Preliminary Report on Hypothalamic Hyperphagia in Ruminants

Abstract

Sustained hyperphagia and subsequent rapid weight increases were induced for the first time in ruminants (goats) by lesions in the ventromedial areas of the hypothalamus. This finding supports the concept that the ventromedial area is functioning in the regulation of food intake of ruminants, although the stimuli monitored may be different from those in monogastric animals.

 Destruction of the ventromedial hypothalamic nuclei is followed by hyperphagia in the rat (5), in the mouse (9), in the monkey and cat (1), and in the rabbit (4). In a recent review of the possible role of the ventromedial area in the regulation of food intake, Mayer (8) summarized evidence that in monogastric animals the ventromedial nucleus is a satiety center which probably acts through temporary inhibition of feeding centers situated in the lateral area of the hypothalamus. The ventromedial area also appears to have some control of gastric contractions and gastric juice secretions.

In ruminants, the nature of digestion is very different, as the diet under natural conditions has a high content of cellulose and hemicellulose and a low caloric density. The role of the components of the gastrointestinal tract differs so markedly from that in monogastric animals that it was by no means evident that a satiety area similar to that seen in monogastric animals should exist or that this function should reside in the ventromedial area of the hypothalamus. Holmes and Fraser (6) reported that ewes, following apparent bilateral electrolytic lesions in the ventromedial nuclei, did not exhibit hyperphagia, striking weight increases, or extreme fat deposition.

While Baile and Mayer (3) observed temporary hyperphagia in goats following injection of small quantities of pentobarbital, a neural depressant, into the cerebral ventricles, thus affecting the ventromedial areas adjacent to the third ventricle, this is at best circumstantial evidence that inactivation of satiety areas situated in the ventromedial region causes the hyperphagia. More direct evidence that the...
hyperphagia in goats was a result of inactivation of the satiety centers is that, later, eating was induced by bilateral injections in the medial hypothalamus of 2.4 mg pentobarbital sodium (2). We report that the induction of ventromedial lesions can cause sustained hyperphagia and increased body weight in goats.

A series of mature goats was surgically prepared with electrode guides which enabled functional placement of electrodes in the hypothalamus as described by Baile, Mahoney, and Mayer (2). Bilateral electrolytic lesions were induced in hypothalamic areas in the anesthetized goats. Daily feed and water intakes were determined. The goats were fed ad libitum during pre- and post-lesioning periods a concentrate ration containing approximately 9% fiber, and 200 g per day of a grass hay.

Of the goats lesioned, the two to be discussed in some detail showed what has been found to be rather typical changes in feed and water intakes and body weight. Other goats in our laboratory showing similar responses following lesioning are being more intensely studied, and histological locations have not, of course, been determined. Goat 46, following a series of electrode placements and stimulations, quickly increased its body weight to about one and one-half its initial stable body weight. Figure 1 shows body weight, feed intake, and water intake. The feed and water intakes are shown as means of 28 days. The weight of Goat 46, which had been constant for two months, almost doubled during three months. Although the feed and water intakes were not measured prior to lesioning, the animal was fed ad libitum the same diet. After about two months of hyperphagia and hyperdipsia, feed and water intakes decreased.

Figure 2 shows the locus of the only lesion found in the hypothalamus at the time Goat 46 was sacrificed. The lesion included the ventromedial nucleus on the animal’s left side. Electrolytic lesions were caused bilaterally in the hypothalamus of Goat 43. Figure 3 shows the weight, feed intake, and water intake data of this goat. The data are grouped in two pre-lesion, one hyperphagic, and two post-lesion periods. The goat ate the 200 g of hay daily throughout the test. The feed intakes (grain only) of the two pre-lesion periods and the two post-lesion periods are less than that of the hyperphagic period (p < .01). The actual productive energy available increased more than the feed intake data would indicate; the animal was always very active and friendly.
prior to the lesion, but immediately after became quite docile and apathetic to her surroundings. The water intake and rate of weight change during the hyperphagic period were higher than those during the other periods (p < .05). Figure 4 shows a lesion located in the hypothalamus of Goat 43; lesions were found in both ventromedial areas.

There are several possible reasons our results are not in agreement with those of Holmes and Fraser (6). There could be a species difference, but this seems unlikely. It is possible, if the authors used young sheep, that the growing lamb, like the immature rat, would not respond to hypothalamic lesions. However, it seems more likely that they used mature sheep. They tested their sheep only on a roughage diet which could be so dilute calorically that volume would limit intake, and intake could not be increased further by destruction of the ventromedial area.

As in monogastric animals, stimulation of the lateral area of the hypothalamus of goats causes indiscriminate eating (7), and destruction of the ventromedial area causes hyperphagia in goats as we have demonstrated. These facts suggest that the role of these areas in ruminants is similar to that in monogastric animals. It may well be that the metabolic trigger of the ventromedial area is different in ruminants.

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Fig. 3. Body weight, water intake, and food intake of Goat 43. The hypothalamic lesion was caused on Day Zero.

* All means not noted by the same letter are different (p < .01).

Fig. 4. Photomicrograph showing lesion in left ventromedial area of hypothalamus of Goat 43. The arrow indicates the site of the lesion.
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References


Preliminary Report on Feeding Activity and Hypothalamic Temperature in Goats

Abstract

Hypothalamic temperature change has been suggested as a stimulus for satiety in ruminants, perhaps mainly because they have a high heat increment immediately following a meal. An experiment was designed to determine the relationship between feeding behavior and body temperature changes. A needle thermistor was implanted in the medial hypothalamus of each of two goats. The goats were tested under the following conditions: water ad lib. (control); feeding period after a 20-hr fast (grain); 1,000 g of grain force fed through a ruminal cannula; 250 ml of 1.0 M acetic acid injected intraruminally; and 2 ml/min of 2.5 M NaAc injected intraruminally for two hours. The hypothalamic temperature decreased following eating, the force feed, and both acid and the salt injections. The fact that hypothalamic temperature does not increase during a large meal or a force feed is evidence that short term satiety in ruminants is not normally a function of hypothalamic temperature. The feed intake depression shown to follow intraruminal acetate injections is apparently not a result of an increased hypothalamic temperature.

The thermostatic theory of regulation of food intake implies that animals eat to keep warm or to keep the hypothalamus at a constant temperature (14). It has been suggested that a thermostatic satiety mechanism, first postulated for monogastric animals (9), would have particular relevance in ruminants (6, 10, 13) because of their high heat increment of feeding (8). It was found that maximum heat production occurs under certain conditions during the first hour after eating is initiated (12). The thermostatic hypothesis appeared to be confirmed by the experiment of Andersson and Larsson (2), who caused changes of the temperature in the anterior hypothalamus, which contains the sensors and controllers of body temperature. They were able to induce eating by cooling this area in a satiated goat, and inhibit eating by warming this area in a hungry goat. However, the temperature changes were so extreme (cooling 9°C and warming 8°C) as to be entirely unphysiological. Such extreme cooling may have simply inactivated the neighboring ventromedial hypothalamic satiety centers, while the extreme warming may have caused anorexia due to pyrexia.

Experiments conducted with rats (1, 11) showed that there was a significant increase in brain temperature of the order of 0.6 and 1.0°C, respectively, while an animal ate. The size...