Silicosis reflects a failure in adequate control of occupational dust exposure. The disorder is associated with radiologic nodular pulmonary opacification which, in its chronic form, predominates in the upper lung zones. Chronic silicosis generally occurs after prolonged low-intensity exposure to dust containing crystalline silica. Such low-intensity exposure causing simple silicosis has often been considered not to cause pulmonary impairment. More recent data have shown chronic simple silicosis to be a significant lung disease. In a previous study of the same population of gold miners, pulmonary dysfunction was found to be associated with silicosis and to increase in proportion with the degree of silicotic nodule profusion. There are now data to suggest that the silicosis is associated with emphysema, which may contribute to the lung dysfunction. This study examined the progression of lung dysfunction in a cohort of gold miners with and without silicosis. This study shows that the progression of lung dysfunction is related to the initial extent of the silicosis and substantially exceeds that predicted by the earlier cross-sectional study.

**Materials and Methods**

The study was conducted in the goldfields of the Free State Province of South Africa. The development of the cohort of 1,197 older gold miners with and without silicosis has been described previously. In summary, a sampling frame was created by selecting men whose routine screening chest radiographs showed changes in keeping with silicosis and matching each of these men with a similarly aged man without silicosis whose radiograph was read on the same day by the same radiograph reader. Men were selected consecutively from the sampling frame, in the order in which they were entered in a ratio of five with silicosis to two without silicosis until the predetermined end of the study period. Five years after the entry of the men into the cohort, it was...
established that 950 (79%) were currently employed as miners. These 950 men did not differ from the total cohort in terms of their original category of lung nodule profusion, length of service on the mine, or smoking history. They were listed according to the nodule profusion on their initial radiograph. They were then sorted by employee number (which had previously been shown to have no relationship to length of or nature of service).

A sample was then created by including all men with category 3 nodule profusion, every third man with nodule profusion 2/3, and every fourth man in the remaining nodule profusion categories. The resultant sample included 267 men who were invited to attend for follow-up assessment with a short questionnaire, lung function tests, and chest radiograph. The questionnaire included questions about their current occupation, their smoking status, and the presence and extent of dysnea, cough, and sputum production. The lung function tests were done according to the standards of the American Thoracic Society\(^8\) by one technician who had done their original lung function tests.

The equipment used was the same as that used for their original tests (Morgan Transfertest Model A incorporating an S-L dry rolling seal spirometer and a flow-volume differentiater [P.K. Morgan; Chatham, UK] linked to a Medical Graphics analogue-digital convertor [Medical Graphics; St. Paul, Minn] and an Apple 2e computer with Medical Graphics operating software). Tests included spirometry and measurement of lung diffusion by the single-breath method.

A single posterior-anterior 125-kV chest radiograph was obtained after completion of the lung function tests and questionnaire and was submitted to the two readers who had read the original chest radiographs. The reading of the radiographs for nodule profusion was done by each reader, independently, with reference to the original radiographs or to the lung function or questionnaire data, using the standard radiographs and instructions of the International Labour Office.\(^9\) If the two readings of the radiograph were not identical and only one subcategory of nodule profusion separated the readings, that subcategory was accepted. When the readings were not identical and no subcategory separated the readings (eg, 2/2 and 2/3), the interpretation of one or other of the readers was accepted depending on whether the subject’s employee number was odd or even. When more than one subcategory separated the readings, a joint reading of the two readers was accepted. To facilitate analysis, the radiographic nodule profusion was converted into a continuous variable ranging from 1 to 10 (no radiograph was read as 0/¬ or 3+/ ) with 1 equal to International Labour Office category 0/0 and 10 as 3/3. Silicosis was defined as nodule profusion of ≥1/0. The computerized tuberculosis register for the miners was scanned to determine whether any of the men in the sample had developed tuberculosis since their entry to the cohort.

Analysis of the data was performed by linear regression to assess the impact of age, cumulative smoking history, initial lung function, initial presence and extent, and progression of silicosis, tuberculosis, and continued exposure to the mine environment on current lung function. Additional evaluation of continuous variables was by analysis of variance\(^10\) and of categorical variables by \(\chi^2\) analysis.

**RESULTS**

Twenty-five (9%) of the 267 men selected for this follow-up study were unable to participate as they were away from the mine on leave. The remaining 242 men did not differ from the total cohort of 1,197 men with regard to their age or length of under-ground exposure. The smokers in the sample had a smoking history that was similar to that of the cohort but the proportion of never-smokers was 24% in the sample and 37% in the original cohort (\(\chi^2\) df1 = 12.7, \(p=0.005\)).

The follow-up assessment occurred approximately 5 years since their entry to the cohort and a mean period of 4.5 years (SD, 0.55 years) after their initial assessment. Thirty-two of the men were no longer working underground. Thirty-nine of the 59 men without silicosis on entry remained free of silicosis. One man with category 1 nodule profusion initially was not thought to have silicosis at follow-up. On average, the degree of nodule profusion on the chest radiograph had increased by one subcategory in the 4.5-year period. In 32 men, the new radiograph reading suggested a reduction in nodule profusion; in 59, the readings were unchanged; and the readings indicated an increase in nodule profusion in 151 of the subjects. A total of 202 men had silicosis on their follow-up chest radiograph (183 on the initial chest radiograph); 40 (78) with category 1 nodule profusion; 120 (73) with category 2; and 42 (32) with category 3 nodule profusion.

