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Effects of isoprenaline on gastric acid secretion and mucosal blood flow during stimulation by pentagastrin or feeding

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Histamine-induced gastric acid secretion in dogs may be either increased or decreased by infusions of isoprenaline. In conscious dogs small doses (about (0·2 μg/kg)/min) increased and large doses (about (2 μg/kg)/min) decreased secretion; whereas in acute experiments in anaesthetized dogs the only effect observed was an increase of histamine-induced secretion (Curwain, Endersby & Holton, 1971). Since acid secretion is more readily depressed when it is induced by means other than histamine (Harries, 1957) we have investigated the effect of isoprenaline on secretion stimulated by infusions of pentagastrin and by feeding (that is by endogenous gastrin).

In eleven experiments on four conscious dogs with Heidenhain pouches, infusion of isoprenaline (0·05–2 μg/kg)/min decreased acid secretion induced by pentagastrin (5 μg/kg)/hour. In three experiments in acute anaesthetized dogs isoprenaline (0·2 μg/kg)/min also decreased pentagastrin-stimulated secretion. Isoprenaline (0·06–0·25 μg/kg)/min also decreased the (gastrin induced) secretion in response to feeding in seven experiments in the four Heidenhain pouch dogs. On no occasion was an increase of acid secretion observed when secretion was induced by these means.

The effects of isoprenaline on mucosal blood flow was studied in five experiments on two of the Heidenhain pouch dogs using the aniline clearance method (Curwain & Holton, 1971). Mucosal blood flow decreased when acid secretion (induced by pentagastrin) fell in response to isoprenaline but the ratio of blood flow to secretion increased. When noradrenaline (0·1–0·5 μg/kg)/min was used to decrease mucosal blood flow and acid secretion induced by pentagastrin or feeding, the ratio of blood flow to secretion remained unchanged (five experiments in two dogs).

We can conclude from these experiments that the inhibitory effect of isoprenaline on pentagastrin (or gastrin) induced secretion is not secondary to a fall in mucosal blood flow and is probably due to a direct antagonism between isoprenaline and the secretory mechanism initiated by gastrin. We cannot exclude the possibility that the inhibitory action of noradrenaline is secondary to mucosal vasoconstriction.

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