TABLE II
World Production of Asbestos*

YEAR	NO. TONS
1880	500
1900	35,000
1920	200,000
1930	450,000
1950	1,300,000
1960	2,300,000
1963	3,200,000
1964	3,540,000
1965	3,600,000

*Adapted from Multiple Sources

asbestos which is used for the most part in products which are unlikely to become airborne. The largest amount—one can only guess at the percentage—goes into asbestos products which, once applied do not disintegrate, such as building materials and floor tile. A fair estimate might be that some 10 to 15% of the asbestos in the approximately 3,000 end-products containing this mineral, e.g. brake lining and asbestos textiles, may by deterioration, weathering, heating, etc., become airborne. However, there are no studies of asbestos concentrations in ambient air. Indications that asbestos may be carried considerable distances in the airstream[7] are based upon: (1) Analysis of deposits on the surface of the earth, snow, etc.; (2) evidence that significant numbers of people with mesothelioma worked or lived near asbestos mines or plants; and (3) evidence that a significant population has asbestos bodies in their lungs at autopsy. But as yet the type, amount and particle size of ambient airborne asbestos has not lent itself to systematic detection and identification. It should also be borne in mind that asbestos deposits have mineral outcrops and hence there is a strong possibility that asbestos in ambient air may be a natural phenomenon and not only a result of increased industrial activity.

Clinical experience indicates that the appearance of asbestosis (pulmonary fibrosis) is dose-dependent. Children have died of asbestosis and cor pulmonale after a few years of an overwhelming exposure "within large shipping bags, trampling down fluffy amosite asbestos which all day long came cascading down over their heads."[8] Sluis-Cremer[9] gives an average in one factory in South Africa of 6 years at work before asbestosis was manifest. Jacob and Anspach,[10] in Dresden, determined an over-all average of 30 years before the asbestosis became disabling. X-ray changes are induced in most cases after 20 years' employment as an insulator (Selikoff, p. 147). One of our cases showed evidence of the fibrotic process on lung biopsy some 22 years after a heavy exposure of less than 2 years' duration.

TABLE III
Workers Exposed to Asbestos in the United States
(1963 Census of Manufacturers—Dept. of Commerce)

RAW PRODUCT MANUFACTURING	NO. WORKERS
Asbestos Products (1963)	19,516
Asbestos Paper Products (1963)	11,874
Asbestos Mines & Mills	497
Subtotal	31,997
USE OF MANUFACTURED PRODUCTS	
Insulation Workers (1967)	18,000
Approximate Total U.S. workers exposed	50,000+

While long latency is usual and progression of the fibrotic process may continue long after the exposure has ceased, there is little information forthcoming regarding quantitative data on the dose which each individual was exposed to. Lung clearance studies indicate a marked difference in the effects of short, heavy dust exposures as compared to minor, low exposures. Heavy dust inhalation incites an acute flammatory reaction and stasis in the respiratory bronchiole and adjoining alveolar ducts and may provide a nidus for a continuing effect.

That asbestosis, i.e. the pneumoconiosis, does not present a major problem for the future is mostly due to the fact that the occupational population at risk, which is difficult to determine exactly, is still quite small and can never reach large numbers (Table III).

While members of the construction industry, for example plasterers and some automobile workers, e.g. brake repairmen, may have greater exposures than insulators working directly with asbestos products, it is safe to say that the kinds of exposure experienced in industry will never affect any sizable group in the general or working population.

The threat from asbestos, however, lies in its possible association with lung cancer; and the expectation that there will be no increase in the mortality from this disease among exposed workers is not so sanguine. Cancer of the lung occurs more frequently in those with occupational exposure, whether or not they develop fibrosis, and this evidence is amply substantiated (Table IV). Two aspects are not known, the pathogenic factors and the dose of asbestos which produce this increased susceptibility. We do not know whether asbestos has properties of primary carcinogenicity or whether its effect is entirely synergistic. That other factors must potentiate the asbestos exposure has been postulated by Selikoff and Hammond[11] who show that asbestos insulators who smoke have about 90 times the incidence of lung