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SILICOSIS AND A FEW OF THE OTHER PNEUMOCONIOSES: OBSERVATIONS ON CERTAIN ASPECTS OF THE PROBLEM, WITH EMPHASIS ON THE ROLE OF THE RADIOLOGIST*

CALDWELL LECTURE, 1957

By EUGENE P. PENDERGRASS, M.D.
PHILADELPHIA, PENNSYLVANIA

Eugene Wilson Caldwell

This is the occasion of the Caldwell Lecture and before I begin my address, I want to express to our president, Dr. Wendell G. Scott and to the members of the Program Committee my sincere appreciation for the honor that they have conferred on me in asking me to present this lecture before the American Roentgen Ray Society. I did not have the privilege of knowing Dr. Caldwell, but those who did have testified on many occasions of his ideals and achievements. This Society has been most fortunate in having many who have contributed much toward the advancement of medicine in general and of radiology in particular. Our heritage is one of which we justly can be proud. I have the feeling that Dr. James T. Case and others who were responsible for the inauguration of the Caldwell Lecture, not only wished to honor Caldwell, but in addition hoped that this special event might become a symbol by which we could honor all of our members and those in collateral disciplines of investigation and in industry who during their lifetime added their bit to our specialty.

As evidence of this premise, the senior Dr. William A. Evans[18] wrote the following: "The early second period of pioneering in American roentgenology was graced by several who by virtue of their noble character and unusual professional attainments have been canonized by those who continued in the work for which these made the supreme sacrifice. One who will change the whole direction of his life current to follow an ideal conceived after comparative success in a given endeavor, must possess unusual attributes. Such was Eugene W. Caldwell of New York. Already a master mind in the field of electrical engineering and physics, upon becoming interested in Röntgen's work, he early appreciated its importance both medical and otherwise. The better to serve its advancement in the field of human relief, Caldwell qualified himself as a physician. Thus trained in two highly technical and scientific spheres, he began a career which added greatly to the efficiency and completeness of roentgen equipment and accessories and its proper adaptation for demonstrating various medical and surgical lesions."

Dr. Evans goes on and describes the work of others and concludes with the following statement: "With Leonard, these three, Caldwell, Dodd, and Kassa-

* From the Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania. Presented at the Fifty-eighth Annual Meeting of the American Roentgen Ray Society, Washington, D. C., October 1–4, 1957.

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bian, succumbed to roentgen injuries incident to their constant studies. The American Roentgen Ray Society, conscious of their beneficent influence, honored Leonard by the establishment of the 'Leonard Prize,' an award granted at intervals for meritorious studies in some phase of roentgenology and commemorates the influence of Caldwell by the 'Caldwell Lecture'. . . . "

THE subject of my address is concerned with certain aspects of silicosis and some of the other pneumoconioses. Many of you know of my interest in this problem. This is the fourth time that I have used this subject in a named lectureship—the Preston M. Hickey Memorial Lecture[65] before the Wayne County Medical Society, April 6, 1942; the Russell D. Carman Memorial Lecture[66,67] before the Minnesota State Medical Association, June 12, 1950; and the Ross Golden Lecture[69] in New York, March 21, 1955, before the New York Roentgen Society.

My own experience on the effects of dust inhalation on the lungs dates back to 1919, when following World War I, Dr. Henry K. Pancoast re-embarked on the studies which he, Dr. T. Grier Miller and Dr. H. R. M. Landis began in 1916.[52] Together, we carried out many studies over the succeeding years. Dr. Pancoast was the first radiologist in this country and one of the few in the world who early made a real effort to learn something about pneumoconiosis. In so doing, he developed many friends outside of radiology in the fields of public health, pathology, chemistry, physics, engineering, law, and industry. For me, meeting and getting to know others interested in dust hazards has been a most rewarding experience.

Anyone who takes the time to read a little about the pneumoconioses cannot help but be impressed with certain facets of the total problem. Some of these are of real importance to the radiologist. Most of the diagnoses begin with an interpretation of the roentgenograms. A radiologist with a broad background of diagnostic training and experience can be a tremendous asset in the care that these dust-affected workers receive because there are many diseases that produce shadows in the lungs that simulate silicosis and asbestosis. There is no part of radiology in which there is a greater opportunity for contributing real service to the health of the community and of the nation. The retrospective studies of chest roentgenograms made on industrial workers provide a most satisfying experience, not only in studying the natural history of the pneumoconioses, but other pulmonary and cardiac diseases.

Over the years I have talked with young men who have trained with us in an effort to stimulate interest in the field of the pneumoconioses, and only a few have responded. For the most part, radiologists have been satisfied with a casual acquaintance with the subject. The information that these men are qualified to render is oftentimes inadequate. Because of such indifference, internists, chest physicians, chest surgeons, pathologists and others are now rendering interpretation of roentgenograms in many places. We, as radiologists, have a very definite obligation in improving this situation.

GENERAL ROENTGENOLOGIC CONSIDERATIONS

The proper roentgen interpretation of pneumoconiosis is dependent on; (1) a knowledge of the anatomy of the chest and the physiologic problems associated with its anatomic constituents; (2) a thorough familiarity with normal fluoroscopic and roentgenographic appearances and their permissible variations; (3) an understanding of the histology of the lungs and especially the lymphatic system; and (4) a clear perception of the pathology of pneumoconiosis and of all conditions which may simulate its roentgenographic appearances.

In many instances, the radiologist is expected to render an opinion from a single roentgenogram with or without an adequate history. Under such circumstances, one has to do the best he can. A report

should include the conditions that should be considered in arriving at the final or presumptive diagnosis.

Although I have had the opportunity of studying roentgenograms of collective surveys done by the Public Health Service, or some other like agency, my experience has been concerned largely with three groups: (I) *The individual who arrives in a general hospital* usually with a complaint relating to some system or organ in the body other than the lung; (II) *the individual who is seeking compensation* for pneumoconiosis; and, (III) *workers in an industry where there is a known or unknown dust hazard* in certain of its operations.

I and II. The roentgen studies for the *general hospital patient* and the *individual who is seeking compensation* can be considered together, because the studies are essentially the same. These include a fluoroscopic and roentgenographic study in addition to all of the aforementioned history review, physical and laboratory examinations. It has been my practice to take a supplementary history myself, making a real effort to learn everything about the individual's total environment.

The fluoroscopic and roentgenographic studies should be planned to obtain good records of any lesions, and to demonstrate, if possible, any roentgen evidence of disability. In pneumoconiosis, one of the problems of disability has to do with pulmonary ventilation.

Ventilation is a cyclic process of inspiration and expiration in which alternately fresh air enters the respiratory tract and an equal amount of pulmonary gas is exhaled.[4] An adequate ventilation should be accomplished without an undue expenditure of energy. It is important to define several terms[15] in order to avoid confusion:

1. Tidal volume (depth of breathing) is the volume of gas inspired or expired during each respiratory cycle.
2. Residual volume is the volume of gas remaining in the lung at the end of a maximal expiration.
3. Total lung capacity is the amount of gas contained in the lung at the end of a maximal inspiration.
4. Vital capacity is the maximal amount of gas that can be expelled from the lungs by forceful effort following a maximal inspiration.

There are many anatomic abnormalities which reduce the vital capacity and which can be demonstrated in roentgenograms of the chest.[2] These pertain to: anomalies in development of vertebrae or ribs,[13] scoliosis, deformities of the thoracic cage due to fractures, thickened pleura, and space-raking lesions within the chest.[4] Lesions affecting the position and mobility of the diaphragm may reduce seriously the vital capacity.[4] Changes in the interstitial tissue caused by edema, fibrosis, or granulomas are associated sometimes with reduction in total lung volume. Roentgenograms should be made in inspiration and in expiration without changing the position of the tube. Roentgenograms in each lateral view should also be obtained.

The patient's chest should be studied fluoroscopically in the erect, horizontal, prone and supine positions with horizontal and vertical beams, and in each decubitus position during quiet and forced respiration, and after coughing and sniffing. Fluoroscopy during the Müller (sniffing against a closed glottis) and the Valsalva maneuvers (straining against a closed glottis) is informative. This gives one the opportunity to determine the movements of the thoracic cage; the position of the mediastinal structures in all projections and during all phases of respiration; the pulsation of the heart and great vessels; the presence or absence of disturbed ventilation. It is well known that obstructive emphysema sometimes is seen best during fluoroscopy.[19]

While the fluoroscopic examination during forced respiration cannot provide an absolute figure of the dynamic reserve of the lungs, one can get a good estimate of the movement of air by observing the movement of the diaphragm and thoracic cage.[4] If fluoroscopy is combined with vital

capacity spirometry, the radiologist will improve his clinical judgment.[4] If the patient performs a maximal breathing capacity test, and there is limitation of rib and diaphragmatic excursion, one will be able to obtain an explanation for the reduced function.[4] The upper respiratory tract and pharynx should be included in the fluoroscopic study.

The number of roentgenograms and the technique to be used should be decided after fluoroscopy. A minimum study should include roentgenograms in inspiration and expiration; in each oblique view (5 to 15 degrees); in each lateral view; and a posteroanterior roentgenogram with a Potter-Bucky diaphragm. It may be decided to do laminagraphic studies and rotational laminagraphy, selective angiography, bronchography, or some other study.

In approaching a comprehensive roentgen examination, one should keep in mind that the radiologist has an opportunity to make a real contribution. For example, although lung volumes are easily measured by the pulmonary physiologist, they vary so widely in healthy persons that figures obtained for pulmonary capacity do not necessarily reflect the presence of pulmonary insufficiency.[3] The most important aspect of pulmonary ventilation is the amount of air that reaches the alveoli and the uniformity with which it is distributed to them.[3] Any condition where air is prevented from reaching the alveoli in sufficient amounts results in hypoventilation with its consequences of anoxemia, CO_2 retention, and respiratory acidosis.[3] Conditions such as asthma, asbestosis, silicosis, other pneumoconioses and emphysema are only a few that produce hypoventilation.

Variations in alveolar ventilation/blood flow ratios may be diagnosed by exclusion in anoxemic patients. Hypoventilation can be eliminated by simple clinical tests (lung capacity, roentgenograms for anatomic changes). Impaired diffusion of O_2 across the alveolar capillary membrane can be measured by more elaborate physiologic tests (CO_2 uptake).[3]

Structural changes in the alveolar walls may produce anoxemia in the presence of normal alveolar ventilation and alveolar blood flow by imposing a barrier to the passage of O_2 across the alveolar-capillary membranes.[3] This occurs in individuals with pulmonary fibrosis, pulmonary emphysema, and other conditions such as some of the pneumoconioses; also mitral stenosis in an advanced stage. It can be detected clinically, by determining that arterial O_2 saturation falls during exercise and is restored to normal values during the inhalation of O_2.[3]

These are only a few examples illustrating an intriguing opportunity for radiologists to participate in a program of correlating radiologic data with physiologic studies. Barden and Comroe,[4] and Wright[5] have demonstrated the possibilities. It now behooves those of us who are located in institutions where there are good physiologic departments to do our part. In improving ourselves, it is not unlikely that real progress may be made in the detection of pulmonary dysfunction.

III. *Workers in an Industry-Dust Hazard Known or Unknown.* With the industrial group, a different program is necessary. It is essential that the employers of workers in any industry or process known to involve exposure to injurious dusts engage the services of a good clinician. He must be familiar with the plant operation and must know the workers—their occupation in the plant and their home environment. Such a physician is held in high esteem by the workers. This is important. Also the services of an industrial hygienist for periodic conventional dust counts are most helpful.

Whenever possible, the roentgenograms of employees should be made at the plant. This keeps the worker on the job, and one is more likely to obtain roentgenograms of good technical detail once a year. When the studies have to be made away from the plant, not infrequently serial studies will be interrupted for several years at a time.

Usually a good history is obtained, a

careful physical examination is made and the laboratory studies are limited to a test for syphilis, a blood cell count, a urine examination and a sedimentation rate determination.

The roentgen studies have varied over the years from stereoscopic and lateral exposures to single posteroanterior roentgenograms. At present I am planning to recommend a new program for new employees with the view of trying to test certain techniques for demonstrating changes in lung dynamics. It is hoped that the following studies can be made on all new employees:

1. Posteroanterior roentgenogram during inspiration. Posteroanterior roentgenogram during expiration. The tube level should remain the same.
2. Posteroanterior roentgenogram with expiration and inspiration recorded on the same film as a double exposure. The tube level should not change for the two exposures. The expiratory phase of respiration should be exposed first, otherwise one will not get good records of the two positions of the domes of the diaphragm and the ribs in inspiration and expiration.
3. Right lateral roentgenogram of the chest in inspiration and expiration. The tube should be kept at the same level.
4. For the succeeding three years, a single posteroanterior roentgenogram of the chest is to be recommended.
5. The fifth year studies will be the same as the pre-employment examination.
6. The full study will be repeated at five-year intervals thereafter, with single posteroanterior roentgenograms during the intervening years, unless some abnormality develops. It is hoped that such studies will compensate for the lack of opportunity to make careful fluoroscopic examinations.

As stated before, except for individual cases and a few collective studies on anthracite and bituminous coal miners, pottery workers, iron miners, foundry workers and asbestos workers, my experience has been limited to a study of a single industry in which the dust hazard is essentially pure quartz in a finely divided state. The workers are middle class Americans and the great majority are white people. This study has extended over twenty-two years.

In many of the individual cases, we have been able to obtain previous roentgen studies, making it possible therefore to learn something of the natural history of the disease.

For some years, Tuddenham,[80] one of my associates, has been working on basic concepts of visual physiology as it concerns the radiologist. His work has been helpful in explaining some of our deficiencies. Some of these include: the avoidance of outside major glare sources striking the retina; working in a room with low lights instead of complete darkness or bright illumination; and the use of diminishing lenses. The viewing room has 60 viewing boxes extending over one wall so that the roentgenograms of a complete case can be set up at one time. With double reading we think this plan reduces the number of errors in detecting abnormalities and changes from year to year. Looking at several roentgenograms, then removing those and replacing them with others of the same case, oftentimes leads to errors. With few exceptions, more than one radiologist studies the roentgenograms at the same time. This is most helpful in that it, on many occasions, prevents one from overlooking not only small but perfectly obvious variants or lesions. I have said repeatedly that if I were working by myself in a small community, my technician or helper of any category would be invited to participate in my studies at the time of reporting. Every radiologist has had or will have a member of his nonprofessional staff call to his attention a shadow that was not evaluated in a report that has been prepared to be sent to the referring physician.

SIMPLE SILICOSIS

The characteristic lesion in silicosis is a

FIG. 1. Early silicotic nodule showing the collection of epithelioid cells which look like the tubercle formation of tuberculosis. (Courtesy of Dr. Arthur J. Vorwald, Wayne State University, Detroit, Michigan.)

