FILE NAME: Owens Illinois Library (OWL)

DATE: 1955

DOC#: OWL040

DOCUMENT DESCRIPTION: Several Articles Related to Illness from Asbestos

A. M. A.
ARCHIVES OF
Industrial Health

VOLUME 11
1955

PUBLISHERS
AMERICAN MEDICAL ASSOCIATION
CHICAGO 10, ILL.

value of which is
riate summing of
sion capacity, and
upon its solubility
sorbed through the
rate which is pri-
e or another of the
pendent of at least
the determinations
es it becomes pos-
of V_e, D, and B_e,
tilation, diffusion,

y makes use of
which is retained
entilation, is only
d flow, and is in-
) acetylene, which
o ventilation and
dent of diffusion,
which is retained
and diffusion and
w. These are in-
hysiologically ac-

dure for measur-
ptake and calcu-
re described, and
eries of subjects
ory impairment)

by the method of
ulmonary funcl-
lly described and
obtained of the
lation, gas trans-
lveolar wall, and
ibilities for deter-
itude of respira-
istinguishing be-
, circulatory, or
suggested.
l for calculating
spiratory system
l carbon dioxide
by partitional

m a greater va-

procedure. For these, a compact, portable apparatus is needed, and methods of gas analysis are required which will permit the measurements of the three test gas concentrations in one test gas mixture. In this way, simultaneous values of V_e, D, and B_e can be obtained in a single test, at rest or under exercise.

Mr. James Brown constructed and assembled the apparatus used in this study. Messrs. Edward McCloskey and Donald Ross assisted in the development of test procedures and in the conduct of many of the tests. Arrangements for the coal miners who came as test subjects were made through the cooperation of Drs. Leslie Falk and Edward Lebovitz, of the Health and Welfare Fund of the United Mine Workers of America. Several of our associates also served as test subjects.

REFERENCES

1. Kety, S. S.: Theory and Applications of the Exchange of Inert Gases at the Lungs and Tissues, Pharmacol. Rev. **3**:1, 1951.

2. Haggard, H. W.: Absorption, Distribution and Elimination of Ethyl Ether: I. Amount of Ether Absorption in Relation to the Concentration Inhaled and Its Fate in the Body, J. Biol. Chem. **59**:737, 1924.

3. Fowler, W. S.: Lung Function Studies: III. Uneven Pulmonary Ventilation in Normal Subjects and in Patients with Pulmonary Disease, J. Appl. Physiol. **2**:283, 1949.

4. Fowler, W. S.; Cornish, E. R., Jr., and Kety, S. S.: Lung Function Studies: Analysis of Alveolar Ventilation by Pulmonary N_2 Clearance Curves, J. Clin. Invest. **31**:40, 1952.

5. Robertson, J. S.; Siri, W. E., and Jones, H. B.: Lung Ventilation Patterns Determined by Analysis of Nitrogen Elimination Rates: Use of the Mass Spectrometer as a Continuous Gas Analyzer, J. Clin. Invest. **29**:577, 1950.

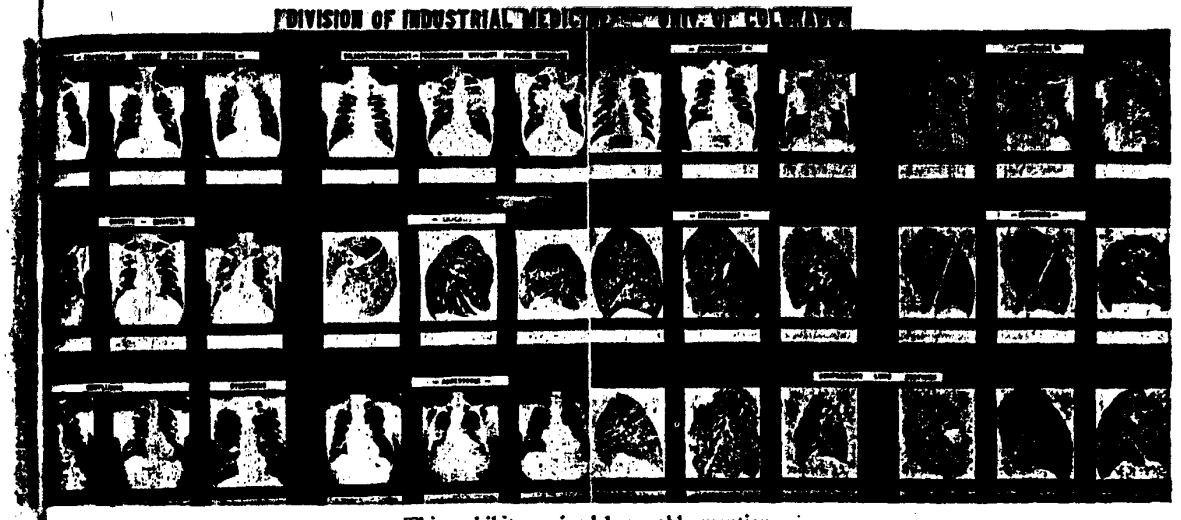
6. Hatch, T. F.: Carbon Monoxide Uptake in Relation to Pulmonary Performance, A. M. A. Arch. Indust. Hyg. **6**:1, 1952.

7. Rahn, H.; Mohney, J.; Otis, A. B., and Fenn, W. O.: Method for Continuous Analysis of Alveolar Air, J. Aviation Med. **17**:173, 1946.

8. Hatch, T.; Cook, K. M., and Palm, P. E.: Respiratory Dead Space, J. Appl. Physiol. **5**:341, 1953.

9. Silverman, L.; Lee, G.; Plotkin, T.; Sawyers, L. A., and Yancey, A. R.: Air Flow Measurements on Human Subjects With and Without Respiratory

Vol 11, 1955



This exhibit received honorable mention.

Scientific Exhibits

PNEUMONOCONIOSIS

ROBERT F. BELL, M.D.
AND
JAMES J. WARING, M.D.
DENVER

THIS EXHIBIT represents a collection of some of the representative teaching material on pneumonoconiosis utilized at the University of Colorado Medical Center. It is presented to the practicing physician for his examination because at any one location he may have had only a rare opportunity to see and compare various types of pneumonoconiosis. Many large films of the pneumonoconiosis and other roentgenograms of diseases resembling pneumonoconiosis were available for examination on the view box.

Shown as a scientific exhibit of the Section on Preventive and Industrial Medicine and Public Health at the 103rd Annual Meeting of the American Medical Association, San Fran-

W

. F. V., 425
. J. J., 159
rby, J. H., 102, 413
k. M. L., 473, 479
E. E., 305
J. D., 422
H. B., 11
i. J. P., 403, 408
G. W., 196
H. N., 457

Y

i. S., 413
D., 280
r. W. J., 280

955

No.	Month
4	April
5	May
6	June

SUBJECT INDEX TO VOLUME 11

The following Index contains an alphabetical list of significant subjects presented in this volume. Abstracts are indexed by category under the heading "ABSTRACTS." Books reviewed are listed alphabetically by title under the heading "BOOKS." Obituaries and death notices are indexed under the heading "DEATHS."

ased on human experience, but in most cases they are calculated, using iments. The value of Q is considered to be the microcuries of a radio total body that will deliver 0.3 rem per week to the critical organ, i. e., radioisotope that results in the greatest over-all damage to the body. ncentrations of the radioisotopes in air or water that if used exclusively ill lead to an accumulation, Q, in the body. In most cases considered, a few weeks. MPC values for air and water to be used in an emergency dose of ionizing radiation is delivered to tissue in close proximity to an specific activity that becomes fixed in living tissue. In setting the MPC ld like to know if such localized radiation increases the probability of

NUCLEAR SC. ABST.

RADIATION INJURY. LOUIS H. HEMPELMANN and JOSEPH G. HOFF- clear Sc. **3**:369-392, 1953.

ld reactions of the human body to ionizing radiations are described. es, the first part of the paper deals with the description of acute radia- owing this, a brief appraisal is made of the possible lines of therapeutic ggested by current animal experimentation. The last part of the paper high to subacute doses and describes late tissue reactions which arise at

FROM THE AUTHORS' SUMMARY.

DUST. H. I. MILLER JR., Heat. Pip. & Air Condit. **26**:109-113 (Sept.)

he problems in designing heating, ventilating, and dust-control facilities large uranium producing plant operated for the Atomic Energy Com- e. The purpose of this article is to indicate the application of standard o the control of dust and atmospheric pollution in an unusual process degree of control is necessary.

FROM THE AUTHOR'S SUMMARY.

TECHNIQUES. SAUL J. HARRIS, New York State Dept. Labor Month.) 1953.

examples of industrial applications of radiation-producing equipment Also included is a general discussion of ionizing radiations, including feguards.

A. T. ROSSANO JR., New York.

IONS FOR NUCLEAR POWER PLANTS. H. HURWITZ JR., Nucleonics 54.

g the energy released in a nuclear accident and for evaluating the dispersed fission products resulting from such an accident are reviewed.

NUCLEAR SC. ABST.

M THE USE OF DENTAL X-RAY UNITS. W. E. NOLAN and H. W. **61**:625-696 (Oct.) 1953.

ntists, technicians, and patients during dental roentgenography were ions for safe procedures are presented.

NUCLEAR SC. ABST.

D RESPIRATORY PROTECTION IN RADIOISOTOPE WORK. G. W. CHANAN, Publication AECU-2821, U. S. Atomic Energy Commission, Service, Oak Ridge, Tenn., Jan., 1953.

iated with the use of radioactive isotopes in the laboratory is dis- asures to prevent contamination are emphasized. The various forms nd mechanisms influencing their stability in the air are discussed. imum permissible air contamination values, the pathway of inhaled er the respiratory system, and the characteristic absorption, deposi- on pattern of contaminants. The characteristics of various respiratory

OCCUPATIONAL DISEASES OF THE LUNGS

Recent Trends in Industrial Health

A. J. LANZA, M.D., New York

On behalf of the Institute of Industrial Medicine, may I express my appreciation of the opportunity to participate in this rather unique meeting? May I also express my admiration for the work which Dr. Marble and his associates in the Massachusetts Medical Society have done in achieving the turnout that we see here today?

Industrial medicine is not static. Research goes on and clinical knowledge progresses, so that all of us continue to learn more and more. That being the case, it does not behoove any of us to be too dogmatic. We must learn to adapt our thinking to the changes in our knowledge and, if necessary, to change our minds. When I was a boy, they used to have a saying, "It was only a wooden Indian and a certain kind of fool who wouldn't change his mind." And occasionally we have to make up our minds that we have to change our minds.

This matter of occupational diseases of the lungs is not a simple one. It is very complicated and, as in all chronic diseases, presents a number of variables, the effect of which we have not always learned how to evaluate. We have learned a good deal about silicosis. We have learned something about asbestosis. Those are the two main forms of pneumoconiosis which cause disability and sometimes death. At the same time we must always remember that in spite of the enormous amount of research that has been done in most of the civilized countries of the world on the subject of silicosis—almost enough to fill an ordinary library—we still do not know what is the nature of the action of the silica particle upon pulmonary tissue. Nor do we know why it is that the silicotic is more susceptible than the normal person to tuberculosis.

Likewise, for instance, in asbestosis we do not know what is the nature of the action of the asbestos particle upon the pulmonary tissue. The late Dr. Gardner propounded the theory that, contrary to the action of silica, the action of asbestos was largely mechanical. That may be so. On the other hand, it is only fair to state that there is a considerable body of opinion that does not go along with that. Diagnosis is not always too easy and calls for all the skill that we can muster with respect to getting a careful occupational history, as well as the clinical and roentgenological evidence that may be available. It is incumbent upon us to be very careful and very precise in our use of terms. All too commonly people are apt to use these various terms signifying different types of pneumoconiosis as interchangeable, which is not correct. These different types of pneumoconiosis, like silicosis and asbestosis and siderosis, are different diseases; and if there is any sloppiness in our use of terms, we only confuse ourselves and make the matter of diagnosis and the just treatment of the individual worker who may be claiming compensation difficult.

