which often contain intra- and extracellular eosinophilic infiltrations, hemorrhages, angiectases, and intravascular endothelial "thrombus-like" knobs; these resemble in many respects the nasal polyps of chronic and allergic rhinitis and display thus a certain likeness with the morphological manifestations characteristic of chronic allergic-hyperergic reactions.

The allergic-carcinogenic concept offers a plausible explanation for the variations in individual reactivity (predisposition: acquired and congenital) and thus for the varying length of latency or exposure time observed as well as for the frequent multiplicity of aniline tumors, and their site and extent in the bladder. The existence of a polyallergy to chemically related compounds makes it easier to understand that any one of a group of aromatic amines may start or continue a carcinogenic reaction in the vesical mucosa. Such chemospecific immunologic interrelations may indicate the manner by which normal or pathologic metabolites of similar chemical structure and of known growth stimulating quality (skatol, indol) may possibly continue an allergic-carcinogenic reaction initiated in the bladder by the exogenous chemical agents, and, thereby, may cause the development of vesical tumors many years after the cessation of occupational exposure to the aromatic amines.

Ullmann contended that aniline, as well as other hydrocarbons, exert an inhibiting effect upon the cellular ferments (catalase) which are supposed to prevent the unrestrained growth of cells (Bloch, Bristol). Present knowledge, in regard to the relation of intracellular enzymes to cellular growth activity, is by far too uncertain and controversial to consider this explanation as plausible. For instance, Wiley could not demonstrate any effect upon the respiratory index of rat tissue (liver, kidney) exposed to a solution containing beta-naphthylamine or removed from animals treated with this compound (Barcroft-Warburg method). Prolonged subcutaneous administration of beta-naphthylamine to dogs also had no significant effect upon the glucose, glutathione, calcium or phosphorus content and the pH of the blood (Wiley).

These investigations have not provided definite information as to the exact nature of the carcinogenic agent nor the mechanism of its action on the cells of the bladder mucosa. It remains doubtful whether these substances are directly or indirectly carcinogenic, i.e., whether they elicit a neoplastic response in a predisposed tissue or produce a conditioning of the mucosa so that other substances may induce the tumorous response (Latzko). In reference to these considerations, Scheele (1926) pointed out that, in general, substances which produce a cancerous predisposition are also carcinogenic (tar).

There may be mentioned in this connection a proposal made by Stallybrass, who suggested an investigation of the combined action of amino bases and arsenic in the production of bladder tumors on the assumption that the effects of the two substances may be synergistic. Such an experiment appears to be appropriate in view that prolonged ingestion of small amounts of arsenic