disaccharidase which does not respond to thyroxine during suckling period. These observations suggest that the hormonal control of the postnatal development of the different disaccharidases is not identical for all of them, trehalase being affected only by cortisone.

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Changes in efferent activities of the gastric vagus nerve by administration of glucose in the portal vein

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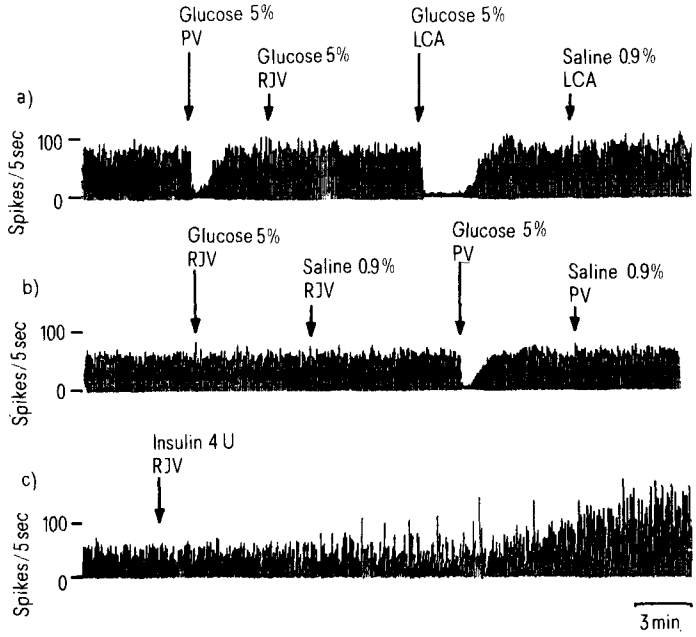
Summary. Injection of glucose in the portal vein, as well as in the left carotid artery, brought a transient decrease in efferent discharges of the gastric vagus nerve, whereas venous injection of regular insulin brought a gradual increase in the discharges.

It has been generally accepted that changes in the glucose concentration of the blood influence gastric secretion of acid which is attributed to the neural glucose sensitive mechanism in the central nervous areas[2-5]. However, behavioural and electrophysiological studies provide good evidence for the existence of neural glucose sensitive mechanism in the hepatoportal areas[6-8]. From these data, we speculated that there might be a close relationship between the neural glucose sensitive mechanism and secretion of gastric acid. Recently, we observed glucose injection in the portal vein that effected efferent activities of the gastric vagus nerve.

Material and methods. Experiments were made on 31 male rats weighing about 300 g under anesthesia with Pentobarbitone 45 mg/kg, i.p. Rectal temperature was kept at about 38 °C. The nerve innervating the stomach was dissected into a fine filament and covered with a mixture of vaseline and liquid paraffin. Discharges were taken from the cranial cut end of the nerve by means of silver electrode, amplified by an R-C coupled differential amplifier. The discharge was integrated after conversion of spikes to standard pulses through a window discriminator and was displayed as vertical deflections. 5% glucose and 0.9% saline solutions were injected through catheters placed in the portal vein, in the cardiac side of the right jugular vein and in the cranial side of the left carotid artery. Glucose was diluted with distilled water. The amount of the injection was fixed at 0.2 ml, so that no appreciable change in discharges was produced by control injection of the 0.9% saline.



a, b Effects of glucose injection into the portal vein, into the right jugular vein and into the left carotid artery upon the efferent activities of the gastric vagus nerve. PV: portal vein, RJV: right jugular vein, LCA: left carotid artery. *c* Effect of insulin administration i.v. upon the efferent discharges. Arrows indicate the time of injection.

