

has been shown that with the increased demands of vigorous exercise, blood flow through the lungs may be increased two and one-half to three times without an increase in normal artery pressure.^{56,57} It is doubtful whether in the normal individual any increase in pressure ever occurs except under conditions of maximal stress.⁵⁸ This physiologic enlargement of the pulmonary arterial tree may be interfered with in silicosis by three mechanisms: (1) decrease in actual size of the bed by direct obliteration of blood vessels;^{58,54} (2) limitation of distensibility of the vascular bed by perivascular fibrosis of arterioles, capillaries, or venules; and (3) vascular spasm secondary to local anoxia-caused ventilatory disturbance, or diffusion inadequacy, which appears to be a local anoxic effect not mediated by reflex neurological pathways.⁵⁸

c. *Clinical Approach to the Determination of Disability.* The clinical approach is obviously limited. The usual disability evaluation must be undertaken in situations where malingering is a frequent factor. Reliance upon objective tests is usually obligatory, but even with this limitation, clinical observations may provide valuable information. The presence of wheezing, dyspnea, or cyanosis is significant. Exercise may elicit one or all of these symptoms, which may not be present at rest. Conversely, patients claiming inability to work may be able to walk with the examiner on the level or up a flight of stairs while conversing on normal conversation without coughing, gasping, or pausing for breath. The lack of value of such other physical findings as barrel-chest, wide costal phrenic angle, kyphosis, use of accessory muscles, impaired expansion, movement *en bloc*, tympanic resonance, impaired liver dullness, impaired cardiac dullness, absent apical impulse, and impaired physical sounds in measuring pulmonary emphysema is shown by Fletcher.⁵⁹ To quote: "With regard to the clinical diagnosis of emphysema, my conclusion is that it cannot be made with any confidence, (at least by a single observer) except perhaps in the most advanced cases, so that there is little hope of the clinician being able to diagnose the early stages. For this reason, turn to the objective methods of the physiologist to help him in his perplexity. It must be pointed out that disability in silicosis is usually due to emphysema."

d. *Roentgenology in the Study of Disability.* Roentgenological studies for determination of disability in silicosis can be used in two ways: (1) determination of the stage of the disease and (2) detection of the presence or absence of emphysema. Note: Measurement of diaphragmatic excursion by the use of

⁵⁶ J. G. Hickam and W. H. Cargill, Effect of exercise on cardiac output and pulmonary arterial pressure in normal persons and in patients with cardiovascular disease and pulmonary emphysema, *J. Clin. Invest.*, **27**, 10 (1948).

⁵⁷ R. L. Riley, A. Himmelstein, H. L. Motley, H. M. Wiener, and A. Courmand, Studies of pulmonary circulation at rest and during exercise in normal individuals and in patients with chronic pulmonary disease, *Am. J. Physiol.*, **152**, 372 (1948).

⁵⁸ G. Liljestrand, Regulation of pulmonary arterial blood pressure, *Arch. Internat. Med.*, **81**, 162 (1948).

⁵⁹ J. Gough, C. M. Fletcher, J. C. Gilson, and P. D. Oldham, Discussion of diagnosis of pulmonary emphysema, *Proc. Roy. Soc. Med.*, **45**, 576-586 (1952).

inspiratory-expiratory films appears to be of some value in determination of emphysema.⁶⁰

Correlation of degree of lung dysfunction with extent of the disease is a matter of considerable controversy. Wright⁴⁹ feels that there is a direct correlation, since discrete nodular silicosis is, with rare exception, entirely compatible with the working capacity adequate to determine full employment," but that "conglomerate shadows" indicate disease that is often seriously disabling. Bruce,⁶¹ Rossier, Bucher and Wiesinger⁶² agree that only in Stage III silicosis is marked disability found. Peterson, Peterson and Startup,⁶³ Motley, Lang, Gordon and Haddock,⁶⁴ and Motley^{50,64} present evidence to show that there is no correlation between radiologic appearance and degree of disability. It is possible to reconcile the diametrically opposed observed viewpoints by questioning the criteria for disability in the latter series. Differentiation between dysfunction as opposed to disability also is not made. The sufficiency of the allowance made for age and the degree with which some series have been weighted with disabled men are open to question. Probably the ultimate answer will await pulmonary function testing of large groups of persons of all ages exposed to silica and similar groups not exposed to silica.

e. *Pulmonary Function Tests in the Evaluation of Disability.* The number and variety of pulmonary function tests used during the past decade to evaluate disability in silicosis have been tremendous. It is beyond the scope of this chapter to attempt a critique of the entire field of pulmonary function. The reader is referred to several excellent published studies.^{49,50,54,65-67} Details of equipment and performance of individual tests can be obtained from these sources. For the present purposes recent significant work will be summarized.

Motley⁵⁰ in a study of 500 hard and soft coal miners with pulmonary complaints and a history of many years of exposure inside mines, concluded that:

An accurate evaluation of the degree of pulmonary function impairment can be made from the following physiological tests: (1) Ventilation measurements from spirogram tracings (tidal volume, capacity, three-second vital capacity, maximal breathing capacity and the shape of the expiration curve following a deep breath), (2) the degree of bronchospasm present, (3) the residual volume and alveolar nitrogen per cent after seven minutes of oxygen breathing, (4) the arterial blood oxygen saturation at rest and immediately after step-up exercise, (5) the

⁶⁰ Personal investigation of O. A. Sander.

⁶¹ Bruce, *Acta Med. Scand.*, Suppl. 129.

⁶² Rossier, Bucher, and Wiesinger, *Visch. naturf. Ges. Zurich*, 83-92 (1947).

⁶³ J. N. Peterson, J. M. Peterson, and C. W. Startup, *Lancet*, **1**, 147 (1931).

⁶⁴ H. L. Motley, Clinical pulmonary physiology II, *Ind. Med. and Surg.*, **22**, 262-267 (1953).

⁶⁵ Baldwin, A. Courmand, and D. W. Richards, Jr., Pulmonary insufficiency; physiologic and clinical methods of analysis; standard values in normal subjects, *Medicine*, **27**, 242-253 (1948).

⁶⁶ J. H. Comroe, ed., *Methods in Medical Research*, Vol. II. Year Book Publishers, Chicago, 1950, p. 74.

⁶⁷ R. Harvey, M. Ferrer, D. W. Richards, and A. Courmand, Influence of chronic pulmonary disease on the heart and circulation, *Am. J. Med.*, **10**, 719-738 (1951).