compounds and amino-compounds; phosphorus; and arseniuretted hydrogen) are followed, if the patient survives, by a compensatory regeneration of the liver tissue, replacing adequately the amount of liver tissue that has been destroyed. Such livers present a multicentric hyperplasia of liver cells forming rounded nodes of regenerated, functioning liver tissue, separated by broad tracts of fibrosis. There is no distortion of the fundamental lobular pattern. Once the destroyed tissue has become fibrosed and a certain degree of regenerative hyperplasia attained, the lesion becomes static (Stewart).

In the chronic progressive type of cirrhosis of the liver a continuous destruction of liver cells is present, which is followed by a corresponding compensatory hyperplasia of these cellular elements. This process may overstep its bounds and assume the character of an autonomous growth (Muir). The agents, which bring about such alterations, must act, according to Moon, repeatedly and continuously causing the production of diffuse chronic hepatitis. This is indicated by degeneration and necrosis, by regeneration of parenchyma, and proliferation of connective tissue. If injury to the liver parenchyma is severe, continued, and repeated, the repair will be accompanied by fibrosis (Moon). The reparative process does not result in the formation of the ordinary lobular pattern under such conditions. It takes on the form of nodular formations following the type of circulatory disarrangement, which causes the distortion of the architecture of the cirrhotic liver (Laennec's type). Cirrhosis of the liver associated with hepatic siderosis and hemochromatosis belongs to this group of hepatic fibroses, which exhibit a tendency to subsequent primary malignancy of this organ (Stewart). This investigator observed in a series of 151 cases of hemochromatosis, collected from various sources, 15 cases of hepatic cancer (9.93 per cent).

From this rather well-established interrelation between cirrhosis and primary carcinoma of the liver, it may be deduced that some of the causative exogenous agents operative in the production of hepatic fibrosis are directly or indirectly responsible for the development of hepatic malignancy. This carcinogenic effect represents a direct action of a carcinogenic component in the cirrhotic agent upon the liver cells, or the cirrhotic process results in the development of metabolic disturbances in the liver function, which give rise to the production of endogenous carcinogenic agents.

Since no information exists in this respect for the majority of known, exogenous cirrhotic agents with possible occupational significance, it is necessary to list and discuss briefly the various cirrhosis-producing factors whether they have shown carcinogenic qualities or not. Although proof of the cirrhosis-producing properties of many of these agents is based on experimental evidence, an occupational or general environmental exposure to a great number of them can be demonstrated. In fact, there exists a limited amount of casuistic information supporting the actual occurrence of cirrhosis of the liver of industrial etiology.