degree and duration of asbestos exposure. Table 2 shows that the smoking groups were similar in age. With respect to duration of asbestos exposure, nonsmokers and current smokers were similar but exsmokers averaged eight years more of work at the plant, this difference being statistically significant. With respect to cumulative asbestos dose, the same pattern appeared: current smokers were similar to nonsmokers, but exsmokers had an average index which was 40 to 55% higher than the average index for the other two groups. Although these differences may be important, they are not statistically significant by two different methods of analysis.

When the men were distributed according to both cumulative dose of asbestos and cigarette habit, the prevalence of plaques read by W.W. varied with respect to both factors (Table 3). The data suggest that the prevalence of plaques is high in exsmokers at any asbestos dosage but that in nonsmokers and current smokers the prevalence increases with increasing asbestos dose. Unfortunately, cross-classification by the two factors results in cells with very small numbers. However, if the data in the two lower asbestos dose categories are combined, then plaques were found in 6 of 21 nonsmokers and current smokers compared with 6 of 7 exsmokers. This difference is statistically significant at the 0.05 level using significance limits for the fourfold table test based on the hypergeometrical distribution.* The findings were similar for plaques read by L.C. but the differences were not as clear-cut and were not statistically significant at the 0.05 level.

Table 3. — Prevalence of Pleural Plaques (Read by W.W.) by Estimated Cumulative Dose of Asbestos and Cigarette Habit in Men Aged 40 or Over.

Asbestos Dose and Cigarette Habit	No. of Men in Group	No. of Men with Plaques
Asbestos index under 20		
Nonsmoker	5	1
Current smoker	8	2
Exsmoker	1	1
Asbestos index 20-59		
Nonsmoker	3	1
Current smoker	5	2
Exsmoker	6	5
Asbestos index 60+		
Nonsmoker	4	2
Current smoker	6	5
Exsmoker	7	5
Total	45	24

Discussion

Several investigators have noted an association between pleural thickening and smoking in workers exposed to asbestos. In a large survey of English naval dockyard workers exposed to chrysotile, amosite, and crocidolite, P. C. Harries et al[2] found pleural thickening in 2.7% of 5,552 nonsmokers, 4.9% of 12,798 smokers, and 6.4% of 4,990 exsmokers. The prevalence of pleural thickening tended to fall slightly with increasing cigarette dosage.

In recent published reports of the studies of these dockyard workers, Rossiter et al[3,4] have provided more details for segments of this population. In a sample of 1,200 men who were aged 50 to 59, pleural thickening was separated into diffuse disease and plaques.[3] Only the presence of plaques was related to the duration of exposure to asbestos and there was an excess of plaques in exsmokers (29.5%) compared with the prevalence in smokers (23.9%) and in nonsmokers (23.1%). The prevalence of pleural calcification was different: 8.8% among nonsmokers, 3.5% among smokers, and 6.1% among exsmokers. A nine-year (1966-1975) follow-up study of 253 dockyard workers[4] found increases in the prevalence of diffuse pleural thickening, almost entirely limited to those men who had stopped smoking after 1966, and of pleural plaques, with the greatest increase (from 10% to 23%) in smokers, but little change in the prevalence of calcified plaques.

Hillerdal[5] reported a study of pleural plaques in chest x-ray surveys of the general population in the Swedish county of Uppsala. Of 492 men with plaques, 354 were interviewed and it was found that 79% were or had been smokers. The expected frequency in men of the same age and occupation is 55%. In a survey of 197 Swedish asbestos workers, Hedenberg et al[6] found pleural plaques in 30% of 103 smokers and 19% of 94 nonsmokers.

Thus, there is general consistency between the reports from England and Sweden and the data reported in the present study, especially with respect to exsmokers in those studies in which these have been separated from current smokers. Unfortunately the studies cited from the literature have no information on the possible confounding of this relationship by degree of asbestos exposure. The data in this study suggest that there may be some confounding. While the association between the prevalence of plaques and smoking was somewhat stronger than that between plaques and cumulative asbestos dosage, it must be remembered that the estimates of dosage were crude. It is uncertain whether the finding of 40 to 55% greater asbestos exposure in exsmokers could account for a prevalence of plaques which was more than double that in the other groups. Conclusions are restrained by the small number of workers in this investigation. A clear-cut answer to the question as to whether the association between plaques and smoking is spurious will require a much larger research effort, preferably in a cohort study.

The hypothesis that smoking is a factor in the pathogenesis of asbestos-related pleural plaques is a plausible one despite the fact that the mechanism by which inhaled asbestos fibers produces fibrous plaques in the parietal pleura is unknown. Smoking is detrimental to various aspects of pulmonary function although the effect on the clearance of particulate matter is not clear.[7] A recent study by Cohen et al[8] shows, by the use of inhaled magnetic dust (magnetite), that long-term dust clearance is impaired in smokers: 50% retention compared to 10% in nonsmokers. It might well be that once smoking has damaged the clearance mechanisms, cessation of smoking could be more harmful in this type of defense than continued smoking because the short-term effect of cigarette smoking is to enhance deep bronchial clearance transiently in some people[9] and smokers may become dependent on this effect to maintain clearance. This

*[illegible] Geigy Scientific Tables, 8th Ed. pp. 120-121, 1962.