenchymal or sarcomatous, epithelial or tubolopapillary and mixed; additionally some tumors are poorly differentiated. Mesotheliomas are often diagnosed because of their typical gross appearance with spread along serosal surfaces, encasing of the lung by a continuous layer of tumor and failure to demonstrate a primary lesion in the lung. However, the same gross appearance may result from bronchogenic or metastatic tumors. These histologic problems explain why thoracenthesis or needle biopsy rarely proves diagnostic. In most of our cases a small open thoracotomy was required but thoracoscopy may be a less invasive alternative.

Mesotheliomas have not responded to any form of therapy. Pleuro-pneumonectomy or "debulking" entail high mortality and no improvement. Concerning recent chemotherapy, at Sloan-Kettering there were only 3 responses among 111 trials; and mean survival of treated and untreated cases has been the same: 9.1 vs. 9.6 months (10) and in our series it was 15.8 months for untreated vs. 12.4 months for chemotherapy.

V. Bronchogenic Carcinoma:

Case reports have accumulated since 1934 but it was not until 1955 that Doll (11) presented epidemiologic evidence

of an increased prevalence. Although acceptance came slowly it is now concluded beyond any reasonable doubt that commercial asbestos is a cause of human lung cancer, though other agents such as tobacco, may enhance its effect. The relative risk (RR), a number that represents the observed divided by the expected number od deaths from a given disease, tabulated by Backlake (2) and by McDonald (12) for 18 major cohort studies has varied from 1 (no increased risk) to 17, with the lowest values, 1 to 5, in miners and millers and the highest, 8 to 17, in pipecoverers and insulation workers. The interaction of smoking to asbestos exposure as risk factors is most important in clinical practice. Lung cancer develops only very rarely in nonsmoking asbestos workers whereas the risk from smoking and asbestos is more than additive, and more likely multiplicative. A relationship to intensity of exposure has been difficult to demonstrate because dust concentrations were rarely measured until 10 or 15 years ago. However the best available data suggest that the dose response relationship is essentially linear, but steeper for asbestos manufacture than for mining. The increased risk appears at about 20 years after first exposure and reaches a peak at 30 years, although obviously there is an interaction between age and lung cancer mortality.

The question of causal relationship or attributability is not a great problem with asbestosis or hyaline plaques which are specific for asbestos nor even for mesothelioma when there has been significant exposure. With bronchogenic carcinoma the situation is quite different in that only 1-2% of all cases can be related to asbestos exposure. The probability that a given case of lung cancer is due to asbestos exposure is based primarily on two factors: the intensity of exposure and the time since first exposure. For example, data of Enterline et al (13) indicate no increased risk (RR = 1.2) for asbestos workers who were exposed to less than 10 million particles per cubic foot (mppcf) and who were first exposed less than 20 years ago, whereas RR rose to 4.7 for those who were first exposed more than 30 years ago to more than 10 mppcf. The time since first exposure can be identified whereas in clinical practice the intensity of exposure almost invariably must be inferred from clinical and histologic evidence.