An average of 78% of the men complained of a cough and 62% complained that their cough was productive of sputum. There was no relationship between the presence of cough and/or sputum production and the presence or degree of silicosis. Cough and sputum production, however, were associated with past or present smoking: cough was noted by 57% of nonsmokers and 85% of past or present smokers (\(p=0.0003\)) and sputum production by 41% of nonsmokers and 70% of smokers (\(p=0.00007\)). However, a complaint of dysnea was not related to smoking history but was less common in men without silicosis (25%) and more common with increasing silicotic nodule profusion (31% in category 1, 38% in category 2, and 56% in category 3: \(\chi^2\) df3 = 8.9, \(p=0.03\)).

Lung function declined in excess of that expected for their increased age in 212 of the 242 subjects. Loss of FEV\(_1\) was an average of 75 mL/yr for the whole sample. Men who did not have silicosis on entry lost 37 mL of FEV\(_1\) per year, and men with silicosis on entry lost 87 mL of FEV\(_1\) per year. The loss of lung function varied directly with the degree of nodule profusion (Table 1 and Fig 1). There was no significant difference in the initial FEV\(_1\) or in the annual loss of FEV\(_1\) between the men (210) who continued to work underground and the men (32) who moved to nondust occupations. No excess loss could be attributed to smoking; the average cumulative consumption of the smokers (ex-smokers and current smokers) was 13.7 pack-years and the 134 men who had continued to smoke had smoked an
average of 2 pack-years since their initial assessment. The miners (54) who had developed pulmonary tuberculosis since entering the cohort had an additional 29 mL/yr loss of FEV₁ and the influence of tuberculosis on lung function persisted after controlling for category of silicosis. After controlling for these variables and for the initial FEV₁, the degree of nodule profusion on the entry chest radiograph remained a significant determinant of the follow-up FEV₁ (p=0.0005).

**DISCUSSION**

It was not possible to examine the entire cohort of 1,197 men in this unfunded study nor did resources exist to include the men who were no longer employed as miners. The men who were selected for this follow-up study were chosen in a random fashion after stratification into their original silicosis nodule profusion category. The sample studied did not differ with regard to any of the measured characteristics from the members of the cohort who remained employed as miners. The men who had left the industry were older than the men who remained but were otherwise apparently similar. It is probable given their age, the duration of their work as miners, and the high level of unemployment that prevailed at that time that the men who had left were more disabled than those who had remained at work. It is thus unlikely that the results of this study overrepresent the pulmonary dysfunction of the whole cohort.

The initial cross-sectional study of this cohort suggested that a loss of approximately 8 mL of FEV₁ per year would be expected from continued exposure to the dusty mine environment, which would be equivalent to approximately 32 mL/yr adding the expected loss of 24 mL/yr of aging according to the Crapo et al. regression equation used in this study. The findings in the present study approximate this expectation with the 37 mL/yr loss of FEV₁ in the men who did not have silicosis on entry. Graham et al. reported an average annual loss of 30 mL of FEV₁ in their cohort of Vermont granite workers. Hnizdlo attributed 10 mL loss of FEV₁ per year to continued dust exposure in a longitudinal study of gold miners that is similar to the estimated 13 mL/yr loss after adjustment for age in the men without silicosis on entry to the present study. Malmberg et al. attributed 0.38%/yr loss of FEV₁ to silica dust exposure in granite crushers, which is remarkably similar to the 0.35%/yr loss of predicted FEV₁ in the men in the present study who did not have silicosis.
Thus, it would seem that the miners without silicosis lost lung function to a degree that conforms with that reported by others and predicted by the earlier cross-sectional study.3

The striking finding in this study was the extent of the additional loss of lung function related to the presence and degree of silicosis on entry to the cohort. This observation held true for the men who, at their follow-up evaluation, had silicosis uncomplicated by tuberculosis or by the development of large opacities and who thus, by definition, had simple chronic silicosis. Men with silicosis suffered a loss of FEV₁ that exceeded the loss in the men without silicosis on entry by 210 mL over the follow-up period of 4.5 years. On average, their silicosis had progressed by one subcategory (eg, 2/1 to 2/2) during the period of their follow-up: based on the results of the cross-sectional study,3 one would have expected a total FEV₁ decrease of 53 mL for this average increase in nodule profusion. The actual decrease of FEV₁ apparently attributable to their silicosis was fourfold greater than this estimate.

The fact that the study failed to show a loss of lung function in relation to smoking probably reflects the small consumption of those who continued to smoke: an average of 2 pack-years in 4.5 years. It also suggests that the study was not sensitive to minor changes in lung function, which emphasize the significance of the changes associated with the initial presence and degree of chronic, simple silicosis.

In conclusion, this study of a sample of a cohort of older gold miners reexamined 4.5 years after their initial assessment has shown a substantial loss of lung function attributable to the presence and degree of silicosis. Silicosis in these miners increased by an average of one subcategory of nodule profusion during the 4.5 years. Men who had withdrawn from dusty occupations showed no difference in rate of lung function decline in this short period of follow-up when compared with men who had continued to work underground. Men who had developed pulmonary tuberculosis suffered from an additional loss of lung function after controlling for category of nodule profusion.

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