While it is true that we have a fair knowledge of the action of silica particles and asbestos particles upon the pulmonary tissue

Chairman, Institute of Industrial Medicine, New York University—Post-Graduate Medical School.

Read in the Symposium on Occupational Diseases of the Lungs, sponsored by the Massachusetts Medical Society in cooperation with the Institute of Industrial Medicine of the New York University

under factory or manufacturing conditions, occur as pure homogeneous particles, because there are often other dust particles among them. As pointed out years ago in researches at Saranac, these other dusts present along with silica modify the action of the silica particles. So we have to approach this whole subject with a considerable degree of humility, because there is so very much concerning these diseases about which we know very little. Our only hope here lies in the progress in the type of work that you will hear described later in the afternoon by Dr. Wright.

The pathologists and physiologists are the only ones who hold the key that may unlock the door to further knowledge of what really goes on in the lungs of persons who breathe the various kinds of dust that we find in our working places. So far our estimation of this disability consists mostly of rule-of-thumb methods, and if they can be combined with a certain amount of common sense, we hope that the standards of dealing with claimants for disability may be of a moderately high order.

Again let me emphasize that there is a great deal of work to do. There is a great deal of knowledge that we do not have. You will hear much more on that from our distinguished speakers this afternoon.

Pathology of Asbestosis

KENNETH M. LYNCH, M.D., Charleston, S. C.

While the asbestos industry may date its beginnings back into antiquity and while the condition now known as asbestosis may have occurred through a long period of time, the industry as we know it is new, and the health hazard related to it is of recent knowledge. In fact, the term asbestosis was not entirely acceptable as recently as about 30 years ago, while up to that time any thought of ill effects of occupational exposure to asbestos dust tended to confuse the resulting disease with silicosis. Parenthetically, perhaps it is permissible to express an opinion that before we have finished with the study of the class of pulmonary disease attending exposure to dust in industry the whole chapter will need to be revised.

It is within the time of experience of some who are still concerned with the study of certain unsolved problems in the subject that there has been established this distinct variety of pneumoconiosis. During the same period, and probably largely because of disclosure of the hazard, there have been improvements by the industry to protect workers, so that, so far as my observations go, the atmospheric conditions in asbestos mills are greatly different from the conditions of 30 or more years ago. Even though improvements have been made and although knowledge of the course of the disease has come largely from examination of workers whose employment went back into the very dusty conditions of earlier times, it should not be assumed that the hazard has been eliminated. Harmful asbestosis is still occurring.

Asbestosis is the product of the inhalation of asbestos dust in sufficient amount and during a sufficient period of time; but under working conditions, either now or formerly, there is no uniformity in degree of disease among workers, even under similar exposures. Apparently some persons in a group working in the same environment will be severely affected, while others will be less affected, and some will escape harm, and this for reasons unknown. Furthermore, the time required for similar quantitative exposures to produce like grades of asbestosis apparently varies with individuals.

While in our efforts to establish protection of the health of industrial workers we have set up certain standards concerning atmospheric dust concentrations, these are of a general nature rather than specific, and may have given a false sense of security. The very dusty atmospheres surrounding the workers in this as well as in certain other industries under former conditions would certainly be more harmful than the relatively clean environment to be found in a present-day careful operation, but we may need to establish something more than merely the particle counting and measuring practices now in vogue if we shall have dependable safety controls.

The particulate matter in asbestos-plant dust is composed of broken fibers of crystalline silicates. When inhaled, most of it is expelled, but the quantity of fragments of lengths up to 100μ or more that may reach the terminal respiratory units is remarkable. As compared with the material deposited in the lung in silicosis and in certain other con-

President, Medical College of South Carolina.

Read in the Symposium on Occupational Diseases of the Lungs, sponsored by the Massachusetts Medical Society in cooperation with the Institute of Industrial Medicine of the New York University

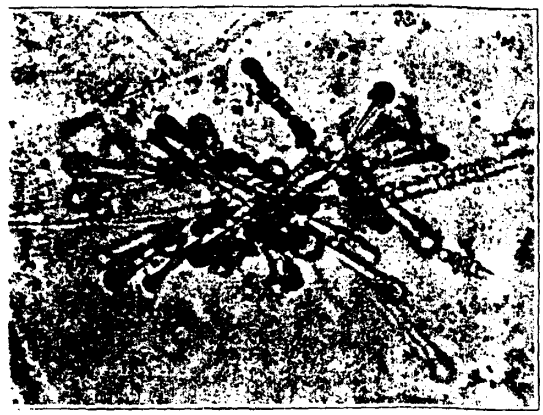


Fig. 1.—Asbestosis bodies in sputum concentrate; reduced from a photomicrograph. × 705.

terial apparently exerts physical rather than chemical influence. At least it now appears that while free silica is in a crude sense chemically poisonous to living tissues when deposited within them asbestos crystal fragments may traumatize living tissues by their physical qualities. Hence the present belief that it is the larger particles that do the damage here, as contrasted with the harmful effects of the smaller particles in silicosis.

It is fitting to that conception that, possibly by a protective mechanism, the asbestos fragment remaining in the lung becomes enveloped by foreign-body giant cells and covered by a smooth coating, apparently of colloidal

Fig. 2.—Grade III fibrosis, with asbestosis bodies, phagocytes, and black granular pigment in reduced alveoli; reduced from a photomicrograph. × 280.

nature and of tissue fluid or cell origin. This process transforms the naked asbestos crystal into a golden or brownish object of a variety of architectural shapes, called the asbestosis body. This characteristic body is formed in the alveolus of the lung. It may be found in the sputum of a subject with asbestosis, as a confirming diagnostic finding, and in the fluid extracted from the lung. Because of their size, comparatively few asbestosis bodies, and only the smaller ones, may be transported in the lymphatic system to be deposited in lymphoid deposits within the lung, while still fewer reach the hilar or mediastinal nodes.

The limitation of physical opportunity for entry of the asbestos fragment into and transportation by the draining lymphatics seems to explain another characteristic difference between silicosis and asbestosis. The small free particles of silica readily penetrate the aveolar walls and lung tissues, aided or unaided by phagocytes, and are mainly deposited in the lymphoid depots along the drainage system, thus setting up focal reaction and characteristic nodular fibrosis, whereas asbestos fragments stay mainly where they land in the alveoli, particularly in the vestibular areas of the lobules, so arousing a more uniformly distributed reaction and usually nonnodular or diffuse fibrosis, although there is evidence that they may penetrate the tissues also. As alveoli become obliterated and the lung architecture becomes distorted, the asbestosis bodies become embedded in fibrous patches and masses, to remain of much the same appearance indefinitely, although in the older scars there appears to be some change in them and they may then absorb the basic-staining dyes.

Just what composes the first part of the reaction to asbestos fragments deposited in alveoli is subject only to deduction at the present. If we assume the primary effect to be from physical injury (traumatism) of

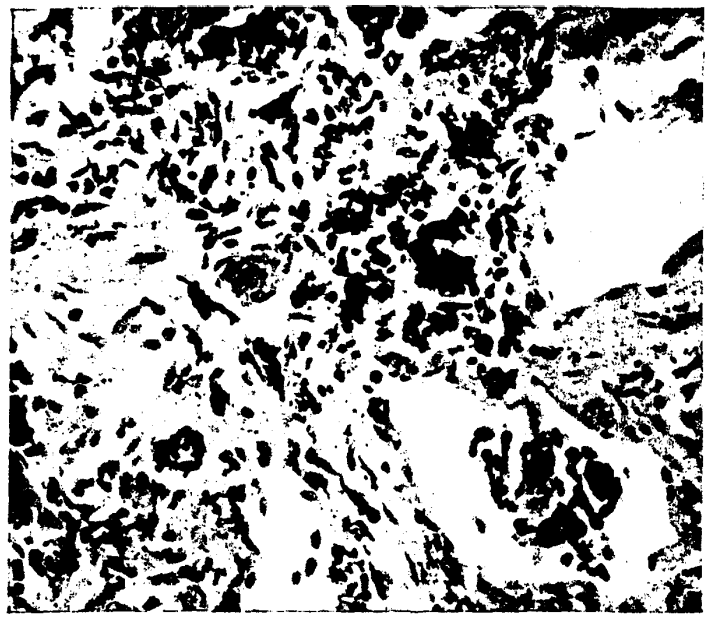


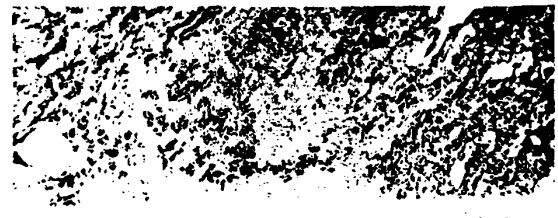
Fig. 3.—Grade IV fibrosis of lung in asbestosis, with asbestosis bodies and granular material in alveolus and fibrous area. × 280.

their exposure, even before there is evidence of fibrosis. It is also consistent with the early "ground-glass" changes in roentgenographic lung fields.

As the condition progresses—ordinarily through years of continued exposure—and while alveolar fluid may continue to accompany the giant cell and colloidal envelopment of the fragments of asbestos crystals as long as fresh deposition continues, permanent change in the form of fibrosis takes over and proceeds. This fibrosis is essentially the same as will occur from any relatively innocuous but irritating presence of solid foreign material located within living tissues. There is some evidence that it may be halted by removal from exposure to continued asbestos inhalation, but after its formation it cannot be expected to be reduced, and when it has reached a disabling grade the disability may be expected to be permanent. In the advanced be encountered, of course, varies with the grade of fibrosis. While that condition tends to be consistent with the degree and duration of exposure to the dust, the causative exposure and the resulting fibrosis do not always agree. Merely the fact that a person has worked in an asbestos plant does not justify an assumption that he has acquired asbestosis of a clinical grade.

In some of our cases the finding of small numbers of asbestosis bodies in the alveoli

Fig. 4.—Advanced Grade IV asbestosis: obliterating hyaline fibrosis.



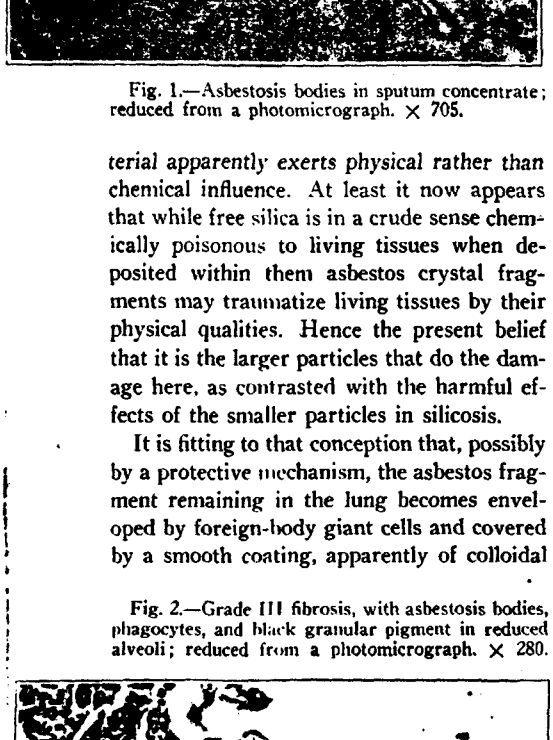
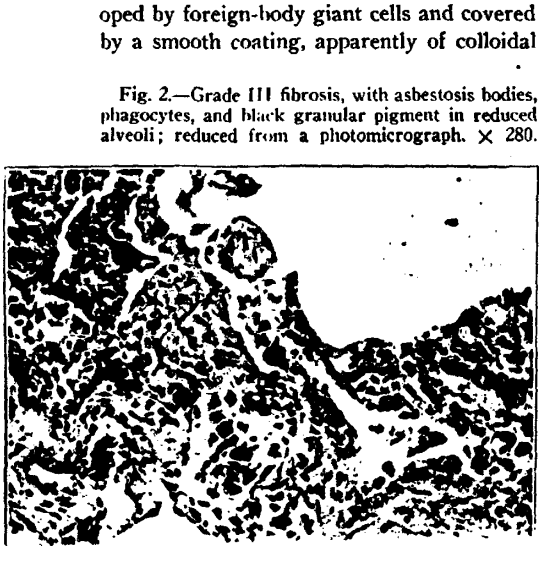


Fig. 1.—Asbestosis bodies in sputum concentrate; reduced from a photomicrograph. × 705.

terial apparently exerts physical rather than chemical influence. At least it now appears that while free silica is in a crude sense chemically poisonous to living tissues when deposited within them asbestos crystal fragments may traumatize living tissues by their physical qualities. Hence the present belief that it is the larger particles that do the damage here, as contrasted with the harmful effects of the smaller particles in silicosis.