REFERENCES

1. Enterline PE:Proportion of cancer due to exposure to asbestos, Branbury Report No. 9, Quantification of Occupational Cancer, Peto R and Schneiderman M (eds.), Cold Spring Harbor Laboratory,Publishers, 1981, pp 19-36
2. Becklake MR: State of the Art:Asbestos-related diseases of the lung and other organs:their epidemiology and implications for clinical practice. Am Rev Resp Dis 114:187,1976.
3. Murphy RLH Jr, Ferris BG Jr, Burgess WA et al:Effects of low concentrations of asbestos:Clinical, environmental, radiologic and epidemiologic observations in shipyard pipecoverers and controls. New Eng J Med 285:1271, 1971.
4. International Labour Office:Guidelines for the use of the ILO International Classification of Radiographs of Pneumoconioses. Revised Ed. 1980. International Labour Office Occupational Safety and Health Series No. 22 (Rev. 80), I.L.O. , Geneva, Switzerland (Also in Med Radiogr & Photogr 57:2, 1981).
5. Selikoff IJ: The occurrence of pleural calcifications among asbestos insulation workers. Ann NY Acad Sci 132:351, 1965.
6. Jacob G and Bohlig H. Roentgenological complications in pulmonary asbestosis. Fortschr RÖentgenstr 83:515, 1955.
7. Epler GR, McLoud TC and Gaensler EA: Prevalence and incidence of benign asbestos pleural effusion in a working population. JAMA 247:617, 1982.
8. Wagner JC, Sleggs CA and Marchand P: Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. Br J Industr Med 17:260, 1960.
9. McDonald JC and McDonald AD: Epidemiology of mesothelioma of estimated incidence. Preventive Med 6:426, 1977.
10. Lewis RJ, Sisler GE and MacKenzie JW: Diffuse, mixed malignant pleural mesothelioma. Am Thor Surg 31:53, 1981.
11. Doll R: Mortality from lung cancer in asbestos workers. Br J Industr Med 12:81, 1955.
12. McDonald JC: Asbestos and lung cancer:Has the case been proven? Chest 78:Number 2:Suppl 374, 1980.
13. Enterline PE, DeCoufle P and Henderson V: Respiratory cancer in relation to occupational exposures among retired asbestos workers. Br J Industr Med 30:162, 1973.

E. NICHOLAS SARGENT, M.D.

THE ILO 1980 CLASSIFICATION SYSTEM (FACTS AND FALLACIES)

TERMINOLOGY: The definition of pneumoconiosis is clouded and its casual use has resulted in confusion. Under the ILO Classification System it is defined as the tissue reactions to dust inhalation and dust was meant to be an aerosol of solid inorganic particulates. (Coal is organic!!) The term "pneumoconiosis" often brings to mind aspects related to compensation and litigation. The term "occupational" also serves to remind physicians of the significance of the disease in question in a particular patient group. "Environmental" lung disease has also been suggested. The best method of defining and classifying pneumoconiosis for medical purposes should rest on morbid anatomical changes and thus it will embrace a variety of lung disorders. However, the term pneumoconiosis is so well entrenched in its usage for the past century, that its elimination is not realistic. Parkes suggests that pneumoconiosis should be defined as a non-neoplastic reaction of the lungs to inhaled mineral or organic dusts and the resultant alteration in their structure (excluding asthma, bronchitis, and emphysema).

ILO 1980 CLASSIFICATION SYSTEM: In the clinical practice it is customary for physicians to describe the radiographic findings associated with the pneumoconioses in a non-quantitative narrative form. However, when the information is to be used epidemiologically (or to evaluate pulmonary disability) the reporting must be more quantitative. The 1980 classification system is the latest in a series of classifications with numerous modifications since 1930. It is designed to permit codification of all types of pneumoconioses including coal workers pneumoconiosis, silicosis and asbestosis. A codification of the technical quality of the film is also now part of the system. To enhance consistency in the application of the classification of the system the use of standard radiographs for comparison is required. These films as well as a detailed description of the classification system may be obtained from the American College of Radiology, 6900 Wisconsin Ave., Chevy Chase, Maryland, USA, 20015 or by writing to the International Labor Office in Washington, D. C., USA or Geneva, Switzerland.

SMALL OPACITIES: With respect to the pulmonary findings, the system divides the opacities into two categories: small and large with each defined in specific quantitative terms. An opacity, of any size or shape which is larger than 10 mm in diameter, must be classified as a "large" opacity and not as a small opacity. With respect to small opacities, two shapes are recognized (small rounded or small irregular). For each shape the opacity is graded into three categories depending on their diameter and width. In the new scheme two letters must be used to denote the predominant size and shape.

The size and shape of small rounded and small irregular opacities have epidemiologic and clinical significance. Small irregular opacities occur following asbestos dust exposure, and usually begin at the lung bases. Small rounded opacities following coal and/or silica dust exposure are found radiographically in the upper and middle zones of the lung before they are observed in the bases. (Small irregular opacities may occur with coal and/or silica dust exposure but they are relatively

uncommon and are usually associated in conjuction with more rounded opacities.) Small rounded opacities by themselves are not seen with asbestos dust exposure.

