showed no evidence of fibrosis or other cellular response. The same has been shown to be true for barium and tin deposits.[43]

Gross examination. Except with prolonged and intensive exposure, the pleural surfaces of lungs with benign pneumoconiosis show only focal and linear collections of pigment that are soft in consistency and not raised above the surrounding tissues. On cut surfaces, gross examination reveals rounded flecks of pigment 2 to 3 mm. in diameter, but a lens shows finer linear deposits in the interlobular septa and in the outer walls of the blood vessels. The entire lung may be colored by pigment as in the black lung of soft-coal miners, but no fibrosis is found unless there has been exposure to free silica. There may be small patches of emphysema in or adjacent to pigmented areas. Scars of healed infections differ from the usual scars by the presence of pigment.

Microscopic examination. If death occurs during exposure, dust particles are found free in the peripheral air spaces or ingested by alveolar phagocytes that are found adherent to alveolar walls and in loose areolar tissue about the blood vessels, especially the arteries, and in the interlobular septa. These last two linear deposits are referred to as perilymphatic deposits because of their close relationship to the lymphatic trunks. The bronchi show relatively little dust; when any is present, however, it is found in the connective tissues just beneath the epithelium. If there has been no recent exposure (years), the intrapulmonary dust tends to be removed from the air spaces and deposited along the lymph trunks; in severe exposure large amounts of dust-filled phagocytes may remain in the alveoli. Nearly all pure substances other than silica cause little cellular reaction. Coal and some silicates, especially mica, may cause minor irritation without producing fibrosis. Unless the linear reaction is fibrous and caused by free silica, there is no altered susceptibility to tuberculosis.

b. Discrete Nodular Silicosis: X-ray examination. This form of dust disease is characterized in the x-ray film by enlarged and dense hilum shadows and small discrete nodular shadows, uniformly distributed throughout all parts of both lungs with the possible exception of small emphysematous areas in the costophrenic region. In the absence of infection, the nodules are of uniform size, usually not exceeding 4 mm. and seldom more than 6 mm. in diameter. The nodules are sharp with well-defined borders (see Figure 6).

Gross examination. The pleurae are studded with slightly elevated, grayish nodules 2 to 3 mm. in diameter, and around these there may be a flat zone of black, gray, or brown pigment. Pleural adhesions may or may not be present. The lungs are stiffer than normal but crepitus is present. There are palpable shotty nodules. The cut surfaces are seeded with black or gray nodules, 2 to 4 mm. or

[43] A. Arrigoni, Pneumoconiosi da bario, *Med. d. lavoro*, **24**, 461–468 (1933); E. P. Pendergrass, Roentgen diagnosis of pneumoconiosis and silicosis, *Am. J. Roent. and Rad. Ther.*, 48, 571–594 (1942) (Baritosis, p. 580); E. P. Pendergrass and A. W. Pryde, Benign pneumoconiosis due to tin oxide, *J. Ind. Hyg. Toxicol.*, **30**, 119–123 (1948); F. Barták, M. Tomecka, and O. Tomíček, Stannosis, *Casop. lék. ces̆k*, **87**, 915–932 (1948); C. C. Dundon and J. P. Hughes, Stannic oxide pneumoconiosis, *Am. J. Roent. and Rad. Ther.*, **63**, 797–812 (1950).

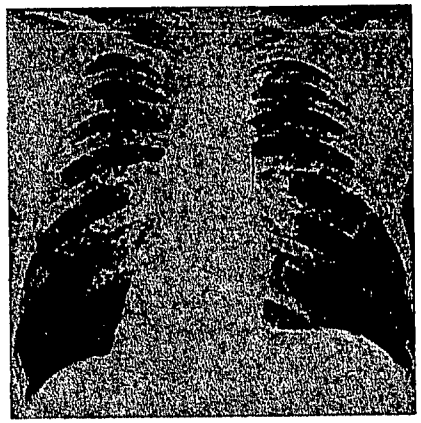

Figure 6. Classical nodular *silicosis* of moderately well-developed degree in sandstone wheel grinder. Only slight shortness of breath on exertion. Note large and dense root shadows.

rarely 6 mm., in diameter. The edges of the nodules are well defined, but a lens shows pigmented strands radiating from their periphery. There is little confluence except where scars or infection are present. Gross emphysema may or may not be present, but this condition may be apparent only with microscopic examination. In differentiation from perilymphatic pigmentation there must be definite pleural nodulation in an appreciable amount. Some nodules may arise along the peripheral branches of the pulmonary arteries. The tracheobronchial lymph nodes are at first enlarged and firm, later smaller and extremely hard. Sections of these nodes reveal dense leatherlike tissue, which is silky in texture because of the decrease in collagenous connective tissue.

Microscopic examination. Microscopic examination shows layers of dense hyaline collagenous fibers with nuclei so compressed as to be almost invisible at times. The borders are clear cut, with no exudation. There may be pigment either about the periphery or in focal points within the nodule itself. The nodule may be calcified, occasionally to the point of bone formation in its central portion. Nearly all nodules are associated with branches of the pulmonary arteries, as demonstrated by wax reconstruction of the arteries; they may form spherical or spindle-shaped masses around the arteries. Pigmentation occurs about the lymphatic trunk just as in nonspecific pneumoconiosis, but there is always some fibrosis. The air spaces immediately adjacent to the nodules are distorted and frequently dilated. There may or may not be emphysema. The alveolar walls between nodules may be entirely normal.

c. Modified Silicosis. Modified nodulation results from breathing dust containing free silica mixed with other minerals. Although a great deal is not known about mixed dust reactions, there have been sufficient examinations of the lungs of