

roentgenographic changes present? Does he have complicating tuberculosis or other infection? Is he incapacitated or disabled by his condition? The first two of these questions are usually answered by the examining physician. The real question is the third question. Is the man disabled? An incorrect answer will be an injustice to employee or employer. In the past, attempts to solve the disability problem in silicosis were based chiefly upon the clinical approach and roentgenographic studies. For obvious reasons, clinical appraisal of a given individual's pulmonary disability is limited to objective signs and principally to observation at rest and during the performance of exercise tests. Roentgenographic studies, in addition to determining the stage or extent of disease, were used to ascertain the presence or absence of emphysema. Currently, physicians are eagerly accepting the objective assistance offered by pulmonary function tests. The use of these more accurate aids must be accompanied by knowledge of their limitations. One of the major unsolved problems is the degree of dysfunction beyond which all individuals will be disabled. Do individuals with simple nodular silicosis have more disabling emphysema than persons in the general population without silicosis? It is probable that satisfactory answers must await lung function testing of large groups of people with and without silicosis.

b. Pathologic Physiology and General Principles. Based upon recent significant studies,⁴⁹⁻⁵² the following simple classification of the types of pulmonary insufficiency is useful: (1) ventilatory pulmonary insufficiency, which is concerned with defective air movement into and out of the lungs; (2) distribution difficulty, which is concerned with alveolar ventilation and perfusion relationships; (3) diffusion difficulty, which is concerned with passage of carbon dioxide and oxygen between capillary and alveolus; (4) circulatory insufficiency, which is concerned with reduction in the absolute size and distensibility of the pulmonary vascular bed.

Ventilatory insufficiency.⁵³ Silicosis may interfere with pulmonary ventilation in several ways. By simple proliferation, it may encroach upon the space that would normally be occupied by functioning lung tissue. Fibrous tissue that undergoes shrinkage may give rise to overdistension of adjacent normal lung tissue. The degree to which the involved portions of the lung can be expanded will be limited. It is possible that by suitable location it may also limit the expansibility of interposed normal lung tissue. There may be interference with air flow by

⁴⁹ G. W. Wright, Disability evaluation in industrial pulmonary disease, *J. Am. Med. Assoc.* 141, 1218-1222 (1949).

⁵⁰ H. L. Motley, The use of pulmonary function tests for disability appraisal and human evaluation standards in chronic pulmonary disease, *Diseases of the Chest*, 24, 237-250 (1953).

⁵¹ R. Austrian, J. H. McClement, A. D. Renzetti, K. W. Donald, R. L. Riley, and A. Courmand, Clinical and physiologic features of some types of pulmonary diseases with impairment of alveolar-capillary diffusion, *Am. J. Med.*, 11, 687-685 (1951).

⁵² R. L. Riley, Pulmonary gas exchange, *Am. J. Med.*, 10, 210-219 (1951).

⁵³ G. W. Wright and G. F. Filley, Pulmonary fibrosis and respiratory function, *Am. J. Med.*, 10, 642 (1951).

limiting the degree to which airways are enlarged both as to diameter and length during the inspiratory phase of respiration. There may be narrowing of the smaller airways of a spastic⁵⁴ or permanent type such as to cause expiratory obstruction with the development of the characteristic pattern of obstructive emphysema. These abnormalities are evidenced by reduction of total lung capacity and maximum breathing capacity.

Distribution difficulty. Silicosis may interfere with even distribution of inspired air among the millions of lung alveoli by:⁵⁴

1. Regional limitation of lung distensibility.

2. Regional obstruction of air passages either by spasm⁵⁵ or fibrosis.

3. Regional loss of elasticity.

Uneven air distribution results in overventilation of some alveoli and underventilation of others.

Silicosis may affect the distribution of venous blood among the capillaries as well as inspired air among the alveoli. This concept visualizes the distribution of blood having blood and gas components.⁵²⁻⁵⁵ Uneven blood distribution results in overperfusion of some alveoli and underperfusion of others. Effective shunting of pulmonary artery blood away from diseased and improperly functioning alveoli through properly functioning ones may act as a beneficial compensatory mechanism. The importance of this phenomenon has been inadequately appreciated.⁵⁸

Techniques suitable for analyzing the relationship of volume and speed of blood flow to alveolar size and ventilation have not as yet been fully explored.⁵⁹

Diffusion difficulty. In addition to disturbances in the ability of the respiratory apparatus to move air into and out of the lungs, there is evidence^{49,51} to suggest that in some cases of silicosis there may be interference with the direct transfer of oxygen and carbon dioxide between the blood and the alveolus. Proliferation of the connective tissue supporting the alveolar vascular bed may not only increase the distance that must be traversed by each molecule of gas but may also change the character of the medium. Under these circumstances (alveolar capillary block), even though the pressures of oxygen and carbon dioxide (P_{O_2} and P_{CO_2}) in the alveoli may be normal, the venous blood brought to the alveoli will not be completely oxygenated. Since carbon dioxide passes through the alveolar pulmonary membrane easier than oxygen, the transfer of oxygen will be retarded and to a greater extent than that of carbon dioxide.^{53,54} It is also important to remember that the effect of a localized diffusion difficulty may be minimized or nullified if there are enough other areas of normal functioning lung to which the blood may be shunted.^{53,54}

Circulatory insufficiency. In the normal individual the pulmonary circulation system is characterized by low pressure (29/9) and extreme distensibility. It

⁵⁴ L. H. Comroe, Interpretation of commonly used pulmonary function tests, *Am. J. Med.*, 10, 356-373 (1951).

⁵⁵ H. L. Motley, B. Gordon, L. P. Lang, and P. A. Theodos, Impairment of pulmonary function in man with silicosis, *Arch. Ind. Hyg. and Occupational Med.*, 1, 133-159 (1950).