lungroots and lymphnodes, and in the spleen the possible means of transportation to be via the lymph- and blood routes. The latter seems rather unlikely in view of the spleen's minimal involvement and the unlikely possibility of long-fibred asbestos dust being carried through the bloodstream. Transportation of asbestos dust via the lymphogenic route from the lung across to the upper abdominal lymphnodes would meet with little success although, according to my own observations, this does occur with regularity in cases of silicosis.

The most likely possibility is that of asbestos fibres penetrating from the lung directly into the pleura and later on the diaphragm since the right dome of the diaphragm showed gross changes from tumors. This ability of asbestos fibres to penetrate the pleura, only confirms Wedler's publication regarding deposist in the pleura observed at regular intervals in asbestosis along with epithelial desquamation, fibrinous stratification, coalescence and crust formation, as well as formation of primary pleural tumors for which, once again, positive proof was submitted by Weiss showing asbestos bodies in tumor tissue. In the case, as presented here, one can, therefore, reasonably assume that the diaphragm was also penetrated by asbestos needles and that, a small portion of the inhaled dust, was able to enter the abdominal cavity in this direct way. The pleuritis exsudativa accompanied by calcified crust formation observed in 1947 lead to the assumption that, substantive asbestos dust deposits had already been present in the abdominal cavity for some time which brought us closer to an answer regarding the question of latency, i.e. the time period between exposure to asbestos dust and formation of carcinoma of the lung.