For coal workers the dust content of the lung correlates well with the number of small rounded opacities of the p and q variety but not well with the small rounded opacities of the r variety. The p opacities tend to have pulmonary function abnormalities related to lower gas transfer of carbon monoxide, i.e. a decrement in gas transfer or alveolar capillary diffusion abnormalities. The irregular opacities of asbestos dust exposure do not correlate well with the quantity of dust found in the lung. However, abnormal pulmonary functions are more closely related to irregular opacities than they are to rounded opacities. The u opacities that occur with asbestosis have a much worse prognosis and have the most abnormal types of pulmonary function.

PROFUSION: This term refers to the concentration or number of small opacities per unit area observed within the lung fields. Twelve categories of profusion are recognized. The quanity of dust in the lungs (incinerated) correlates well with coal and silica in relation to the profusion but the quantity of asbestos dust in the lungs or asbestos fibers does not correlate with the profusion of irregular opacities. However, of all the characteristics of small opacities requiring codification, profusion is the most important for it is the best indicator of the seriousness of any disease that may be present.

In advanced cases of pneumoconiosis there is usually no question radiographically regarding the diagnosis of the disease. However, when only small opacities are present which are few in number (i.e. low profusion) interpretation can be quite difficult. This is because small opacities can occur in a wide variety of situations both normal and abnormal, as well as in pneumoconiosis. For example, as individuals become older, periodic respiratory infections often result in pulmonary fibrotic changes that appear radiographically as small irregular opacities. These changes are particularly prevalent in cigarette smokers. Individuals who suffer from chronic congestive heart disease, in time develop fibrotic findings in the lung that may be confused with the early stages of pneumoconiosis. Many pathological conditions quite unrelated to dust (e.g. other interstitial diseases) at various times in there courses, manifest themselves radiographically by the appearance of small opacities. Thus it is evident that the radiographic findings in early pneumoconiosis are not so characteristic that their interpretation can be counted on to be unequivocal.

Although this twelve point scale of profusion implies a high degree of quantification for the recording of profusion levels, it must be pointed out that the definition of major profusion categories on which the scale is based, is rather non-specific. Hence, when the profusion levels of series of radiographs are evaluated by a group of physicians, substantial differences of opinion can be expressed. (Physicians generally have the greatest difficulty in separating a series of radiographs into normals and abnormals when profusion levels are nearer the divisions of 0/1 and 1/0.) A given physician will therefore exhibit some inconsistency in his or her codification of profusion. Because of the difficulties that exist in the interpretation of chest radiographs, it is perhaps not surprising that

inconsistencies arise when a number of physicians independtly evaluate a series of radiographs or when the same individual physician evaluates the series a number of times. However, it should be pointed out that such inconsistencies are unavoidable and indeed characteristic not only of radiographic procedures but all clinical testing (including history taking, physical examinations and physiological tests) due to uncertainties inherent in all methodologies in which human judgement is a factor.

COMPENSATION: For coal workers using the ILO Classification System the Department of Labor, Employment Standards Administration uses a profusion of 1/0 to indicate disability and casusality with ten or more years of coal mining. Thus with ten years or more of coal mining a 1/0 or better gives a miner a continuing disablity pension plus the survivors benefits even though he may or may not have been disabled. Great difficulities arise in the low levels of profusion (where one reader might call it a 1/0 and another reader might call it 0/1 for awarding compensation.

An additional characteristic of small opacities that must be codified in the ILO 1980 Classification System is the extent of the pulmonary disease. To record this parameter, the lung fields are divided into six zones, three on each side corresponding to the upper, middle and lower thirds of the lung fields. The new classification system combines roundness and irregularity of opacities but does not differentiate as to their individual location. No distinction is made between rounded or irregular opacities or the location of the opacities relative to compensation or eligibility. A miner with asbestosis dust exposure with small irregular opacities at the bases, could qualify for benefits if this person worked in a coal mine (in spite of the fact that he may have no rounded opacities due to coal or silica in the upper lung zones).

