# GASTRIC SECRETORY ACTIVITY IN ISCHAEMIC HEART DISEASE: A POSSIBLE PATHOGENETIC MECHANISM*

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## 1. FAT TOLERANCE IN ISCHAEMIC HEART DISEASE

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The feeding of fat to patients with ischaemic heart disease results in a more extensive and more prolonged lipaemia than in age-matched controls. The differences are greatest in the later phases, i.e. 6 and 8 hours after the meal. For this reason it has been held that the more prolonged lipaemia is due to an insufficiency of clearing factor in patients with ischaemic heart disease. *In vitro* clearing experiments, however, have not confirmed this.[1] It was emphasized that the degree of lipaemia at any one time was the algebraic sum of processes of absorption of fat into, and processes of clearing of fat from, the circulation. Attention was therefore focussed on the possibility that absorptive differences underlay these different postprandial lipaemic responses. In a study on matched pairs,[2] the lipaemia was more prolonged in the group with ischaemic heart disease when fat was fed orally, but when it was administered intravenously, identical lipaemic curves were seen in both patient and control groups.

This study, together with other recent studies, has focussed our attention on the possibiiity that the nature of the disordered fat metabolism in ischaemic heart disease may be determined in the gastro-intestinal tract. It was therefore decided to compare the gastric secretory activity in patients with ischaemic heart disease with that of controls.

## 2. GASTRIC LIPOLYTIC ACTIVITY

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Cantone, Rulli and Rossi[3] fed gastric mucosal extract to man and noted a decrease in plasma lactescence. Whether or not the human stomach has lipolytic activity is a matter of controversy.[4,5] Experiments were designed to test this. It was found that, on incubating human gastric juice *in vitro* with a safflower-oil emulsion, considerable clearing occurred as measured by the percentage change in optical density over one hour at 37°C. This clearing activity was not as marked as with pancreatic juice, but pancreatic lipase was inactive in a pH range below 6. All studies, therefore, were conducted at a pH below 6 to exclude the possibility of activity owing to regurgitation of pancreatic juice.

The other possibility was that the clearing observed was solely the result of acid hydrolysis of fats. Tests were therefore carried out on gastric juice before and after heating to 100°C. The boiled gastric juice was still able to clear the emulsion, but the degree of clearing was less than the unheated portion. This could imply that optimum gastric lipolysis is dependent upon both acid and a lipase acting concurrently. Further evidence in favour of the presence of a lipase was that the basal acid secretion, measured in mEq. per l., correlated with the lipolytic activity, but did not correlate following the injection of histamine. While the acid secretion invariably increased after histamine, an increase in lipolytic activity did not necessarily follow.

## 3. GASTRIC SECRETORY ACTIVITY IN PATIENTS WITH AND WITHOUT ISCHAEMIC HEART DISEASE

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Results of the augmented histamine test,[6] carried out in 32 patients with unequivocal evidence of ischaemic heart disease, were compared with those obtained in 20 controls of similar age and body weight and without symptoms referable to the gastro-intestinal tract. The basal secretion and the response to maximal histamine stimulation (MAO) of the controls compared well with figures for healthy people elsewhere,[7] but the patients with ischaemic heart disease had a significantly ($p<\cdot01$) lower mean acid output with regard to both basal secretion and MAO. Basal gastric lipolytic activity, as measured above, followed the same trend. It was concluded that gastric secretory activity was less in patients with ischaemic heart disease, certainly in the basal state.

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