lular tight contacts have been reported for rabbit sinus node[12-15].

The most important investigations for the present study are the measurements of the space constant; however, they differ considerably. The space constant measured by Bonke[3] in planar preparations of the sinus node was 465 μm, whereas Seyama[6] found in the quiescent nodal strips 828 μm. In the former experiments the polarizing current was supplied from an extracellular suction electrode, in the latter it flowed ahead through the sucrose gap. These discrepancies may be due to the very complex geometry of the sinus node. However, the comparison of the cell length and space constant (even Bonke's value) in the sinus node 20 μm[1,2] and 465 μm[3], with the respective values for ventricular muscle, 125 μm and 880 μm[16], suggest the low resistance intercellular coupling between the sinus node cells.

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Luminal gastrin does not activate rat stomach histidine decarboxylase[1]

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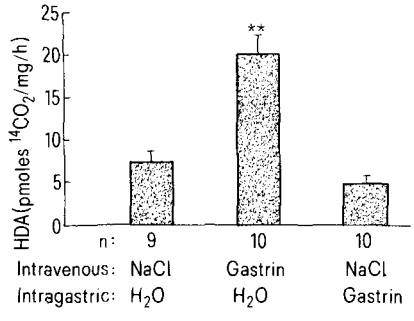
Summary. Fasted rats have a low gastric histidine decarboxylase activity. I.v. infusion of heptadecapeptide gastrin for 2 h raised the enzyme activity. Intragastric perfusion with the same dose of gastrin and for the same period of time did not reproduce the effect of circulating gastrin. It is concluded that luminal gastrin, in contrast to circulating gastrin, does not activate rat stomach histidine decarboxylase.

Immunoreactive gastrin is said to be released into the gastric lumen in several species[2-7]. There have been speculations that luminal gastrin may be of physiological importance and that it may exert some of the functions that have been attributed to circulating gastrin, i.e. stimulation of the parietal cells[5,8] and trophic effects on the digestive tract[9]. Another function of circulating gastrin is to activate the histamine-storing endocrine cells in rat gastric mucosa[10]. This activation is reflected in an increased activity of the histamine-forming enzyme, histidine decarboxylase[10]. The activity of this enzyme is known to reflect the serum gastrin concentration[11], and exogenous gastrin is a potent stimulus for enzyme activation in fasted animals. The present study was designed to clarify whether luminal gastrin can mimic the effects of circulating gastrin on the gastric histidine decarboxylase activity.

Adult male Sprague-Dawley rats were used. They were fed a standard diet of food pellets and tap water. Prior to experiments the rats were deprived of food but not water for 48 h in individual cages with wire mesh bottoms. Synthetic human gastrin I 1-17 (15-leucine) was purchased from Fluka AG, Basle, Switzerland, and stored at -20 °C. Fresh solutions were prepared daily by dissolving the gastrin in sterile saline (0.9%) or deionized water (as specified). The biological activity of gastrin-17 (15-leucine) has been ascertained. It is said to have more than 80% of the biological activity of the natural 15-methionine variety[12].

Under chloralose anesthesia (1.2 ml 5% solution i.p.) the abdomen was opened through a midline incision. A flanged polyethylene catheter (Portex Ltd, Hythe, England), 2 mm in diameter, was inserted through the rumen and secured with a purse string suture. An i.v. cannula (No. 10, Portex Ltd) was inserted into the external jugular vein. Infusions were made using constant rate Harvard infusion pumps. The stomachs were perfused either with deionized water or with gastrin-17 dissolved in deionized water. The dose given was 10 μg/kg · h. The pH of the gastric perfusate was 6.2. The intragastric infusion rate was 3.6 ml/h, a rate that produced no visible distension of the stomach. The rats received also i.v. infusions of either sterile saline or gastrin in saline, 10 μg/kg · h. The rate of i.v. infusion was 2 ml/h. After 2 h the animals were killed by exsanguination, the



Histidine decarboxylase activity (HDA) in rats with gastric perfusion and i.v. infusion. Mean ± SEM. ** denotes $p < 0.005$ compared with controls (rats not given gastrin). The number of animals in each group is indicated.

stomachs were removed, opened along the greater curvature and rinsed in ice-cold saline. The mucosa of the oxyntic gland area was scraped off and homogenized in ice cold 0.1 M phosphate buffer, pH 6.9–7.0, to a final concentration of 100 mg (wet wt)/ml.