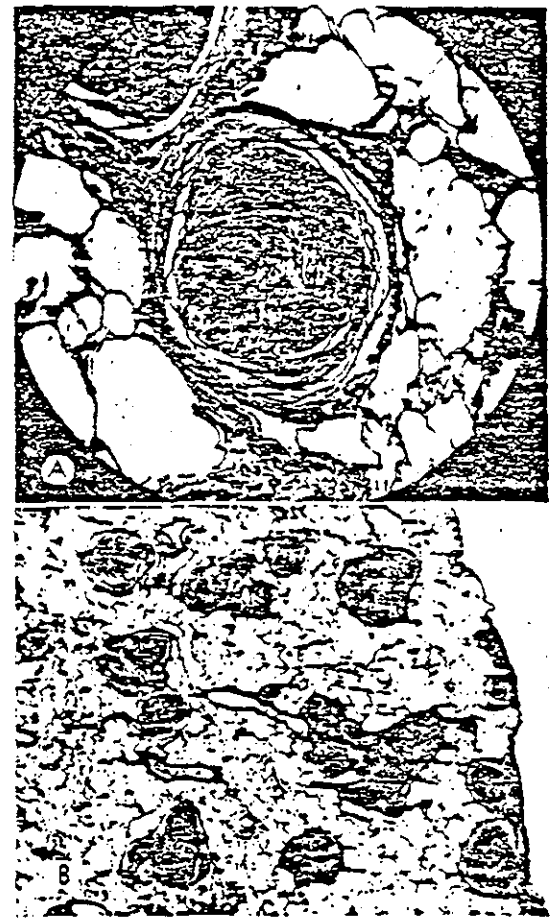


FIG. 2. (*A*) Silicotic nodules showing the hyalin-collagen fibers. Note that these nodules are often adjacent to each other. The nodules have varying shapes and are not cylinders of tissue; consequently, they do not appear as well-defined circular shadows on the roentgenogram. They do not produce characteristic roentgen shadows that allow one to differentiate such shadows from the shadows of other conditions such as siderosis or miliary tuberculosis solely by the roentgen examination. (*B*) Larger field of the same case as *A*. Note the varying sizes of the nodules and their distribution not only in the lung parenchyma, but also along the visceral pleura. (Courtesy of Dr. Arthur J. Vorwald, Wayne State University, Detroit, Michigan.)

circumscribed nodule of hyaline fibrosis (Fig. 1 and 2). The earliest lesions are invisible or are recognizable only microscopically or with a magnifying glass.[24] These parenchymatous nodules may continue to develop and ultimately reach a size of 3 to 4 mm. in diameter. Pathologically, the nodules have well-defined borders except when there are accumulations of nonsiliceous dusts.

The earliest roentgen findings that I accept as evidence of simple silicosis are the small, multiple shadows, 2 to 6 mm. in diameter, more or less uniform in density, which do not disappear in a roentgenogram made with slight rotation. Shadows that disappear with slight rotation are likely to be vascular (Fig. 3, 4 and 5). The blood vessel shadows are denser and their borders are more sharply defined than are those of nodular shadows. The shadows of the silicotic nodules are usually distributed along and between the vascular channels and the bronchial tree of both lungs, and, on the roentgenogram, at times, they may be limited largely to one lobe. Not frequently, even though a bilateral distribution of the shadows occurs, does one see them in the apical, peripheral and lower portions of lung fields.

The roentgen appearance of the shadows of the silicotic nodules is not characteristic and not as diagnostic as one would wish (Fig. 6, 7 and 8). Some are round, some oval, some irregular, some large and some small. Most of the lesions have a uniform density, but some have shadow densities in

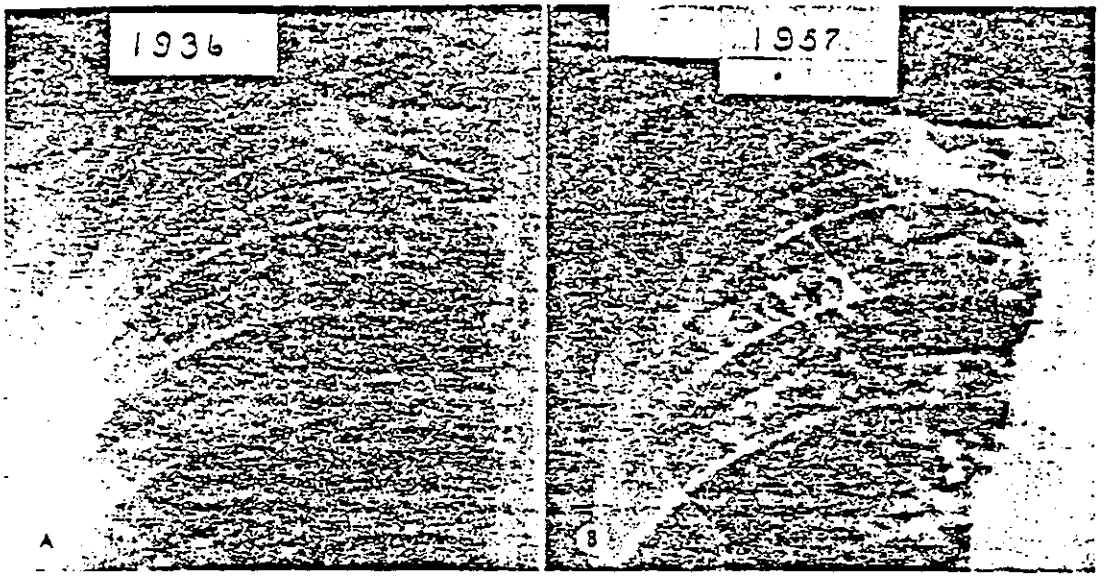


FIG. 6. (*A*) Early silicotic nodulation in a pure quartz worker in 1936. (*B*) Conglomerate silicotic lesion in the same individual twenty-one years later. This worker is asymptomatic and has been so for several years, so his lesion probably falls in the category of simple silicosis.

seen on the roentgenogram. This observation apparently is true even when men are working under similar conditions in the same industry, and, up to the present time, there is no adequate explanation.

When nodulation occurs, the nodules may or may not show progressive changes. In a few cases that have been followed for fifteen years or longer, I have seen the pattern of nodulation replaced by massive shadows, with either complete or almost complete disappearance of visible nodular shadows on the roentgenogram.

One may or may not be able to demonstrate a definite enlargement of the hilar lymph nodes.

Another lesion generally included under the classification of simple silicosis is the small *conglomerate lesion* (1 to 5 cm. in diameter). Such lesions may result from a combination or coalescence of discrete nodules, or the lesion, primarily, may occur as such.

Conglomerate lesions are usually localized and do not occur in the same portion of the lung in every patient. On the other hand, roentgenographic studies of patients with such lesions frequently show them in the upper half of the lung fields (Fig. 6). Microscopic examination of the tissue from such areas often reveals no evidence of infection (Fig. 9). The nodules seem to be closer together than in other portions of the lung; they are less uniform in size, and they are embedded in a matrix of diffuse, fibrous tissue having the same characteristic appearance as that forming the nodules themselves.[28]

The shadow of the conglomerate lesion in simple silicosis is often difficult to distinguish from that found in silicosis with infection. From the roentgenologic standpoint it is impossible to differentiate in a single examination, whereas serial examinations may or may not show slight changes in the extent of the shadows of lesions complicated by an active infection. Roentgenograms made with a Potter-Bucky diaphragm and/or laminagrams made in the erect posture provide additional information which is helpful in evaluating the presence or absence of an infection.

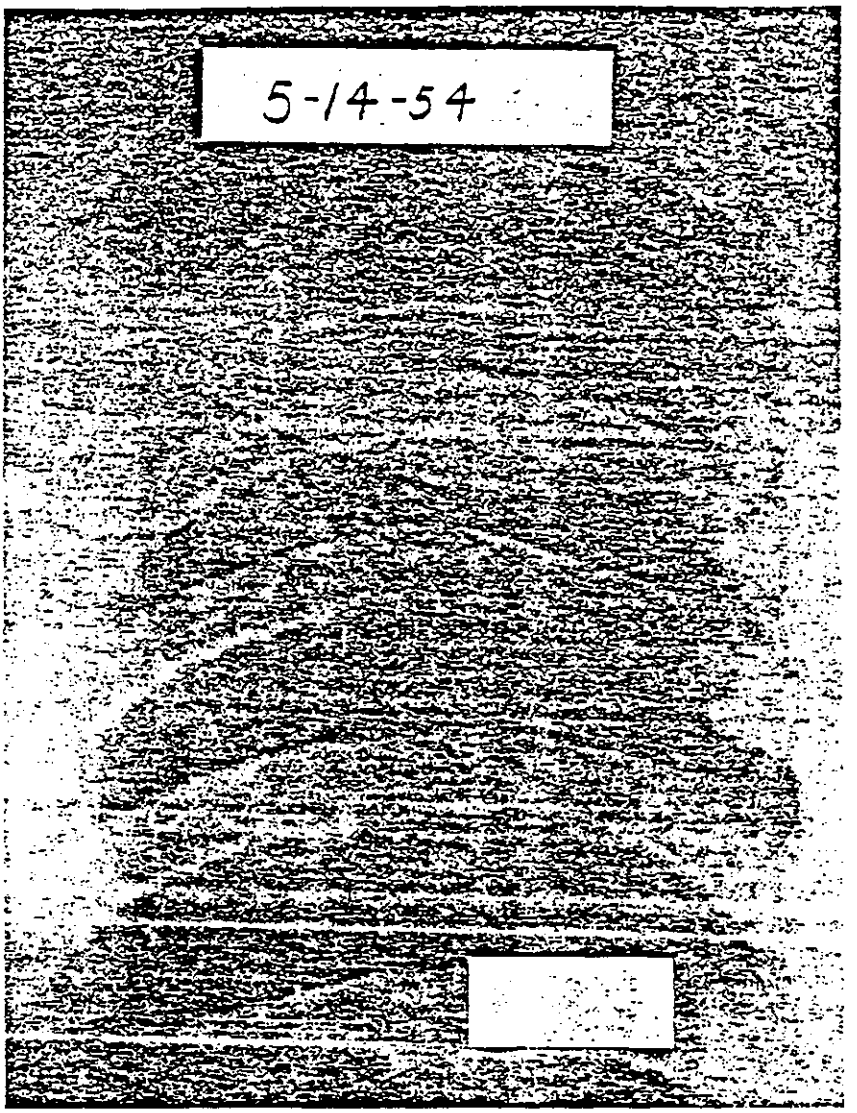


FIG. 7. Sarcoidosis of the lung in an individual twenty-six years of age. Note the pattern of the lesion in the right upper lobe. It could be mistaken for a pattern of a silicotic process. This case was proved by a scalene biopsy.

Including the conglomerate lesion under the category of simple silicosis is only justifiable in those instances when repeated examinations over several years fail to show any roentgen evidence of change. The conglomerate lesion in simple silicosis is not a thick dense lesion, but irregular, thin, having its longest diameter in the vertical position. If, after all of the above qualifications have been considered, there is still doubt as to the true nature of the lesion, my plan is to regard the lesion as one of silicosis with infection either quiescent or chronic.

The presence of well developed emphysema may or may not be observed in simple silicosis. One cannot be certain whether the emphysema is due to silicosis or not, especially if the pattern of nodulation is slight. When well advanced, it is relatively easy to diagnose with either the fluoroscop-

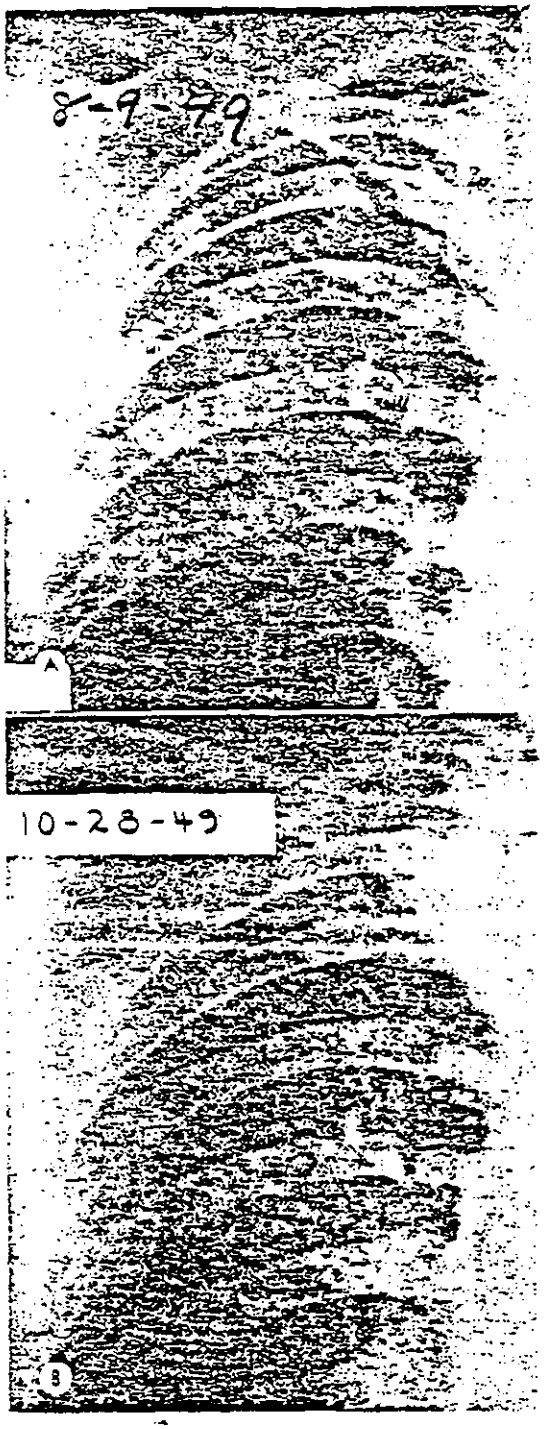


FIG. 8. (*A*) Hemosiderosis in a man, thirty-seven. On August 9, 1949, there was evidence of a pattern of nodulation in the upper lobes of both lungs, the right being shown here. (*B*) This examination was made two months later. Note the tremendous increase in the pattern of nodulation. Shortly after this, the individual was operated on and following a commissurotomy, died from a pulmonary embolus. Microscopic studies showed the presence of hemosiderosis.

ic or roentgenographic examination. The barrel chest; the restricted movement and flattening of the domes of the diaphragm; the increased size of the preaortic space; the increased volume of the lungs seen in the heavily exposed roentgenograms made in the erect and horizontal positions; the loss of fine lung detail in conventional exposures; the widening of the interspaces; and the demonstration of bullae in supervoltage roentgenograms are some of the criteria for diagnosis.

In focal emphysema of slight to moderate degree, careful fluoroscopy with well accommodated eyes sometimes gives one the best lead. Increased pulsation of the pulmonary artery may be present. Overexposed conventional and kyphotic roentgenograms are helpful. In many instances, we believe that emphysema can be demonstrated by supervoltage roentgenograms

FIG. 9. Modified silicosis in a conglomerate mass from a hematite miner. One does not see the well-defined silicotic nodules, such as shown in Figure 2. (Courtesy of Dr. Arthur J. Vorwald, Wayne State University, Detroit, Michigan.)

better than by other roentgen procedures.

Enlargement of the hili is very difficult to demonstrate with certainty. Overexposed or supervoltage roentgenograms in the posteroanterior, oblique and lateral views are necessary. Laminagrams and rotational roentgenograms are helpful. Comparison with previous roentgenograms provides the most reliable information if the roentgenograms are exposed well enough to differentiate between vascular and lymph node shadows.

SILICOSIS WITH INFECTION

In this group are included all cases of silicosis with detectable evidence of infection. It is not always possible in the living patient to determine whether the infection is active or inactive even in instances when conventional clinical and laboratory examinations are available. Under such circumstances, mistakes will occur, but if one exercises good judgment, the affected person can be protected by taking the necessary precautions.

The lesions (either some or all) described as occurring in simple silicosis may be modified by infection. Other lesions that may be found include: cavities (usually thick walled)—tuberculous in origin; lesions occurring as a result of anemic infarcts; massive lesions; mottling; soft nodulation; distortions of the tracheobronchial structures; bronchiectasis; Reichmann's "rainstorm" streaking, distorted vascular pattern; mediastinal displacement; extensive calcification in the hili, paratracheal region and in the lung fields; various degrees of emphysema and bleb formation; pleural thickening, interlobar and peripheral; pleural collections; pneumothoraces; and deformations (flattening, peaking, individualization of costal components and adhesions) of the domes of the diaphragm. The radiologist is rarely able to predict whether the infection is due to the tubercle bacillus or some other organism. One suspects, however, that in the majority of instances the superimposed infection is tuberculosis, for, as stated by Gardner,[25] the postmortem examinations showed an element of tuberculosis in 60 per cent of the cases studied by him. Other infections and infestations include: staphylococcic and Friedländer's pneumonia; histoplasmosis; and fungus diseases.

