tunity is afforded for any carcinogenic substances, possibly contained in the digested material, to exert their specific action upon the mucosa. Such an effect should manifest itself most in those parts in which the fecal matter moves very slowly or remains stationary for prolonged periods, depending upon the normal or pathological functional status of the bowel (spastic or atonic constipation). These considerations provide a plausible explanation for the considerably higher frequency of cancers in the large intestine compared with that in the small intestine, and for the relative distribution of these tumors in the different parts of the large bowel. The incidence increases rapidly with the approach to the anus and is highest in the sigmoid and the rectum (Körte; and Petermann and Anschütz: 297 colonic carcinomas: 47 in cecum; 22 in ascending colon; 19 in hepatic flexure; 44 in transverse colon; 31 in splenic flexure; 10 in descending colon; 124 in sigmoid. Kaufmann: 123 carcinomas of large bowel: 36 in colon; 28 in sigmoid; 51 in rectum).

The hemorrhoidal area of the anal region, with its sluggish blood supply, does not create a favorable soil for the development of rectal malignancy (as contended by Behan). On the contrary, rectal hemorrhoids figure rarely in the carcinogenesis of this part of the bowel (Ewing; Kraske; and Hayden and Shedden), in spite of the frequent irritation and injury to which the anal region and the protruding, hemorrhoidal nodes are exposed. A similar parallelism exists between the functional dynamics and the distribution of tumors in regard to the urogenous tract, in which the ureter takes the place of the small intestine, and is rarely the site of neoplasia.

III. PRECANCEROUS LESIONS

Congenital Polyposis. Several aspects of the developmental history and the local distribution of certain intestinal, precancerous lesions, the polypous adenomas, are in accord with the conceptions developed, regarding the factors controlling the causation and localization of intestinal malignancy. Intestinal, adenomatoid polyps are uncommon during the first decade and are usually single during this period (Erdmann and Morris). They become more frequent in the following decades and are then often multiple (Hullsiek; and Fansler). The age distribution of a series of 93 cases was as follows (Hullsiek):

Years:	1-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80
Cases:	5	23	21	23	11	4	5	1

While the presence of a hereditary factor was demonstrable in an appreciable proportion of the cases observed (Jüngling; Hullsiek: 11.1 per cent; Schüttler: 17.5 per cent; Döring (1907); Port (1897); Wechselmann (1910); Thorbeke (1914); and Lockhart-Mummery and Dukes (1939): 50 to 60 per cent), the absence of this condition among new-born babies (Feyrter), its rarity during the first decade, and its increase in frequency and multiplicity during subsequent years show rather definitely that these adenomatous pro-