It is fitting to that conception that, possibly by a protective mechanism, the asbestos fragment remaining in the lung becomes enveloped by foreign-body giant cells and covered by a smooth coating, apparently of colloidal

Fig. 2.—Grade III fibrosis, with asbestosis bodies, phagocytes, and black granular pigment in reduced alveoli; reduced from a photomicrograph. × 280.



nature and of tissue fluid or cell origin. This process transforms the naked asbestos crystal into a golden or brownish object of a variety of architectural shapes, called the asbestosis body. This characteristic body is formed in the alveolus of the lung. It may be found in the sputum of a subject with asbestosis, as a confirming diagnostic finding, and in the fluid extracted from the lung. Because of their size, comparatively few asbestosis bodies, and only the smaller ones, may be transported in the lymphatic system to be deposited in lymphoid deposits within the lung, while still fewer reach the hilar or mediastinal nodes.

The limitation of physical opportunity for entry of the asbestos fragment into and transportation by the draining lymphatics seems to explain another characteristic difference between silicosis and asbestosis. The small free particles of silica readily penetrate the aveolar walls and lung tissues, aided or unaided by phagocytes, and are mainly deposited in the lymphoid depots along the drainage system, thus setting up focal reaction and characteristic nodular fibrosis, whereas asbestos fragments stay mainly where they land in the alveoli, particularly in the vestibular areas of the lobules, so arousing a more uniformly distributed reaction and usually nonnodular or diffuse fibrosis, although there is evidence that they may penetrate the tissues also. As alveoli become obliterated and the lung architecture becomes distorted, the asbestosis bodies become embedded in fibrous patches and masses, to remain of much the same appearance indefinitely, although in the older scars there appears to be some change in them and they may then absorb the basic-staining dyes.

Just what composes the first part of the reaction to asbestos fragments deposited in alveoli is subject only to deduction at the present. If we assume the primary effect to be from physical injury (traumatism) of alveolar linings and septa, we may assume fluid accumulation. It may constitute the first or acute reaction, and that fits with the physical evidence of increase of lung fluid that

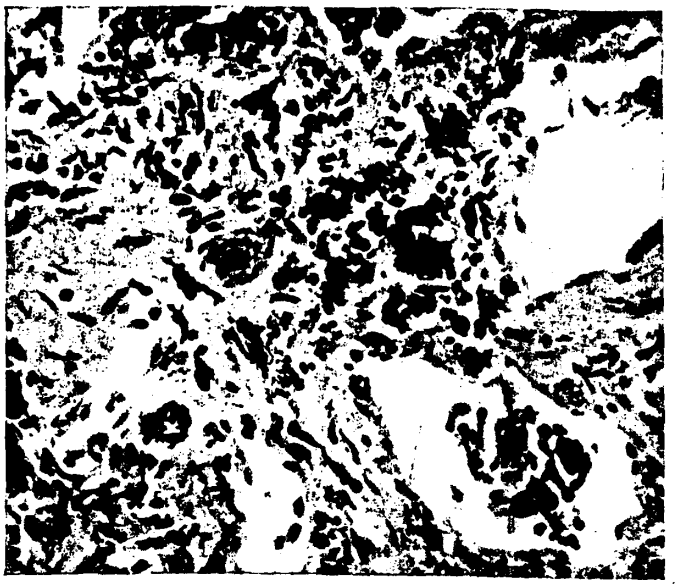


Fig. 3.—Grade IV fibrosis of lung in asbestosis, with asbestosis bodies and granular material in alveolus and fibrous area. × 280.

their exposure, even before there is evidence of fibrosis. It is also consistent with the early "ground-glass" changes in roentgenographic lung fields.

As the condition progresses—ordinarily through years of continued exposure—and while alveolar fluid may continue to accompany the giant cell and colloidal envelopment of the fragments of asbestos crystals as long as fresh deposition continues, permanent change in the form of fibrosis takes over and proceeds. This fibrosis is essentially the same as will occur from any relatively innocuous but irritating presence of solid foreign material located within living tissues. There is some evidence that it may be halted by removal from exposure to continued asbestos inhalation, but after its formation it cannot be expected to be reduced, and when it has reached a disabling grade the disability may be expected to be permanent. In the advanced stage it causes respiratory embarrassment and finally circulatory difficulties resulting in increased right heart burden.

The gross condition of the lung, as seen be encountered, of course, varies with the grade of fibrosis. While that condition tends to be consistent with the degree and duration of exposure to the dust, the causative exposure and the resulting fibrosis do not always agree. Merely the fact that a person has worked in an asbestos plant does not justify an assumption that he has acquired asbestosis of a clinical grade.

In some of our cases the finding of small numbers of asbestosis bodies in the alveoli

Fig. 4.—Advanced Grade IV asbestosis: obliterating hyaline fibrosis.

with preservation and at times exaggeration of the linear markings.

Stage II—Second-Degree Silicosis.—The nodules are from 2 to 3 mm. in diameter and are of sufficient size to obscure the linear markings to a great extent.

Stage III—Third-Degree Silicosis.—The nodules are more than 3 mm. in diameter. Small areas of coalescence may be seen.

B. SILICOSIS WITH CONGLOMERATION

This is probably the result of interaction of an infectious organism, not necessarily the tubercle bacillus. It is conceivable that there is no relationship between the silicotic nodule and the infectious element.

C. SILICOSIS WITH TUBERCULOSIS

This may occur as the result of an infection superimposed upon progressive and still active silicosis or of such an infection superimposed upon an old and already stabilized silicotic process.

Diffuse obstructive emphysema is known to be one of the complications of silicosis. It is seldom clinically manifested in cases of simple discrete nodular silicosis, but conglomerate silicosis is almost always complicated by it in some degree. I do not mean to imply that chronic pulmonary emphysema is never detected in a person who has discrete nodular silicosis but to raise a question as to the underlying cause when the two are associated. Is the emphysema directly related to the pulmonary deposition of quartz, or is it an entirely independent process?

Persons with diffuse obstructive emphysema have a reduced maximum breathing capacity and an increased residual air. It is felt that important knowledge concerning the actual residual air/total volume ratio can be obtained from an adequate chest fluoroscopy and from films made in full inspiration and full expiration. It would appear logical that the area enclosed within the silhouette of the thoracic cage at full inspiration should bear a relationship to the total volume of air within the lungs and that the silhouette of the thoracic cage at full expiration should

A short time ago Dr. George Wright made a study of 134 cases that showed a good correlation of residual air/total volume with area of roentgenologic expiration/area of roentgenologic inspiration, the coefficient of correlation being 0.772, with a standard error of 0.02 (Fig. 3). Therefore it seems permissible to say that when diffuse obstructive emphysema is a complication of the silicotic process fluoroscopic examination of the chest and a chest roentgenogram made in full expiration should furnish information as to the presence of pulmonary function impairment. The moderate and severe forms should not be difficult to identify, but it is not easy to be definite about persons with slight impairment. I know of no way in which we can determine accurately the degree of diffuse obstructive emphysema other than by employment of pulmonary functional studies.

The discrete generalized nodular pattern, as visualized in the chest roentgenogram of a person who has had an adequate exposure to quartz, reflects the unit pathological lesion of silicosis—a nodule of hyaline fibrous tissue. This roentgenological finding does not imply the presence of pulmonary disability or that the abnormal densities will be progressive in nature (Figs. 4*A*, 4*B*, 5*A*, and 5*B*). At the present time there are no statistical data available, to my knowledge, which would confirm a general impression that all cases, or even the majority of cases, of simple silicosis are progressive. One such study has recently been started and will be reported on at a later date.

Not infrequently the roentgenologic differential diagnosis of siderosis and silicosis is said to be a source of considerable difficulty. One of the main features, other than the occupational history, is that in simple silicosis there is always accentuation of the hilar regions, in contrast to the normal appearance of the lung roots in a person who has been exposed to iron oxide only (Figs. 6*A* and 6*B*). If a worker has had adequate pulmonary deposition of both quartz and iron oxide, as is the case in some industrial occu-

was merely incidental. The lung in such an instance exhibits nothing else of that connection. Usually the occupational story reveals that the person worked in an asbestos plant perhaps even for several years but some time previously. Merely for recording purposes, we have designated the condition as Grade I.

Grade II is also an incidental finding. It means an estimated thickening of alveolar walls and perivascular fibrosis, neither being remarkable, together with asbestosis bodies in alveoli and scattered through the fibrous framework. The lung of that grade may be somewhat coarsened in texture but may not appear grossly abnormal.

Grade III is associated with definite respiratory difficulty, but up to this phase apparently progress of the disease may be halted by removal from exposure, and the condition remains in status quo. It is not per se a fatal state. The anatomical grading is based upon autopsy examination when death has occurred for another reason. The lung in this case is of coarsely honeycomb fibrous texture, nonelastic and distended. This quality is general, although not entirely uniform. Particularly in the upper parts there are patches of distorting indefinite scarrings, and there is a grayish marbled cast on section. The pleura may be thickened and adhesions may be found, but in some instances it may be quite normal.

Grade IV is the advanced phase which will terminate fatally on its own account if some other death-dealing condition does not intervene. The subject becomes increasingly, and finally absolutely, disabled in respiratory and circulatory functions.

The lungs in this final phase are tough, coarse, inelastic, fixed in expansion, grayish marbled in appearance, and generally, but irregularly, in an extreme state of fibrous induration. Various distorting scarrings occur, particularly in the upper parts. Although such scarrings are patchy rather than nodular, they sometimes resemble, both grossly and microscopically, the lumps and nodules of silicosis. The pleura may be thick, even cartilaginous in toughness, and the cavity partly or entirely obliterated, but not inevitably. Inasmuch as pleural fibrosis is not invariable, it appears that its occurrence is as a complication and not as a part of the primary disease.

Roentgenologic Aspects of Silicosis and Asbestosis

LEONARD J. BRISTOL, M.D., Trudeau, N. Y.

A complete roentgenologic study of the chest should include fluoroscopic and roentgenographic examination. Both of these procedures lend aid in evaluating the anatomy of the chest, some of the physiologic aspects of respiration, and the presence or absence of abnormal shadows. It is imperative that one be aware of variations seen in the healthy chest before an attempt at accurate interpretation is undertaken. The lack of understanding of such variations and roentgenograms of poor quality are frequent causes of incorrect diagnosis.

The normal lung structure is produced by the pulmonary artery and its branches, and these are visible from the hilum to the periphery. These branches divide and subdivide, forming finer and finer ramifications. The injection of a contrast medium into the pulmonary artery of a normal excised inflated lung will render an excellent illustration of how the massive contrast-filled vessels stand out sharply against the air-containing alveoli and bronchi (Fig. 1).

