

abdominal wall (bruise or contusion) and to the stomach (appearance of gastric distress soon after the accident; bloody vomitus or tarry stools); 2) continuation of gastric symptoms following the trauma until definite evidence of a gastric neoplasm appears; 3) this preparatory interval must last at least six months; 4) the post-mortem examination must show residues of the preceding trauma (old hemorrhages, blood pigments in abdominal and gastric walls, adhesions and splinters or bullets lodged in the gastric wall in or near the carcinoma). If a medico-legal verdict should be rendered, favouring the presence of causal interrelations between the injury and the subsequent neoplastic disease, this decision rests upon probable relations, which are far from certain. The anatomical features of beginning gastric cancers and their general etiology are incompatible with a purely or principally traumatic origin. It may be conceded that an epigastric trauma may aggravate a pre-existing gastric carcinoma (Borrmann; Ewald; and Kissinger).

V. EXPERIMENTAL CANCER OF THE STOMACH

The experimental production of gastric cancer in animals has been accomplished by the use of various agents. Some are identical with those suspected of being operative in the causation of gastric neoplasia in man by occupational exposure (Klein and Palmer).

Tar and Crude Oil. Cancer of the stomach in mice was reported by Bonne following the application of tar to the skin. The percentage of mice developing gastric tumors depended upon the site and duration of tarring. The incidence was highest when the perioral and perinasal region of the skin was painted with tar, affording a greater opportunity for the oral ingestion of this substance. The papillomas produced were single or multiple, carcinomas were rare. Similar observations were made in mice exposed to cutaneous applications of tar by Koose and Cordes; and Walteman. Koose and Cordes believe that these remote effects of tar are not caused by the chemical itself, but by a bacteriophage-like agent, which penetrates the tarred skin and spreads in the body producing cancer in the stomach.

The development of gastric hyperkeratoses and papillomatosis in the forestomach of rats and mice was noted by Buschke and Langer; and Fischer, respectively, following the resorption of tar through the rectum. This conclusion was based on the observation, made by Fischer, that similar changes were found in the stomach of mice when tar was incorporated into the diet. This local intragastric action of tar was confirmed by Schabad and Ménétrier, respectively, who introduced tar into the forestomach of mice and rats, and by Brancati, who found papillomas and carcinomas of the stomach in mice and rats kept on a diet of milk and tar. Rats fed with a mixture of tar, lanolin, aniline oil, and toluylenediamine three to four times weekly developed in one case an adenocarcinoma of the stomach with metastases to the regional lymph nodes and liver. Other rats of this series showed retroperitoneal sarcomas and myeloid metaplasia of the spleen and lymph nodes (Voronoff and