On the other hand, musk mallow and castor oil plant leaves extended the larval period greatly, to nearly twice that of larvae fed on lettuce (table 1).

With respect to larval mortality, it was observed that the short larval duration was associated with low larval mortality (table 1); when the mortality rate increased, the larval duration was extended.

As regards the pupal stage, it was observed that under a constant temperature of 27 °C and 60% relative humidity the duration of this stage, for pupae developing from larvae fed on different diets, varied considerably according to the host plant used. The shortest pupal duration was 11.83 ± 0.6 days in the case of pupae developing from larvae reared on lettuce, while the longest one was 23.57 ± 0.62 days in case of pupae resulting from larvae fed on castor oil plant leaves (table 1).

On the other hand, the pupal weight was significantly affected by the nutritional value of the larval diet. Pupae developing from larvae fed on kidney bean pods and cotton plant leaves gave rise to heavier pupae (378 ± 0.01 and 337 ± 0.07 mg/pupa, for both diets respectively). The present finding is in agreement with the data obtained by Dhandapani and Balasubramanian[4], who found the heaviest pupae of *H. armigera* on cotton. The minimum pupal weight was obtained from larvae reared on dwarf mallow leaves, it was 273 ± 0.08 mg/pupa.

Castor oil plant leaves which had a retardation effect on the developmental duration gave pupae of normal wight; 312 ± 0.01 mg/pupa (table 1).

2. Effect of larval diet on the activity of the emerging adults. The data obtained in table 2 clarify that the duration of the ovipositional period, as well as the life span of the adults, was remarkably influenced by the different larval food. Maximum longevity was shown by larvae reared on kidney bean pods and minimum longevity by those reared on castor oil plant leaves.

The larval food also has a significant effect on the reproductive potentiality of the emerging moths (table 2). Females which emerged from larvae fed on kidney bean pods laid the highest number of eggs (698.34 ± 12.9 egg/female), followed by moths from larvae fed on cotton plant leaves. The lowest average number of eggs was recorded from moths which emerged from larvae reared on castor oil plant leaves.

Statistical analysis of the data revealed that kidney bean pods and cotton plant leaves significantly affected the reproductive potentiality of the emerging females. This may be due to the nutritional constituents, especially protein, in these plants[4,6]. The presence of high quantities of water and sugar in cotton plant leaves[7] may induce the larvae to consume more food. Thus heavy pupae were obtained and the reporductive potential of the females increased.

Therefore, the order of preference of the different hosts can be arranged as follows with regard to the different biological aspects: kidney bean pods = > cotton plant leaves > lettuce ≫ musk mallow ≫ castor oil plant leaves = dwarf mallow.

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Mechanism of intercellular synchronization in the rabbit sinus node[1]

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Summary. A strip of tissue cut from the sinus node, 0.5 mm wide, was studied in a sucrose gap chamber. With the gap width of 0.6–1.5 mm the 2 ends of the preparation showed nonsynchronous activity. Synchronization could be re-established by a shunt resistor, 15–100 kΩ, connected a cross the gap, suggesting that synchronous firing of the sinus node requires local circuit currents.

Despite the fact that sinus node cells are coupled electrically[3-6], the mechanism of the synchronization remains unclear[7,8]. Thus we attempted to find out whether or not local circuit currents could bring about synchronization between 2 parts of a sinus node strip separated by a sucrose gap.

Methods. Spontaneously active strips, 4–6 mm long and 0.5 mm wide, were cut from the rabbit sinus nodes parallel to the crista terminalis. The distance between crista terminalis and cut line was 2–2.5 mm. Such preparations comprised the pacemaker cells almost exclusively[6], and were verified electrophysiologically (fig. 1). Each strip was mounted in a 3-compartment chamber, tyrode – sucrose – tyrode. The gap width could be changed between 0 and 2 mm. Perspex plates of 0.09 mm in thickness separated the compartments. The preparation was pulled through 0.5-mm holes drilled in these plates, and the holes made watertight with silicone grease. Both ends of the strip protruded freely into the outer compartments perfused with Tyrode solution (NaCl 147 mM, KCl 2.7 mM, $CaCl_2$ 1.8