In the conventional postero-anterior projection the branches of the pulmonary artery overlap one another and present a definite network-like pattern. In viewing a single chest roentgenogram there is much overlapping, and unless we are aware of what these "white shadows" represent, when they are linear or sometimes nodular in character, we may incorrectly diagnose them as indicating a lesion. The appearance of blood vessels in the frontal view varies greatly in normal persons. Usually the older the person the heavier the linear markings, possibly due to sclerotic changes in the pulmonary arteries. Bronchial asthma and frequent respiratory infections are also known to cause heavy linear shadows. The roentgenogram of the healthy chest presents not one pattern but a series of merging patterns covering a wide spectrum, ranging from that with very minimal fine markings to one with manifest coarse linear shadows. Therefore, without supportive clinical evidence an apparent increase in the vascular pattern per se can have little specificity in roentgenologic diagnosis of pulmonary disease. In all in-

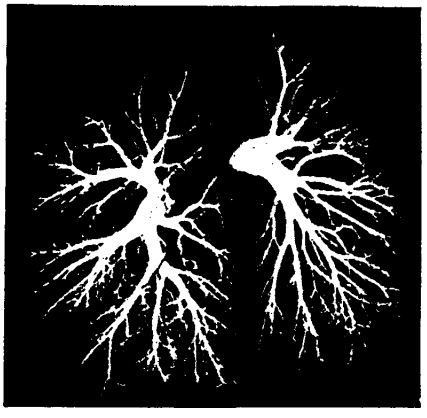


Fig. 1.—Excised inflated lung. The pulmonary artery has been injected with iodized oil-U. S. P. (Lipiodol). Pulmonary branches divide and subdivide, forming the normal lung structure portrayed by a chest roentgenogram.

Director, Department of Radiology, Trudeau-Saranac Institute.

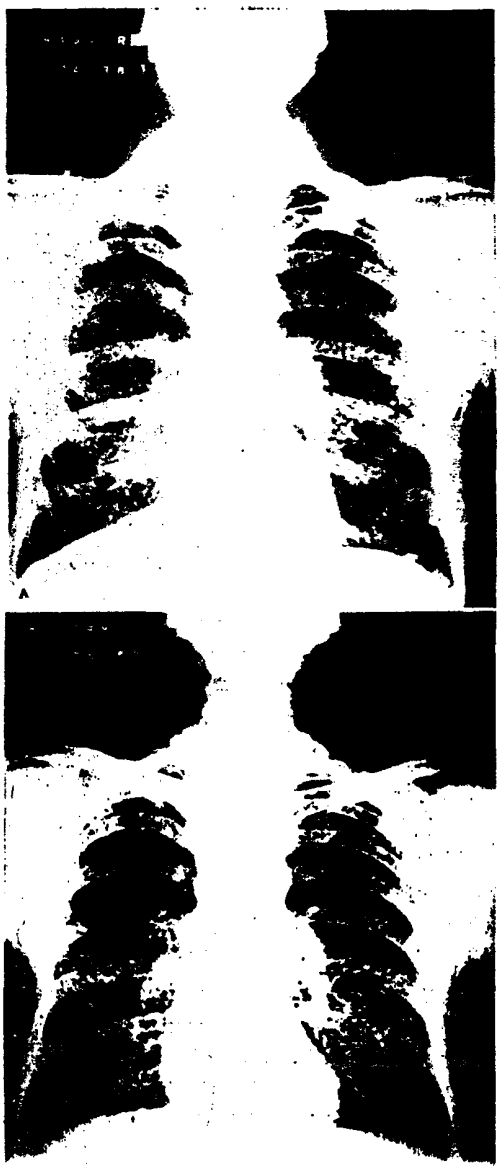
Read in the Symposium on Occupational Diseases of the Lungs, sponsored by the Massachusetts Medical Society in cooperation with the Institute of Industrial Medicine of the New York University—Post-Graduate Medical School, Boston, Oct.

ry of exposure
and (2) the
discrete gen-
licosis, with

miner whose
te generalized
ified as simple
17 years later
of the x-ray
rking without

conglomerate areas of increased density in the complicated forms. We have come to recognize three roentgenologic types: (*a*)

Fig. 5.—This man had Stage II simple silicosis in 1938, without symptoms (*A*). The x-ray changes are characterized by discrete generalized nodulation and definite enlargement of the lung roots. Twelve years later (*B*) there has been no change in the degree of discete nodulation, but in the interval there has developed a slight degree of coalescence of the nodules in the right outer second interspace. In 1950, the date of this roentgenogram, the subject had no respiratory complaints and was gainfully employed.



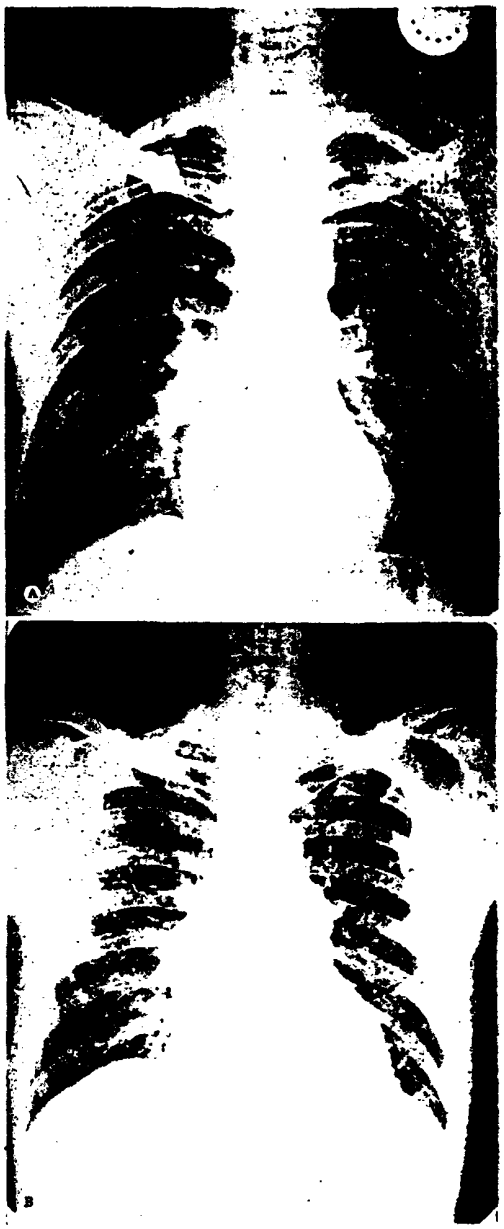


Fig. 6.—(*A*) Simple nodular silicosis, Stage I. Note generalized discrete nodulation and enlargement of both lung roots. (*B*) This patient has been exposed to iron oxide fumes only. The characteristic nodular pattern is visualized, but the lung roots are of normal size and position.

simple silicosis; (*b*) silicosis with conglomeration; (*c*) silicosis with tuberculosis. Following is a useful classification.

A. SIMPLE SILICOSIS

Stage I—First-Degree Silicosis.—The nod-

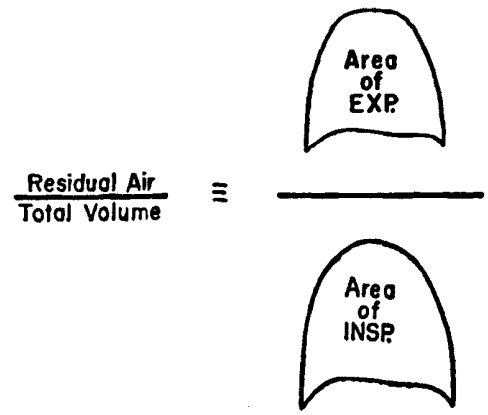


Fig. 2.—The area enclosed within the silhouette of the thoracic cage at full expiration bears a relationship to residual air, and the silhouette of the thoracic cage at full inspiration bears a relationship to the total volume of air within the lungs.

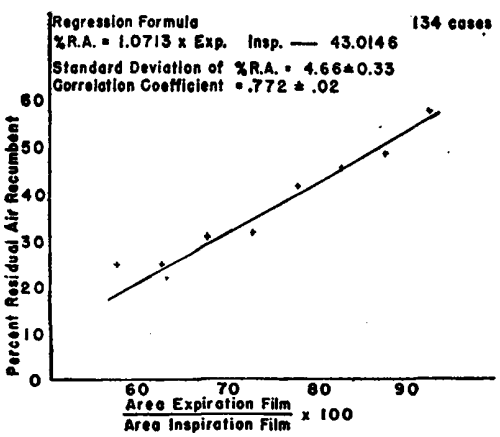


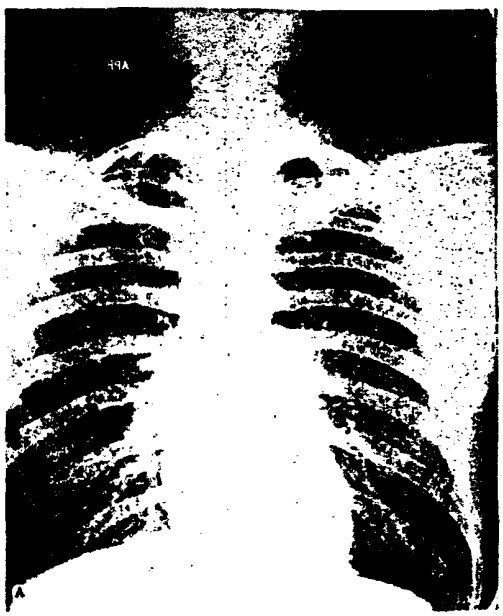
Fig. 3.—Regression formula demonstrates excellent correlation between ratio $\frac{\text{area expiration}}{\text{area inspiration}}$ and per cent residual air. Coefficient correlation is 0.772, with a standard error of ±0.02.

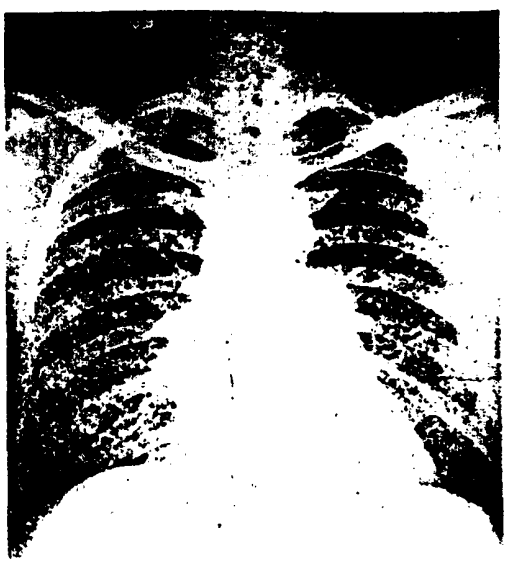
gen changes in relation to the clinical history, physical findings, and laboratory data. Far too often this aspect of roentgenologic diagnosis is overlooked, and the outcome may be most embarrassing.

Silicosis is the result of the interaction of free crystalline silica and lung tissue. Silica has a specific effect, causing proliferation of connective tissue and the formation of parenchymal silicotic nodules. Microscopically the nodules are seen to be composed of hyaline collagen fibers and are evenly distributed throughout the lungs. The diagnosis

criteria: (1) an adequate history of exposure to free crystalline quartz silica and (2) the roentgenologic demonstration of discrete generalized nodulation in simple silicosis, with

Fig. 4.—This man is an iron-ore miner whose chest roentgenogram showed discrete generalized nodulation in 1933(*A*). It was classified as simple silicosis, Stage I. In the same chest 17 years later (*B*) there has been no progression of the x-ray changes, and the man has been working without complaints.





result of the reaction of free crystalline silica on the pulmonary tissues.