Some of the detailed changes occurring in the respiratory and cardiovascular structures as demonstrated by the roentgen examination in silicosis with infection[16,28,63–67,70] are summarized below.

TRACHEA

The tracheal shadow may be in a normal position, especially if a tuberculous process or some other infection is superimposed upon an already established silicotic process. In other instances the trachea is displaced backward and/or to one side. Frequently, there are calcified lymph nodes in the paratracheal region. They may be irregularly or completely calcified. Some have an eggshell type of calcification (Fig. 14). At one time, we thought calcification was largely due to tuberculosis. Now we know that histoplasmosis, coccidioidomycosis and possibly other infestations and infections may produce calcification that looks like tuberculosis.

When calcification is incomplete, we suspect that the process is not completely healed.

HEART AND AORTA

On occasions, evidence of cor pulmonale is observed in the advanced silicotic patient. Many clinicians[16] and others have regarded this condition as a complication of silicosis. The relationship between cause and effect is difficult to establish in this instance, and some cardiologists who have given considerable thought to the question find themselves unable to express an opinion. Many of us who have been interested in silicosis for years have not been impressed with the incidence of cardiac complications. On the other hand, it is possible that with increased use of modern cardiac studies in the well-advanced silicotic patient, more evidence of heart changes will be found. But even if such evidence is

found, the cardiologist and the general pathologist know that in the older age group of men not exposed to harmful dust, who are studied at autopsy, cardiac complications are not uncommon.

DOMES OF THE DIAPHRAGM

If considerable emphysema is present, the domes of the diaphragm may be depressed and limited in their movements. At times it is necessary to have the patient cough or sniff in order to demonstrate, roentgenoscopically, evidence of diaphragmatic excursion.

Another abnormal appearance is multiple peaking of the domes of the diaphragm. It may be impossible to differentiate the peaking caused by pleural adhesions from that due to inelasticity of certain structures of the lung. Both causes may be present. Many bizarre appearances of the domes result from emphysema and contractions secondary to lesions within the lung and pleura.

HILI

The shadows of the hili may or may not be within normal limits. They may be enlarged or may be partly or totally obscured by larger shadows produced by lung lesions. Each hilus may or may not be displaced upward, laterally or backward by contracting scars of infected silicotic processes in the lungs. The roentgen appearance of the hili may present a rather homogeneous shadow as contrasted to the normal. There may be calcified lymph nodes in one or both hili. The lymph nodes either may be densely calcified, irregularly calcified, or the calcium may be deposited in such a way as to produce an egg-shell appearance. Calcification of lymph nodes is regarded as evidence of tuberculosis, histoplasmosis or some other process. If the lymph nodes are not completely calcified, they are thought to be the seat of an infection that is not completely healed. Such evidence is not, as a rule, thought to be significant enough to justify recommending that the individual be removed from his particular occupation unless there are other modifying influences.

On the posteroanterior roentgenogram, one not infrequently sees a large shadow which may be unilateral or bilateral. For years these shadows were thought to be due to enlarged lymph nodes, and in some instances were treated as lymphoblastoma. Now we realize that they may represent a lung lesion (massive) which has migrated to the midline and its shadow is superimposed on the hilus, thereby posing a diagnostic problem unless one has previous roentgenograms which demonstrate the natural course of the lesion.[58]

LUNGS

The lung changes vary considerably. The lesions include: nodulation, soft nodulation, conglomerate and massive lesions, emphysema, emphysematous blebs, bronchiectasis, abscesses and cavities.

The term "linear markings" is not employed by us. I presume that those who recommend the term use it to describe shadows in the peripheral portion of the lung and not for the shadows of the vascular markings. Such shadows are so ill defined that we prefer not to classify them.

Nodulation. The shadows of nodulation are similar to those described under simple silicosis and cannot be demonstrated by a roentgen examination in every case of modified silicosis with infection. This is particularly true in certain industries such as hard coal mining and granite cutting. As already stated, the roentgen evidence of the pattern of nodulation disappears in some instances when massive lesions develop.

Soft Nodulation. The shadows of soft nodulation are much larger than those produced by nodulation and mottling, but are smaller than the conglomerate shadows. The description of such roentgenologic shadows is provided in order to emphasize a perinodular cellular reaction observed by the pathologist.

Massive Shadows. These shadows vary from 5 to 20 cm. in size. Some are round, some oval and some wedge-shaped. Some

involve an entire lobe or more than one lobe (Fig. 10). These lesions are usually due to extensive areas of fibrosis and are found anywhere. Frequently, they occur adjacent to interlobar fissures and involve either the basal portion of the upper lobes or the apical portions of the lower lobes or the process may extend across the interlobar fissure and involve both lobes. In lateral views of the chest, lesions are often seen lying posteriorly. Rotational laminagrams are helpful in demonstrating the relations of such lesions. With overexposed roentgenograms, laminagraphic studies or roentgenograms made with the Potter-Bucky diaphragm, it is possible to show some of the details of a massive lesion, such as distorted and dilated bronchi, emphysematous changes, effect on the vascular structures, cavities and areas of calcification and caseation.

At one time most of the massive lesions were thought to be due to silicosis with infection, but Riddell,[24] Gardner,[25] McCloskey[26] and others question whether this is always true. Besides infection and vascular occlusion, Gardner[25] indicated that a "third essential factor may be minerals other than free silica."

On conventional studies, air shadows in massive lesions simulate the appearance of cavities, but with laminagraphic examination in the erect posture, dilated major bronchi sometimes are found to be the cause of the air shadows (Fig. 11*A* and 11, *B–D*).

In conventional roentgenograms and laminagrams, at times, one sees shadows extending downward more or less perpendicularly from the massive lesions toward the domes of the diaphragm. These are blood vessels and have been called Reichmann's "rainstorm" streaks (Fig. 12). These vessels are particularly striking when filled by an opaque material (pulmonary angiography). The position of the vessels is due to the contraction of the lung structures produced by the massive lesion.

With selective angiography, one can now

FIG. 10. (*A*) *Anthracosilicosis in a fifty-seven year old man. The massive lesions involve the upper portion* of the lower lobes and they seem to have extended across the interlobar fissures into the lower portion of the upper lobes. This individual had shortness of breath, emphysema and cough. The costal expansion and contraction was good and there was no delay on inspiration and expiration. The expiratory movement of the domes of the diaphragm was quite prolonged (over ten seconds). (*B*) The lateral roentgenogram shows the position of the massive lesions. These lesions lie posteriorly.

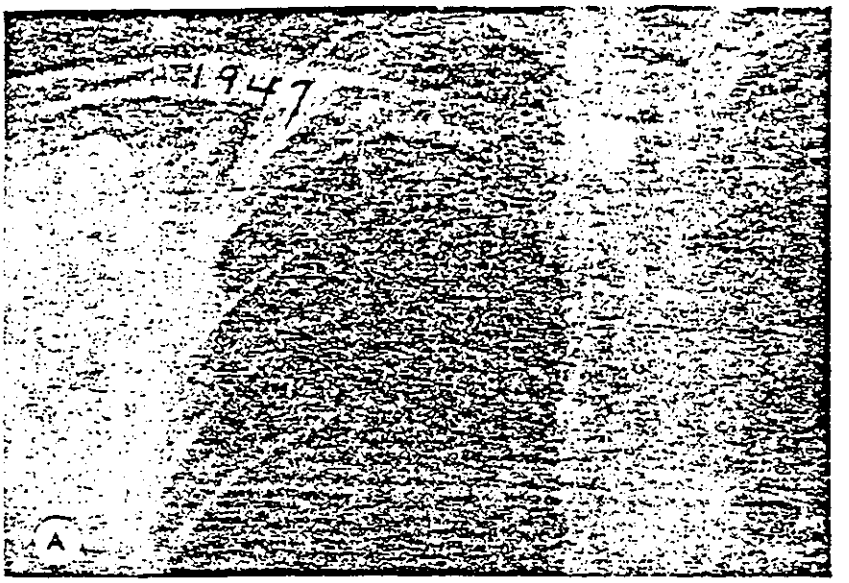


FIG. 11. (*A*) Conglomerate mass in the right upper lobe (arrows) in a young man who worked in an industry which had a pure silica dust hazard. In 1945, the chest was normal. In 1947, several months before this study, he had acute pneumonia. The roentgen manifestations of silicosis developed very rapidly thereafter.

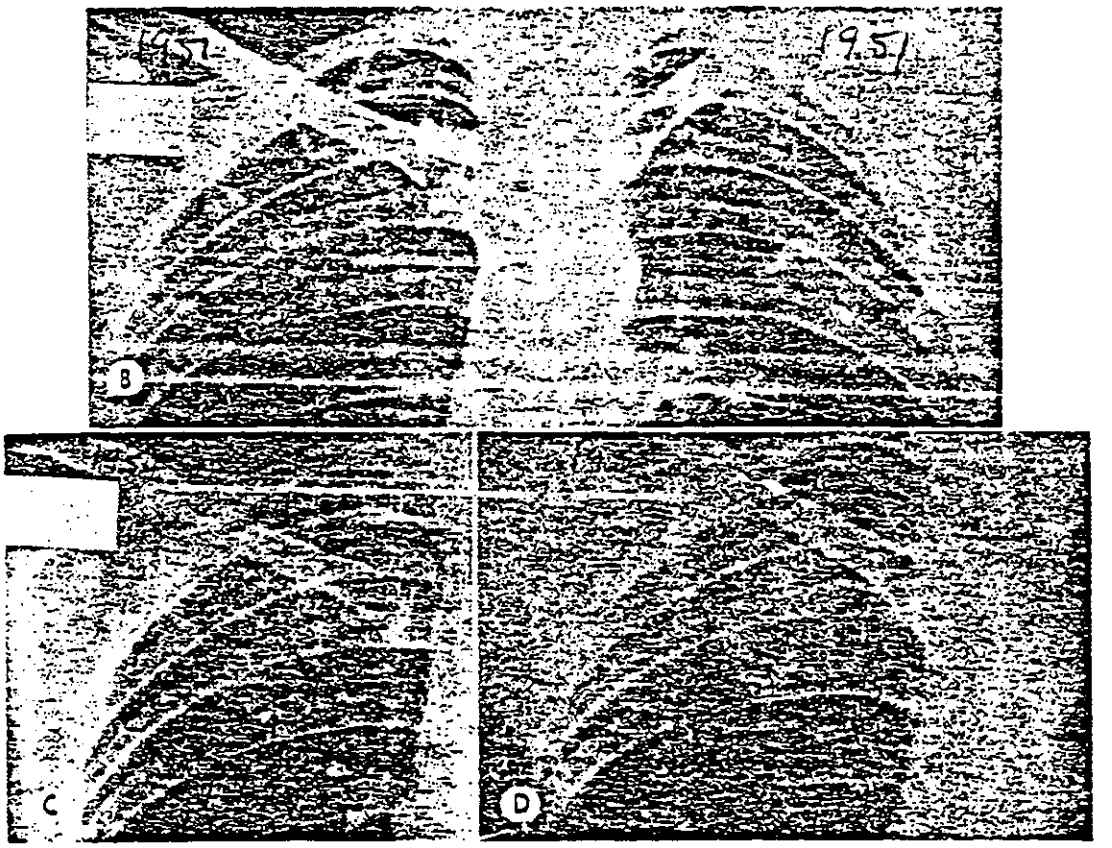


FIG. 11. (*B*) Roentgenogram made in 1951, four years later. There are conglomerate masses in each upper [illegible]. (*C*) March, 1954. A small cavity is developing in the lower portion of the conglomerate mass (arrow). (*D*) April, 1955. The cavity is larger. No tubercle bacilli have been demonstrated. A presumptive diagnosis [illegible] anemic infarct has been made, but frequent examinations for tubercle bacilli are continuing.

FIG. 12. (*A*) The appearance of the blood vessels as seen in 1936 in a person thirty-eight years old. The blood vessels in the left lower lobe are ill-defined. Compare these with *B*. (*B*) Note the stringy shadows of the blood vessels seen in the left lower lobe. This occurs after the contraction of the left hilus upward by the fibrosing lesion in the left upper lobe and produces what has been described as Reichmann's "rainstorm" streaks. These shadows are cast by the blood vessels. Same case as *A* which is regarded as tuberculosilicosis, involving each upper lobe.

demonstrate in the living patient, the fibrosing effect of the massive lesion on the surrounding blood vessels. Some vessels are completely obliterated; in others, the lumen is variously encroached upon (Fig. 13). Vorwald[81] tells me that in those patients with partial encroachment upon the lumen, but not complete obliteration, dyspnea is more dominant. This is a very interesting observation. It may be a corollary to that of the partial encroachment upon the lumen of the renal artery (Goldblatt's kidney); *i.e.*, where one may find hypertension with partial, but no elevation of blood pressure with complete obliteration.

FIG. 13. Endarteritis obliterans occurring in a silicotic person with a conglomerate mass which was an anemic infarct with liquefaction necrosis. (Courtesy of Dr. Arthur J. Vorwald, Wayne University, Detroit, Michigan.)

Selective angiography will show displacement of vessels in patients who have emphysematous blebs and bullae, and in localized pneumothoraces and pleural collections.[56]

Calcification in massive lesions is regarded as evidence of tuberculosis or histoplasmosis (Fig. 14). Sputum examinations and skin tesrs may be helpful in making the diagnosis.

We have observed a migration of massive lesions toward the hilus in certain instances (Fig. 15).[58] Just why this observation was not recorded years ago may be explained on the paucity of serial examinations and a failure to put all of the roentgenograms on

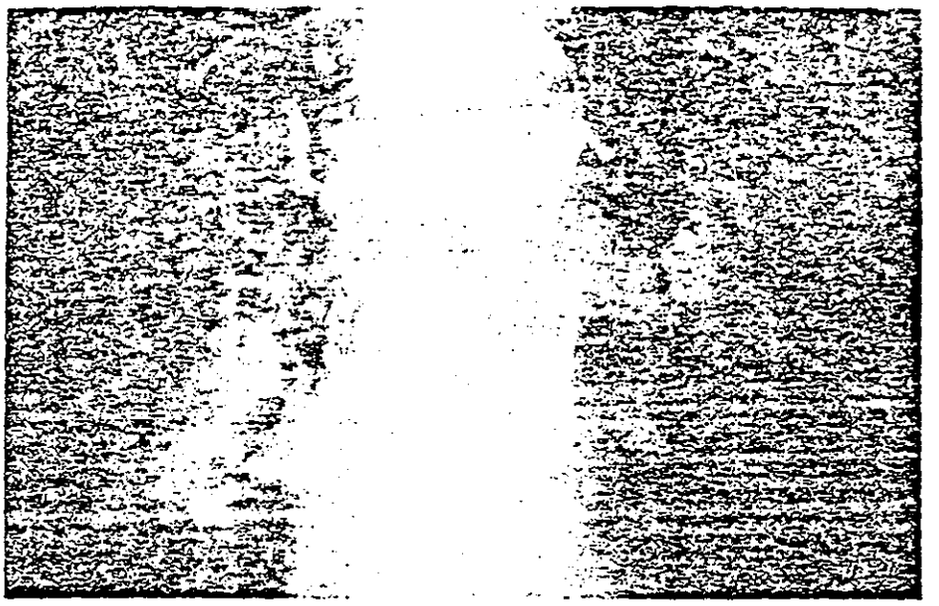


Fig. 14. Tuberculosilicosis or histoplasmosilicosis in a sixty-seven year old individual who has been followed for over twenty years. The calcification in the massive lesion has gradually increased over the years. This roentgenogram shows some of the types of calcification that may occur in histoplasmosis or tuberculosis.

the view boxes at one time. In other words, if one looks at the roentgenograms of serial studies one at a time, the slow migration will be missed. In some of the workers with lesions near the midline, in our first study, it was possible to get earlier roentgenograms which when compared with later ones provided a clue as to what was happening.

