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## Relations between gastric, serum gastrin, parietal cell antibodies and the HLA-system.

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## CHAPTER 10

### SUMMARY

In Chapter 1 the problems to be studied are discussed;

A. In a normal population

- the prevalence of achlorhydria and the reliability of the serum gastrin concentration as its marker.
- the validity of the concept of a type A and B gastritis in achlorhydria.
- the relation between the basal gastric pH as a parameter for the gastric secretory capacity and the basal serum gastrin concentration.
- the relation between parietal cell antibodies and two parameters of gastric function: the basal gastric pH and serum gastrin.

B. In a family study of achlorhydria:

- the prevalence of achlorhydria with and without parietal cell antibodies in the relatives.
- the association of antigens of the histocompatibility system with achlorhydria and pernicious anemia.

In Chapter 2 the organization of the study, the patients and the methods are described.

Chapter 3 deals with the prevalence of achlorhydria in a normal population and the reliability of the serum gastrin concentration as its marker.

In a middle aged group of normal subjects the prevalence of achlorhydria and the value of the determination of the basal serum gastrin as an indirect method to demonstrate it were studied. Achlorhydria was found in 12 out of 366 males (3%) and 2 out of 198 females (1%); 13 of these 14 (93%) had an elevated serum gastrin. In 11 persons a moderately elevated serum gastrin -less than two times normal- was found in absence of achlorhydria. In seven of them hypochlorhydria was demonstrated. Of 540 persons with a normal serum gastrin one had achlorhydria. Serum gastrin appeared to be fairly reliable as a marker for achlorhydria.

In Chapter 4 the relation between the basal serum gastrin concentration and the basal gastric pH are discussed.

In a normal middle aged population the basal serum gastrin concentration and basal gastric pH were determined. Persons with achlorhydria were excluded, and in 550 non-achlorhydric persons the relation between basal gastric pH and serum gastrin was studied. Persons with a basal gastric pH higher than 6 had significantly higher serum gastrin levels. The explication of this phenomenon is thought to be the impaired inhibition by acid of the gastrin secretion by the G-cell, due to hypochlorhydria. The mechanism is the same as seen in hypergastrinaemia in achlorhydria.

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Chapter 5 discusses the merits of the separation of chronic atrophic gastritis with achlorhydria in the types A and B.

The applicability of the concept that chronic atrophic gastritis can be subdivided in type A and B was tested in hospital patients and normal subjects with proven pentagastrin-refractory achlorhydria. Classification was based on the determination of the basal serum gastrin and parietal cell antibodies. Of 59 hospital patients with achlorhydria 71% could be classified as belonging to either type A or B, for 29% the criteria for neither type were fulfilled. Of 14 asymptomatic achlorhydric found among 564 normal persons, five could be classified as having a type A gastritis, and one as a type B gastritis. In 8 (53%) persons an elevated serum gastrin was found in absence of parietal cell antibodies, representing an intermediate type of atrophic gastritis. Because one third of the hospital patients and more than half of the persons with achlorhydria in a normal population had to be classified as belonging to an intermediate type, the discrimination between type A and B atrophic gastritis in achlorhydria seems to be of only theoretical value.

Chapter 6 deals with the relation between parietal cell antibodies and basal serum gastrin. Parietal cell antibodies, basal gastric pH and serum gastrin were determined in 544 normal subjects. They were grouped regarding parietal cell antibody fluorescence score (negative, weakly positive, positive). Serum gastrin appeared to be significantly higher in subjects with these antibodies, as well in the group with a weakly positive as with a positive fluorescence score. A statistically significant difference between the groups as to serum gastrin

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was not present.

This might be explained by the recent finding, that parietal cell antibodies seem to block the gastrin receptors on the cellular membrane. This would influence the feedback mechanism between the secretion of acid and gastrin, and cause a higher basal serum gastrin at a constant basal gastric pH.

In Chapter 7 a family study in relatives of achlorhydric subjects is discussed. In 207 first degree relatives (parents, siblings, children) of 40 persons with achlorhydria serum gastrin and parietal cell auto-antibodies (PCA) were used to detect persons with achlorhydria. In 53 relatives an elevated serum gastrin or parietal cell antibodies or both were present. These 53 were considered to be suspect for achlorhydria. Data about acid secretion could be obtained in 33, of whom 21 had achlorhydria. Of the remaining 20 relatives who did not consent to gastric acid sampling, about 5 may have had achlorhydria. A grossly impaired vitamin B12 absorption was found in 2 subjects. Both subjects still had a normal haemoglobin. No difference in the prevalence of achlorhydria was found between relatives of probands with parietal cell antibodies (15/145;10.3%) and of probands without these (6/62;9.7%). In the same family achlorhydria was found with and without PCA. An elevated serum gastrin was more reliable as a marker for achlorhydria than a positive PCA fluorescence-reaction.

Chapter 8 is about the serologically defined HLA-antigens in relation to pernicious anaemia. In 86 first degree relatives of 10 pernicious anaemia patients the HLA-A, B and C antigens were typed. In all fami-

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lies more than one pernicious anaemia patient was present. The prevalence of each antigen among patients and relatives was compared with a normal control population. No differences in the prevalence of any of the antigens between the families and the normal controls was found. This was in agreement with reports of similar studies, in which a relation between HLA-A,B or C and pernicious anaemia was not evident.

In Chapter 9 the lymphocyte defined antigens of the HLA system are compared in pernicious anaemia patients and normal controls. For this study the pernicious anaemia patients discussed in Chapter 7 were combined with patients from a study of the Netherlands Red Cross Blood transfusion Service, Amsterdam. An association of pernicious anaemia with HLA-DR2 was found. A relative risk of 5.93 was calculated. Since these data were obtained in a rather small group of patients, this result has to be interpreted with caution.