Asbestosis is a form of pneumoconiosis resulting from prolonged inhalation of asbestos fiber. It is a chronic disease, with diffuse pulmonary fibrosis which takes years to develop. As in other occupational diseases of the lung, the chest roentgenogram is the cornerstone in establishing the diagnosis. Of course, it goes without saying that an adequate history of exposure to asbestos fiber is also a requisite criterion.

It would be wise, before proceeding to the roentgenologic classification of asbestosis, to caution about making statements as to pulmonary functional impairment—"disability"—from the x-ray. An expiration film or chest fluoroscopy is not of much help in these patients, because their lungs usually empty very well. When functional impairment is present, it is usually a problem of a "tight lung" in contradistinction to the diffuse obstructive emphysema seen in complicated forms of silicosis. Physiological study of such cases usually reveals a decreased maximum breathing capacity, a small total volume, and no increase in residual air. Dr. George Wright has performed complete physiological functional studies on many men from the asbestos industry. When we correlated his findings with the x-ray, it was found that some persons with slight roentgenologic changes had definite functional impairment. This, as you will remember, was not the case in simple discrete nodular silicosis.

As in silicosis, the roentgenologic changes in the lungs of persons with asbestosis can be divided into varying stages of development, as follows.

ASBESTOSIS—STAGE I

There is present an extremely fine network of increased densities at both bases, radiating from the cardiophrenic angles toward the costophrenic sulci. There may or may not be a superimposed granular pattern. The middle thirds of the lung fields may also be The portions of the lung involved have a hazy appearance (Fig. 7).

ASBESTOSIS—STAGE II

The roentgenologic picture is more dense. There is a beginning decrease in the clarity of the cardiac silhouette. The densities extend to the periphery of the middle and the lower thirds of both lung fields. These parenchymal changes are sometimes referred to as having a "ground-glass" appearance. The lung roots are generally accentuated (Fig. 8).

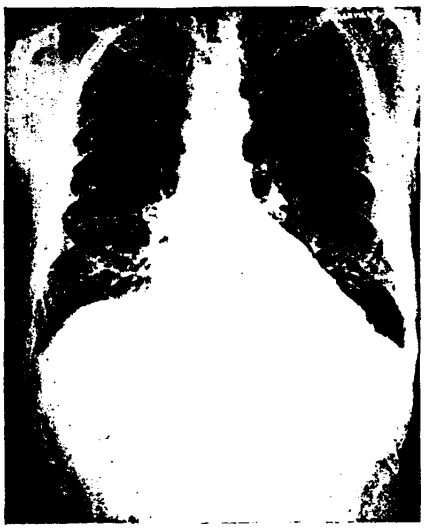


Fig. 7.—Asbestosis, Stage I. Note fine network of increased densities at both bases. Midlung fields and apical regions are clear. Heart borders are clearly defined.

ASBESTOSIS—STAGE III

Considerable shadowing of the middle and the lower thirds of both lung fields is noted, which can extend to the upper thirds. The outlines of the cardiac silhouette are difficult or impossible to differentiate against the densities within the lung parenchyma—the so-called "shaggy heart" (Fig. 9).

It is not especially important whether we have some difference of opinion as to which x-ray stage of asbestosis a patient has. The

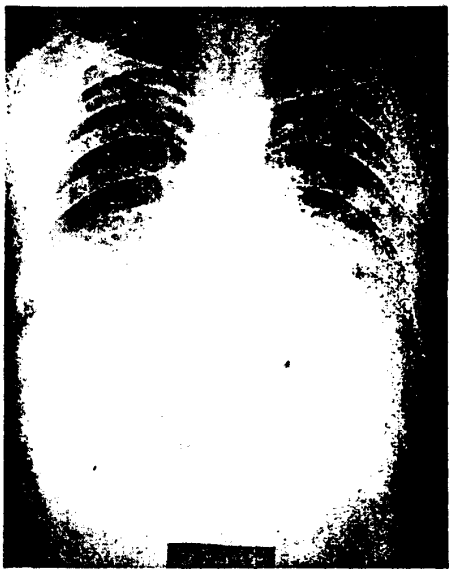


Fig. 8.—Asbestosis, Stage II. Basal areas of increased density are more pronounced than in Stage I. There is slight involvement of midlung fields but complete clearness of the apices. Note beginning loss of definition of right cardiac border.

nosis and of the presence or absence of pulmonary functional impairment as the result of the pulmonary deposition of asbestos fiber. The diagnosis is most difficult in cases which demonstrate only a very slight departure from the usually accepted normal chest roentgenogram. These cases are classified as doubtful or uncertain. Usually stereoscopic films of good diagnostic quality are most helpful in such situations. At any rate, it would seem mandatory that these cases be closely followed by means of serial chest x-rays. Fortunately, this problem does not confront us too often.

This discussion does not represent a complete synopsis of the problem of pulmonary asbestosis and silicosis. It was the intention only to demonstrate various roentgenologic manifestations of the lung abnormalities incident to these conditions. However, one would be remiss if mention was not made of the fact that these fibrotic diseases may give rise to cor pulmonale. The right ventricular enlargement is best demonstrated in oblique views of the chest and by fluoroscopy. It is enlargement without gross alteration of the cardiac silhouette on the posteroanterior projection.

CONCLUSIONS

Silicosis and asbestosis result from the inhalation of sufficient quantities of free crystalline quartz silica and asbestos fiber.

When pulmonary reaction occurs as the result of deposition of either dust, a characteristic, though different, roentgenological pattern is manifested in the chest x-ray.

An x-ray classification of both conditions is employed according to the extent of the characteristic shadows.

Simple silicosis is usually not accompanied by any pulmonary functional impairment, whereas the complicated forms almost always demonstrate some departure from the normal.

The functional impairment in silicosis is due to diffuse obstructive emphysema. A chest fluoroscopy and/or chest roentgenograms taken in full expiration aid in evaluating the presence or absence of this complication.

Not all cases of asbestosis have a breathing problem. When it is present, it is due to a

Fig. 9.—Asbestosis, Stage III. Basilar areas of increased density and involvement of midlung fields are more striking. Characteristic shadows are now extended to involve the upper thirds. Cardiac silhouette has assumed typical "shaggy" appearance.

"tight lung." Expiration films are not of much value in determining the presence or absence of respiratory impairment in these cases, as there is no increase in residual air.

Unlike silicosis, we found some persons with slight x-ray changes indicating asbestosis with definite functional impairment present.

DISCUSSION

Dr. M. C. Sosman, Boston: Dr. Bristol has presented a very complete and scholarly paper concerning the demonstration and identification of the various forms of silicosis and asbestosis by roentgenological examination. The problem, however, is quite different when attempting to make a diagnosis of silicosis or asbestosis in some of the patients seen in a general hospital. Dr. Bristol's patients were x-rayed particularly for silicosis or asbestosis, and in many of them there was a definite history of exposure. In many of the others there was a known pulmonary disability, and the possibilities of pneumoconiosis were automatically considered. By contrast, most of our patients are sick, but they have a much wider variety of disease, and the demonstration of the abnormality causing the symptoms is often difficult, with its identification even more arduous.

Many of our patients have shortness of breath, cough, fever, or loss of weight. It may be due to disease elsewhere in the body, heart disease or any other conditions than industrial disease affecting the lungs. But it is surprising how many times the various pulmonary diseases and conditions can imitate one another.

At our institution we had chest x-rays taken of 8,000 patients last year, and there were only 3 in whom we could conclude, with any certainty, that their illnesses were due to occupational pneumonitis. However, we can see many slight pulmonary changes and noncharacteristic shadows, such as Dr. Bristol has demonstrated in the early stages of silicosis and asbestosis, but with no symptoms or signs and no history of industrial exposure. At this point I would like to take up the gage of battle which Dr. Chapman flung at me in introducing me. Dr. Bristol has just stated that in evaluating x-ray films one must consider the patient's history as well as make a thorough physical examination. Now I submit to you, gentlemen, that, as a physician practicing radiology, I cannot remember a single case in which the physical examination changed the diagnosis which had been made from the x-ray findings, the history, or the laboratory results, or a combination of the three. In other words, I feel that it is a waste of the physician's time to listen to the patient's lung and then decide, in view of what he thinks he hears, what the x-ray shadows mean. However, I would not urge you to stop doing it, for it impresses the patient and gives the physician time to think what might possibly be wrong and what kind of an x-ray examination should be made first.

As to the exact problem presented today, may I show you a series of slides, any one of which could be interpreted as various stages of silicosis or asbestosis if the conditions of exposure were adequate and if the diagnosis was not otherwise known or available? May I add at this point, and stress its importance, that I firmly believe the x-ray examination of the chest is only half of the diagnosis; the other half must be a history of adequate exposure. We are not justified in making a diagnosis from x-ray films alone or from the history alone, but we can do it from the combination of the two.

[Dr. Sosman then showed a series of slides, in each case pointing out the similarity between the condition demonstrated and similar lesions which could be due to silicosis or asbestosis. He pointed out that it was easy to demonstrate symptom-producing lesions in the lungs in over 95% of the cases but that it was difficult to identify them from the x-ray examination and the history in more than 75%. He included the following: carcinoma of the lung, simulating the conglomerate stage of silicosis; metastatic carcinoma, duplicating the nodular stage of silicosis; retrograde lymphatic permeation from the retroperitoneal abdominal area simulating early streaking of silicosis; various granulomatous diseases, infections, parasites, effects from inhaling toxic materials; lipid pneumonitis, scleroderma, multiple infarcts, chronic passive congestion, especially from mitral stenosis; idiopathic pulmonary fibrosis, diffuse bronchitis and bronchiectasis and sarcoidosis, in addition to several rare conditions just recently identified.]

Conclusion: 1. X-ray examination is mandatory in all occupations where there is any possibility of exposure to disease-producing materials, with primary films at employment and routine films at least once a year thereafter.

2. In many conditions seen in the general hospital, silicosis has to be considered, but it is actually diagnosed in only a very small fraction of the total number of patients seen in a general chest clinic in such an area as metropolitan Boston.

Functional Abnormalities of Industrial Pulmonary Fibrosis

GEORGE W. WRIGHT, M.D., Cleveland

I have been asked to discuss the abnormalities of function that occur in certain of the pulmonary diseases of occupational origin. Knowledge concerning the derangements of function, characteristic of each specific disease, will assist materially in making a correct diagnosis. Information concerning the degree to which function has been deranged, consequent to the disease, will assist in proper evaluation of the physical disability arising out of the pathological process. Time will not permit me to discuss all the occupational pulmonary diseases; therefore, I propose to discuss chiefly silicosis and asbestosis. These two diseases offer a nice contrast and are representative of the abnormalities of function that one meets in other types of industrial pulmonary disease.

Since this audience includes a larger proportion of lay than medical persons, it seems to me advisable that I spend some time in describing as simply as I can the function of the respiratory and cardiocirculatory apparatus. I trust that my medical associates will forgive me if I, for the purposes of rapid comprehension, simplify the processes which we know are, in reality, quite complex. The human body is an internal-combustion machine which obtains its energy by burning carbohydrates and fat. In the process, oxygen is utilized and carbon dioxide is produced. The rate of burning is proportional to the rate of energy expenditure; hence, oxygen utilization and carbon dioxide production are augmented or diminished in proportion to energy expenditure. The oxygen utilized is obtained from, and carbon dioxide, in turn, is discharged into, the atmospheric air. Since combustion goes on deep in the tissues of the body, some arrangement must be made for transporting oxygen to, and carbon dioxide away from, the active tissues. This transportation is performed by the blood stream which contains oxygen and carbon dioxide in physical solution and combined with chemical substances. It is readily understandable then that, as energy production is augmented during increasing work, more and more transport of these chemicals must take place. This is accomplished by increasing the blood flow to the active parts, the increase in blood flow being effected by an increase in the activity of the heart, the organ that pumps blood through the vascular channels of the body. The rate of exchange of oxygen and carbon dioxide between the body and the atmospheric air varies approximately tenfold, depending upon the rate of physical energy utilization.

