Silicosis	Silicosis with infection
No change noted until extensive fibrosis has taken place, when there may be an increase in tactile fremitus generally.	When infection is extensive, tactile fremitus is increased and occasionally friction fremitus may be elicited. Extensive thickening of the pleura or pneumothorax may produce a decrease or absence of tactile fremitus over affected areas.

Percussion. There is usually an impairment of the percussion noted over the whole chest but unless one is particularly observant, this may not be detected.

Silicosis	Silicosis with infection
Since impairment in resonance is general over all lung areas, it is difficult to demonstrate until advanced fibrotic changes have developed. Decrease in diaphragmatic excursion may sometimes be revealed by percussion.	Increase in loss of normal resonance. When massive areas of fibrosis have developed this may amount to absolute dullness over areas involved. Amphoric resonance may be elicited where there is pneumothorax. Increased diaphragmatic excursion may sometimes be shown in advanced infection.

Auscultation. Breath sounds are usually decreased in intensity and the character and prolongation of expiration is readily noted.

Silicosis	Silicosis with infection
Decrease in breath sounds general and more marked as the condition progresses. Subcrepitant râles, which clear up after coughing, are occasionally heard.	Usually heard, some of the following: persistent post-tussic crepitant and subcrepitant râles; coarse rhonchi associated with productive coughing, wheezing; crepitant râles increased by exertion and coughing; amphoric breath sounds over cavities and areas of pneumothorax; pleural friction rub occasionally.

Part of the symptomatology associated with silicosis may be due to pathological processes other than fibrosis. Filley, Hawley, and Wright,[46] using isolated perfused lungs of guinea pigs found that colloidal silica produced bronchial constriction, although soluble silica did not. This at least suggests that part of the disability in silicosis may result from bronchial constriction. The inability of soluble silica to cause bronchial constriction, however, raises a question as to the validity of the assumption that this same reaction occurs in workers exposed to silica.

6. *Diagnosis of Silicosis*

To make a definite diagnosis of silicosis, each of these three criteria must be fulfilled: (*1*) history of adequate exposure to dust containing free silica in particle sizes below 10 μ; (*2*) exposure to such dust over a period of years depending on the concentration of free silica in the dust; (*3*) a nodular x-ray pattern with

[46] G. F. Filley, J. G. Hawley, and G. W. Wright, *J. Ind. Hyg. Toxicol.*, **27**, 37 (1945).

shadows enlarged and increased in density. The history of exposure has become of increasing importance because of the many other conditions that now are known to simulate nodular silicosis by x-ray. Hamlin[47] has found more than [illegible] conditions or diseases that cannot be differentiated from silicosis by x-ray alone.

In evaluating the past dust exposures, it is essential to start with the very first gainful employment, including part-time jobs during the school years. Many instances could be cited of significant dust exposures on such part-time jobs in the remote past that were the only explanation for x-ray lung changes found much later in life. Also, precise details of each occupation must be covered. It is not enough, for example, to elicit a history of foundry work in the past. It is important in determining the type of foundry, the type of castings poured, the size of the castings, the method of cleaning the castings, and the precise occupation in the foundry. It is not enough even to know that one of the past jobs was grinding. Where was the grinding done? Any other jobs in that area that caused dust or fume exposure? If so, what were they? What type of castings were being ground? How had they been cleaned before grinding? What abrasive was used in the grinding wheels? The only dusts to which iron and steel grinders may be exposed when their castings are free from molding sand are iron and the artificial abrasive, aluminum oxide, in which case they could not have silicosis. Silicon carbide (SiC), which is carborundum, may be mildly fibrogenic, but aluminum oxide never is.[48]

Even after the most detailed occupational history and x-ray film evaluation, occasionally it is impossible to differentiate silicosis from sarcoidosis. It then becomes necessary to make special tests for sarcoidosis, such as serum protein determinations to see if the a/g ratio is reversed, the Kveim skin test, and liver function tests. Even these may be inconclusive, in which case a surgical lung biopsy must be resorted to. These have been done with increasing frequency in recent years. Danger of complication is practically nil, and the results usually are conclusive when the surgeon makes a careful selection of an involved area.

7. *Evaluation of Disability in Silicosis*

Statement of the Problem. To both management and labor, silicosis at one time was nearly synonymous with total disability. It has now become clear that uncomplicated, first-stage, discrete, nodular silicosis is notorious for its lack of symptoms. Even relatively advanced stages of silicosis may be unaccompanied by disability. In fairness to both employer and employee, it is necessary to go beyond simple diagnosis. In examining a patient with roentgenographic evidence of silicosis, an attempt must be made to answer certain questions. Does he have a history of sufficient exposure to free silica dust to account for the

[47] H. H. Hamlin, Chest conditions simulating silicosis, *Ind. Med.*, **15**, 376–381 (1946).

[48] O. A. Sander, Foundry workers' pneumoconiosis, *Arch. Ind. Hyg. and Occupational Med.*, **10**, 512–521 (December, 1954).