LARGE OPACITIES: The large opacities which represent a lesion greater than 1 cm are divided into three categories depending on their greatest diameter or length. The finding portends a much more serious prognosis and is usually associated with abnormal pulmonary functions. Many of these patients will progress with abnormalities resulting in progressive massive fibrosis, marked emphysema, distortion and destruction of the lung with eventual death from cor pulmonale. Contrary to the ILO Classification, by definition according to the American Society of Pathologists, the lesion must be at least 2 cm in diameter to classify as progressive massive fibrosis. Difficulties arise in the classification system in that other disease processes (e.g. neoplasm, tuberculosis, etc.) may appear as "large opacities" on the x-ray. Without the clinical, the laboratory, and without previous films, large opacities of disease processes not due to pneumoconiosis in some cases may be classified erroneously.

PLEURAL ABNORMALITIES: The 1980 Classification System goes into great detail in reference to pleural thickening. Particularly it is known that asbestos dust inhalation (unlike coal and silica) results in abnormal pleural thickening with abnormal pleural shadows which are quite characteristic in many instances (i.e. plaques). Thus, the system requires that the site (chest wall, diaphragm and costophrenic angle) and the extent of the thickening be recorded separately. It is recognized that

this thickening can be observed either in profile (edge on) or en face (face on). However, for pleural thickening observed face on, its presence can be recorded but its thickness cannot be measured. When the pleural thickening is observed in profile its width can be measured and the ILO System recognizes three grades of width. Measurements are quite difficult in many instances because the same plaques are frequently seen with both a profile and en face image. Thus greater numbers of plaques may be recorded erroneously. The measurements are at best a very rough approximation of the width and extent of the actual plaque.

While circumscribed localized pleural thickening in the form of a plaque is quite characteristic, the ILO System also recognizes that "diffuse" pleural thickening does occur with asbestos dust exposure. Although it is non-specific, the length and width of the diffuse pleural thickening is also graded using the same measurements that one uses for circumscribed pleural plaques.

Standard films provided for the classification of the pleural changes need to be revised. The standard films are quite inadequate in that they do not differentiate between diffuse pleural thickening or pleural plaques in a clear definitive manner. Pleural plaques are more characteristic than diffuse pleural thickening but there is an in between "gray area" where one cannot be certain that we are dealing with confluent plaque formation rather than diffuse pleural thickening. Pathologically diffuse pleural thickening of asbestos dust exposure generally is quite extensive and involves the costophrenic angle. The characteristics of "diffuse" pleural thickening following asbestos dust exposure have not been clearly defined radiographically. One questions if it should be called "diffuse" (on some of the standard films) when its extent is only a few interspaces. Arbitrarily, I would not call it "diffuse" unless it also obliterates the costophrenic angle (plaques generally do not obliterate the costophrenic angle). A revision of the Standard Films (increasing the numbers) to run the whole range of pleural abnormalities is required. Indeed fat and muscle shadows are quite troublesome and examples of these shadows should be included in standard films.

It is also recognized that radiographically only 15% of the pleural plaques associated with asbestos dust exposure show recognizable calcification, (approximately 85% will show calcification pathologically). Calcification when it occurs on the diaphragm, the chest wall, the mediastinum and the pericardium is recorded separately. A localized calcification which is seen due to asbestos dust exposure should be classified as a plaque, even, if the surrounding soft tissue component is not seen on the single PA view. This needs further clarification in the 1980 Classification System.

While it is emphasized that the simple PA chest radiograph is the keystone of all radiographic examinations of the chest, there are occasions when a more extensive examination is necessary. For example, pleural thickening can be more confidently detected and diagnosed most easily when it is seen in profile. Therefore, when localized thickening exists, as it frequently occurs in the case of asbestosis, it is desirable to take oblique views and lateral views of the chest in an effort to bring the lesions into profile for more accurate measurements, (and to exclude other "simulating" lesions). Computed tomography is particularly valuable: in differentiating ill defined pleural shadows and demonstrating additional plaques and

calcifications, demonstrating the extent of circumferential diffuse pleural thickening, demonstrating emphysema, in substantiating interstitial disease and even honeycombing, and demonstrating the extent of suspected mesothelioma.