After noticing the migration of the massive lesions, I studied the work of Lubert and Krause on lobar collapse and bronchopulmonary segments,[46,48] and learned that they were observing migration in lung lesions. These authors[48] state: "The lobes do not merely shrink to become smaller replicas of themselves. As they become smaller they change from a three-dimensional, pyramidal or conical figure to approach a two-dimensional triangle and flatten along the mediastinal and parietal pleura. With continuation of the collapsing force, they move along the curved surface of the chest wall toward the hilus, the direction of the movement depending on the natural configuration and position of the lobe and the presence or absence of modifying factors." The observation of migration has been helpful, and explains the pathologic physiology simulating the shadows of greatly enlarged hilar lymph nodes.

Kirby[45] has operated on and removed 9 massive lesions, the operations being performed because of a diagnosis of suspected carcinoma of the lung. The lesions were unilateral single masses usually found in hard coal miners, without a pattern of nodulation (Fig. 16 and 17). A similar appearance was seen in quartz workers. All of the lesions proved to be silicotic, without evidence of carcinoma.

The roentgen diagnosis in the first of Kirby's patients was "probable carcinoma." On that basis, the patient had a lobectomy. I was upset over the roentgen diagnosis because the lesion had the appearance of a silicotic massive lesion. When the second patient came along, I was asked to give an opinion. On the basis of a single lesion and marked encroachment on the lower lobe bronchus, a diagnosis of carcinoma was made. At operation, the lesion

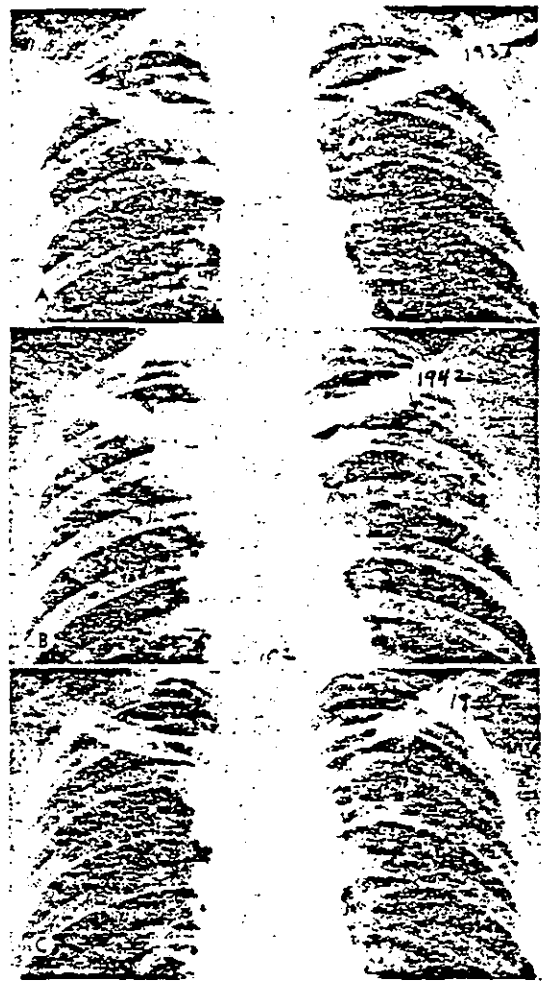


FIG. 15. (*A*) Silicosis with infection in an individual twenty-seven years of age. This examination was made in 1937. Note the lesions in both upper lobes as indicated by arrows. (*B*) Silicosis with infection in a thirty year old individual who was exposed to essentially pure quartz. Note the lesion in the left upper lobe which is close to the periphery. The lesion in the right upper lobe has migrated toward the mediastinum. This examination was made in 1942. (*C*) Roentgenogram taken in 1957. The lesions in the upper lobes are continuing to migrate toward the hilus. The one on the right side is scarcely seen at this time. The one on the left side has migrated considerably since the previous examination.

proved to be a silicotic mass. Approximately one year later, the follow-up roentgen study of the chest showed a large mass in the upper lobe. It was thought that a carcinoma was missed at the first operation so the patient was re-operated upon. A silicotic mass was found at the second operation. A recent examination, seven years after operation, showed no massive lesions.

The development of lung masses following operation is thought to be an aftermath of lung trauma. Because of that observation, I have been somewhat concerned about lung biopsy. The experience of Theodos *et al.*[79] and Effler and his collaborators[17] in over 100 individuals who had lung biopsies done under local or general anesthesia shows that the procedure is safe and quick and that an accurate diagnosis can be obtained.

If Vorwald is correct in his impression that massive lesions with partially occluded vessels may be a dominant factor in causing dyspnea, one should consider removal of such lesions if by selective angiography one can demonstrate evidence of partial occlusion of the vessels.

We are now fully aware that nodulation is not necessary in order to make a diagnosis of silicosis in an unilateral massive lesion in a coal miner. The thoracic surgeons are not as convinced of that fact as we are at this time. Krause and Lubert[46] made an observation which should be considered when making a diagnosis of carcinoma. They feel that the presence of a shadow of unknown etiology in an anterior segment should impart a feeling of special urgency because in these instances the possibility of neoplastic disease is somewhat greater. Although many of the massive silicotic lesions are posterior, we have found them in the anterior segment of the upper and lower lobes.

Emphysema. Emphysema may be focal or generalized. Gough,[31] Fletcher,[19] Gilson and Hugh-Jones,[27] and other English investigators,[10,11,22,32,50,61,63,73] in excellent studies, have re-emphasized the difficulties in determining the presence or absence of emphysema or its extent, when present in coal miners. We will consider emphysema under another heading.

In pure quartz workers emphysema is present, but is not the major factor that one

Fig. 16. Anthracosilicosis in a fifty-seven year old coal miner simulating a carcinoma of the lung. This individual had a twelve year history of cough and dyspnea. There was a 22 pound weight loss in two years. Bronchoscopy had revealed an extensive obstruction of the right main bronchus, with blood stained secretions. The Papanicolaou smear was regarded as negative. A roentgen examination in November of 1948 showed a mass at the right hilus. There was no roentgen evidence of nodulation. Laminagrams of the right hilus area showed evidence of encroachment on the lumen of the right main bronchus. The patient was operated on and the mass was found to be anthracosilicosis.

sees in the coal miner. Gough[21] expressed more concern as to the effect of emphysema on the heart than one usually hears about in this country. Dyson,[16] in his comments about the heart, was talking largely about his experience with coal miners.

When focal emphysema is present, its diagnosis is not easy, as mentioned previously. The supervoltage roentgenogram is sometimes helpful in showing focal emphysema along the peripheral portions of the lung.

Bleb and Bullae Formations. These can readily be seen in most instances. If very large, they will displace and distort the vascular and bronchial trunks.

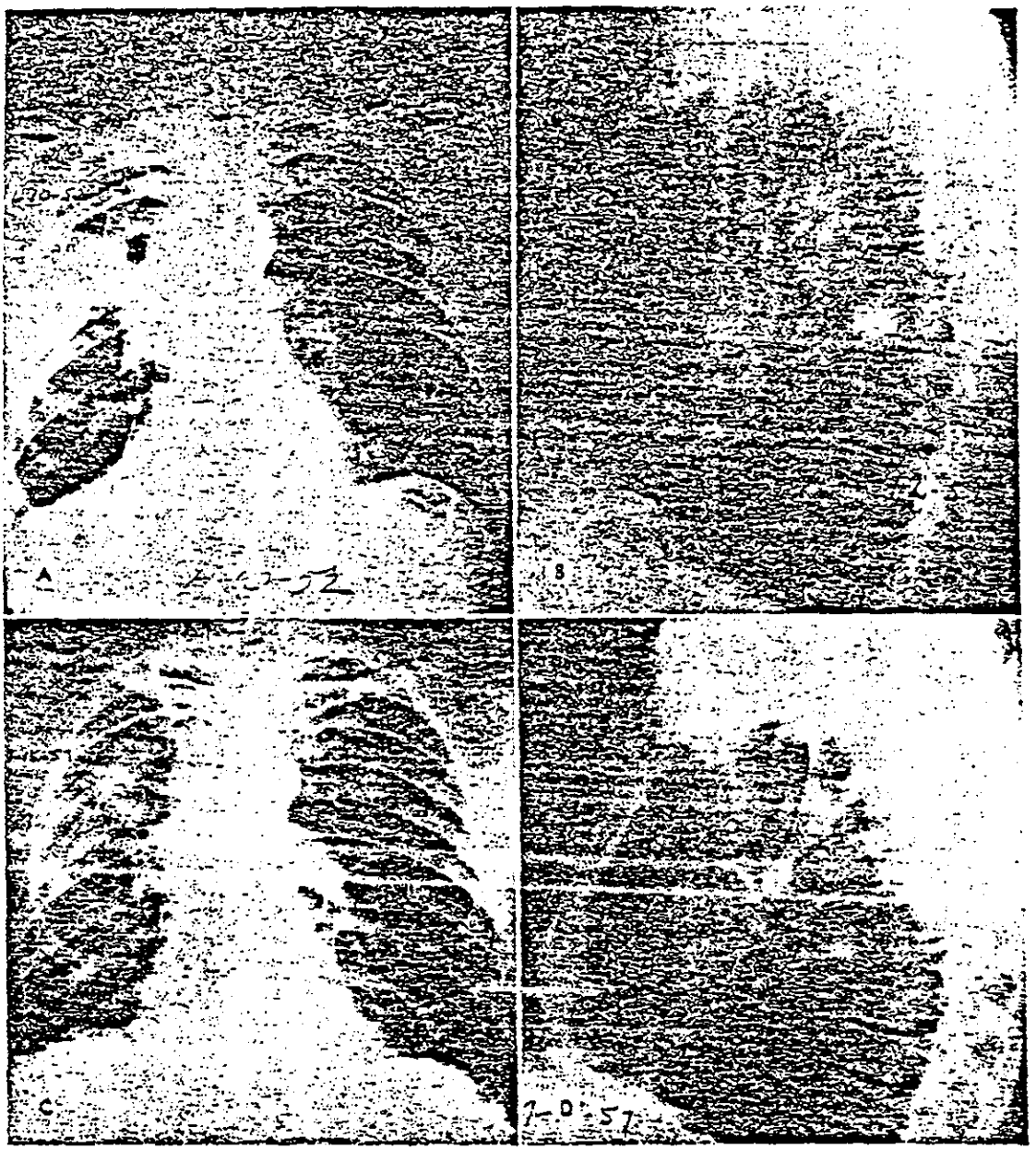


FIG. 17. (*A*) *Same case as Figure 16, four years later* (November 10, 1952). There is a mass in the right upper lung field which from the roentgen standpoint was interpreted as anthracosilicosis. The surgeons thought it was a carcinoma. (*B*) Lateral roentgenogram, showing the position of the mass. The lesion was removed and found to be an anthracosilicotic mass. (*C*) This roentgenogram was made in July, 1957. There has been no return of the anthracosilicotic mass. The shadow in the right upper lung field is due to the appearance of the ribs following thoracotomy. There is no roentgen evidence of anthracosilicosis at this time. (*D*) Lateral roentgenogram made in July, 1957.

Bronchiectasis. This, when present, may be demonstrated by erect and horizontal laminagraphy or by the injection of a water-soluble opaque medium. The bronchiectasis may or may not be due to the silicotic process. If there is no causal relationship, the bronchiectasis is often observed in the left lower lobe, but it may be present anywhere.

Abscesses. Following pneumonia or metastasis from infected lesions elsewhere, abscesses, when complicating a silicotic process, act very much like abscesses in nonsilicotic lungs, except that they may be resistant to antibiotic therapy. If the inside of the abscess wall undergoes epithelization, it becomes chronic with intermittent inflammatory reactivation. When this occurred, lobectomy or some other surgical procedure had been performed in our cases.

Cavities. These are most often due to a tuberculous process, but are also seen in carcinoma. The walls may be thin or thick.

Where the silicotic process is quiescent, the cavity may be thin walled as is seen in tuberculosis without a complicating silicosis. When the silicotic process is active, the wall is often thick, irregular and shaggy. I have not had enough experience with the use of antibiotic therapy in these cases to have an opinion as to their response to treatment.

PLEURA

Occasionally, one sees a pneumothorax complicating silicosis. In such instances the extent of any pleural thickening may be studied. Empyema and other pleural lesions may occur. Since the onset of antibiotics and sulfonamide therapy of lung infections, one rarely sees empyema in the silicotic patient.

TUBERCULOSILICOSIS AND SILICOSIS WITH TUBERCULOSIS

The roentgen manifestations of tuberculosilicosis are protean and yet distinctive.[70]

Only rarely is one fortunate to see the evolution of the entire process in a given patient. It would seem, however, that the pathologic evidence is sufficient and that adequate numbers of cases have now been followed by means of serial roentgenograms over a period of years to warrant a presumptive diagnosis of tuberculosilicosis[4] either upon the visualization of nodulation with concentration, coalescence or conglomeration, or, when the involvement is bilateral, upon the demonstration by themselves of large areas of conglomerate or massive fibrosis. Commonly, these areas of conglomerate fibrosis continue over the years to increase slowly in size, incorporating within themselves more and more silicotic nodules from adjacent portions of the lungs, until finally one may have as the end-result either single or multiple, unilateral or bilateral massive shadows of fibrosis (Fig. 18). As healing takes place, calcification often is observed in heavily exposed roentgenograms. The trachea, hili, and other mediastinal and lung structures may be displaced by scar contraction, especially if the lung lesion extends to and involves the pleura. One may see evidence of varying degrees of emphysema. The changes may be so extensive as to obscure the details that are associated with either tuberculosis or silicosis. As these individuals are not toxic, it is thought that the emphysema accounts for the disability, but disturbed blood supply may also be a factor.

As indicated previously, some of these lesions undergo cavity formation. It should be borne in mind that histoplasmosis may simulate a tuberculous process so far as calcification is concerned. We have observed extensive changes take place over a period of three to five years in some patients who were diagnosed as having tuberculosilicosis, but in whom no recognizable tuberculosis was found at the postmortem examination. Limited reservation must, of necessity, be entertained, however, until the case for or against nontuberculous infection as the etiologic agent is definitely proved.

