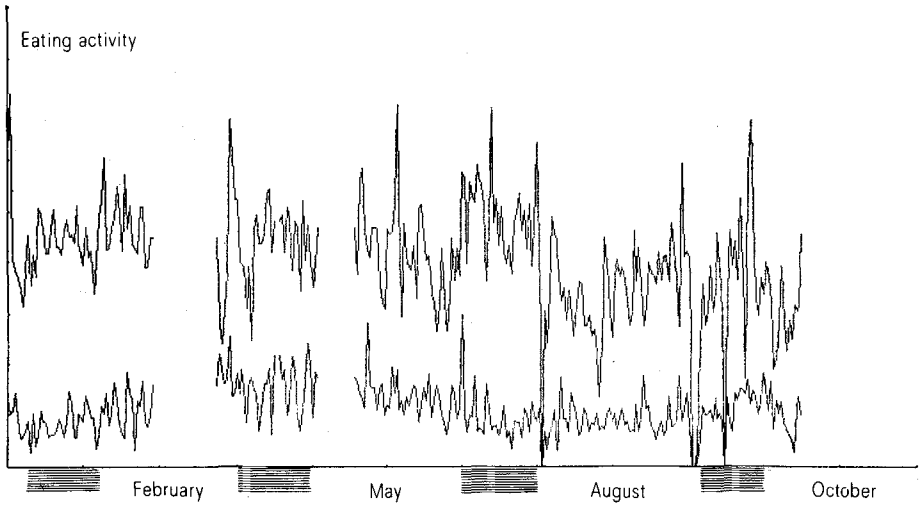

Fig. 3. Total amount of food consumed per light (lower graph) or dark period plotted against time for 10.5 months. The hatched areas indicate the periods during which the data presented in the table and figure 2 were obtained. The observed 5 day fluctuations in eating activity may run parallel with the estrous cycle. In addition a longterm fluctuation - peak in March/April, dip in August - can be observed.

simple food hoppers, recording of the eating activity of rats is very feasible. Automatic data sampling and storage as well as suitable computer evaluation of these data makes the method a valuable and reliable one for the investigation of eating behaviour in rats.

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Amylase secretion from rat parotid glands as dependent on co-operation between sympathetic and parasympathetic nerves[1]

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Summary. A slow, long-lasting 'degeneration secretion' from the parotid gland was brought about in anaesthetized rats by section of the auriculo-temporal nerve 16–19 h in advance. This parasympathetic background activity greatly increased the secretion of amylase elicited by sympathetic nerve stimulation.

Although sympathetic nerve fibres surround the acini of most salivary glands, the salivary flow response to electrical stimulation of the cervical sympathetic trunk is mostly remarkably small, even when the pronounced vasoconstriction elicited at the high stimulation frequencies required for secretion is prevented pharmacologically[2]. Experiments on dogs and cats show, however, that the flow is much increased, and otherwise subliminal frequencies become effective, when the sympathetic nerve is stimulated while the gland is exposed to a slow stream of parasympathetic impulses, corresponding to the physiological situation in the waking state[2,3]. In these experiments, only the volumes of saliva produced were taken into account, but recent observations on the parotid gland of the rabbit indicate that also the secretion of amylase on sympathetic stimulation is promoted by simultaneous parasympathetic activity[4].

After section of the auriculo-temporal nerve in rats, acetylcholine release from the degenerating axons causes a period of 'degeneration secretion' of saliva from the parotid gland; it starts after 14–22 h and lasts for 7–8 h[5]. In the present experiments, this was made use of to provide the slow, prolonged parasympathetic background activity on which sympathetic stimulation was superimposed to evoke secretion of amylase. As in the rabbit, but not in the dog or cat, the parotid gland of the rat is rich in amylase, which is secreted particularly in response to sympathetic stimulation[6,7].

Methods. In 10 male rats of a Wistar strain (weights 290–400 g) the right auriculo-temporal nerve was cut in ether anaesthesia and 16–19 h later chloralose (50 mg/kg) was given i.v. after induction with ether. Anaesthesia was then maintained by injecting pentobarbitone (2–5 mg/kg) i.v. when required. A tracheal cannula was inserted and the right parotid duct cannulated. Drops of saliva of a size of 10 µl were recorded and collected in samples of 12–14 drops each, which were stored frozen until analysed for amylase[8]. The cervical sympathetic trunk was exposed, placed on a bipolar electrode and stimulated at 0.3, 0.5 and 1.0 Hz, using a pulse duration of 2 msec and supramaximal intensity. Stimulation at 1.0 Hz was repeated after i.v. injection of atenolol 2 mg/kg. The 10 rats all showed degeneration secretion, but only in 6 of them was it sufficiently rapid, constant and long-lasting to allow collection of all the stimulation samples and interposed controls, altogether 10–12 samples, occupying a time period of 3.5–4.5 h.