Aliquots (0.5 ml) of the homogenate were incubated with 4×10^{-4} M 1-^{14}C-L-histidine (1.3 mCi/mmole; New England Nuclear) in the presence of 10^{-5} M pyridoxal-5-phosphate and 5×10^{-4} reduced glutathione at 37 ° C for 1 h under nitrogen. Total reaction volume was 0.53 ml. The enzyme reaction was stopped by acidification and the amount of $^{14}CO_2$ produced was determined as described in detail elsewhere [11,13]. Enzyme activities are expressed as pmoles $^{14}CO_2$ produced per mg tissue (wet wt)/h. Comparisons between groups were made using Student's t-test for unpaired observations. Differences with a p-value of less than 0.05 were considered significant.

3 groups of rats were examined. Control rats received an i.v. infusion of saline and simultaneously an intragastric perfusion with deionized water. In a 2nd group the rats received an i.v. infusion of gastrin and an intragastric perfusion with water. In this group gastric histidine decarboxylase activity was markedly increased compared with the control rats. A 3rd group of rats received the same dose of gastrin in the gastric perfusate whereas the i.v. infusion was saline. In this group the enzyme activity did not differ from that seen in the control group. The data are summarized in the figure.

Data are accumulating that upon stimulation of the gastrin cells gastrin is released into the lumen of the stomach as well as into the circulation[2-7]. Whether this phenomenon is physiologically significant or not remains to be established. There have been suggestions that luminal gastrin may exert a biological action from within the lumen of the gut[5,9]. In the rat, activation of histidine decarboxylase in the oxyntic mucosa is one of the functions of gastrin[11]. As expected, i.v. infusion of a large dose of gastrin raised the histidine decarboxylase activity. Intragastric administration of gastrin, however, failed to reproduce this effect. This could mean either that the endocrine cells containing the enzyme do not respond to gastrin from the lumen or that they are inaccessible to gastrin from the lumen under the conditions of the experiment. However, it should be realized that the failure of luminal gastrin to reproduce the effect of circulating gastrin on histidine decarboxylase does not necessarily mean that other target cells are inaccessible or non-responsive to luminal gastrin.

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Effect of sudden reductions of the arterial blood pressure on the mean diastolic coronary resistance

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Summary. At the beginning of a 10 sec arterial haemorrhage, vascular elasticity induces an increase of mean diastolic coronary resistance. Then, the increase is counteracted by the relaxation of the vascular musculature, which causes a coronary hyperaemia when, after the haemorrhage is arrested, the vascular wall is stretched by a sudden though slight increase of blood pressure.

The value of the mean diastolic coronary resistance (MDCR) is a reliable index of the coronary vascular resistance (CVR). It can be calculated from the ratio of the mean diastolic aortic blood pressure to the diastolic coronary flow.

In addition to metabolic, humoral and nervous factors, both vascular elastic distensibility and a myogenic mechanism take part in the regulation of CVR[2-8]. Since the elastic and the myogenic mechanisms are affected by the transmural coronary pressure, this research was planned to investigate how they interact upon the regulation of MDCR during and after abrupt reductions of the aortic blood pressure (ABP) in the absence of corresponding changes of the baroreceptor activity. In a previous study it was seen that abrupt but transient falls of ABP can be effectively produced by inducing and then stopping a brief multiple arterial haemorrhage[9].

Materials and methods. In 8 open-chest dogs under general barbiturate anaesthesia and artificial ventilation, the probes of 2 electromagnetic flowmeters were placed around the ascending aorta and the left circumflex coronary artery in order to record stroke volume (SV) and coronary flow (CF) respectively. ABP was derived by means of a plastic catheter connected with an electromanometer and pushed into the aorta via the left common carotid artery.

To produce a multiple arterial haemorrhage, 3 silicon tubes filled with heparinized saline solution and clamped on 1 side, were inserted into the right common carotid and the 2 femoral arteries.