Silicosis with tuberculosis may occur in either of *two* forms, the clinical course and roentgen behavior of which are as distinc-

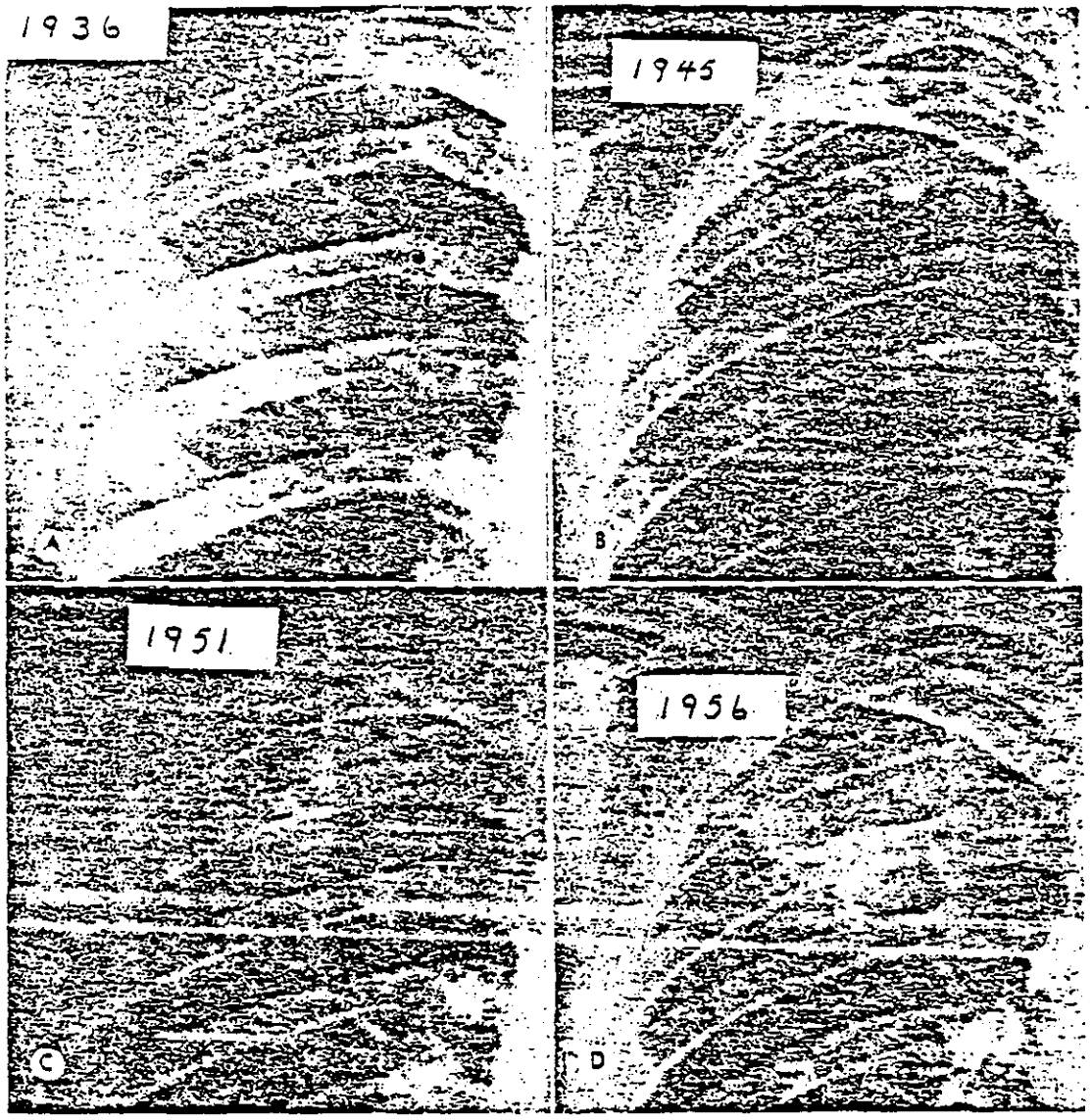


FIG. 18. Silicosis with infection, possibly tuberculosilicosis. (*A*) Appearance of right upper lobe in a silica worker, thirty-eight years of age in 1936. His chest was thought to be healthy. (*B*) This examination was made in 1945. A conglomerate shadow has appeared since early modulation developed in 1937. (*C*) Roentgenogram made in 1951. Note the progressive increase in the size of the lesions. (*D*) Roentgenogram twenty years after the individual first developed this lesion. Note the calcification in the hilus. We have never found any clinical or laboratory evidence of tuberculosis. He has not been skin tested with histoplasmin. This series illustrates the development of a massive lesion in a single individual who has had exposure to quartz dust with very little if any other dusts in the atmosphere.

tively different as is their pathology.

In the first group are those in which there is a fresh infection, either new or arising from the reactivation of a latent focus, superimposed upon a progressive and still active silicosis. The general tendency is toward uncontrollable extension to death before there is opportunity for the chronic

...nges of tuberculosilicosis to occur. Rarely, several years may elapse before ... symptoms supervene.

In the second group are the unusual but ...stent cases in which silicosis and tuber... occur together but act independent... The infection, observed by means of ...al roentgenograms, is seen to behave ...pically as it does in nonsilicotic sub...ts.[7] The tuberculosis develops upon a ...kground of an already stabilized silico... in which the quartz particles are pre...ably completely isolated within their ...ous nodules and thus exert no effect on ... superimposed infection. We have then ...ple the coexistence of silicosis and tu...losis within the same patient, but ...ence modification or acceleration of ei... disease process by the other.

Recently, we have observed another ...e. The patient was a relatively young ... with widespread disease, which devel... very rapidly. The distribution of the ... was similar to that seen in miliary ...erculosis. The patient had tuberculosis ... the larynx. Hoarseness was the chief ...plaint. The patient did not have any ... nor any signs of toxicity, but he had ...sitive biopsy and his sputum was posi... for tubercle bacilli.

Chadwick and Pope[8] in discussing tu...losis of "yesterday and today" call ...tion to the falling morbidity and mor... rates. The incidence of tuberculous ...tion and tuberculous disease is grad... decreasing. Cooper[14] states that this ...rvation carries over into the silicosis ...p. Factors such as better nutrition, ... housing, less crowding, and isolation ...ven cases of tuberculosis are responsi... Cooper[14] does not know of any good ...ence to show that there is decreased ...ence of the tubercle bacillus. He states ... some phthisiologists have emphasized ... the race as a whole shows improve... in resistance. Vorwald *et al.*,[15] in ... studies of the effect of BCG* vaccina... in guinea pigs with silicosis, demon...ate conclusively that the BCG strain ... Calmette-Guerin.

used in their study can produce progressive and fatal pulmonary tuberculosis in guinea pigs whose tissue resistance has been modified by the inhalation of quartz dust. I know of no study of this type among silica workers.

RAPIDLY DEVELOPING SILICOSIS

This type of silicosis[25] is not seen very often. When it occurs, there are two causative factors that seem responsible—gross overexposure to finely divided silica and a complicating infection, especially tuberculosis. On two occasions, I have had the opportunity to study serial roentgenograms in rapidly developing silicosis; one group was exposed in drilling a tunnel and the other while bagging silica sand. I have not seen a single case of this type within the past ten years.

The early roentgen appearance is that of a diffuse haze in the lower lobes, especially on the right side. The roentgenogram is not too informative, but the fluoroscopic study reveals some limitation of costal and diaphragmatic expansion.

In the more advanced lesions, there is evidence of lobar and/or lobular consolidation. In the chronic cases, the lower lobes show chronic scar contraction and the lung ventilation occurs largely in the upper lobes.

PULMONARY PHYSIOLOGY STUDIES IN SILICOSIS

Wright,[16] in summarizing his observations on pulmonary physiology in silicosis, states that the earliest form of silicosis, generally classified as simple discrete nodular silicosis, is attended either by no recognizable physiologic alterations of function of the cardiorespiratory apparatus, or, at most, by very slight ones. It is not surprising to me that such a statement is made, even though it may be modified when more information is available. The lungs have tremendous powers for compensation in health and disease.

When conglomerate silicosis develops, one usually finds alteration of pulmonary

function.[54] The maximal breathing capacity is usually reduced, the residual volume is increased, and the oxygen ventilation equivalent may be above normal.[55] The findings are like those found in diffuse obstructive emphysema. This type of abnormality can be demonstrated by fluoroscopy and roentgenography, but Wright[56] considers that for practical purposes it should be said that in all stages of silicosis, the severity of impairment of respiratory function bears only the grossest correlation with the extent of the disease found in the roentgenogram. This is not discouraging—there is an opportunity for the physiologist and the radiologist to pursue further investigative work.

ASBESTOSIS

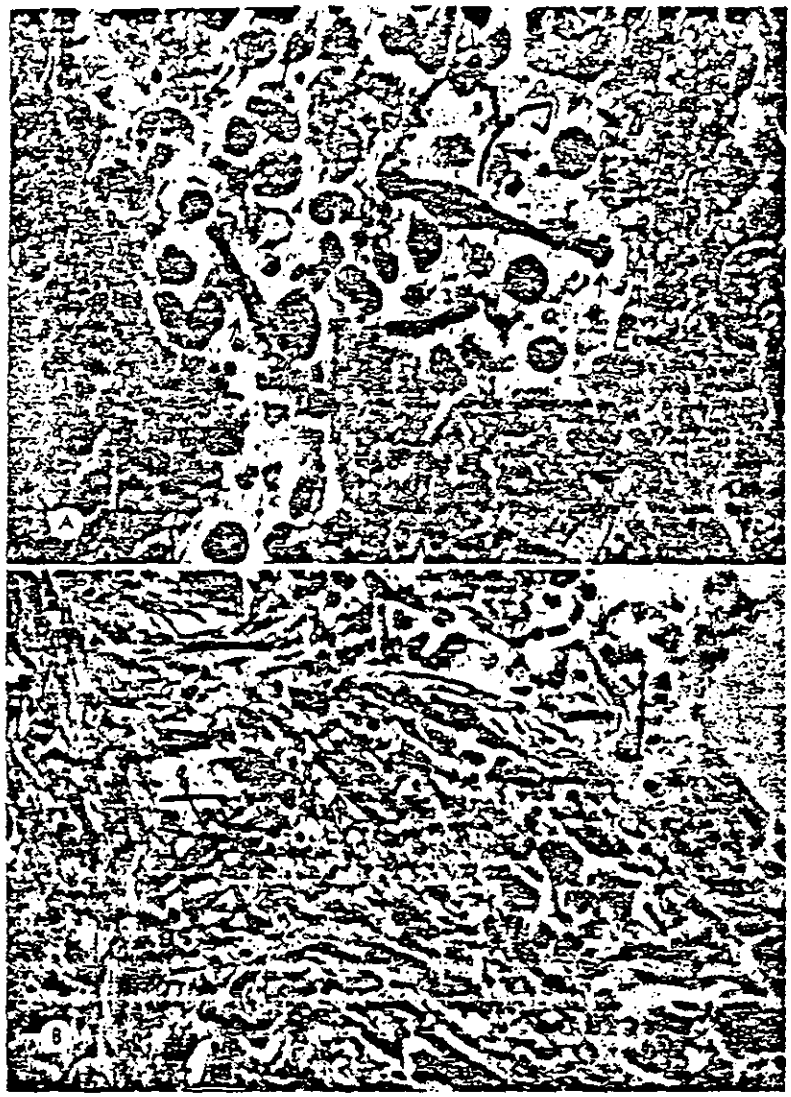
The roentgen findings in asbestosis are seen first in the lower half of the thorax. This form of pneumoconiosis is notoriously difficult to diagnose during its so-called early stages. In fact, the patient may have symptoms of what in this country is often diagnosed a slowly-resolving virus pneumonia evidenced by weakness, chronic cough, but no fever. The roentgen findings in such patients after the disappearance of the pneumonia are essentially within normal limits so far as the fluoroscopic and roentgenographic observations are concerned. Yet, in such individuals, the microscopic studies of lung biopsies show thickening of the walls of the alveolar sacs and the respiratory bronchioles, and excellent examples of well-formed asbestos bodies (Fig. 19). My experience, therefore, is not in accord with that of Cartier[2] who says that "no cases of asbestosis of clinical importance have been diagnosed by the pathologist without having been detected anteriorly by the roentgenologist." I must hasten to say that I have not had lung biopsies on mine workers, but have had a few microscopic studies of lung biopsies on mill and textile laborers and on an individual whose work was concerned with covering pipes with asbestos for insulation purposes.

In answer to a query[13] as to the presence of asbestosis in a 1952 roentgenogram of a fifty year old man who had worked from 1940–1945 covering pipes with asbestos insulation, it was suggested that the exposure would have had to be much more severe in order to produce asbestosis. After seeing and examining a well-advanced and proved case of asbestosis in a man whose occupation was covering pipes with asbestos material, I find it difficult to express an opinion on just how much and what kind of exposure is necessary to produce asbestosis. For years, workers in covering pipes have not been thought of as working in an unusually dangerous dust hazard. The patient, referred to above, was a self-employed individual, intelligent, and although he knew asbestos dust could produce lung damage, he felt that he was not being exposed to enough asbestos dust to hurt himself. The dust hazard in this instance was produced by sawing and fitting the coverings (Fig. 20).

A careful fluoroscopic and roentgenographic study should be made of all workers going into the asbestos industry. A number of these workers are women, so one has to be concerned with the confusing shadows of the breast in women and heavy pectoral folds in men. Stereoscopic roentgenograms are helpful. Roentgenograms exposed in full inspiration and in full expiration in the posteroanterior and right lateral views, and posteroanterior views in each decubitus position will greatly assist as a baseline for future comparison in order to detect the earliest roentgen evidence of deviation from the normal. Oblique views (15 to 25 degrees) are most helpful in demonstrating pleural changes.

For the most part, the roentgen signs are not recognizable until the fibrosis has extended to and involved the visceral pleura. Early visceral pleural changes are not seen except at the interlobar fissures and then only when one makes special exposures in the cephalic or caudal views centered along the interlobar fissures (Fig. 21).

I have found the expiratory posteroanterior roentgenogram helpful in studying



...(A) Several asbestos bodies in the lung. Note the various shapes (arrows) and the irregular deposit ...and the asbestos fiber. Some asbestos bodies have terminal globular swellings and transverse markings. ...There are two large collections of asbestos bodies. (Courtesy of Dr. Arthur J. Vorwald, Wayne State ...versity, Detroit, Michigan.)

...stosis; in fact, sometimes it seems to ...ide more reassuring evidence of dis... than is obtained on the inspiratory ...genogram.

The earliest roentgen evidence of asbes... is a presumptive limitation of dia...matic and costal expansion and roent...graphic evidence of restricted move... of the vascular shadows (elongation ...separation of the truncal shadows). Such findings must be presumptive unless pre-employment studies are available for comparison. Retrospective studies are invaluable in asbestosis. Oftentimes the roentgen evidence of asbestosis is seen best in the left lower lobe. Just why this occurs is not known.

Gradual disappearance of the lateral costophrenic sulci is a helpful diagnostic sign in asbestosis and is interpreted as evidence

