effluent dialyzate fluid show that this is mainly due to a high protein loss into the peritoneal cavity, which obviously cannot be compensated by de novo synthesis. The reason for the high protein leakage in these small laboratory animals is not known. It is suggestive that the high mortality rate after 48 h of dialysis with Sterofundin® may be connected with the hypoproteinemia. Yet, 48 h after dialysis with Peritosteril HK® no animals had died, although serum protein was reduced to a similar degree. Therefore the properties of the dialyzate fluid seem to be essential for survival. The composition of Sterofundin® and Peritosteril HK® differ in many respects including concentration of cations and types of anions as well as pH-values and osmolarity. At present it is a matter of speculation which parameters may be most important in regard to the mortality rate.

It can be concluded from our experiments that dialyzate fluids of the type of Peritosteril HK® should be used for continuous dialysis in experimental studies with rats and guinea-pigs, and that protein must be replaced during long-term treatment to avoid hypoproteinemia and hemoconcentration.

Under these circumstances, the method described is a simple, quick and cheap technique for peritoneal dialysis in small laboratory animals. It was successfully used in the treatment of experimental acute pancreatitis in more than 300 rats[11].

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Changes in gastro-intestinal serotonin content associated with fasting and satiation

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Summary. Rats fasted for 24 h were fed for 3 h, after which time food was removed. Food intake decreased serotonin levels in the stomach and duodenum by 30 and 40%, respectively. These changes persisted for about 3 h. Food intake did not change tryptophan content in the stomach, while, in the duodenum, tryptophan level rose by 100% at the end of the feeding period and remained elevated for about 9 h.

High concentrations of serotonin (5-HT) are present in the gastro-intestinal tract of all vertebrates. This amine is mainly localized in the enterochromaffin cell system of the mucosa[1,2], although small concentrations have been found also in the intramural nervous system of the gastro-intestinal tract[3,4]. It has been suggested that 5-HT has a stimulating action on gastro-intestinal motility and, therefore, that this monoamine might function in the modulation of the peristaltic reflex[5–7]. Modifications in the level of this monoamine have been obtained with the use of diets free from, or lacking in, tryptophan[8,9], by modifying the normal intestinal flora[10,11], or by administering drugs acting on monoamine metabolism[12,13].

This report shows that gastro-intestinal 5-HT content undergoes changes associated with fasting and satiation.

Materials and methods. Experiments were carried out with male Wistar rats, initially weighing 150–180 g. The rats were housed 3 per cage in wire-bottom cages at a room temperature of 24 °C with reversed light-dark cycle: lights on from 22.00 to 10.00. They had access to water ad libitum but were trained to consume their normal food intake in a period of 3 h: food was presented ad libitum at 11.00 and removed at 14.00. Experiments were carried out after 3 weeks of training. At this time, each rat ate an average of 18.6 ± 0.5 g of the diet per day. Rats were fed

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Changes in gastro-intestinal serotonin and tryptophan levels associated with fasting and satiation

Duration of fasting time interval after last meal, h	Stomach Tryptophan (μg/g)	Serotonin μg/g)	Duodenum Tryptophan (μg/g)	Serotonin (μg/g)
24	7.35 ± 0.18	1.31 ± 0.18*	12.42 ± 0.24*	4.33 ± 0.12*
9	7.39 ± 0.29	1.25 ± 0.31*	18.21 ± 0.29*	4.74 ± 0.18*
3	7.41 ± 0.26	0.91 ± 0.24	24.23 ± 0.21	3.08 ± 0.15
0 (at the end of feeding period)	7.02 ± 0.15	0.86 ± 0.10	23.91 ± 0.07	2.97 ± 0.09

Each point is the average ± SE of 16 animals. Animals fasted 24 h were allowed to eat for 3 h after which time food was removed (zero time). The duration of fasting (h) reported indicates the intervals between food removal and death. * $p < 0.01$ compared to the value for 0 h of fasting.

with Mil rat Chow pellets from Morini Laboratories, S. Polo D'Enza, Italy.