PULMONARY FUNCTION: The radiograph is currently the best available clinical method of diagnosing pneumoconiosis and it is the simplest and relatively accurate method of assessing the degree of involvement of the lung for coal workers pneumoconiosis and silicosis. However, its limitations may result in false negative findings early in the disease, and in some discrepancy in the degree of involvement. Good film technique with experienced readers are essential to keep the discrepancy to a minimum.

Radiographic methods primarily record anatomical structure and with very limited exceptions they do not record function. The information provided by the chest radiograph on the structures of the lung and on the pathological changes that may exist within them, is much greater than information on how the lungs may actually be functioning. The chest radiograph is better in evaluating pathological characteristics of a disease and it must be used with caution in assessing any disability that may have resulted from the disease. These limitations of the chest radiograph in the evaluation of pulmonary impairment are not difficult to understand. In pneumoconiosis, the disease particularly in its early stages, frequently is confined to small portions of the lungs (e.g. the upper lobes) and in such instances large segments are relatively unaffected. The unaffected regions are likely to function reasonably well and therefore, regardless of how extensive the involvement may be in the diseased zones, pulmonary function may not be significantly impaired. On the other hand, there are times when the disease from the beginning involves much of the lung parenchyma, with fibrotic changes that may not be impressive radiographically, but because they are so widespread they may impair function and cause disability relatively early.

In coal workers pneumoconiosis, the presence of small opacities correlates better with the amount of dust retained in the lungs than it does with lung function. In both simple coal workers pneumoconiosis and silicosis there is little in the way of a relationship between the radiographic features and impaired lung function. Only in Stages B and C conglomerate silicosis and coal workers pneumoconiosis does radiographic category relate to lung function. Pathological correlation is good and in most cases the roentgenographic characteristics are such that the findings are essentially diagnostic.

Relative to asbestosis the greater the past cumulative dust exposure, the higher the probability of progressive pulmonary fibrosis. The progression of small, irregular opacities depends upon average and cumulative asbestos dust exposure. Thus the patient should be advised that based on this possibility, even small increments in cumulative dust exposure will increase the risk for future progression of pulmonary fibrosis. The progression of abnormalities, (i.e. small irregular opacities) correlates impressively with progressive pulmonary function decline.

On the other hand, progression of pleural abnormalities is primarily dependent on time, and is not likely to be influenced by modest additional average or cumulative exposure. Unlike the lung changes, progression of

pleural thickening and pleural plaques is related to length of exposure and time since first exposure, but not to average or cumulative dust dose. Thus, the findings are consistent with the common clinical and radiologic observations of long latency of pleural effects (particularly calcification), the frequent finding of extensive pleural abnormalities in the absence of parenchymal disease, and the correlation of progressive parenchymal fibrosis (asbestosis) with heavy dust exposure. Diffuse pleural thickening is more likely to cause abnormal pulmonary function. Pleural plaques unless very extensive do not cause pulmonary function decrements. The patient might still be advised to continue in his occupation without any change if no other jobs were available.

The physiological limitations of the chest radiograph should in no way cause a lowering in one's mind of its value (either from a clinical or public health standpoint), in the evaluation of persons suffering from pneumoconiosis. Its objectivity in reliably assessing the pathological anatomy, short of a tissue specimen is unequalled.