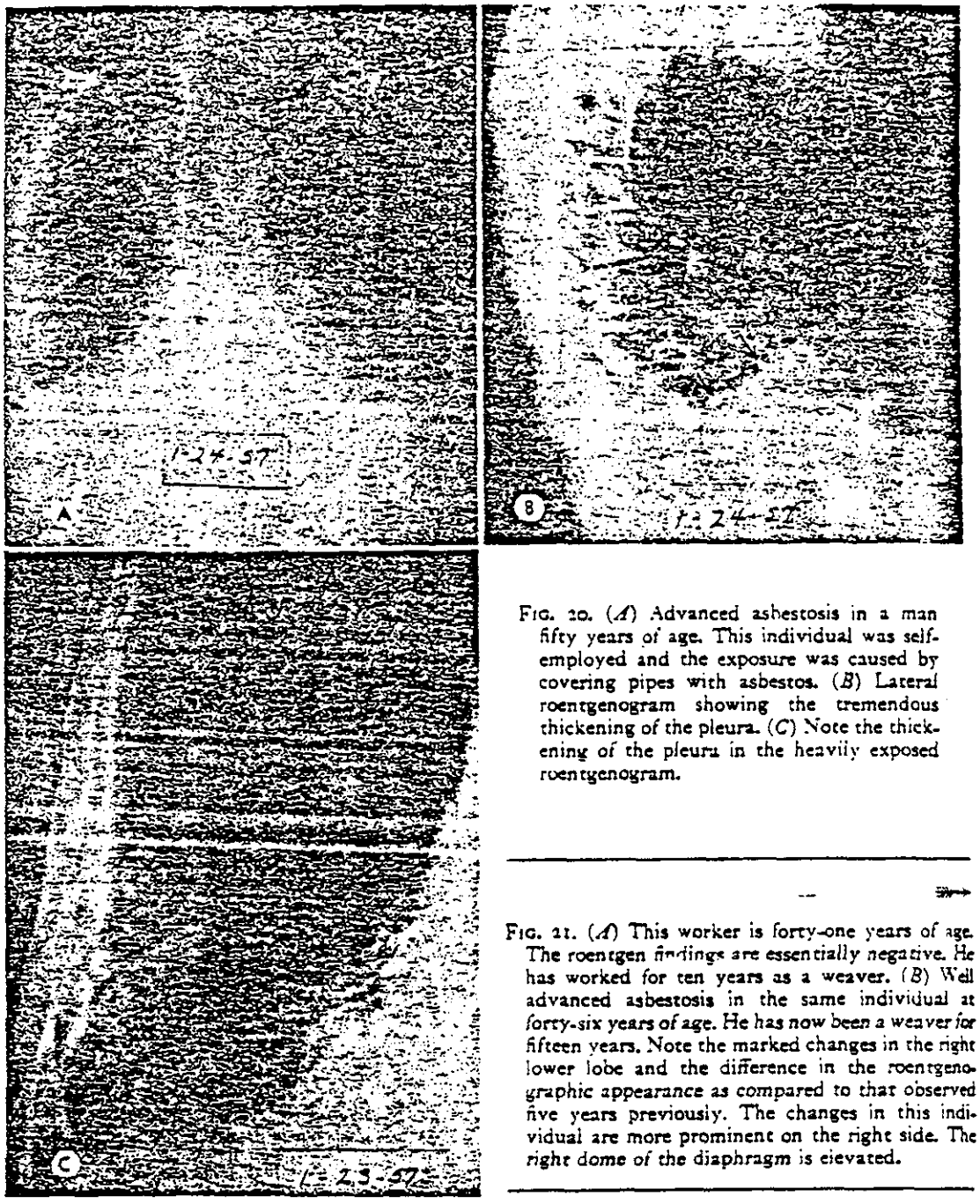
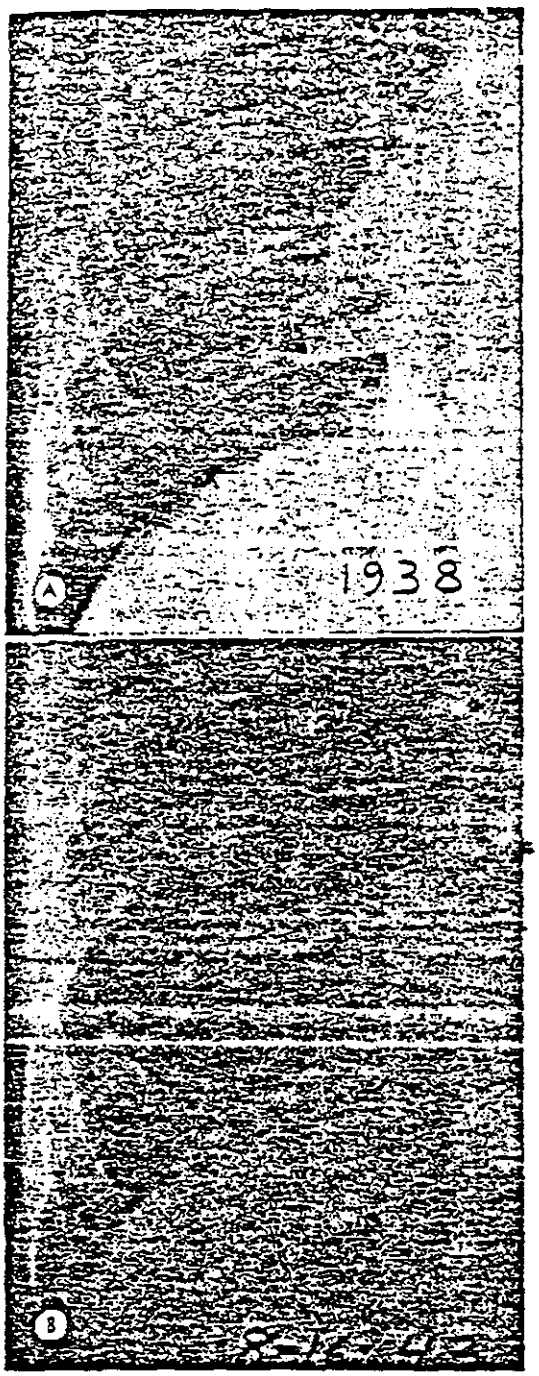


FIG. 20. (*A*) Advanced asbestosis in a man fifty years of age. This individual was self-employed and the exposure was caused by covering pipes with asbestos. (*B*) Lateral roentgenogram showing the tremendous thickening of the pleura. (*C*) Note the thickening of the pleura in the heavily exposed roentgenogram.

FIG. 21. (*A*) This worker is forty-one years of age. The roentgen findings are essentially negative. He has worked for ten years as a weaver. (*B*) Well advanced asbestosis in the same individual at forty-six years of age. He has now been a weaver for fifteen years. Note the marked changes in the right lower lobe and the difference in the roentgenographic appearance as compared to that observed five years previously. The changes in this individual are more prominent on the right side. The right dome of the diaphragm is elevated.

of pulmonary and pleural involvement. The sharp outline of the cardiac silhouette gradually becomes ill-defined due either to the blurring shadows of superimposed structures or thickening of the visceral pleura (Fig. 22). My explanation for the exaggerated pleural thickening in the lower lung areas is that the pleura seems to show greatest thickening in those areas where there is greatest mobility of the structures.



Possible trauma exerted by the fibers on the surrounding structures during respiratory movements is the real factor, not only in causing pleural thickening, but also in producing prominence of the left lower lobe markings; the cardiac pulsation may act as an additional factor.

In the advanced stages, the diaphragmatic excursion is markedly disturbed; the lower lung fields have an appearance of hypoventilation plus a leather-like or ground glass density. All structures, including the borders of the cardiac silhouette, are ill-defined (Fig. 23). The pleural shadows are greatly thickened, the thickening being less in the upper portions of the chest. The upper lung fields are ventilated, possibly hyperventilated, at times giving one the impression of emphysema. Asbestosis may produce shadows of a nodular pattern or irregular, stringy shadows not only in the lower lobes but in the middle or upper lung fields. The vertical diameter of the chest (first rib posteriorly to level of domes of diaphragm in inspiration) is reduced. Frequently, the elevation of the left dome is more pronounced.

Pulmonary Physiology in Asbestosis. Wright,[55] in his studies of asbestosis, showed that there is little and oftentimes no impairment of ability to ventilate the lungs in the typical asbestotic person, as measured by maximum breathing capacity. There is a slight to moderate reduction in total lung volume.[55] It should be possible to show this by fluoroscopy and with roentgenograms by demonstrating the restriction of thoracic and diaphragmatic movement. When lung fibrosis is extreme in extent, the maximum breathing capacity is limited, but fluoroscopy shows little or no impairment to emptying of the lung.[77,85] Gas studies of O_2 and CO_2 show an impediment to the passage of oxygen across the alveolar membrane, and during exercise the person with asbestosis overbreathes.[85] In spite of these and other complementary observations, Wright[85] indicates that it should be emphasized that his data show that an individual retains his average capacity for work in the presence of incomplete oxygenation of the blood. Wright,[85] in his discussion on asbestosis in connection with correlation of the roentgenologic and physiologic pulmonary studies, states—"that

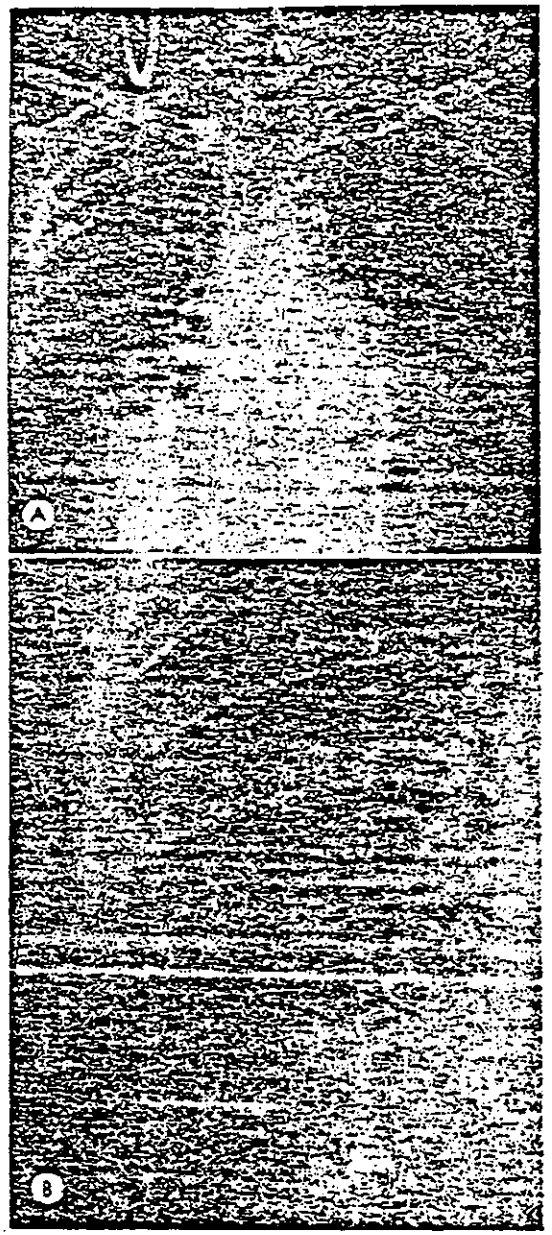


FIG. 22. (*A*) Asbestosis in a fifty-four year old man who worked as a laborer for four years operating a shaker which removed dust from the asbestos fiber. There was a mechanical ventilator in a large room with three windows and a door. This individual was in normal health until August, 1945 when he developed a dry hacking cough and dyspnea on exertion. There was a dull substernal pain. One can see evidence of fairly advanced changes in both lower lobes. There is definite thickening of the pleura on both sides with some obliteration of the costrophrenic sulci. (*B*) The close-up study shows the marked changes in the right lower lobe. Movement of the domes of the diaphragm was fairly good in spite of the extensive lung changes. It was difficult to demonstrate good cardiac pulsations because of the overlying shadows in the lung field. The roentgen kymographic studies showed fairly good amplification of the pulsation of the heart.

three situations can exist: (1) physiologic abnormality without any definite roentgenologic abnormality, (2) roentgenologic abnormality plus physiologic abnormality, and (3) roentgenologic abnormality without any physiologic abnormality."

Tuberculosis and Asbestosis. My experience has led me to believe that asbestosis does not predispose or render a person more susceptible to tuberculosis. When tuberculosis does occur in an individual with asbestosis, its roentgen manifestations are essentially those of one who has had no complicating disease (Fig. 24). I am aware that there is difference of opinion about susceptibility in asbestosis and suspect that other factors may be of major importance.

COAL WORKERS' PNEUMOCONIOSIS

The pathologic studies of Hart *et al.*,[26] Gough,[30] Heppleston,[28] Gilson,[25] and the roentgenologic and epidemiologic studies of Fletcher and his scientific associates[21,22] have been responsible for a new concept in pneumoconiosis. If Collis[12] could examine the pathologic and epidemiologic data that have been collected by Gough and his colleagues, I feel sure that he would modify the statement contained in his scholarly Milroy Lecture which reads as follows: "some dusts, such as coal, it is true, not only appear to have no power of producing pneumoconiosis, but even may possess some inhibitory influences on phthisis; . . ."

Re-orientation concerning the pneumoconiosis problem among the anthracite and bituminous coal workers has been difficult for many of us, who, for years, have been disciples of those who believed preponderantly that disability was the result of the fibrogenetic effects of finely divided silica. Kammer[42] feels that we in America should re-assess the whole problem of the miner. I

...ld be inclined to extend the re-assess-...nt to workers in other industries—two ...amples being the talc workers in the ...te of New York, and the gold miners in ...stern Australia.[1]

The work of Gough and his colleagues is ...t important in another respect. There ...e many well-trained young men in indus-..., public health, and medicine, who have ... techniques which extend beyond the ...rs of the microscope; beyond the previ-... concepts of acceptable dust counts for ...ealthy atmosphere; and, beyond old ... on environment. Lastly, but not ..., they cannot be as completely dedi-...d to silica as I found operative in my ... thinking. The future for better work-... and living conditions should be bright.

It should be emphasized that many of ... coal miners in this country have had ...sures to silica from hard rock mining ... the pathologic process in their lungs ... not correspond to that under discus-... Their disease is one of a modified sili-..., *i.e.*, anthracosilicosis. That form of ...moconiosis is not being considered

...al workers' pneumoconiosis is not a ... condition. It occurs largely among ..., who work at the coal-face, but it oc-... elsewhere also, even among those load-... the coal into the ships.[28]

In the pathologic studies of Hart *et al.*,[36] ...gh,[37] and Heppleston,[38] it was noted ... the condition is pathologically distinct ... silicosis. The lungs contain large ...ntities of coal dust, which is aggre-... into foci about the respiratory bron-...les. The fibrosis is sparse, the coal dust ... held in a fine mesh of reticulin fibrils, ... in appearance. The second finding ... that the pathologic process was the ... in every coal field, suggesting that ... is nothing biologically peculiar about ...structure. The third finding was that the ...se has two forms. First, simple pneu-...oconiosis, which is characterized by small ... of coal distributed throughout the ..., these foci being surrounded by small ... of emphysema, so-called focal em-...ma. Secondly, infective pneumoconi-

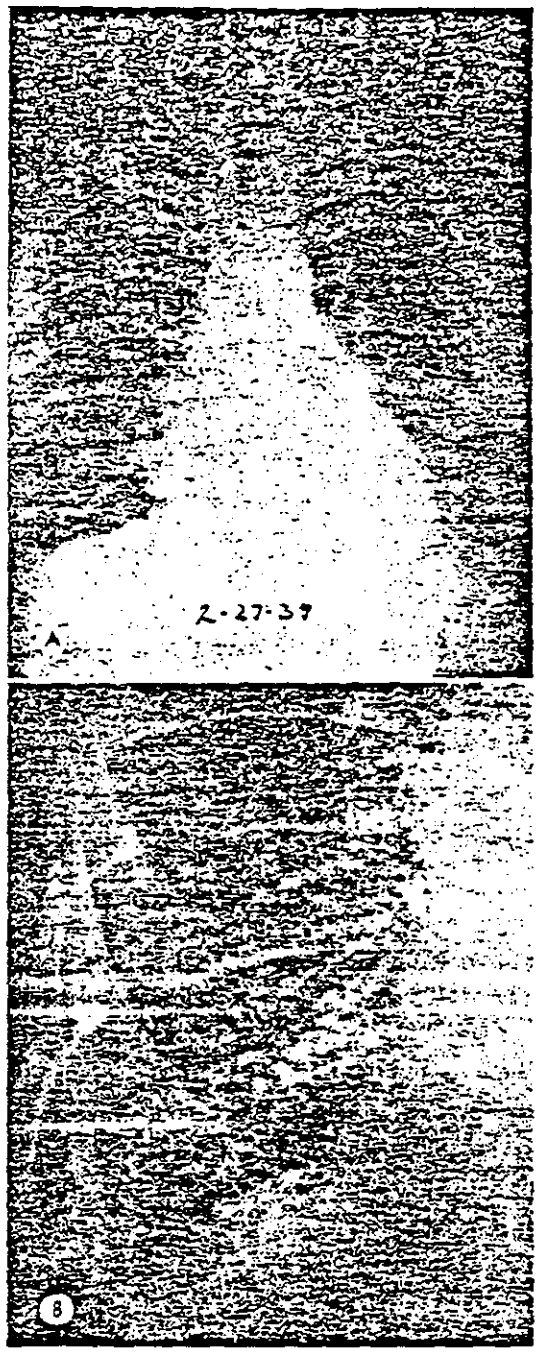


FIG. 23. (*A*) Well advanced asbestosis in an individual fifty-three years of age who had worked for over thirty years with asbestos. He had clubbing of the fingers, chronic cough, dyspnea, anorexia and fatigue. Note the pattern of the disease throughout both lungs, especially in the left lower lobe. (*B*) The close-up study illustrates very well the changes in the lung and shows the thickening of the pleura along the right lateral chest wall.

