and also, though not to the same degree, in the second group. In one worker atrophic pharyngitis had developed. The number with laryngeal changes appeared to have increased only in the first-mentioned group. That chronic changes of the upper respiratory tract may appear also after brief but heavy dust exposure is borne out by the fact that so many workers (particularly three of them) still had symptoms after having left the factory (two even had bronchopneumonia) and showed significant changes also at the final examination.

As pointed out by all previous authors, bronchial disorder is the essential feature of the disease. Most of the workers in the present series complained, at least periodically, of symptoms in the form of a cough with or without sputum, wheezing sounds in the chest and frequently shortness of breath. During the worst period the bronchial symptoms were distributed as follows among the 36 workers: no cough, 0; cough with no sputum, 22; cough with copious sputum, 14; wheezing sounds in the chest, 31; shortness of breath, 27.

The frequency of these complaints is all the more evident on comparison of the exposed workers and the controls, 4 per cent of whom showed "much coughing," and 24 per cent, "shortness of breath more marked than normally."

The cough was described in varying terms. In some cases it was said to be violent, particularly at night, with attacks of choking. In many cases it was worse in the evening or in the morning; in others, no worse than "a tickle in the throat." In the daytime, for example, at the examinations, many of the patients had a more or less violent and harsh cough, which appeared to me to be rather characteristic. When the cough was attended by expectoration, the product was generally viscid and mucoid, sometimes more copious, but not blood-stained, and appeared mostly in the morning. No severe hemoptysis was recorded. In many cases rhonchi predominated ("wheezing like a loud siren"), particularly at night, causing slight shortness of breath, and audible as sibilant rales on auscultation. The shortness of breath was most noticeable on exertion. Bronchoscopy, which was performed in four cases, showed no severe changes (histologically, in one case, very mild chronic bronchitis).

As already mentioned, the particle size of the factory dust indicates its ability to pass far down the respiratory tract, most probably as far as the alveoli. Formerly this was considered possible for particles of 10.5μ, or perhaps slightly bigger (McCrae,[10] 1912), whereas according to Hatch[11] (1948, 1950), only particles below 3μ should be able to reach the alveoli. As 22 per cent of the factory dust had a particle size of 8μ or smaller, possibilities seem to exist for the appearance of lung complications.

Acute changes in the lungs, manifested in edema or "pneumonitis," are caused by several metals in the form of dust. Besides a purely chemical corrosive process (chemical pneumonitis, caused, for example, by beryllium, nickel, cadmium and osmium) there are probably several other factors to be considered. A definite inflammatory reaction may prepare the way for secondary infection. It is also reasonable to presume that an infection may be promoted by chronic changes

10. McCrae, J.: The Ash of Silicotic Lungs, The South African Institute for Medical Research, Johannesburg, 1913.

11. Hatch, T., and Hemeon, W. C. L.: Influence of Particle Size in Dust Exposure, J. Indust. Hyg. & Toxicol. **30**:172, 1948. Hatch, T.: Paper read at the Sydney Congress, 1950, to be published.