size on successive films. Ultimately central rarefaction, interpreted as cavitation, may develop with subsequent evidence of infection in other parts of the lung. Gardner[44] presents the following working hypotheses with regard to conglomerate lesions:

1. In silicotuberculosis the infection may heal so completely that its tuberculous origin is no longer recognizable. Such an outcome has been observed in experimental animals exposed to ferruginous chert and infected with attenuated tubercle bacilli.

2. Conglomerate lesions in the upper lung fields, particularly when they are bilateral, are in the great majority of cases due to an underlying tuberculosis in healed or latent form. In persons not exposed to dust, bilateral disease in this location proves to be tuberculous in 96 to 98 cases out of every hundred, and even unilateral disease can safely be considered tuberculous in 90 per cent of cases.

3. Large isolated or bilaterally symmetrical conglomerations in the lower lungs should be considered tuberculous unless this origin can be excluded. In that case the possibility of organizing pneumonia due to Friedlander's bacillus or other organisms should receive consideration.

4. The character of the fibrosis resulting from the combined activity of an infectious organism and silica dust with or without other minerals may suggest a relationship between the time of exposure to dust and the development of the infection.

(a) When the silicosis is already established before tuberculosis supervenes the infection localizes in and about the nodules which retain their form but increase in size. Bronchial obstruction *caused by the infection* may produce widespread atelectasis with a consequent approximation of the nodules in the involved area. In such cases the outlines of large masses of nodules in the resultant conglomeration are distinct and clearly defined.

(b) When silicosis and chronic tuberculosis have developed simultaneously the nodular character of the conglomerate fibrosis is less obvious. Nodules are present but they are surrounded by a matrix of more diffuse fibrosis produced by the action of the silica upon the organizing pneumonic process.

(c) When the scars of a healed infection are already present before employment in a dusty industry, the inhaled particles accumulate in particularly large quantities in their immediate vicinity. Continued accumulation in the localized area produces many nodules which are small because they are closely packed together but typically spherical in outline because there is no activity in the underlying infectious process to produce diffuse fibrotic reaction.

Many patients with conglomerate silicotic fibrosis are dyspneic, but the toxic symptoms of the associated infection may be absent. This is reasonable since healed tuberculosis does not cause symptoms and even active infections may be so well encapsulated by dense fibrous tissue that no tissue reaction is possible.

5. *Silicotuberculosis*

The relationship between silica exposure and the development of tuberculosis has been well demonstrated in the United States as well as in other countries where there has been such exposure. One of the most remarkable demonstrations of the relation of tuberculosis to silica dust exposure is found in the work of Mavrogordato,[45] illustrated graphically in Figure 11.

[44] L. U. Gardner, "Pathological Anatomy," in B. E. Kuechle, ed., *Fourth Saranac Laboratory Symposium on Silicosis*. Employers Mutual Liability Insurance, Wausau, Wis. (1930).

[45] A. Mavrogordato, *Pub. S. African Inst. Med. Research*, Report No. 19 (1926).

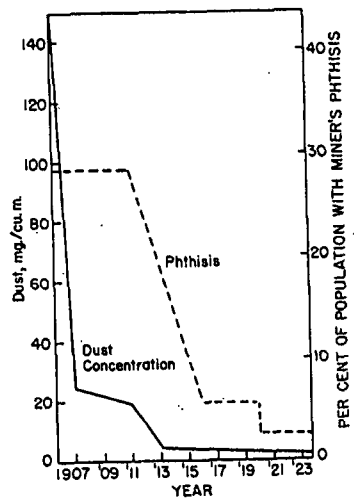

Figure 11. Dust control and silicosis in South Africa.

The symptomatology and clinical findings in silicosis and in silicosis complicated by tuberculosis (Figure 12) have been summarized in the National Silicosis Conference Report of the Committee on Medical Control and these have been condensed by Lanza[19] as follows:

SUBJECTIVE SYMPTOMS: *Dyspnea*. The complaint most frequently mentioned is shortness of breath. Depending upon the extent of the involvement, this varies from slight

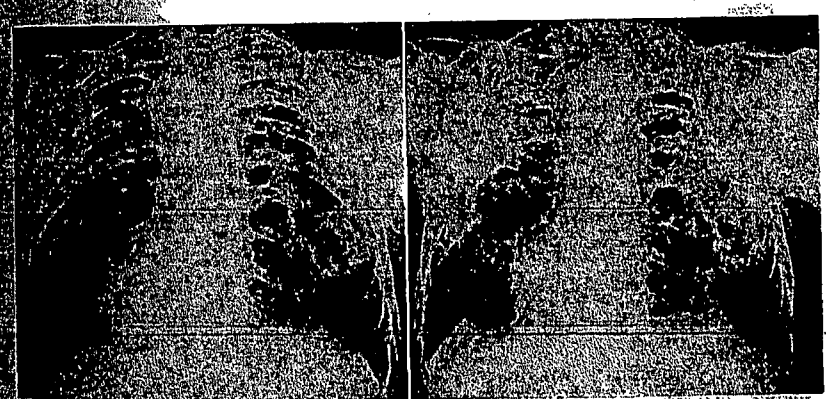

Figure 12. Progressive *silicotuberculosis* and emphysema, with tubercle bacilli never found in numerous sputum and gastric cultures. Marked dyspnea and impaired lung function. Patient died in 1948 of right heart failure.