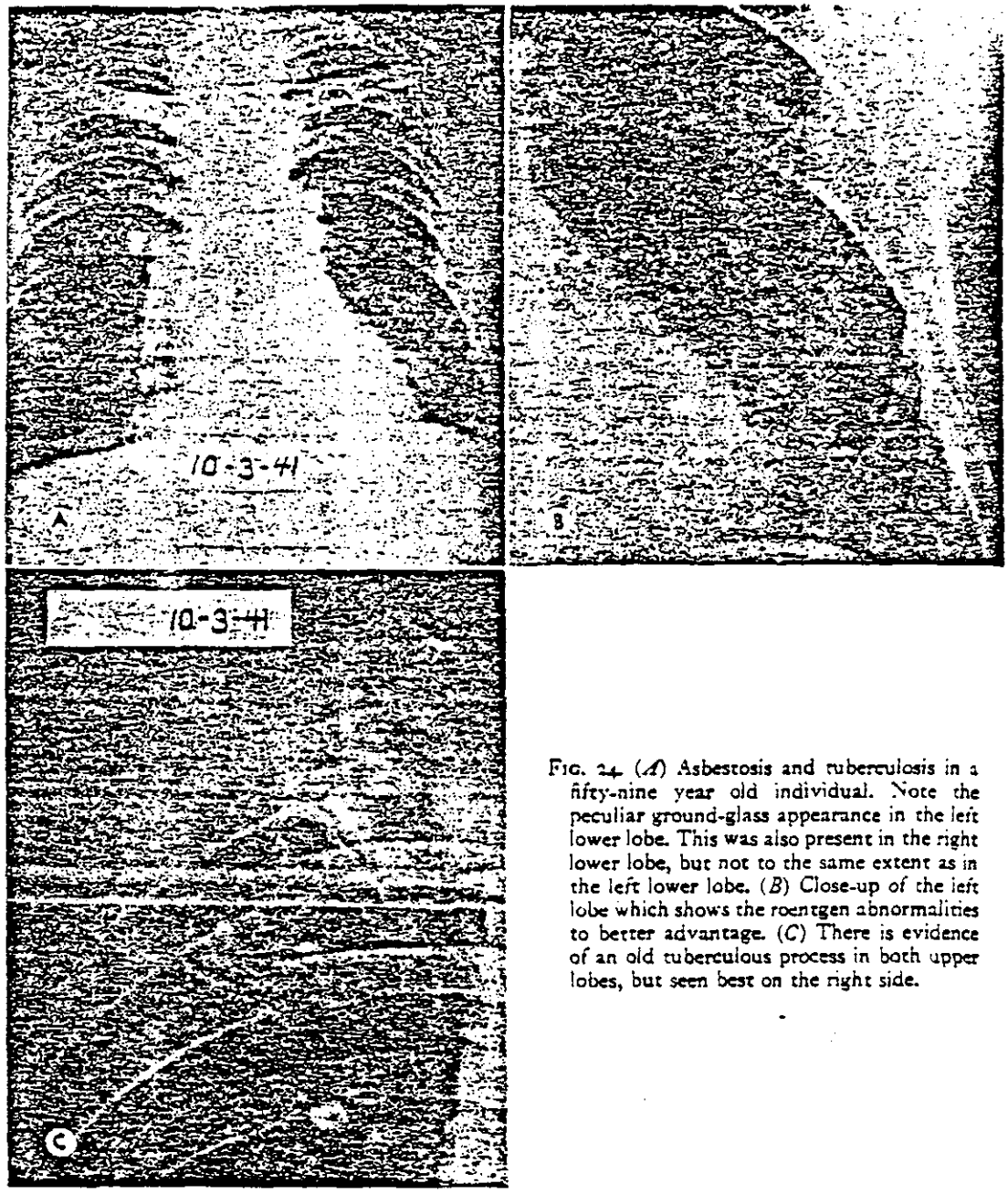


FIG. 24. (*A*) Asbestosis and tuberculosis in a fifty-nine year old individual. Note the peculiar ground-glass appearance in the left lower lobe. This was also present in the right lower lobe, but not to the same extent as in the left lower lobe. (*B*) Close-up of the left lobe which shows the roentgen abnormalities to better advantage. (*C*) There is evidence of an old tuberculous process in both upper lobes, but seen best on the right side.

osis, which starts as a collagen fibrosis within a few coal foci and subsequently enlarges and coalesces to form a dense mass of fibrous tissue occupying much of a lobe or even a whole lung. This second form is thought to be due to tuberculosis superimposed upon a lung heavily laden with coal dust.

The roentgenologic manifestations are classified as follows:[20] *Simple pneumoconiosis* refers to those cases having minute shadows throughout both lungs. Simple

pneumoconiosis is subdivided into three categories of increasing profusion of the discrete shadows. *Complicated pneumoconiosis* refers to those cases having shadows of greater size. Complicated pneumoconiosis is subdivided into four categories: (*A*) Shadows occupying at least one anterior rib space, generally more or less circular, 1 cm. in diameter and of uneven density; (*B*) dense shadows more extensive, better defined and more homogeneous than category *A*, and occupying less than three anterior rib spaces; (*C*) similar shadows extending more than three anterior rib spaces; and (*D*) shadows extending more than three anterior rib spaces plus gross distortion of the pulmonary architecture.

There are two disease hypotheses. The studies suggest that man's environment causes differences in the natural history of simple and complicated pneumoconiosis. *Simple pneumoconiosis* only appeared and progressed as such in those who were exposed to coal dust. *Complicated pneumoconiosis* appeared and progressed in those who left the mine; it did not appear to any extent in those who had less simple pneumoconiosis than would be classified in the second category.

In surveys on coal miners, McCallum and Browne[24] found in the roentgenographed group that the roentgen evidence of pneumoconiosis increases in prevalence with rising age and with length of time spent at facework. Between twenty-six and forty-five years of age, nearly 8 per cent had simple pneumoconiosis and the percentage of men affected rose sharply between thirty-six and forty-five years of age. These two age periods correspond roughly to from one to ten years and eleven to twenty years at the face. They observed that the length of time at the facework is more important in producing roentgen signs of pneumoconiosis than is the period of time working in the pit. These authors believe that the men with pneumoconiosis should be allowed to continue work at the face only under medical supervision.

Clinical studies[15,17,31,32,78] on coal workers in this country continue and in at least two communities,[47,52] the influence of our English colleagues has become manifest. The use of the "standard set" of diagnostic roentgenograms is being employed.

In studies concerned with the relationship between *tuberculosis* and *pneumoconiosis* in coal miners, Cochrane[9] suggests "that a little coal dust retained in the lung has a definite therapeutic effect, whereas larger accumulations increase the attack and mortality rates from tuberculosis." It is felt that with "standard film guides,"[54] the roentgenologic diagnosis of tuberculosis can be made with reasonable consistency and that shadows characteristic of tuberculosis can be distinguished in roentgenograms from those of simple and complicated pneumoconiosis.

The attack rate of the re-infection type of tuberculosis is found to be related to the severity of the associated simple pneumoconiosis[50] and is higher than in the general population. It seems to be reduced in cases of complicated pneumoconiosis.

For comparative purposes, it is of interest to note that Wall,[83] in a study of 100 autopsied cases of anthracosilicosis, found pulmonary tuberculosis in 3 cases, as judged by histopathologic criteria and examination for acid-fast bacilli on stained smears. The majority of the patients died of heart disease, with cor pulmonale accounting for one-half of the cardiac deaths.

Pulmonary Function and Roentgenologic Correlation. Gilson and Hugh-Jones,[27] in 1949, undertook an investigation with the object of determining the precise cause of breathlessness in a group of coal workers and relating its severity to the roentgen changes in the lungs. Their results showed that the main disability from pneumoconiosis, excessive breathlessness on exertion, cannot be distinguished by functional tests from breathlessness caused by chronic non-industrial pulmonary disease. This observation renders it difficult to allocate in an individual the proportion of breathlessness caused by industrial exposure.

In England, the National Insurance (In-

dustrial Injuries) Act (1946) attempts to make such an allocation. Under its regulation there are two separate issues in relation to the disability benefit for pneumoconiosis: *first, its diagnosis,* "has this man got pneumoconiosis within the meaning of the Act?"; secondly, the disability assessment, "to what extent is the man disabled and what proportion of his disability is due to pneumoconiosis?"

Under the Industrial Injuries Act in England, the definition of pneumoconiosis is based on pathology and during life the diagnosis is based on the occupational history and so-called "characteristic" roentgen appearances. Unfortunately, the correlation between roentgenologic and pathologic findings is not perfect. But, the studies of Gilson and Hugh-Jones[27] emphasize two aspects concerning the roentgen diagnosis. First, miners with no roentgen evidence of pneumoconiosis are, on the average, as breathless for their age as men with *simple pneumoconiosis and more* breathless than non-miners, particularly the elderly. This suggested that such miners might logically receive benefit for their disability. Secondly, functional emphysema, which increases with the ascending roentgenographic categories, is part of the same pathologic process described for pneumoconiosis. Under those circumstances, it is regarded as illogical to give extra disability benefit for "emphysema" to disabled men who have the roentgenographic changes of pneumoconiosis.

In *simple pneumoconiosis* there was no average increase in breathlessness on exertion with increase in roentgenographic abnormalities. However, there was an increase in breathlessness normally associated with age, old men being proportionately more breathless than young men whatever the degree of change.

In *complicated pneumoconiosis,* the breathlessness was nearly always severe and increased with the roentgenographic changes, and was augmented even more by advancing age than in simple pneumoconiosis.

In assessing the extent of disability, it is necessary to consider all disabilities as they relate to "loss of health, strength and power to enjoy life." The extent of breathlessness was the main disability to receive Gilson and Hugh-Jones' consideration, and the one which we as radiologists are most concerned about. Their studies indicated that a combination of the standard exercise test and some determination of the maximum ventilatory capacity gave the best objective measure, but that it was probably unsuitable for the Pneumoconiosis Panels. Gilson and Hugh-Jones suggest that possibly the simple procedure of measuring diaphragmatic movement and level is well enough related to maximum ventilation to allow a reproducible division of subjects into 4 or 5 grades pertaining to breathlessness. Their most discriminating test permitted the division of range of breathlessness between normal and extreme disability into nine steps. These authors indicated that there are clear differences between the average degree of breathlessness for each roentgenographic stage and the normals at the same age. They stated: "This 'average' increase of breathlessness at each x-ray stage might justifiably be regarded as that due to pneumoconiosis, and a case could be made out for allotting benefit for 'disability due to pneumoconiosis' simply upon the x-ray-stage at a given age, and this is the most practicable and logical conclusion to draw from our results."[27] The work of Gilson and Hugh-Jones is a magnificent contribution and goes a long way to show the possibilities in this field.

Mobility of Pneumoconiotic Deposits. In 1918, Haythorn[37] noted that in an anthracotic lung there was always a large number of free pigment-bearing cells during the early stages of pneumonia. This observation, as well as his continuing interest in the subject, was somewhat esoteric to me —a young radiologist, because it was thought to be impossible for one to demonstrate the roentgen manifestations of the results of migration of alveolar phagocytes loaded with dust particles. The clinician,

On the other hand, observed that the coal miner continues to have a black sputum for a long time after he gives up work in the mine. Such observations, as well as clinical improvement of some workers after leaving dusty occupations are to be regarded as significant even if one does not become interested in basic physiologic processes.

Fortunately, there are investigators[24,25] who do appreciate the need for basic work with the excursionary activity of alveoli, the mechanism by which dust is removed from the interior of the lung, and the implications that such knowledge may contribute in solving some of the problems of pneumoconiosis.

I now believe that the radiologist may be able to make a minor observation concerning the mobility of pneumoconiotic deposits, possibly in welders and workers in other opaque dusty occupations such as barium and tin oxide workers. This opinion is based on the observation of a considerable disappearance of the pattern of hemosiderosis in mitral heart disease following commissurotomy. Follow-up chest roentgenographic studies on individuals like welders, after they are removed from their dust exposure, may provide interesting data on what can be expected from the roentgenologic standpoint.

PNEUMOCONIOSIS AND RHEUMATOID ARTHRITIS (CAPLAN'S SYNDROME)

In 1953, Caplan[6] called attention to an association of unusual massive lesions in the lungs of coal miners with rheumatoid arthritis. He found an increased frequency of massive lesions in these arthritic persons and, in some, the massive lesions were similar in that they were multiple, round, well defined and distributed rather evenly throughout the lungs. In others, the roentgenologic appearance resembled the usual massive lesions of infective pneumoconiosis. The lesions were not associated with constitutional symptoms nor unusual respiratory distress. Caplan does not suggest that all coal miners with pneumoconiosis and rheumatoid arthritis show a characteristic roentgenologic appearance.

Subsequent to Caplan's observations, others have made additional studies.[25,26,32] Gough, Rivers and Seal[33] have reported on the pathologic findings of the lungs of 14 Welsh coal miners with rheumatoid arthritis and biopsy lung specimens from 2 additional cases. They concluded: "Nodules having a distinctive gross appearance correspond with the radiographic round opacities described by Caplan (Fig. 25). These 'rheumatoid' pneumoconiotic nodules contain necrotic collagen and dust. Tuberculosis was present in several cases. A non-specific inflammation was also found. This is believed to be the 'rheumatoid' component."

Miall[34] has carried out an epidemiologic survey of rheumatic arthritis in a complete community of 9,430 males over fifteen years of age in South Wales and among other things observed that there was a significantly high prevalence of massive fibrosis and tuberculosis among miners with arthritis. Price and Skelton[32] have described widespread nodular lesions in the lungs of a woman, aged fifty-eight, with rheumatoid arthritis. At necropsy, scattered focal lesions were found in the lung, in which there was an active subacute inflammation of the arteries and bronchioles.

The foregoing comments are only a few of the reports relating to some aspect of the possible implication of Caplan's observation (Caplan's syndrome).

We have not made any particular study on the subject of rheumatoid arthritis and pneumoconiosis, although we are interested in any factor which may have a modifying influence on the lesions of pneumoconiosis. Therefore, in an informal manner, we have been collecting roentgen studies on individual workers and patients who have rheumatoid arthritis and pneumoconiosis. I am not prepared at this time to do more than say that I have seen roentgenograms of individuals exposed to relatively small amounts of coal dust and silica which demonstrated lesions similar to those described by Caplan and others. In some of the lesions there was evidence of calcium

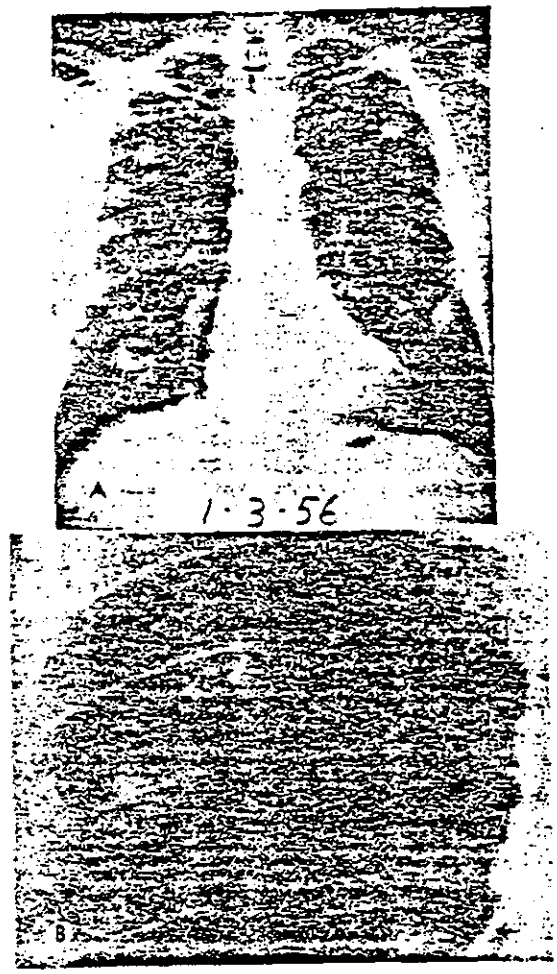


FIG. 25. (*A*) Caplan's syndrome (coal workers' pneumoconiosis with rheumatoid arthritis) in a fifty-six year old individual. This person has been exposed to coal dust for seven years and he has a well advanced rheumatoid arthritis. The diagnosis was made by roentgen examination and has not been confirmed by lung biopsy. (*B*) Close-up of lesions in the right upper lobe. The lesions look very much like malignant metastatic nodes, but this individual has been under observation for some six or seven years and we believe that malignancy can be excluded on the basis that they have not changed in size.

deposition. Whether the calcium was resultant from tuberculosis, histoplasmosis or some other necrotizing process is uncertain (Fig. 25).