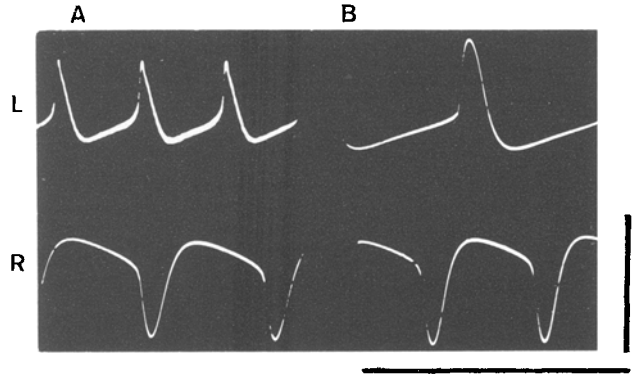

Fig. 1. Control transgap action potentials recorded from 2 different strips (A and B). L, activity of the left-hand side of the strip, the right-hand side being depolarized by KCl. R, activity of the right-hand side of the same strip, the left-hand side being now depolarized by KCl. Time: 1 sec, voltage: 50 mV.

mM, $MgCl_2$ 1 mM, $NaHCO_3$ 11 mM, glucose 5.5 mM). All the solutions were oxygenated with 95% O_2 + 5% CO_2 and kept at 30 °C. Action potentials were recorded by means of extracellular Ag/AgCl electrodes placed in the outer compartments. The sucrose gap could be shunted by a resistor connected to the electrodes as in the experiments of Barr et al.[9].

Results. Out of 118 preparations only 33 were suitable for the present experiments. These strips produced pacemaker-shaped action potentials at both ends (fig. 1). The electrical activity of one end of an individual preparation was visualized when the other end was depolarized with an isotonic KCl solution. Such strips were active at a single rate, 0.8–3.6 Hz, when all compartments, including the gap, were perfused with Tyrode solution. Other strips showed conduction blocks and were probably damaged during the experimental procedure.

Synchronous activity was recorded when the width of the sucrose gap was 0.5 mm (5 experiments). Sucrose gaps of 0.6–0.9 mm (24 experiments) resulted in asynchronous transgap action potentials with peak amplitude 15–38 mV. However, in the majority of experiments only incomplete blocks were observed (e.g. 2:1 block in fig. 3). Complex discharge patterns were caused by the interference of the 2 intrinsic rates of the 2 end portions of the strip (figs 2 and 3). Asynchronous activities could be synchronized by a shunt resistor connected across the gap. The maximal values of shunt resistances for successful synchronization in various experiments were 15–100 kΩ (figs 2 and 3).

In six experiments with 0.8 and 0.9 mm gaps even 2 kΩ shunts did not synchronize the strips. On the other hand, it was possible to get synchronization by shunting in 2 cases with a 1-mm and in 1 case with a 1.5-mm gap (figs 2 and 3).

In 4 additional experiments with 0.7- and 0.8-mm gaps intracellular action potentials were recorded by means of 2 glass microelectrodes. In these strips, synchronization could also be re-established by the shunt resistors, 20–60 kΩ, connected across the gap (fig. 4).

The application of hyperosmotic sucrose (600 mM) instead of isotonic (300 mM) invariably made the synchronization impossible within 10–20 min. The peak transgap action potentials dropped to 10–15 mV. The transgap resistance increased 1.38- to 2.1-fold in respect to isotonicity, e.g. the mean transgap resistance of 0.8-mm gaps during iso- and hyperosmotic perfusion were 146 ± 19 kΩ and 233 ± 24 kΩ respectively (6 experiments). These effects were reversible within 3–10 min upon re-introduction of isotonic sucrose solution.

Discussion. The results obtained suggest that local circuit currents are responsible for synchronization. A direct proof of this statement is the synchronization of 2 different rates evoked by the shunt resistor connected a cross the gap. An additional support is provided by the experiments with hyperosmotic sucrose solution which increased the transgap resistance and thereby made synchronization impossible.

Thus, one may assume that in the sinus node, like other cardiac tissues[9-11] there are low resistance intercellular junctions which allow a flow of current sufficient for synchronization. It should also be noted that such intercel-