Animals were killed by decapitation: one group at the end of the feeding period (fed animals), the other groups at different intervals thereafter (fasted animals). Stomach and duodenum were quickly dissected, cleaned in distilled water, blotted with filter paper, frozen in liquid nitrogen and stored at −30 °C until analyzed. Tissues were homogenized with a Polytron tissue homogenizer (Kinematica Gmbh., Luzern, Switzerland). Tryptophan and serotonin were assayed fluorometrically as previously described[14,15].

Results. The changes of 5-HT content occurring in the stomach and duodenum after feeding are shown in the table. The levels of 5-HT declined by 31 and 40%, respectively, at the end of the feeding period, remained at this level about 3 h and had returned to the fasting levels 9 h after food removal. In the stomach, the level of tryptophan, the 5-HT precursor, was unchanged in fasted and fed rats. On the contrary, in the small intestine, tryptophan levels rose by 100% at the end of the feeding period and remained elevated for more than 9 h. This increase most likely reflects the absorption of the digested proteins.

Discussion. The present study demonstrates that 5-HT concentrations in the stomach and duodenum are influenced by food intake. Fasted rats have higher levels of gastro-duodenal 5-HT than fed rats. It is possible that the decline in the gastro-duodenal content of 5-HT following food intake might be due to the release of the amine in situ and, therefore, into circulation. Consistently, Buldring and Crema[16] observed both in vitro and in vivo that, by exerting pressure on the gastro-intestinal mucosa, serotonin is released into the intestinal lumen.

On the other hand, a degradation of 5-HT by the action of monoamine oxidase does not seem to occur to a considerable extent in the gastro-intestinal wall, since we found that 5-hydroxyindoleacetic acid levels in this tissue were below the sensitivity of our method of assay (10 ng/g of tissue[14]) both in fasted and fed animals. The observed changes in the serotonin content were not associated with changes in the level of gastro-intestinal tryptophan. In fact, the levels of tryptophan did not change after feeding in the stomach; while, in the duodenum, they underwent changes in the opposite direction to intestinal 5-HT.

Experiments are in progress in our laboratory to clarify whether serotonin levels are affected by gastric distension, by some food constituent, or are controlled by the CNS in a reflex manner; moreover, to evaluate the physiological significance of our findings.

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Effect of nerve stimulation, denervation, and duct ligation, on kallikrein content and duct cell granules of the cat's submandibular gland

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Summary. Various procedures which reduce or deplete the kallikrein content of the cat's submandibular gland correspondingly reduce the number of apical granules in the striated duct cells. The kallikrein content is greatly reduced after chronic parasympathetic but not after sympathetic nerve section which suggests that the parasympathetic innervation is required for synthesis or storage of this enzyme.

The kallikreins, or kininogenases, are widespread enzymes belonging to the larger group of serine proteases[3,4]. The cellular and subcellular location of kallikrein has been studied in the salivary gland by several groups of workers[5–8] since it may have a bearing on its physiological significance in this organ where its role has been a subject of much speculation and discussion[9,10]. Whereas all workers agree that kallikrein is located in secretory granules, the earlier results suggested its location in acinar granules[5,11]. More recent work suggests that it is located in granules of the granular tubules and striated ducts in the rat[12] and in striated ducts of the cat[7,8]. The present experiments which correlate the kallikrein content of the cat's submandibular gland with associated microscopic changes, lend support to the thesis that kallikrein is localized in small granules in the apical region of the striated duct cells. They also indicate that the sympathetic innervation which is most effective for the stimulation of kallikrein secretion[13] is unnecessary for the synthesis and storage of this enzyme whereas parasympathetic denervation results in almost complete failure of the gland to synthesize or to store kallikrein.

Methods. For acute experiments involving nerve stimulation, cats of 1.5–4.5 kg and of either sex were starved for 16–20 h, and anaesthetized with chloralose (80 mg/kg^{-1} i.v.) after induction with chloroform. The chorda lingual and cervical sympathetic nerves were exposed and stimulated electrically at 10 and 20 Hz respectively and saliva was collected as described previously[13]. For operative procedures with recovery, sodium pentobarbital

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