REFERENCES

1. Becklake MR, Fournier-Massey G, McDonald JC, et al: Lung function in relation to chest radiographic changes in Quebec asbestos workers. 1. Methods, Results and Conclusions. Bull. Physio-Path Resp. 6:637-659, 1970.
2. Fitzgerald, Carrington CB, Gaensler EA: Environmental lung disease. Med Clin of North America 57:593-622, 1973.
3. Lyons JP, Ryder RC, Campbell H, et al: Significance of irregular opacities in the radiology of coal workers pneumoconiosis. Br J Ind Med 31:36-44, 1974.
4. Morgan RH: Decision processes and observer error in the diagnosis of pneumoconiosis by chest roentgenography. Am J Roentgen 117:757-764, 1973.
5. Parkes WR: Occupational Lung Disorders (2nd ed) Pub. Butterworth & Co. London, 1982.
6. Public Health Service, Department of Health Education and Welfare: Specifications for federal examinations of underground coal mines. Federal Register 43:33713-33720, 1978.
7. Sargent EN, Morgan WCK: "Coal Workers' Pneumoconiosis" (chapter 15) and "Silicosis" (Chapter 16) in Induced Disease, Preger L (ed) Pub Grune & Stratton, N.Y.,N.Y., 1980.
8. Sargent EN: (ed) Technique for chest radiography for pneumoconiosis. Pub. American College of Radiology, Chicago, Ill., 1982.
9. VanOrdstrand HS: Pneumoconioses and their masqueraders. J Occupat Med 19:747-753, 1977.
10. Weill H: Basis for clinical decision making. Chest 78:382-383, August Supplement, 1980.

ASBESTOS RELATED DISEASE - PLEURAL ABNORMALITIES

E. Nicholas Sargent, M.D.

The pleural abnormalities following asbestos dust exposure include: (1) plaques (circumscribed or localized). (2) diffuse pleural thickening (circumferential or interlobar), (3) effusion, (4) mesothelioma. Pleural changes are more frequently found without associated pulmonary parenchymal abnormalities.

The normal pleural shadow (e.g. pleural stripe, accompanying shadow) has often been called pleural thickening. It consists mainly of internal intercostal muscle bundles and some fat. The parietal pleura is only 10 to 60 micra in thickness, as is the visceral pleura, and thus the normal pleura contributes very little to the shadow. Normally the shadow tapers from the apex and disappears usually below the fourth or fifth rib. Diffuse pleural thickening often follows a gravitational event in the pleural space (e.g. infection or trauma)and usually obliterates the costophrenic angle(casting a shadow which is wider inferiorly and tapers towards the apex).

The characteristic pleural plaque following asbestos dust exposure does not involve the pleural space or the mesothelial layer of the pleura and is subjacent to the parietal pleura. On the PA projection it is found along the costal reflection of the pleura, along the mid third of the thoracic wall. It does not involve the apical regions or the costophrenic angles until it becomes very extensive. Plaques also occur in the areas of reflection of the diaphragmatic parietal pleura (and the mediastinal pleura as well). Parietal pleural plaques occur over bony prominences and tendonous surfaces (thus involving primarily the pleura overlying the central tendon of the diaphragm). Pathologically plaques show great variation in size and shape. They are rarely thicker than 1 centimeter in cross section, until they are very far advanced.

Radiographically in the PA projection the plaques are described as shadows which are seen in "profile" with a sharply defined edge; or en face with ill defined margins. Frequently many of the plaques are projected partially in profile and partially en face. Thus the x-ray images are the result of the size, shape, and location of the plaque, but particularly the shadow will vary with the direction of the x-ray beam and the thickness of the tissue. The use of oblique projections is particularly important not only to confirm suspected questionable shadows on a PA projection but to discover additional areas of plaque formation that are not obvious on the PA projection. By rotating the patient in the oblique projection, plaques which are seen en face can be brought out in profile, and the true cross-sectional width more accurately measured.

Frequently difficulties arise as to whether certain shadows seen on the radiograph may be due to causes other than plaques. On the average it takes 20 or more years for a plaque to form and be radiographically identifiable. It usually takes three to five years for a plaque to change its size or shape. The use of comparison films for progression is helpful in the differential diagnosis. Furthermore, one must be familiar with shadows that simulate plaques.

Interdigitations of the external abdominal oblique and serratus anterior muscle slips cause shadows overlying the ribs. When they are bilateral, symmetrical and equal they are readily identifiable. They tend to disappear on the oblique view. Not infrequently, single muscle slips can cause difficulties. Comparison with old films or future films for

progress is of value as one cannot always be certain that an early plaque is beginning in the area of a suspected muscle shadow. Subpleural fat casts problematical shadows. Computed tomography is helpful. Rib injuries with overlying pleural thickening, iatrogenic and postoperative pleural changes can cause localized pleural thickening. Subpleural metastatic disease, (e.g. myeloma, breast metastases, thymomas, and melanomas) is a consideration; as well as uncommonly metastatic Hodgkin's histiocytic lymphoma and other lymphomas causing localized pleural thickening (can be differentiated clinically).