Adequate gas exchange requires that at some place the blood must flow over a large vascular surface where the medium separating the blood from the outside air is very thin and hence will permit the gases to cross from the blood to the air phase with great rapidity. Such a large vascular bed would be very fragile and subject to great injury if exposed on the outer surface of the body, but man has developed so that this vascular bed is contained within a very strong covering and protecting device, namely, the chest cage. The lungs are this vascular bed through which blood can circulate very rapidly and present an enormous surface to air that can be variably and rapidly refreshed. It is ap-

Director of Medical Research, St. Luke's Hospital.

Read in the Symposium on Occupational Diseases of the Lungs, sponsored by the Massachusetts Medical Society in cooperation with the Institute of Industrial Medicine of the New York Univer-

not exchanged frequently the blood would soon pick up all the oxygen there and replace it with carbon dioxide. In such circumstances, carbon dioxide would back up in the tissues and the tissues would suffer from a lack of oxygen. Lack of oxygen is known, under situations familiar to all of us, to result in a smothering of the combustion processes in any sort of internal-combustion machine. Obviously, therefore, it is necessary that the air within the lungs be rapidly and continuously changed in order to provide an atmosphere inside the lungs which closely resembles the outside air. The act of breathing serves the purpose of exchanging the lung gas with that of the outside air. In summary, the respiratory apparatus is an air pump actuated by movements of the thoracic cage, which contains a large vascular surface designed for the rapid exchange of gas between blood and air. To complete the apparatus, a blood pump, namely, the heart, forces blood through the vascular surface of the lung at a variable and sometimes rapid rate. In addition, of course, the heart also pumps blood away from the lung to the tissues that utilize the oxygen and nutrients contained in the blood itself.

The respiratory and circulatory apparatus does not operate in an unregulated fashion. There is a control mechanism situated in the brain which dictates to the thoracic cage the rate at which air should be moved into and out of the lungs, the rate at which blood will be propelled through the lungs, and the way in which blood shall be distributed throughout both the lungs and the body. This control mechanism is under the influence of many factors. The precise manner in which the body integrates its activities so that respiration and blood flow keep pace with the demands for energy production is incompletely understood and highly complex.

During the past few years, methods have been devised for quantitating some of the physiologic aspects of the respiratory and circulatory systems. It is possible to measure the volume of air contained in the lungs in their fully distended state (total volume) and also in the state of being as empty as one can ume), the difference between these two volumes being the stroke volume (vital capacity) of the respiratory pump. One can also measure the maximum rate at which the respiratory apparatus can move air into and out of itself in repeated cycles (maximum breathing capacity). The response of the respiratory apparatus, in terms of the volumes of air it breathes per minute, to the stress of physical exercise (O_2v, or oxygen ventilation equivalent) can also be measured. A sample of blood drawn from one of the peripheral arteries of the body and subsequently measured for its oxygen and carbon dioxide pressure and for the degree to which the oxygen carriers have picked up a load of oxygen while in transit through the lung gives us an accurate estimate of the effectiveness with which the lung ventilates the blood which perfuses it. It is also possible to measure the partial pressure of oxygen and carbon dioxide in the air of the deeper portions of the lung where gas exchange is taking place between the blood and air phase. The actual physical state of the lungs is now the subject of intensive study in several laboratories. Although we are now able to measure the pressure and quantity of blood flowing through the lungs under resting conditions and during mild exercise, in man we are only beginning to arrive at a solution to the problem of measuring blood flow during heavy exercise. The measurement of potential differences in the heart muscle, as reflected in the electrocardiogram, permits us to learn much concerning the action of the heart itself. Nevertheless, we are still quite ignorant of the details concerning the chemistry associated with energy production in heart muscle, and the same may be said for the muscles that permit us to carry on physical exertion. One can see, therefore, that, although we possess useful knowledge which permits us to recognize derangements of function and to quantitate them, we do not have all the information that we would like to, and there are many questions still unanswered.

Do these various measurements just mentioned supply us with reliable information

physical energy? This question has been under study for a number of years in the Department of Physiology of the Edward L. Trudeau Foundation, and a preliminary report of rather extensive data in this regard has been made. The maximum energy output during exercise of six minutes' duration was measured in a group made up of normal persons and others with various kinds of pulmonary disease. The oxygen uptake during the most intense exercise of which each individual was capable was measured. This maximum O_2 uptake is believed to quantitate the peak capacity of the integrated functions of the respiratory and circulatory systems and to reflect maximum energy output. Numerous physiologic measurements of various aspects of respiration and circulation were carried out in conjunction with the determination of the maximum ability for oxygen uptake. In normal persons and in others with various forms of pulmonary disease, including many with industrial disease, a high correlation between the maximum capacity for oxygen uptake and a combination of the maximum breathing capacity and oxygen ventilation equivalent was demonstrated. The correlation coefficient was 0.904 S. E. 0.025. It is apparent from these data that in those persons who suffer from pulmonary disease a measurement of the maximum breathing capacity and of the response to exercise in terms of ventilation (O_2v) either directly determine or reflect some common factor that determines the ability such persons possess for physical energy expenditure.

What are the vulnerable points of the respiratory system? It is apparent that disease may hinder the motion of the thoracic cage, thus impairing the capacity of the person to move air into and out of the lungs in an augmented fashion. Disease may also cause obstruction to the large or the very small tubes that conduct air into the various portions of the lung. Such obstruction will impair the rate of air flow through the tubes and reduce the capacity of the respiratory apparatus to perform as an air pump. Lung tion or resists deformation during expiration, an alteration that also would reduce the capacity of the respiratory apparatus to pump air. Any of the three alterations just mentioned may cause uneven distribution of gases in the lung, thus interfering with maintenance of an optimum pressure of oxygen and carbon dioxide in the gas phase, and lead to inadequate ventilation of the blood as it perfuses the lungs. Disease processes may cause a proliferation of the tissues that support the blood vessels inside the lungs and in this manner increase the thickness of the membrane that separates the blood from the gas phase, with a resultant impediment to the passage of vital gases across this separating membrane. This, in turn, would lead to inadequate ventilation of blood which perfuses the lung. Disease processes might also destroy portions of the vascular bed of the lung, with the result that the area available to the body for transfer of gases from the blood to the gas phase might be reduced. In this connection, reduction of the vascular bed may also impose a resistance to blood flow through the lung and abnormally increase the work of the heart with consequent damage to that organ. Disease process may also so influence the lung as to cause unusual reflexes to arise from within the lung and thus vary the breathing response during rest or especially during exercise. As mentioned before, disease processes may interfere with the proper ventilation of the blood as it perfuses the lung, with the result that the blood itself will undergo changes in the way of increase in the oxygen-carrying cells, thus causing the blood to become more viscous and in that way increase the work of the heart in the process of pumping blood through the vessels of the lung. Disease processes also produce other changes in the cardiorespiratory apparatus of a very subtle nature, and presently only vaguely recognized, which may contribute materially to disability arising out of pulmonary injury.

We may proceed now to a discussion of the specific way in which the respiratory appara-

investigators throughout the world have shown that the earliest recognizable form of silicosis, generally classified as simple discrete nodular silicosis, is attended either by no recognizable alterations of function or, at most, by very slight ones. It is perhaps surprising to some that a disease process which can be readily seen as manifested in a chest roentgenogram may nevertheless fail to cause any recognizable functional disturbance. The use of the word "recognizable" is deliberately chosen, since it is entirely possible that slight alterations of function are not recognized by virtue of the fact that thus far all studies in diseased persons have been made after the disease has become manifest. We are forced to compare measurements of the diseased person with the average for these same measurements in normal men. If we were able to study persons early in life, prior to the development of the disease, and repeat the studies in the same persons at the time the disease is fully developed in its simple discrete nodular state, perhaps we would recognize some reduction in respiratory function. We have no way, at present, of disclosing that a high normal man might be converted to a low normal by disease. Also, properly sampled groups of diseased persons measured for comparison with groups of normal men have not been studied.

When conglomerate silicosis develops, one usually finds readily recognizable alterations of pulmonary function. The maximum breathing capacity is usually reduced, the residual volume is increased, and the oxygen ventilation equivalent may be slightly above normal. These findings are quite typical of those observed in diffuse obstructive emphysema, and indeed the histological sections of the lungs from conglomerate silicotics show the anatomy characteristics of diffuse obstructive emphysema. When the impairment to movement of air into and out of the lungs is severe, it can be recognized by observation of the limitation of thoracic motion during the act of breathing. More sensitive methods permitting earlier recognition of this obstruction to air flow are [illegible] maximum inspiration and expiration. In contrast to the person with idiopathic diffuse obstructive emphysema, in whom recognizable alterations of effectiveness with which the blood is ventilated are common, most persons with conglomerate silicosis show little or no impairment of blood ventilation at rest, although evidences of inadequate ventilation may appear during moderate or heavy exercise. One may interpret this as indicating that in conglomerate as well as in discrete nodular silicosis the involved areas of lung are perfused either slightly or not at all with blood from the pulmonary artery. Virtually all the blood that flows through the lung appears to traverse normally functioning lung tissue. In many but not all cases of conglomerate silicosis, the pressure required to force blood through the vascular channels of the lung is increased as a result of attrition of the vascular bed. This, in turn, places a strain on the heart, with production of hypertrophy of the cardiac muscle. If the resistance to blood flow is severe enough and lasts over a sufficient length of time or is complicated by pulmonary infection, heart failure may occur.

The physiologic abnormalities observed in the various stages of silicosis fit reasonably well with the histologic abnormalities. As mentioned earlier, the discrete nodular stage is focal. Each diseased area consists of destroyed lung replaced by the granuloma characteristic of silicosis. No air enters these foci, and no pulmonary artery blood perfuses them. In between each focus one sees a narrow zone of dilated alveoli, spoken of as being emphysematous. The physiologic data indicate that these dilated alveoli are too few in the aggregate to recognizably influence function, or they function normally in spite of being dilated. When clinical or physiologic evidence of impairment consistent with diffuse obstructive emphysema is observed in a person with simple discrete nodular silicosis, one must suspect that the emphysema is a chance concomitant disease and would have occurred even though the silicosis were ab[illegible]

tous tissue which, in the aggregate, may comprise a large portion of the total intrathoracic substance. The air-containing lung tissue usually displays the characteristic appearance noted in diffuse obstructive emphysema. Although critical measurements of the total pulmonary capillary bed have not been made, it is obvious that some, and in many cases much, of the vascular bed has been destroyed. In these cases, thickening of the right ventricle, presumably caused by the increase of work required to force blood through the lungs, is often evident.

In summary, it is the experience of all investigators that the simple discrete nodular phase of silicosis is rarely complicated by recognizable physiologic alterations of the cardiorespiratory apparatus. In the conglomerate form of the disease, however, the classical evidences of diffuse obstructive emphysema are relatively common. It is of practical interest that in all stages of silicosis the severity of impairment of respiratory function bears only the grossest correlation with the extent of the disease displayed in the roentgenogram. If the physiologic alterations are slight, there is little impairment of the ability to carry out physical exertion. If, on the other hand, there is a marked reduction in breathing capacity, the degree by which such a person's ability for physical exertion is limited may be equally great. The breathlessness during exertion that restricts the physical work capacity of the conglomerate silicotic is primarily caused by loss of breathing power. Present-day techniques permit us to measure the physiologic alterations with a reliable degree of accuracy.

