

progression of the disease. They use four categories, the first three of which are distinguished chiefly on the basis of the size and extent of the shadow, and the last of which indicates the presence of thoracic distortion and emphysema.

Categories of Complicated Pneumoconiosis

Category A. Shadows occupying at least one anterior rib space, generally formed by the coalescence of more or less circular shadows, more than 1 cm in diameter, with indefinite margins and of uneven density.

Category B. Dense shadows, which are more extensive, better defined, and of more homogeneous density than Category A, but which extend over less than three anterior rib spaces.

Category C. The same type of shadow as in Category B, extending over more than three anterior rib spaces.

Category D. One of the preceding classes of shadows associated with serious distortion of the pulmonary architecture.

In the early stages of progressive massive fibrosis, the background of simple pneumoconiosis always can be seen, but as the shadows grow in extent, and particularly as emphysema develops, the underlying simple pneumoconiosis may become completely obscured. Such cases are rare, however, the vast majority still showing evidence of nodulation even after emphysema occurs.

The validity of Category 1 has been questioned in this country because pinhead shadows can be seen in the chest films of many persons who never have had dust exposure in industry. It has appeared to many that this was the stage that the late Dr. Homer Sampson liked to call the "Stage of Imagination." Fletcher has justified it with controlled studies using seven film readers who had had wide experience in this field. He emphasizes that persons so classified never are disabled. It should be noted also that the Silicosis Board of England does not accept applications for compensation of miners having only a Category 1 classification.

Gough¹⁰⁰ and Heppleston have shown that the pinhead shadows are due to focal collections of coal pigment in the peribronchiolar lymphatics, referred to as coal macules. The mixed type appears to be due to a combination of coal macules and small silicotic nodules, while the nodular type usually is due to definite nodular silicosis. In the complicated groups, the confluent shadows are due to progressive massive fibrosis, which is thought to be the result of the combined effects of the tubercle bacillus and the coal dust. Fletcher thinks of coal dust as modifying tuberculous infection to the extent that many miners would have died of tuberculosis had they not had coal-dust exposure. It is noteworthy that the tuberculosis mortality always has been lower among coal miners than among all-occupied and retired men in Britain. The theory that all progressive massive fibrosis in coal miners is a modified tuberculosis is strongly supported by the evidence, but is unproved.

Gough and Heppleston^{103,104} also have shown that a focal type of emphysema was a characteristic finding in coal workers' lungs. At first this emphysema was thought to be due to a dilatation of the alveoli immediately adjacent to the coal macules. More detailed recent studies suggest, however, that it is essentially a dilation of the respiratory bronchioles and that the alveoli are relatively normal. The pathogenesis of the bronchiolar dilation is thought to be a direct effect of the coal deposits on the smooth muscle in the walls of the bronchioles. When such dilation becomes extensive, increase in the dead air space is thought to impair the ventilatory capacity of the lungs to the extent where exertion produces dyspnea.

This concept of disabling emphysema is in sharp contrast to that which has been generally accepted in this country. Most disabling emphysema is thought to be due to obstruction of the bronchial and bronchiolar airways. Lung function tests and the beneficial effect of bronchodilator aerosol therapy by Motley¹⁰⁵ and others strongly suggest that airway obstruction is the principal cause of chronic diffuse emphysema. It appears likely that even though there may be a significant degree of focal emphysema in a simple pneumoconiosis case, there must still be chronic bronchial obstruction to produce breathlessness. As a matter of fact, both Fletcher and Hugh-Jones have repeatedly stated that miners with simple pneumoconiosis have little or no measurable impairment of lung function when the age factor is taken into consideration, but workers with progressive massive fibrosis may be severely disabled. Death rates from all causes among retired miners in Wales showed that cases of simple pneumoconiosis have the same mortality rate as all men in England and Wales, while there is an increased mortality rate in those with massive fibrosis. This strongly suggests that focal emphysema alone is not a factor in causing right heart strain. Present evidence from the British studies, therefore, seems to indicate that simple coal pneumoconiosis and focal emphysema are not physiologically significant unless markedly advanced. Workers with minimal coal-dust deposits and disabling emphysema may have two separate entities which are unrelated, and other causes for the emphysema must be looked for.

In this country there never has been an industry-wide survey in bituminous mines, except for the study in a Utah mine by the United States Public Health Service in 1941-1942. This showed a low incidence of anthracosis (3.2 per cent) and tuberculosis, and no evidence of any significant degree of emphysema. The only other American studies have been made on miners who think they have dis-

¹⁰³ A. G. Heppleston, The pathological anatomy of simple pneumoconiosis in coal workers, *J. Pathol. Bacteriol.* 66: 235-246 (1953).

¹⁰⁴ A. G. Heppleston, The pathogenesis of simple pneumoconiosis in coal workers, *J. Pathol. Bacteriol.* 67: 51-63 (1954).

¹⁰⁵ L. Motley, L. P. Lang, and B. Gordon, Use of intermittent positive pressure breathing combined with nebulization in pulmonary disease, *Am. J. Med.*, 5, 853-856 (1948).

¹⁰⁶ R. H. Plinn, H. E. Seifert, H. P. Brinton, et al., Soft coal miners' health and working conditions, *U.S. Pub. Health Bull.* No. 270, 1941.