Characteristically calcifications in pleural plaques occur in the center of the plaque. However, calcification is seen only in 15% of asbestos related plaques radiographically, whereas it is found in 85% of the cases pathologically. Calcifications secondary to tuberculosis empyema, hemothorax, and other etiologies which are events that occur in the pleural space, resulting in calcifications which are more medially disposed and are further away from the internal surfaces of the ribs. Tuberculosis tends to be unilateral but can be bilateral. Finding other evidence of old Tbc is helpful. With rib injuries present on the film this leads one more towards the diagnosis of the calcification being due to trauma, particularly if it is unilateral. Other rare causes of calcifications which may simulate plaques are irradiation, mineral oil aspiration, pulmonary infarction adjacent to the pleura, and even calcification of the pleura secondary to scleroderma has been described. Although calcifications in plaques are more commonly bilateral they can be unilateral and a careful history as well as seeking evidence of other disease processes helps in the differential diagnosis.

The calcifications that are seen en face radiographically are varied and can be nodular, linear, circumferential, irregular, pseudovascular, and amorphous. Occasionally when they are very extensive they appear to have a "holly leaf" or "candle wax" appearance. The use of oblique views for defining their extent and width is of great value (also in differentiating calcifications in cartilages and in granulomas). On the PA projection calcifications occur not only in the region of the leaves of the diaphragm and along the thoracic wall, but they can occasionally be found in the mediastinum and even in the pericardium. Oblique views help to show the extent of the calcification along the mediastinal area and the pericardial region. Computed tomography is a much more sensitive method of picking up plaques and calcifications in all involved areas.

The most common finding in surveys of large numbers of patients exposed to asbestos dust is a noncalcified parietal pleural plaque located along the costopleural margin or on the surface of the diaphragm. This does not mean that less frequently one can see associated diffuse pleural thickening with or without plaques; interstitial disease with or without plaques; or pleural effusions with or without plaques. Any of these events can overlap with one another. All findings appear to be dose and time related. Generally, a light exposure results in an asymptomatic patient showing isolated plaques after a long latent period. With a heavy exposure one tends to find more interstitial disease in a shorter time frame. Occasionally, however, with very heavy exposure one can find extensive diffuse plaques with calcification, with a short latent period.

Diffuse pleural thickening is non-specific but when it occurs following asbestos dust exposure, it tends to involve more of the visceral pleura rather than the parietal pleura. Radiographically one cannot differentiate

visceral or parietal pleural thickening except when one finds thickening in the interlobar fissues. Diffuse pleural thickening usually obliterates the costophrenic angle. Computed tomography is particularly helpful in outlining circumferential pleural thickening of the diffuse type which might not be recognized on the plain film. Interlobar visceral pleural thickening does occur less commonly and even interlobar calcified plaque formation can occur.

A rare type of "hyalinosis progressiva maligna" or "hyalinosis complicata" has been described. This may or may not be accompanied by pleural effusions. Diffuse pleural thickening usually occurs unilaterally, but spreads contralaterally over a short period of time. Pathologically, both visceral and parietal pleura are thickened. There is a diffuse exudative pleural "rind" and these patients have been operated on with no evidence of mesothelioma formation. They have a very poor prognosis and tend to die of infections and respiratory failure.

Benign pleural effusions are now recognized with increasing frequency (occurring from 8 to 10 years on the average after the initial exposure). They may be clear or bloody and can be unilateral or bilateral occurring with or without plaque formation. More frequently they result in diffuse pleural thickening but often they disappear with no evidence of any residual pleural changes. However, one must exclude a malignant mesothelioma by careful clinical evaluation and follow-up of the patients. Pericardial effusions and pericardial calcifications have been described.