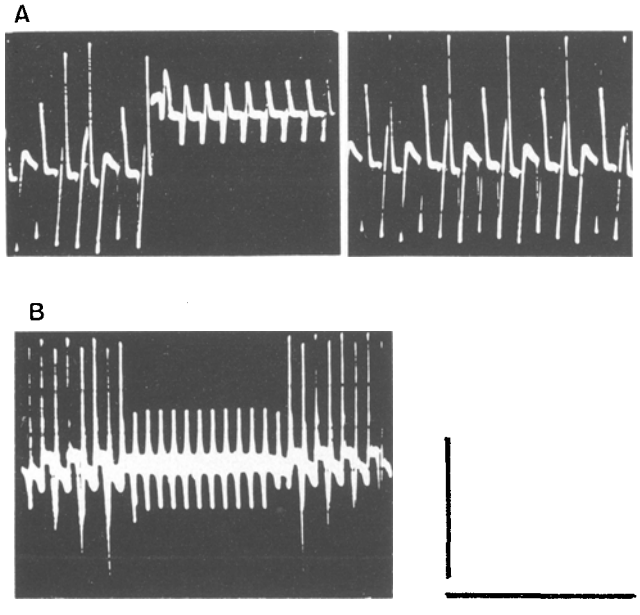

Fig. 2. Transgap action potentials before, during and after synchronization by shunt resistors. Upstrokes mark the activation of the left-hand side of the strip, downstrokes mark the activation of the right-hand one. A, gap 0.6 mm, shunt 45 kΩ. B, another strip, gap 1.5 mm, shunt 51 kΩ. Time: A, 5 sec, B, 10 sec; voltage A, 20 mV, B, 50 mV.

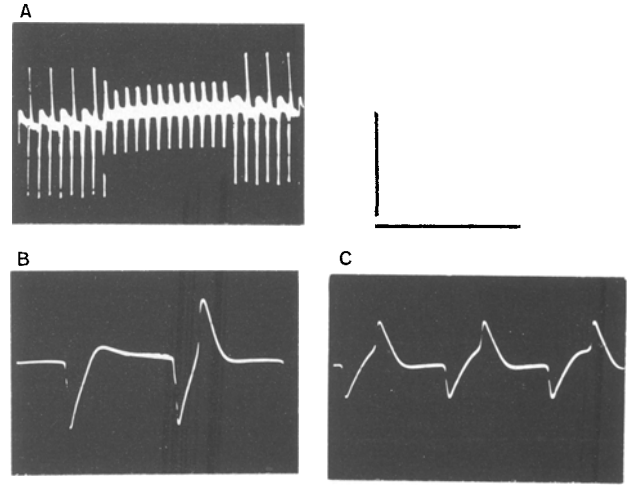

Fig. 3. Transgap action potentials before, during and after synchronization by shunt resistors. A, gap 0.8 mm, shunt 80 kΩ; B and C, the same strip, gap enlarged to 1.0 mm. B, asynchrony, C, synchronization by 68 kΩ shunt. Time: A, 10 sec, B and C, 1 sec; voltage: A, B and C, 50 mV.

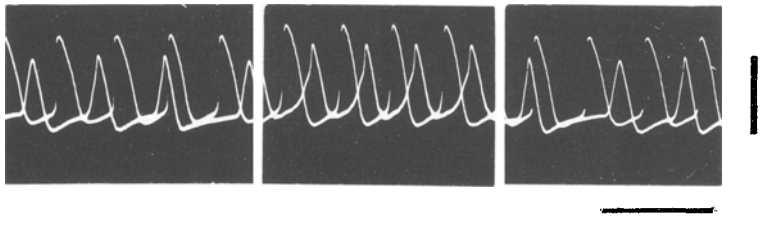

Fig. 4. Intracellular action potentials before, during and after synchronization by 32 kΩ shunt, gap 0.7 mm. Major potentials recorded from the left-hand side of the strip, minor potentials recorded from the right-hand side of the same strip. Note a disappearance of arrhythmia during synchronization. Time: 1 sec, voltage: 50 mV.

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.

1 This study was supported by Polish Academy of Sciences, grant 640/VI.
2 The authors are indebted to Proff. S. Weidmann and W. Trautwein for reading the manuscript and valuable comments.

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

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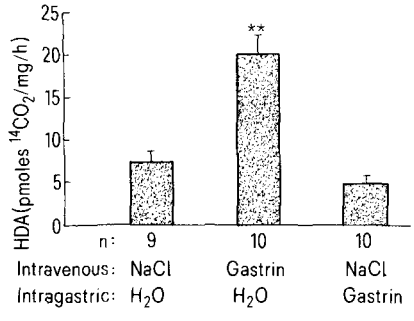
Summary. Fasted rats have a low gastric histidine decarboxylase activity. I.v. infusion of heptadecapeptide gastrin for 2 h raised the enzyme activity. Intragastric perfusion with the same dose of gastrin and for the same period of time did not reproduce the effect of circulating gastrin. It is concluded 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.