Fig. 4. Cross section (10 μm) of pedicle (PD) showing decrease in AF positive granules (arrows) AF × 360.

was reduction in the cell’s size, which measured 30.0 × 20.5 μm with a nucleus of 9.5 μm diameter. During the last phase of vitellogenesis, when the oocytes were fully loaded with yolk spheres and measured from 2.17 to 2.25 mm in length, there is a recorded decrease in AF and PAVB positive granules in the pedicle, (Figure 4) as well as in the A-type cells. Simultaneously, the chorion formation takes place and the stainable granules in the pedicle come to a negligible concentration and some flaky mucoid substance was visible in this region.

It is difficult to trace the source of this AF and PAVB positive granules in the pedicle. But it is definite that the material is not secreted by the cells of the ovarian pedicle, because these cells always responded negatively to AF and PAVB stains. There could, therefore, be two likely possibilities. Either this material is other than the neurosecretory material, since, besides the NSM, a variety of other substances are also revealed by these staining techniques, or this could be an additional storage site for the water extractable material obtained from entire ripe ovarioles acts as an ovarian sex hormone which inhibits the neurosecretory supply to the corpus allatum, and simultaneously stimulates the discharge of neurosecretions from neurosecretory cells of the brain and thereby brings about oviposition. But the same result was not obtained when aqueous washings of freshly laid eggs were injected into other female bugs. The present observations suggest that the probable source of the ovarian sex hormone envisaged by Nayar and Doane might be related to the AF and PAVB positive granules of the ovarian pedicle of D. koenigii and may represent stored neurosecretory material.

Lateral Hypothalamic ‘Feeding’ Sites and Gastric Acid Secretion

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Summary. Electrical stimulation within the lateral hypothalamus which had been effective in evoking stimulus-bound feeding in satiated cats did not produce any significant stimulating effect on gastric acid secretion in the same cats when hungry.

It is generally known that the lateral hypothalamic area contains structures responsible for the initiation of feeding. It is reasonable to ask, therefore, whether the lateral hypothalamic feeding system also controls gastric acid secretion. So far, there have been only a few studies directly dealing with this problem. It was found that gastric acid secretion increased as a result of electrical stimulation or stimulation by 2-deoxy-D-glucose (2DG, a compound known to be effective in stimulating feeding) within the lateral hypothalamus in acute rats. It was also observed that the stimulating effect of i. v. injection of 2DG on gastric secretion in chronic cats was abolished by bilateral lesions in the medial forebrain bundle, a hypothalamic structure known to be involved in feeding reactions.

1 The author wishes to thank Dr. M. I. Grossman for advice and valuable discussion and Dr. D. Novin for critical reading of the manuscript. Thanks are also due to Miss Susan Davis and to Mr. R. Garcia for skilful technical assistance.

3 The author is grateful to Dr. J. D. Elashoff who did the statistical analysis of the results.


5 P. Tiedtlbaum and A. N. Epstein, Psychol. Rev. 69, 74 (1962).


The above data suggest that the lateral hypothalamic structures which govern the initiation of feeding may also influence gastric acid secretion. The present study was undertaken to further explore this possible relationship.

Method. The experiments were performed on adult cats, both male and female. In each animal 2 monopolar electrodes (made of stainless-steel wire, 0.3 mm in diameter, insulated except for 1 mm at the tip) were bilaterally implanted in the lateral hypothalamus. The coordinates of implantation were: All, L3, H -4, according to the stereotaxic atlas by JASPER and AJMONE-MARSAN. A reference electrode was placed in the calvarium over the frontal sinus.

Two weeks after the implantation a test for stimulus-bound feeding was performed. The cat was placed in an experimental compartment and allowed to eat ad libitum. When the animal stopped eating, electrical stimulation (1–3 V, 100 cps, 1 msec dur/imp, given either continuously or with 10 sec on and 10 sec off intervals) was applied unilaterally to the hypothalamic electrode tip. 7 cats in which electrical stimulation had resulted in eating were chosen for the experiment.

In each of these cats a gastric fistula was constructed, as described by EMAS. A few weeks later the experimental procedures started. First, during a few days the cat was trained to stay still in the stand (as shown by EMAS and AJMONE-MARSAN). Then regular sessions were conducted 2 to 3 times a week. The cats were deprived of food for about 20 h before the session. Each session consisted of six 15 min periods of collection of basal gastric secretion. Immediately after the session the volume of each sample was determined and its acidity was measured by titrating a 0.2 ml sample to pH 7.0 with 0.2 N NaHCO₃ with the use of an automatic titrator (Radiometer, Copenhagen). The total acid output was calculated by multiplying the volume of each sample by its acidity. After 5 control sessions, electrical stimulation of the hypothalamic site previously effective in producing feeding, was applied during the next 3 sessions. Stimulation was given with 10 sec on and 10 sec off intervals during the 3rd or 4th period in 2 other cats (GH7 and GH11). Parameters of stimulation were the same as those previously effective in evoking feeding.

After the completion of the experiments, the cats (except GH7 and GH11) were sacrificed and their brains were taken out for anatomical verification of the location of electrodes (Figure 1).

Results. It was found that the cats remained generally quiet during the hypothalamic stimulation, although licking, swallowing and salivation was frequently observed. The acid output was usually highest at the beginning of the session and tended to diminish toward the end of the session during both control and stimulation sessions. Figure 2 shows the diagrams of the mean values of the acid output in the periods before (A), during (B) and after (C) stimulation in stimulation sessions and in the corresponding periods of the control sessions (A', B', C').

Fig. 1. A diagram of a frontal section of the diencephalon, after the atlas of JASPER and AJMONE-MARSAN, showing the sites of stimulation within the lateral hypothalamus. Numbers 1, 2, 3, 4 and 5 inside black circles show the location of the tips of stimulating electrodes in cats GH1, GH2, GH3, GH4 and GH5, respectively. The electrode tip locations for cats GH2 and GH3 were found 1 mm caudal to those shown above. HL, lateral hypothalamus; NHvm, ventro-medial hypothalamic nucleus; MFB, medial forebrain bundle; AH, dorsal hypothalamic area. Other denotations refer to extra-hypothalamic structures.

Fig. 2. The effect of electrical stimulation of the lateral hypothalamic ‘feeding’ sites on gastric acid secretion. Bars represent means ± SE for gastric acid output (μEq) in 15 min periods before (A), during (B) and after (C) the hypothalamic stimulation in 5 stimulation sessions and in the corresponding periods (A', B', C') in 5 control sessions, for each cat (except GH3 in which 2 stimulation sessions were performed only). As shown above, the lateral hypothalamic stimulation produced no significant change or a slight decrease (in cat GH11) in gastric acid secretion.