During the past few years a group of 57 men who had experienced varying degrees of exposure to air-borne asbestos fiber were studied in the Department of Physiology of the Edward L. Trudeau Foundation. A preliminary report of these data was presented at the Seventh Saranac Symposium in September, 1952. What is the status of the respiratory and circulatory systems in a person who has typical roentgenographic and clinical evidences of asbestosis? A study shows rather clearly that in the typical asbestotic there is little, and often times no, impairment of ability to ventilate the lungs, as measured by the maximum breathing capacity. There is likely to be, however, a measurable restriction of the degree to which the lung can be expanded, as evidenced by a slight to moderate reduction in total volume. Of course, when the fibrosis is extreme in extent, the impairment to enlarging the lung may cause an appreciable loss of maximum breathing capacity. Fluoroscopic examination, as well as a comparison of inspiration and expiration films, and measurement of the actual residual volume of the lung show that there is little or no impairment to emptying the lung in such persons. When one measures the partial pressure of oxygen and carbon dioxide in the gas of the lung, one finds that these pressures too are within normal limits. Nevertheless, study of samples of blood removed from the peripheral artery will usually show a rather marked inadequacy of oxygen transfer from the lung air to the lung blood. The partial pressure of oxygen in the arterial blood is usually subnormal, and less than the normal proportion of oxyhemoglobin will be found in the sample. The difference between the pressure of oxygen in the lung gas and in the lung blood is usually quite markedly increased. The partial pressures of carbon dioxide are apt to be within normal limits, both in the lung gas and in the lung blood. These data quite clearly indicate an impediment to the passage of oxygen across the membrane separating the gas from the blood phase in the lung. Carbon dioxide transfer, however, is not appreciably interfered with. During exercise the person with asbestosis is apt to display overbreathing. The volume of air breathed per minute will be considerably greater for a given stress of exercise than would be true in normal persons. This is well demonstrated in measurements of the oxygen ventilation equivalent, which is found to be elevated. The rate of respiration and the pulse rate during exercise are apt to be higher than normal in the person with asbestosis. The

limits the physical activity in some persons with asbestosis resides, in part, in the abnormally high rate of breathing and the abnormally increased volume of air breathed during work. The subnormal oxygen content and the pressure in the arterial blood act as an unusually strong stimulus to breathing. In addition, reflexes originating from the abnormally stiff and relatively uncompliant lung of the asbestotic may add to the stimulus of breathing or even give rise directly to the distressing sensations felt by the patient.

To what extent does the presence of an abnormally low oxygen content of the arterial blood, one of the chief characteristics of asbestosis, presage a limitation of capacity for physical work? We found that some of those persons showing a definite subnormal O_2 content of the arterial blood still possessed a normal maximum capacity for physical exertion. Because we are comparing such a person with the average normal man and not with himself prior to injury, it would be incorrect to say that our evidence demonstrates an absence of impaired maximum capacity for physical work. It should be emphasized that our data show retention of average capacity for work in the presence of incomplete oxygenation of the blood.

These findings in the person with asbestosis stand in rather striking contrast to the abnormalities seen in the typical silicotic. Whereas in the conglomerate silicotic a marked reduction of maximum breathing capacity and an increase of residual air, typical of diffuse obstructive emphysema, are commonly observed, such abnormalities are usually absent in the asbestotic. In the latter, there appears to be little or no impairment of movement of air into and out of the lungs, the chief abnormality observed in regard to thoracic action being that the lungs are resistant to inspiration. Whereas the person with silicosis usually shows no or little abnormality of the transfer of gases between the lung phase and the blood phase, quite the reverse is true in the person with asbestosis. In the latter, inadequate oxygen transfer to blood as it flows through the lung who shows no unusual response to exercise from the standpoint of the amount of air that he breathes, the person with asbestosis breathes considerably more than does the normal man for a given intensity of physical exercise. One can generalize by saying that, although both the silicotic and the asbestotic man develop unusual shortness of breath during physical exertion, the person with silicosis does so chiefly because of a loss of breathing power, whereas the man with asbestosis becomes short of breath primarily because his breathing load, or response in terms of breathing during the exercise, is unusually great. Secondary effects on the heart because of an impediment to the flow of blood through the lung can be manifested in both diseases. In other words, cor pulmonale is a not unusual complication of either conglomerate silicosis or asbestosis.

This contrast of physiologic abnormalities is consistent with what one sees in regard to the abnormal anatomy or histology of the lung in the two diseases. In the silicotic the disease is focal, with large areas of healthy lung tissue remaining in the presence of the scattered nodulation; hence, the relative paucity of physiologic abnormalities. In the conglomerate form of silicosis there is still some healthy lung tissue, but coexistent is a diffuse emphysema, with its airway obstruction and consequent loss of breathing power. Only later, when the ventilation of the air spaces is inadequate, do evidences of improper ventilation of the perfusing blood appear. In well-developed asbestosis the tissue alterations appear to be more widespread than in silicosis, and even when the process is not severe in any one area, a major portion of the lung will be involved. Hence, there is relatively little truly entirely normal lung tissue in the person with clinically manifest asbestosis. The nature of the histologic change is such as to increase the thickness of tissue that must be traversed by oxygen in going from the lung air to the lung blood. The abnormality in asbestosis is chiefly located in the membrane that separates the blood and the gas phase. Because of this and

in asbestosis, the primary or chief abnormality exhibited by the asbestotic patient is impaired oxygen transfer to the blood rather than impaired breathing ability and ventilation. It is true that the histologists have reported emphysema as being present in the tissues of the lung in the typical asbestosis case. Small areas of emphysema undoubtedly do occur, but the massive emphysematous involvement of the lung, which is so commonly observed in conglomerate silicosis, does not appear to be present as a rule in even the severe cases of asbestosis.

What can be said about the relationship between the presence or absence of physiologic abnormalities and the presence or absence of recognizable pathologic manifestations in the chest roentgenogram in asbestosis? Our studies showed quite clearly that examples of asbestosis, clearly recognized by roentgenography but with minimal or absent physiologic abnormalities, do exist. This can only mean that in some (not the rule) instances the earliest roentgenologic manifestations are still focal in nature and that large volumes of normal lung tissue remain through which blood can perfuse in preference to those areas that have been damaged by the asbestosis process. The reverse situation may also be observed, as evidenced by the observation of some persons showing evidences of abnormal physiologic conditions in the arterial blood with no clearly recognizable abnormality in the chest roentgenogram. Two possible explanations exist for this not unusual finding in our series of cases. First, the earliest recognition of asbestosis in the chest roentgenogram is notoriously very difficult, because the earliest manifestation is simply an exaggeration of bronchovascular markings that are normally present in the lung. It may be that in those cases which we observed with evidence of impediment to oxygen transfer across the membrane but without evidences of change in the x-ray we failed to recognize the earliest x-ray abnormalities. The second explanation may be that the disease process is so diffuse in its very earliest stage that there is little or no As a consequence some of the blood must flow through areas of lung tissue in which the membrane has been slightly thickened, thus causing impairment of oxygen transfer to the blood. Since the lung is so uniformly involved by the slight alterations of tissue, there is poor contrast between normal and diseased areas and, hence, no definite abnormal shadow in the roentgenogram. In regard to the correlation between x-ray abnormality and physiologic abnormality in asbestosis, one can state that 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.

What about the duration and intensity of exposure to air-borne asbestos fiber and the subsequent development of roentgenologic or physiologic abnormalities? As one might expect from experience with other pulmonary diseases of occupational origin, there was a gross correlation between the intensity and duration of exposure and the development of abnormal shadows or physiologic changes. However, some persons with prolonged and very intense exposure failed to develop any evidences of either physiologic abnormality or roentgenographic abnormality. Our material fails to give us any evidence as to what the shortest required duration or intensity of exposure is in order to produce a recognizable asbestosis.

It has long been known that both crystalline silica in finely divided form and asbestos fiber when introduced into the lung can produce fibrosis which leads to physical impairment. Although the clinical manifestations of the disease are in many, though not all, respects similar, the physiologic abnormalities are recognizably distinctly different in the earlier stage of the two diseases. In the terminal stage the distinction may not be entirely clear and may lead to confusion. A further difficulty in diagnosis resides in the fact that many workers with obvious pulmonary abnormality have had exposure to

genologically manifest but without recognizable physiologic impairment. An enormous respiratory reserve, which is the birthright of most of us, permits extensive tissue injury and damage with little or no loss of capacity for physical work. This statement of fact in no way should be construed as condonement for needless injury.

DISCUSSION

DR. JAMES L. WHITTENBERGER, Boston: I was pleased to hear Dr. Lanza's remarks; he made a number of remarks I was going to make. Many of us in the field of pulmonary physiology do not understand all that Dr. Wright was talking about, and I personally agree with the statement Dr. Lanza made that in many ways we are in a primitive state so far as our information about industrial pulmonary diseases is concerned.

I read the other day a passage written by Harvey in 1635,[1] in which he discussed the effect on the lungs of the smoke which heavily contaminated the atmosphere of the large industrial cities in England. He believed that the elderly and infirm were most likely to suffer, especially in the autumn of the year when the smoke was inclined to be severest. Even today we are much concerned with the same problem, and I am not sure that we understand it very much better than he did at that time.

Having been shown the large array of pulmonary function tests which Dr. Wright used very skilfully in his study of industrial pulmonary disease, we should ask ourselves the question of the significance of some of these tests. I am not by any means against doing these tests. I think that it is the only way we will learn about these conditions and perhaps learn how little we know about them, but the respiratory system, in spite of the simplified diagram Dr. Wright presented, is an extremely complex system. The interrelationships of ventilation of the lungs, diffusion in the lungs, and circulation throughout the lungs are so complex that it is often extremely difficult to say what is due to impairment of the circulation and what is due to impairment of diffusion or ventilation.

As physicians seeing patients with pulmonary disease which may be related to occupational exposures, I am sure that you would like to know what tests are really feasible outside of the larger hospitals and university centers. I think that the vital capacity, especially if it can be a timed vital capacity, is a measurement which is definitely worthwhile. I think that the maximum breathing capacity, which is a simple test, is very worthwhile.

There is a point here about normal standards. If we use the standards in the literature, it is often difficult to tell what patient is normal or not normal, whereas if we had a vital capacity or maximum breathing capacity taken on the patient himself at some prior time when presumably his lungs were normal, we would have a much better idea whether disease has affected him or not. I don't know how feasible it is in industrial medical practice to get this type of measurement, but I personally think that it would be very worthwhile.

Many of the tests Dr. Wright described depend a lot upon the cooperation of the patient. It is true that arterial saturation during exercise changes independently of any willingness on the part of the patient, but the degree to which he is willing to push himself is very important. The vital capacity and the maximal breathing capacity depend obviously upon voluntary cooperation.

I would like to take this opportunity to agree with Dr. Sosman about the value of the physical examination. I do not know how many of you have read about the tests that were carried on at the Cardiff Pneumoconiosis Research Station in Wales a year or two ago.[2] Some of the men in this outstanding research station were dissatisfied with what they could deduce from a patient's physical examination, and they invited a number of Professor Christie's experts to come from London and examine the same patients. These patients had emphysema or massive pulmonary fibrosis resulting from pneumoconiosis occurring in coal workers.

Since time is short, I will only say that the physicians were dismayed by how often they disagreed on physical signs even in patients with advanced pulmonary disease. They just did not agree at all. So, not only should a physician throw away his stethoscope, but also he may as well not bother to percuss or inspect the patient.

I should not close on such a pessimistic note, as I would like to say that many researchers in this field believe there is hope in the development of objective tests of the type which measure the mechanical properties of the lung, the elastic properties and the resistance to breathing. These studies are relatively in their infancy. They have not been to any great extent applied in patients with industrial pulmonary disease. I will close by saying that we need humility in our study of pulmonary disease, but still we should continue trying.

REFERENCES

1. Missen, G. A. K.: Lethal Aerosol, Letters to the Editor, Lancet **265**:1212 (Dec. 5) 1953.

2. Fletcher, C. M.: Clinical Diagnosis of Pulmonary Emphysema: An Experimental Study, Proc. Roy. Soc. Med. **45**:577-584 (March) 1952.

