Letters to the Editor

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NOTES ON POINTS IN SOME OF THIS WEEK'S LETTERS APPEAR ON P. 1011.

Modes of Stimulation of the Gastric Secretion

THE manner in which gastric secretion is stimulated through the parasympathetic nervous system and by histamine may be better understood by means of combined physiological and histological study.

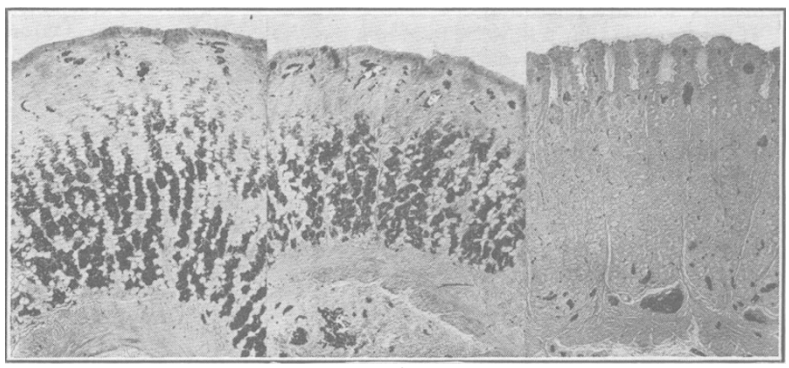
Rhythmic stimulation of the vagi with a weak induction current produces a scanty flow of slightly alkaline, neutral or slightly acid mucous fluid from the stomach. A stronger current provokes a copious secretion of regular gastric juice of high acidity and peptic power and rich in dissolved mucin[1]. If there are in the vagus two kinds of secretory fibres innervating the gastric mucosa, then those which are activated by a weak induction current will have relation chiefly to the surface epithelium cells and perhaps to the mucoid cells. In strong stimulation of the vagi, mucoid, peptic and parietal cells are brought into activity. Under normal conditions, when gastric secretion is produced, for example, reflexly as in 'sham-feeding', the composition of the juice corresponds to that obtained by strong electrical stimulation of the vagi. However, at the end of the secretory period there is an abundant flow of visible mucus, which is much greater than in gastric secretion stimulated by histamine or alcohol[2, 3]. This suggests the participation of surface epithelium mucous cells in certain phases of reflexly provoked gastric secretion.

The exceptionally high peptic power of 'vagus' gastric juice may be attributed to an enormous discharge of granules from the peptic cells, the granules being presumably vehicles of the enzymes[4]. (Compare Fig. 1(*a*)—control—with Fig. 1(*c*)—effect of vagus stimulation—and note the disappearance of the dark-coloured granules from the peptic cells.)

Quite different relations were noted when histamine was administered to an animal. The volume of secretion produced by this drug is no less and sometimes even greater than in experiments involving electrical vagus stimulation or sham-feeding. The acidity of the 'histamine' gastric juice and its total chlorine concentration correspond to those of the 'vagus' juice. This shows that the source of almost all the chlorine is in both cases one and the same[5]. On the other hand, the total organic matter, including the pepsin and dissolved mucin, gradually diminish during secretion. They may practically disappear from the juice if histamine is injected repeatedly[6, 7, 8]. Therefore it seems legitimate to conclude that histamine stimulates the parietal cells only, producing a flow of acid solution, without having any effect on the peptic cells (Fig. 1(*b*))[4]. At the beginning of the secretion on histamine, the acid solution produced by the parietal cells washes out from the glandular tubules the zymogen material which may have accumulated there, presumably during the inactivity of the gland.

Histamine action on the gastric glands is not, however, restricted to selective stimulation of the parietal cells. Experiments in which gastric secretion activated by histamine was followed by sham-feeding or a test-meal showed that histamine definitely diminishes the secretory effect of the two latter agents[9]. Histamine exercises its greatest inhibition on the nervous, that is, 'vagal', phase of gastric secretion.

These and other experiments support the theory that many of the digestive glands are composed of different sets of secretory epithelia. The secretory activity of such glands is not regulated *en masse*, but various nerves (for example, those innervating the submaxillary gland[10]) or chemical agents stimulate or inhibit each set of secretory elements separately. This does not exclude the mutual influence of one group of cells on another through the action of 'chemical messengers'[11, 12]. Therefore it may be concluded that the *qualitative* changes which occur in many digestive secretions under various conditions of stimulation are due to the unequal *quantitative* activity of different groups of secretory cells in a given gland[13, 14, 15].



(*a*) (*b*) (*c*)

FIG. 1. Sections of gastric mucosa. (*a*) Control; (*b*) effect of histamine stimulation; (*c*) effect of vagus stimulation.

The histological part of the investigations quoted in this letter was performed in the Department of Histology, McGill University, under the direction of Prof. J. C. Simpson.

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