Any patient with a history of asbestos dust exposure pleural effusion should be considered as having a mesothelioma until proven otherwise. Characteristically the pleural effusions usually do not cause a shift of the heart or mediastinum. This is usually due to the encompassing pleural tissue "rind". Computed tomography is helpful in outlining the extent of the mesothelioma particularly when used for prognosis during therapy. The incidence of mesothelioma is not related to cigarette smoking as is bronchogenic carcinoma. Mesotheliomas do metastasize both ipsilaterally and contralaterally (usually before the patient dies there are distant metastases). Clubbing is more common with mesotheliomas but mesotheliomas are much more infrequent tumors than are bronchogenic carcinomas.

Pleural plaques do not degenerate into mesotheliomas. A pleural plaque merely means that the patient has been exposed to asbestos dust and is a "marker" for asbestos dust exposure. Bronchogenic carcinomas must always be looked for on the radiograph for any patient in which plaques are found (since the plaques mean asbestos dust exposure). Cigarette smoking and asbestos dust exposure are synergistic in causing bronchogenic carcinoma.

A change which is being recognized with increasing frequency is infolding of the lung associated with plaque formation or other pleural thickening. Infolding of the lung near areas of abnormal pleura is associated with segmental and subsegmental atelectasis resulting in "pseudotumor" formation (which must be differentiated from bronchogenic carcinoma). Characteristically, a relatively wide line extending towards the costopleural margin from the shadow of the "pseudotumor" should make one suspect a possible non-neoplastic situation. Computed tomography, needle aspiration biopsy and comparison with previous films are helpful

in the differential diagnosis. The finding is non-specific. It has also been found following pneumothorax, pleural fluid with tuberculosis, and hemorrhage secondary to trauma. It should be considered in patients known to have been exposed to asbestos dust and careful study to prevent an unnecessary thoracotomy is required.

Family exposure does occur and the finding of plaques in a young adult particularly when calcified, should alert one to the fact that the occupational history should include not only the immediate family, but other relatives and also all areas in which the patient has lived. It should be emphasized that it does not take a heavy expsure to cause a pleural plaque. Exposures of only a few months have been described resulting in plaques after a very long latent period (as much as 25-30 years after a light exposure).

Bilateral pleural plaques will be discovered with increasing frequency when they are particularly looked for on every radiograph. They merely mean that the patient has had asbestos dust exposure and should be correlated particularly with a lifetime occupational and family history. The patients are usually asymptomatic and must be cautioned not to smoke. Lifetime radiographic surveillance for any progressive changes as well as the possibility of a future occurrence of a pleural or parenchymal neoplasm, is indicated.

References

1. Blesovsky A: The folded lung. Brit J Dis Chest 60:19-22, 1966.

2. Fletcher DE, Edge JR: The early radiological changes in pulmonary and pleural asbestosis. Clin Radiol 21:355-365, 1970.

3. Gaensler EA, Kaplan AJ: Asbestos pleural effusion. Ann Int Med 74:178-191, 1971.

4. Kreel L: "Asbestosis and mesothelioma on computed tomography". (Chapter 13) in Induced Disease. L. Preger (ed), Grune & Stratton, N.Y., 231-253, 1980.

5. Mattson SB: Monosymptomatic exudative pleurisy in persons exposed to asbestos dust. Scand J Resp Dis 56:263-272, 1975.

6. Navratil M, Dobias J: Development of pleural hyalinosis in long term studies of persons exposed to asbestos dust. Environmental Research 6:455-472, 1973.

7. Sargent EN, Felton J, Barnes LT: Calcified interlobar pleural plaques, following asbestos dust inhalation. Radiol (3) 140:634, Sept, 1981.

8. Sargent EN, Gordonson J, Jacobson G, et al: Bilateral pleural thickening: A manifestation of asbestos dust exposure. Am J Roentgen 131:579-585, 1978.

9. Sargent EN, Jacobson G, Gordonson JS: Pleural plaques: A Signpost of asbestos dust inhalation. Semin Roentgen 12:287-297, 1977.

10. Sargent EN, Jacobson G, Wilkinson E: Diaphragmatic pleural calcification following short occupational exposure to asbestos. Amer J Roentgen (3) 115:473-478, July, 1972.