Editorial

GASTRIN

The hormone gastrin was first isolated from the antral mucosa in 1905 by Edkins (Edkins 1905, 1906). Its chemical structure was determined as 17-Linearly arranged L-amino acids (Gregory et al., 1964). Gastrin is synthesised by the G-cells found in the pyloric glands of the antral mucosa and the proximal part of the small intestine (Creutzfeldt et al., 1975; Nilsson et al., 1973; Walsh et al., 1975). The release is brought about by mechanical, neural or chemical stimuli acting on the G-cells. Calcium, Magnesium, Epinephrine and L-arginine hydrochloride are some of the chemicals known to cause gastrin release. The chemicals are either transported through the blood, released at nerve endings or contained in substances coming in contact with the G-cells. Proteins, Peptones and Amino-acids are also stimuli causing the production of gastrin (Ganguli 1970). The mechanism leading to an increase in gastrin content by Atropine, in response to feeding and insulin is not known (Farooq and Walsh, 1975). Bombesin, a new product, isolated from frog-skin, has been shown to stimulate gastrin production when injected intravenously.

Suppression of gastrin is brought about by Secretin, Glucagon, Gastric inhibitory polypeptide and acidification of the antral mucosa. Somatostatin, a hypothalamic hormone is found to inhibit gastrin release (Bloom et al., 1974). A definite control of gastrin synthesis by the pituitary gland has been studied in hypophysectomized subjects where significant atrophy of the gastric and intestinal mucosa was observed (Crean 1968). Administration of growth hormone resulted in high levels of gastrin in serum and a reversal of gastro-intestinal atrophy. Growth hormone thus produces its gastro-intestinal effects through gastrin.

Bioassay estimation of fasting serum gastrin levels gave values ranging from 20 to 400 picograms per milli-liter (Berson and Yallow, 1972; Pointner, 1975). The clinical application of this measurement has been found very useful in patients with the Zollinger Ellison syndrome, where hypergastrinaemia is an essential feature (Isenberg et al., 1973; Thompson et al., 1972). A rise in the gastrin level after Secretin administration in such patients, is an important discriminating factor (Isenberg et al., 1973; Thompson et al., 1975).

From the multiple physiological actions of gastrin, the main one is stimulation of acid secretion in the stomach. To enhance Pepsin production and increase gastric mucosal blood flow are also additional functions of gastrin (Walsh and Grossman, 1975). Gastrin is mainly catabolised in the kidney (Booth et al., 1973). Experiments show that it is taken up directly from the peritubular capillary blood and degraded by the renal parenchyma (Davidson et al., 1974; Hall et al., 1973).

An increase in the serum gastrin level causing hypersecretion of acid is encountered only in the Zollinger Ellison syndrome (Gregory et al., 1967). The exclusive satisfactory treatment is thus total gastrectomy for this condition. Patients with duodenal ulcer show normal basal gastrin values, but the response to a standard meal releases more gastrin and more rapidly than normal controls (Becker et al., 1973; Chayvialle et al., 1975). This could be attributed to an abnormal gastrin inhibitory mechanism (Grossman et al., 1976). Other workers have also found no difference in gastrin concentration in the two situations (Brady and McGuigan, 1975). The exact role of gastrin in the diathesis of doudenal ulcer has thus not yet been determined.

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