SILICOSIS AND PULMONARY CARCINOMA

The radiologist in a general hospital is unable to give an opinion on the incidence of carcinoma of the lung. His patients are usually referred for definitive therapy and occasionally for diagnosis. The radiologist is, however, confronted with a diagnostic problem when he makes an examination of the chest of an individual who has silicosis and who has symptoms that may be interpreted clinically as suspicious of carcinoma.

I have seen silicotic persons who have had persistent hemoptysis without definitive lung lesions which one would interpret as either tuberculosis or carcinoma. Subsequent bronchograms have demonstrated stenosed bronchi and at operation the stenosis was the result of fibrosis and scar contraction and not due to carcinoma. On other occasions, I have seen patients with hemoptysis, unilateral massive lesions, and suspicious Papanicolaou smears. No carcinoma was found at operation.

When the silicotic patient has multiple conglomerate lesions with a pattern of nodulation, it is impossible or at least improbable that one can make a roentgen diagnosis early enough for a curative surgical procedure. If there is any doubt as to the identity of the lesion, one should utilize all diagnostic means, including bronchography, selective angiography, and Papanicolaou smears. If one is still uncertain, we recommend surgical exploration.

We have noted that a number of silicotic patients often are referred directly to the thoracic surgeon with a presumptive diagnosis of carcinoma of the lung. These patients have frequently been either hard or soft coal miners. In every instance with 2 exceptions, the roentgen diagnosis has been silicosis with infection. One of the exceptions, diagnosed as carcinoma, showed a narrow and funnel-shaped bronchus surrounded by a mass. The lesion was a silicotic mass. The other exception was a lobar consolidation, typical of atelectasis from a bronchial obstruction. That was a carcinoma.

The physician, in any discipline, who is unfamiliar with some of the vagaries of silicosis and other forms of pneumoconiosis

...ll conclude that a unilateral conglomer... or massive lesion in the absence of a ...ttern of nodulation should be regarded ...carcinoma until proved otherwise. That ...especially true if there is a high index of ...picion for carcinoma of the lung. I have ...nd laminagraphy most helpful when in ...bt about the nature of a massive lesion ...pneumoconiosis. Distortion of bronchi, ...ium deposition, small emphysematous ...eas around the lesions, and possibly other ...all silicotic lesions assist in excluding ...arcinoma as the first choice for diagnosis.

There are two types of carcinoma seen in ...tients with silicosis that frequently are ...diagnosed. One group includes those ...amous carcinomas which become necro... in the center, break down and subse... ently the patient coughs out most of the ...rotic material leaving a fairly thick-...lled, large, oval cavity, which gradually ...reases in size. One rarely, if ever, finds ...berele bacilli in the sputum. The lesion ...y or may not extend to and destroy the ...acent ribs (Fig. 26). Oftentimes, the pa... nt does not complain of much pain, even ...gh, the ribs are eroded. Because of the ...e cavity and rib erosion and silicosis, ...re is an unbelievable urge to call such ...ns tuberculosis. Tuberculous cavities ...y either thin walled or, as so often hap...s, thick walled. The cavity is irregular ...; is rarely larger than 5 to 10 cm. in di...eter. The cavity noted in primary carci...ma of the lung is usually 5 or more cm. in ...meter when first seen. There is prac...ally no reactive lung lesion around the ...l of the cavity.

The second group of carcinomas that ...e likely to be missed includes those that ...elop slowly amid shadows which may ...ve been interpreted as due to silicosis ...tuberculous infection. Carcinoma is not suspected ...tuberculosis is thought to be the pre...sive modifying infection. When the ...inoma has grown large enough, the ...nus is obstructed and atelectasis ...es place. The atelectasis produces a sud... increase in the roentgen manifesta... Such rapid progression of the shadow density in the roentgenogram, rarely, if ever, takes place in tuberculosilicosis.

Lung lesions which have migrated to the hili produce shadows that look not unlike lymphoblastomas or other mediastinal tumors. When dealing with pneumoconiosis, one should adhere strictly to a rule of not giving radiation or chemotherapy for a presumed diagnosis of malignancy. Biopsies for confirmation of the diagnosis are recommended; otherwise, tremendous damage to the tissue might occur.

Under pathologic considerations, comment has been made on the incidence of carcinoma of the lung among silicotic persons, but there are several other studies that should be mentioned. Schoch[73] in an investigation of material from the Swiss accident insurance organization found no etiologic relationship berween silicosis and primary carcinoma of the lung. Wall,[85] in a postmortem study of a series of 100 cases of anthracosilicosis, found 4 cases of primary carcinoma of the lung. James[41] reports that primary lung carcinoma was found at necropsy in 3.3 per cent of 1,827 South Wales coal miners and in 5.4 per cent of 1,531 South Wales non-miners. In 12, the tumor was in the mass, and touching it, in 5. In 7, the tumor was remote. The tumor was similar in the histologic variants, in the age incidence, and in the distribution of metastases as that seen in the non-miners.

ASBESTOSIS AND PULMONARY CARCINOMA

I personally have not studied any cases of carcinoma of the lung in asbestosis. The roentgen studies of the cases that I have seen have not been serial studies, but have been roentgenograms of advanced lesions, such as atelectasis of a lobe, a large mass adjacent to the hilus or a unilateral lesion in an upper lobe. Lynch and Pratt-Thomas[49] indicate that the statistical evidence for a causal relation between lung carcinoma and asbestosis is strong. An opposite opinion is expressed by Jacob and Bohlig.[40] In Dresden, carcinoma among asbestos workers seems to be rare. Four cases of fatal carcinoma of the lung were found in 339

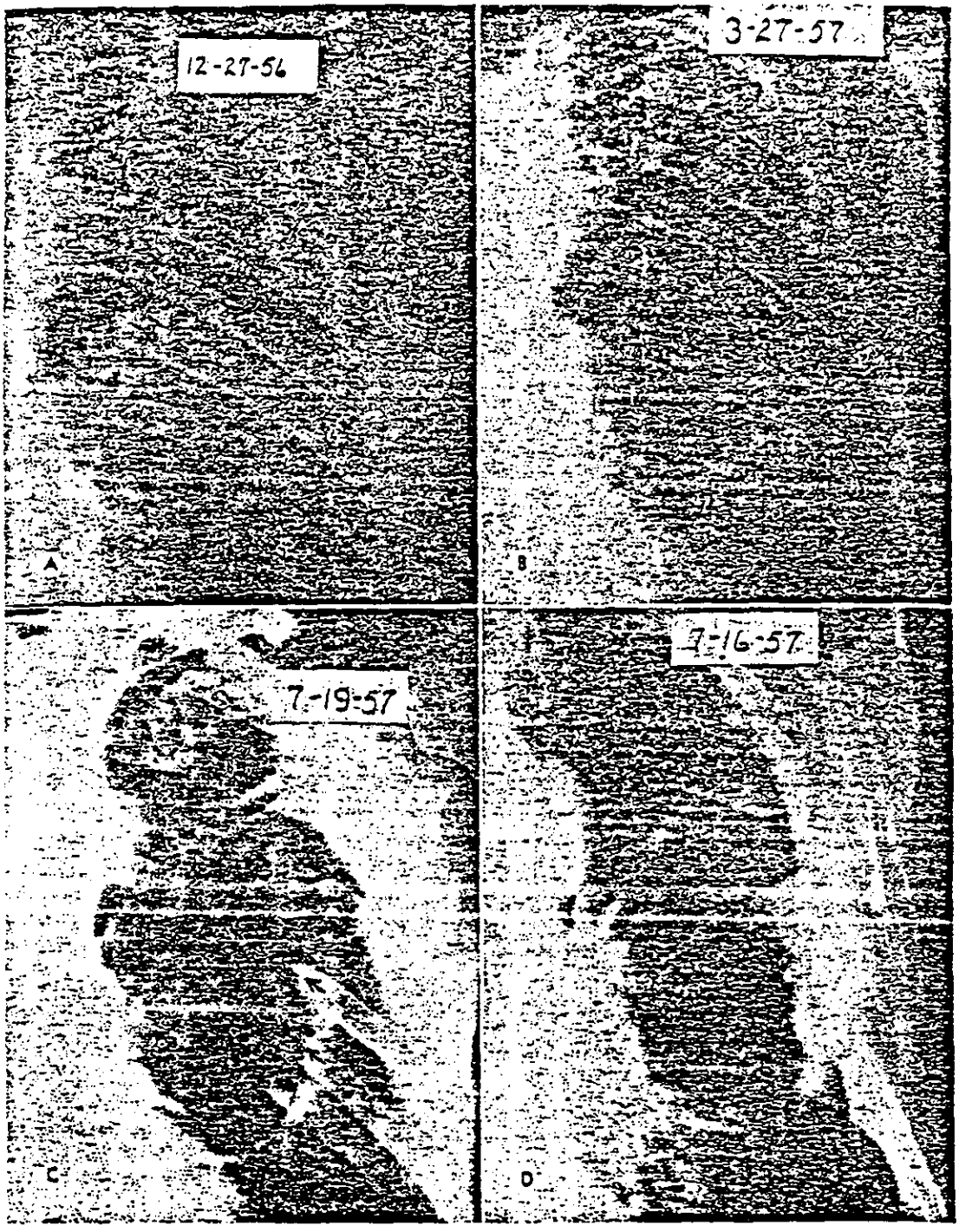


FIG. 26. (*A*) Carcinoma of the left lung in a seventy-one year old white man who had been a coal miner for forty years. He had been in his usual health except for increasing shortness of breath and chronic cough until November, 1956, when he developed pain in the left side of the chest and hemoptysis. A roentgen examination on December 27, 1956 showed a large cavity in the upper portion of his lower lobe. It was thought to be tuberculosis and anthracosilicosis. Note that the ribs are not involved. (*B*) Roentgenogram made in March, 1957. The appearance was essentially the same except that this time the sixth rib was eroded. (*C*) Roentgenogram made in July, 1957. The cavity was larger and portions of four ribs were destroyed. We thought the lesion was a carcinoma which had largely excavated itself. A biopsy confirmed the diagnosis. (*D*) A slightly oblique roentgenogram shows the wall of the lesion adjacent to the thoracic wall (arrows).

asbestos workers from 1930 to 1954. Doll,[15] in a study of 105 autopsies of asbestos workers, found 15 carcinomas of the lung associated with asbestosis. He concludes that lung carcinoma is a specific industrial hazard of certain asbestos workers and that the average risk among men employed twenty years or longer is 10 times that experienced by the general population.

PNEUMOCONIOSIS AND PULMONARY CARCINOMA

Carcinoma of the lung is a major cause of death today; therefore, the relationship of environmental factors to the etiology of lung cancer is receiving increasing consideration. Epidemiologic studies have been carried out in many industries, and are likely to increase, as guide-lines are evolved and a program of confidence is developed between governmental agencies, labor, and industry.

SUMMARY AND CONCLUSION

1. Pneumoconiosis continues to be a health hazard, national and international in scope.

2. There are many dedicated individuals in science, in medicine, in organized labor, in industry, in insurance, in the legal profession, and in a number of agencies in our local, state and national governments who would welcome a united effort to prevent pneumoconiosis and its complications.

3. Laws governing work conditions and workmen's compensation have been helpful, but, organized continuing education is likely to be a better approach in this country because, unlike England, we have 48 states, most of which have different codes of laws. In 1936, the United States Secretary of Labor, Frances Perkins, recognized the need for an organized educational effort to provide industry, labor, medicine, and government with an informed opinion concerning silicosis. After the several committees wrote their reports[57,58,59,60,61] for publication, the committees were discharged. Conditions have changed since the publication of the Perkins committees' reports. Many of the industrial processes elaborating dust are new. Scientists now believe that submicroscopic dusts should receive major consideration. It may be necessary to re-evaluate the concepts concerning the amounts of dust that constitute safe levels. Methods of dust counting may need revision. Development of aerosols for control of dust hazards seems to be increasingly effective. There are many, many other phases of this problem which involve careful consideration by disciplines outside of radiology, but radiology has an important task to perform.

4. The radiologist plays a major role in the diagnosis of pneumoconiosis in the living individual. He assumes a role of increasing importance in the estimation of disability and in the results of rehabilitation.

It is said that the majority of controverted compensation cases involve questions of fact rather than questions of law.[39] Most of the fact cases involve a medical question.[39] Some of the questions are: What is the diagnosis? When does the condition become disabling?[84] When should the employee, subject to exposure, be removed from his employment?[84] When should such employee be deprived of his privilege of continuing at work in the occupation in which he may be engaged?[84] What methods are available to medical science to pronounce that the employee is totally disabled?[84] What sequelae result from a diagnosis of partial disability?[84] Should the employee at that time terminate his employment?[84] These are some of the imponderables with which the physician and the lawyer are faced in their consideration of pneumoconiosis in an individual seeking compensation. It is estimated that workmen's compensation administration is about 80 per cent medical.[39]

The satisfactory operation of the workmen's compensation system depends largely on competent and adequate medical treatment of injured employees, the prompt furnishing of accurate medical reports on injuries and the giving of medical

testimony at hearings on controverted claims. In compensation cases, the physician's responsibility is not confined to the patient but extends to the community, in the persons of the members of the industrial accident commission and to the involved industry." The radiologist will be able to serve the patient and the community better if the profession can agree on guide-lines of diagnosis and extent of disability. This has been done in England and other countries. We should be able to develop working guide-lines for the United States. Guide-lines for diagnosis and disability ultimately would provide essential data for the scientist experimenting with new techniques for humans and the biometrician analyzing epidemiologic and environmental data of communities where there are dust hazards.

It would provide information for the State Pneumoconiosis Panels and industrial accident commissioners which should not be susceptible to as much disagreement among medical experts as obtains now.

Our present method of medical education does not provide for the implementation and the follow-up information that are necessary in controlling pneumoconiosis. Often times, medical practice does not keep pace with new and acceptable concepts, especially, when new data develop within a discipline outside of medicine.

5. Some of the other groups concerned with the problem of pneumoconiosis include: the general physician; the internist; the cardiologist, the pulmonary physiologist; the allergist; the phthisiologist; the thoracic surgeon; the bronchologist; the physical therapist (rehabilitation); the pathologist; the experimental pathologist; the public health officers; members of Bureau of Standards; United States Department of Labor; the industrial hygienists; the air pollution engineers (ventilation, aerosols, etc.); the industrial physicians; the psychiatrist; the representatives of labor, industry and insurance; the lawyers; the physicist; the biometrician; and the toxicologist.

6. It is suggested that the American Roentgen Ray Society initiate through its President and its Executive Council a proposal that the National Academy of Sciences request the Committee on Radiology of the Division of Medical Sciences of the National Research Council to formulate plans for the inauguration of a *National Committee on Pneumoconiosis Protection and/or Control.* A prototype of such a committee is the National Committee on Radiation Protection. The National Committee on Radiation Protection is so constituted that all interested groups are represented. It is a continuing committee which keeps informed on new developments and, with similar committees from other countries, agrees on acceptable methods and standards of good practice. Its reports are published in scientific journals and handbooks and are made available to everyone concerned. These reports are educational and informative, but are not regulatory.

7. Finally, if we as radiologists will accept the challenge presented by the problem of pneumoconiosis and do something constructive about it, we can feel assured that the ideals and conquering spirit of Caldwell and other great pioneers, indeed, will continue to live.

Department of Radiology
Hospital of the University
of Pennsylvania
Philadelphia 4, Pennsylvania

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