Results and discussion. Injection of 5% glucose (0.2 ml) into the portal vein and into the left carotid artery caused a transient decrease in discharges, whereas the right jugular injection of glucose produced no appreciable change. The effects of glucose were more intense when injected into the cranial side of the carotid artery than into the portal vein. The 0.9% saline infusion produced no response in discharges. These results indicate that the portal glucose infusion effects on efferent activities of the gastric vagus nerve may be attributed to the neural glucose sensitive mechanism located in the portal vein, especially since the same doses of glucose infusion into the right jugular vein had no effect on the discharges (figure, a and b). However, after hepatic vagotomy, the discharge response usually associated with the portal injection of glucose was not reproducible. Regular insulin (4 U i.v.) caused a gradual increase in discharges (figure, c). These experiments were conducted repeatedly, and similar results were obtained. We know that hypoglycemia causes excessive secretion of gastric acid. It has been assumed that hypoglycemia acts by stimulating the neural glucose sensitive mechanism in the central nervous areas, because denervation of the vagus nerve abolishes such response to hypoglycemia[2-5]. However, there are many reports of the existence of a neural glucose sensitive mechanism in the hepato-portal areas. Russek suggested glucoreceptors in the portal areas as a result of his behavioural work[6]. Niijima observed afferent hepatic discharge that was inversely related to the glucose concentration in the portal vein[7]. Schmitt also showed the projection of glucose infusion in the portal vein on hypothalamic neuron activities[8].

In our experiments, glucose infusion into the portal vein, as well as into the cranial side of the left carotid artery, caused a transient decrease in the efferent activities of the gastric vagus verve, whereas i.v. insulin administration caused an increase. These results suggest that the neural glucose sensitive mechanism located in the hepato-portal areas plays a role in these responses, and that there might be a neural system which modulates gastric secretion of acid through hepatic vagal afferents and gastric vagal efferents.

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Effects of captivity on glucose tolerance in dogs[1,2]

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Summary. Captivity decreased tolerance to glucose and increased blood serotonin levels in 6 normal dogs investigated. Return to freedom brought normalization in the glucose tolerance test and reverted blood serotonin to control levels.

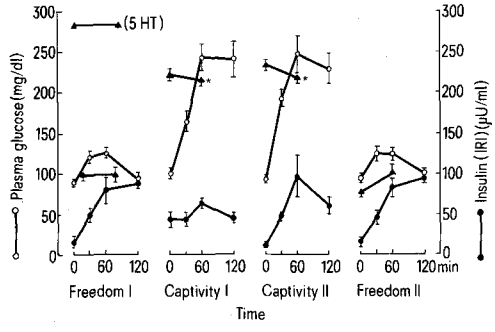
It was found that sulpiride (an atypical dopaminergic blocking agent) but not haloperidol (a classical dopaminergic blocking agent) decreases tolerance to glucose and raises blood serotonin levels both in humans and dogs[4]. Taking into account that these drugs have been successfully used in the treatment of more than 1500 headache and other psychosomatic patients[5,6], and because of some chance findings registered during prior studies, we decided to investigate the effects of captivity on glucose tolerance and on serotonin levels in the blood.

Material and methods. Our protocol included 6 adult mongrel dogs weighing 25–30 kg. Control tests (free state) and captivity state tests were carried out according to plan. When in captivity, animals lived in dog-colony, inside individual barreled boxes measuring $1.25 \times 0.75 \times 0.75$ m. When free (control), animals lived in the garden of our homes, under controlled conditions. During control and captivity periods all animals were fed on a commercial dog food (Perrarina). 4 consecutive (30-day intervals) oral glucose tolerance tests (1.7 g/kg) were performed on each dog. Peripheral blood samples were taken for glucose[7], insulin[8], and serotonin[9] determinations at 0, 30, 60 and 120 min. Whole blood serotonin levels were assessed at 0 and 60 min only. All experiments began at 08.00 h, after 16 h fasting.

Results. Despite the fact that fasting serum glucose levels were normal, captivity reduced the tolerance to glucose, while return to freedom normalized this tolerance (figure). In addition, it was showed clearly that captivity increases blood serotonin levels.

The dogs' behaviour varied according to the experimental conditions. During the first days (3–5) after captivity periods, they were apathetic and did not bark; no responses to environmental stimuli were observed. For instance, they did not react to aggression by other dogs or to children's caresses. However, after those first days, good humour and aggressive manifestations were progressively restored.

Discussion. Captivity decreased tolerance to glucose in the 6 dogs investigated in the present study. Reduction of



4 consecutive oral glucose tolerance test performed in 6 adult mongrel dogs at 30-day intervals. Means±SEM. Serotonin was assayed at 0 and 60 min, only. Changes in 5HT are expressed in percentage. *$p < 0.001$. Statistical significance against zero-mean value at freedom I test (100%).