Asbestosis as Differentiated from Other Pneumoconioses

O. A. Sander, M.D., Milwaukee

The preceding discussion has shown that the roentgenological appearance of a well-defined asbestosis is quite distinctive and that it differs materially from any of the other pneumoconioses. For that reason, I shall not limit my part of the discussion to the differences between this disease entity and the other dust diseases but shall include other conditions of the lungs which may be misdiagnosed as asbestosis.

The first and commonest cause of misdiagnosis is poor film technique. A perfectly normal chest can be made to look like one with definite first-stage asbestosis by slight underexposure, by lack of contrast, and by blurred vascular markings due to too long exposure time. Such films are especially common in overweight persons, the heart shadow usually being horizontal and often presenting a shaggy appearance due to compression of the vascular shadows in the lower lobes. Films lacking proper penetration, sharply defined detail, and lack of contrast should be rejected for the diagnosis of any occupational disease of the lungs but particularly of asbestosis.

Another condition which causes diagnostic trouble at times is emphysema due to any cause, when there are one or more adhesions of the diaphragm due to past pleurisy. The "ground-glass appearance" is very easily read into such a film. When to this are added some retained secretions in the lower lobes, which often are bronchiectatic in emphysema, the misdiagnosis is established.

A pneumoconiosis of which we have become increasingly aware in recent years is that due to excessive deposition of coal dust. It appears quite certain now that free silica is not needed as an essential component of the dust to develop the characteristic changes. The fine lacy character of the shadows plus some emphysema gives a pattern which looks very much like "ground glass" and is easily mistaken for early asbestosis.

All these examples I assume represent patients who present themselves to their physicians with symptoms of dyspnea, with a history of some exposure to asbestos dust, and with chest films of the character I have mentioned. How easy it is to fall into the trap of a gunshot misdiagnosis! Unless other possible causes for the x-ray changes are considered first and a detailed past occupational history is evaluated, along with the character and extent of the most recent dust exposure, another worker who actually needed reassurance will be told that his lungs are full of asbestos dust and that he should quit his trade! From that point on, his symptoms usually increase to a marked degree, and he has developed what to him is a real disability. In my experience, such doctor-induced disability is commoner in some areas than is the disability from the disease itself.

Because of the complete lack of unanimity of opinion about this disease among physicians and because of the need for more clearly defined criteria for diagnosis and advice on continued employment in the trade, the medical and industrial hygiene advisers

Read in the Symposium on Occupational Diseases of the Lungs, sponsored by the Massachusetts Medical Society in cooperation with the Institute of Industrial Medicine of the New York University—Post-Graduate Medical School, Boston, Oct. 28, 1952.

have set up a so-called Air Hygiene Committee. This committee has met on several occasions to determine whether those who are most intimately associated with this disease can agree on the various medical and hygiene phases of the problem.

At the last meeting, each medical member of this committee brought with him sample films of the various stages of asbestosis, including some he had classified as essentially negative. All films were reviewed by the eight physicians who were present. We found fairly good agreement on the more advanced stages of asbestosis but practically none on the borderline degrees. A number of my films from the textile mill in North Carolina for which I am consultant, representing cases which I had classified as "early or first-stage asbestosis," were called "essentially negative" by a number of physicians present who had had long experience with this disease. The reverse also was true; some films I had called negative which others thought represented first-stage asbestosis. The same disagreement was found with the other films which were presented. It was our final conclusion that it is impossible to clearly define a first-stage case and that it can be called "essentially negative" one day and "first stage" the next by the same reader. This was not new. It has been emphasized repeatedly by Pendergrass, beginning in 1938.

Our group finally agreed that little attention should be paid to the first-stage diagnosis, that little, if any, disability has been shown to exist with the borderline stages, that no one should be advised to stop work with these questionable degrees of change, and that workers should be kept at their regular jobs but the dust control should be so improved that their cases will not progress to the stage where everyone agrees that they have asbestosis.

Regarding recommendations of transfer to less dusty or nondusty work, we agreed to the following:

1. Persons under 40, when the diagnosis is clear cut (beyond first stage), should be moved [illegible]

2. Where progression is seen on serial films, regardless of age, less dust exposure is clearly indicated.

3. Exceptions should be made if there will be material improvement in dust control on the present job within a very short time.

I have deviated from my assignment of differential diagnosis, because the diagnostic criteria are so intimately associated with it. Until we have some agreement on x-ray interpretation, the present chaotic state of affairs will continue.

In the differential diagnosis, it is my belief that a new approach is essential, not only with asbestosis but also with nonoccupational diseases as well. Because textbooks in medicine are written by diseases, each followed by a listing of other diseases which must be differentiated from it, the disease itself becomes fixed first in the diagnostician's mind. He says to himself, "This is it," and he pays only cursory attention to the diagnostic criteria of the disease and to the diseases from which it must be differentiated. A more scientific approach would be to list all the conditions and diseases which are compatible with the x-ray pattern which the patient presents and then, after a complete and detailed medical and occupational history, physical examination, and laboratory studies, to see which of the positive findings more closely fit the listed diseases.

I should like to cite a case which undoubtedly would not have been misdiagnosed had this approach been used.

A railroad shop repairman, aged 57, had to stop work because of increasing shortness of breath several years ago. Cyanosis and dyspnea became progressively worse, and he died of anoxia and right heart failure several months ago. His work in the railroad shop included welding, unpacking, and repacking asbestos insulation around locomotive boilers and doing some fitting and grinding. The chest x-ray film showed a diffuse mottled and micronodular pattern in both lungs, and compressed lower lobes due to high position of the diaphragm, with numerous annular shadows of less density scattered throughout both lungs. Asbestosis was diagnosed by the man's physician, because there had been some asbestos-dust exposure, because he had always heard that the x-ray pattern of [illegible]

because it did not look like the siderosis of welders about which he had read something recently. The degree of asbestos-dust exposure was not inquired about (it was spasmodic and minimal). Other diseases and conditions of the lungs were not considered. The annular shadows should have brought emphysematous blebs or air cysts into the differential diagnosis, as well as the granulomatous diseases. Miliary tuberculosis had to be ruled out as well as berylliosis with emphysema. Sarcoidosis also had to be considered, although the annular shadows were against it. After definitive study, the diagnostic possibilities should have been reduced to emphysematous blebs plus pneumonitis or polycystic disease plus pneumonitis. Also, there were none of the characteristics of asbestosis and no pleural involvement.

After postmortem study the diagnosis was clarified—a widespread congenital cystic lung, with interstitial fibrosis due to long-standing infection. The cuboidal and columnar epithelium lining of the cysts is noteworthy. Also, there was complete absence of anything resembling asbestosis bodies, an absence of pleural thickening, and no evidence of any hyaline fibrosis. Even with this clear-cut postmortem evidence, the physician who originally made the diagnosis still believes that this was a case of asbestosis and is sending the tissue slides to various pathologists. He should be convinced soon that he most likely was wrong, which will be most embarrassing to him.

This case, along with many others which could be cited, clearly points up the need to consider all possibilities in cases presenting bizarre x-ray patterns. Each case must have painstaking study, including not only the immediately preceding occupational history but also a history of every job from the first one on. An example is a foundry worker with a chest film which was characteristic of a moderately well-developed asbestosis. Careful study of the occupational environment revealed no possibility of asbestos exposure in this shop. More detailed early occupational history revealed that the man had been a plumber's helper during the late teens and early twenties, doing all the sawing of asbestos pipe coverings, usually in con-

When the most detailed analysis and study will not reveal the true diagnosis, which happens in occasional cases, taking a biopsy specimen of the lung should be considered. This has become a rather simple procedure with little danger of complications. It must be remembered, however, that because of the relatively small piece of lung usually obtained for biopsy, there always is some danger that the diagnostic pathological change will not be revealed.

In conclusion, as with all chronic chest diseases, so especially with asbestosis, all possible causes of the pathology revealed by the chest film must be considered. Poorly studied cases result in misdiagnoses which not only are embarrassing to the physician but which may also cause irreversible harm to the patient. We owe it to our patients and to the medical profession to so thoroughly investigate every obscure case that such unfortunate situations do not arise.

DISCUSSION

DR. KARL T. BENEDICT, West Boylston, Mass.: It is a privilege to substitute for Dr. Harriet Hardy; she has helped me many times, but a substitute is always a "second." The assignment is "the differential diagnosis." I have had no experience with asbestosis and, furthermore, I believe that experience with most pneumoconioses, as seen today, is gained only after 20 to 30 years' study, because it often takes that long to produce such disease.

In spite of the foregoing, I believe I am qualified to make certain remarks, because I have been practicing industrial medicine for 15 years in one of the world's largest artificial abrasive plants. Since 1940, we have taken more than 25,000 chest x-rays on some 5,000 abrasives workers. From 1911 to 1940, except in rare instances, we simply did so-called routine physical examinations.

I agree with Dr. Sosman that the physical examination is not worth very much in these circumstances. Thanks to Phil Drinker and others, dust control is much better in our plants today. In one large plant our dust counts run consistently 3,000,000 to 4,000,000 particles per cubic foot. We handle a great variety of dusts, for that is our business, and safe handling is essential.

Recently at the Seventh Saranac Symposium, several world-renowned authorities on pulmonary diseases attempted to define pneumoconiosis. I shall

group insisted that pneumoconiosis simply means dust in the lung. The other group restricts it to those conditions producing demonstrable disability. The hitch is, Who demonstrates the disability? The roentgenologist sees nodular fibrosis in the x-ray film; that is pneumoconiosis to him. At once, or sometimes much later, the industrial physician and hygienist set about to correct conditions to prevent progress of the pneumoconiosis or development of new cases. Later, though the worker still may not know of his trouble, the family physician has difficulty clearing an ordinary chest cold or the surgeon is faced with anesthesia worries. The occupational disease or the health department specialists in our state government want reports when "injury" has resulted. Compensation and insurance authorities talk about the number of pneumoconiosis cases, based upon those workers whose earnings have suffered. The compensation lawyer appears later in the picture, and alas sometimes the last, the pathologist, may be the first in proving that a case of tuberculosis or cancer is in reality pneumoconiosis.

I believe we all know when the man is suffering from pneumoconiosis, whether we be laymen or physicians. What he wants to know is, What is wrong? What can be done about it? Is he permanently disabled? Is his life shortened?

There can be only one answer: not better diagnosis or differential diagnosis or function studies but adequate dust control. Since different dusts require different degrees of dust control, witness the different effects from inhaling small amounts of aluminum (therapeutically) and beryllium or radioactive dusts. There is, therefore, good reason for you and me to continue to study pneumoconioses and their differential diagnosis. We in the abrasive industry are continuing this research. Specifically, we believe that under reasonable control alumina dust is no problem. You have heard of the "carborundum lung" no doubt; this is an unjust label, as my good friend Dr. Eddy, of the Carborundum Company, well knows, for Carborundum is just a trade name for the fusion product, silicon carbide. Furthermore, just what disease, disability, or pathology silicon carbide per se will produce in man has never been proved conclusively, I believe. It is entirely possible that this lung condition is caused by significant free silica contaminations. Further study is needed, and we intend that it shall be made.

Shaver's disease has been mentioned before. In spite of several years' research tending to prove that alumina fume is the cause, there are those who believe that silica fume is to blame. Industry is somewhat discouraged by the inconclusive nature of these and similar medical studies.

We all know about the problems related by beryllium and radioactive dusts. On the other hand, our concepts of clay and talc pneumoconioses have changed in recent years. And what do we know about zirconia or titania or magnesia or boron carbide or graphite dust inhalation? In our industry organic substances have caused only two minor cases of asthma. And we have seen no cases of cancer of the lung among our abrasives employees.

In conclusion, our goal is not a specific M. A. C.* figure but maximum dust control to eliminate all pneumoconioses. This goal is not easily attainable. Adequate differential diagnostic knowledge will help us to achieve it.

* Maximum allowable concentration.