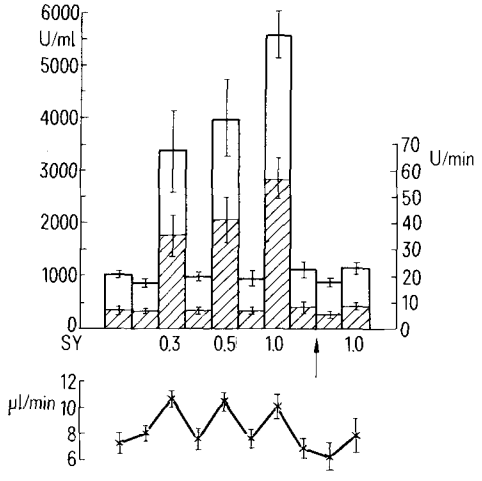
Results and discussion. The results of the 6 experiments are summarized in the figure. The saliva produced during the degeneration secretion contained amylase in a concentration similar to that reported for saliva secreted during electrical stimulation of the auriculo-temporal nerve in normal rats[7]. Superimposed sympathetic stimulation markedly increased the amylase content of the saliva. A frequency as low as 0.3 Hz raised it almost 4 times, from

861±71 to 3374±748 units/ml ($p < 0.02$) (mean±SEM), and at 1.0 Hz it rose from 943±116 to 5573±425 units/ml ($p < 0.001$), i.e. to a value similar to that obtained when the sympathetic nerve was excited at 10 Hz in the absence of parasympathetic background activity[9].

Sympathetic stimulation also increased the salivary flow rate. At 0.3 Hz it rose from 8.1±0.5 to 10.7±0.5 µl/min ($p < 0.01$). The higher frequencies did not further accelerate the flow, probably because of increased sympathetic vasoconstriction. Owing to the increased flow rate during sympathetic stimulation, the output of amylase calculated per unit of time rose even more than the concentration, at 0.3 Hz from 7.0±0.8 to 35.6±7.7 units/min ($p < 0.02$).

The β_1-adrenoceptor blocking drug atenolol decreased the amylase secretion in response to sympathetic stimulation. Previous observations indicate that the β-adrenoceptors of salivary glands belong to the β_1-type[10-14], and in rabbits amylase secretion on sympathetic stimulation is abolished by atenolol[4]. In the present experiments, a small response to stimulation at 1.0 Hz remained after atenolol; the dose of the drug was very likely insufficient for complete block.

In experiments carried out in this laboratory on normal rats of the same strain as the one used here, a sympathetic stimulation frequency of 0.5–1.0 Hz was required to produce a perceptible flow of saliva[15]. The present observations show that by providing a low parasympathetic background activity it is possible to obtain at lower frequencies not only secretion of fluid but also a very marked output of amylase; and already at 1.0 Hz, the saliva thus produced contains as much amylase as that secreted at 10 Hz when the gland is activated exclusively via the sympathetic pathway. As shown originally by Ohlin[16], alimentary reflexes in salivary secretion in rats engage not only parasympathetic, but also sympathetic secretory nerves. Hence it can be concluded that the augmented responses[2,3] demonstrated here, when the 2 types of nerves co-operate, are of physiological significance.



Effects of sympathetic stimulation (SY) on the amylase concentration (open columns) and output/min (hatched columns) during parasympathetic degeneration secretion. Below: salivary flow rate. Arrow: atenolol, 2 mg/kg. Means±SEM. When stimulation samples at 0.3, 0.5, 1.0 Hz and 1.0 Hz after atenolol are compared with their preceding controls, the following p-values are obtained: less than 0.02, 0.01, 0.001 and 0.05 for amylase/ml; less than 0.02, 0.02, 0.001 and 0.01 for amylase/min; and less than 0.01, 0.05, 0.05 and 0.05 for flow rates.

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Naloxone selectively blocks dopamine response of Br-type neuron in *Helix pomatia* L.[1]

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Summary. The present study demonstrates that the potent opiate antagonist, naloxone can selectively block the DA induced inhibition of the bursting activity pattern of the RPal or Br-type neuron. The dopamine inhibitory affect can also be blocked by haloperidol, a established dopamine receptor blocker.

Previous investigations dealing with the molluscan CNS have demonstrated the the physiological and pharmacological characteristics of this system are comparable to the mammalian CNS[3-11]. In *Helix pomatia* the giant identified bimodal pacemaker, Br-type neuron has been shown to receive synaptic inputs[12]. It has also been demonstrated that this neuron is sensitive to the application of various monoamines[12]. Recently, Fuxe et al.[14] reported that naloxone may block dopaminergic transmission in a mammalian system. Therefore, the present study was undertaken to determine if this latter effect exists on a known dopamine responsive cell.

Preparation. Experiments were carried out on the Br-type cell of the snail *Helix pomatia* L. Previously this neuron was termed RPal[15]. The preparation and recording technique have been well documented[13]. Briefly, while being perfused the ganglionic complex was freed from the connective tissue surrounding it. The preparation was then transferred to a 20 ml glass vessel with a open front end so that the cells in question can be identified, positioned and