Preoptic temperature during oral and intragastric feeding*

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Preoptic brain temperatures of rats were recorded during oral and intragastric feeding. Oral ingestion of solid dry food was the only treatment that significantly increased preoptic temperature. Oral ingestion of liquid food and infusion of either liquid food or water directly into the stomach via a cannula failed to produce a significant change in brain temperatures. These data are not consistent with Brobeck's (1951) thermoregulatory hypothesis which predicts that both ingested and infused liquid nutrients should also increase preoptic temperature.

Although extensive work has been done in the area of hunger, the mechanisms by which feeding is initiated and terminated are still not well understood. One proposal that has attracted considerable attention is Brobeck's (1951) thermoregulatory hypothesis. This hypothesis states that the organism's energy equation is balanced by a temperature-regulating mechanism and that food intake is part of the normal control of body temperature. Brobeck's most recent version of thermoregulation (1960) suggests an interaction between feeding and temperature regulation via a central heating mechanism located in the preoptic area of the hypothalamus.

The position outlined by Brobeck makes extensive use of the concept of specific dynamic action (SDA). Ingestion of food initiates metabolic processes that involve chemical reactions associated with digestion. The heat produced during metabolism is called SDA. It is this heat which terminates feeding, and the lack of it, when an organism needs more food, which initiates feeding behavior. Temperature variations in the brain have been recorded by Abrams & Hammel (1964), Delgado & Hanai (1966), and Hamilton (1963b) and correlated with food intake in a manner consistent with this hypothesis.

Grossman & Rechtschaffen (1967) have also recently reported that preoptic temperature increases during food intake, but conclude that these events are not causally related. These investigators implanted thermistors into both rats and cats and noted that eating often continued after a maximum temperature had been reached. It was concluded that food temperature and/or muscular activity involved in chewing rather than SDA determine brain temperature changes.

The purpose of this experiment was to determine whether or not infusion of nutrient substances directly into the

stomach would produce an increase in preoptic brain temperature similar to that produced by normal feeding. A thermoregulatory interpretation of the regulation of food intake would predict an increase in brain temperature during feeding irrespective of the route by which food is introduced into the organism. If, however, muscular activity involved in chewing is the determinant, intragastric feeding would not be expected to increase brain temperature.

METHOD

Four male Sprague-Dawley rats, 100 days old at the time of testing, were implanted with a stomach cannula and a small bead thermistor (YSI, No. 44005). Surgery was performed using barbiturate anesthesia. Following recovery from surgery, each S was placed on a deprivation schedule that permitted access to food for $\frac{1}{2}$ h every 12 h. Water was always available in the home cage.

Each S was intubated in the fundic portion of the stomach. The cannula was brought out of the abdominal cavity and was then passed subcutaneously up the back and externalized through a small incision on the withers. The stomach cannula was constructed of PE 90 polyethylene tubing flanged at the proximal end and fitted with a Teflon washer to facilitate attachment to the stomach. The distal end of the cannula was encased in PE 200 tubing to prevent rupture of the cannula as a result of movements made by S.

After recovery from the cannulation the thermistor was stereotaxically implanted into the preoptic region of the hypothalamus corresponding to De Groot (1959) coordinates (A = 7.6; L = 1.0; H = -1.5). The placements were verified histologically following testing.

The thermistor served as one leg of a Wheatstone bridge circuit. The signal from the bridge circuit was amplified and converted to voltage. This signal was the input to a digital voltmeter and a

printer-recorder. This circuit allowed visual monitoring of temperature and direct digital printout.

During testing, S was housed in an 8-in. square sanded Plexiglas cage with a liquid feeding cup mounted on one wall. The front wall of the cage, adjacent to the wall containing the food cup, was of clear Plexiglas to permit observation of S. This cage was placed inside a sound-deadened box to minimize extraneous stimuli and to maintain a relatively constant ambient temperature of $23^{\circ}\text{C} \pm 1^{\circ}\text{C}$. All tubing and cables were attached to S via a suspension arm and slip ring assembly.

Following adaptation to the 12-h food deprivation schedule, S was placed in the apparatus twice a day for 2 days. During this preliminary training, S was fed and allowed to explore the apparatus.

All Ss received two trials under each of four treatments: oral solid food, oral liquid food, intragastric liquid food, and intragastric water. The oral solid food was placed on the floor of the cage and consisted of 6 g of dry Purina laboratory chow which required handling and extensive chewing by S for ingestion. The oral liquid food was 6 cc of a mixture of whole egg, sucrose and evaporated milk (Teitelbaum & Epstein, 1962). To ingest it, S was required to lap the liquid from the food cup. Intragastric liquid food was 6 cc of the same liquid diet, but it was infused directly into the stomach. Similarly, 6 cc of water were infused into the stomach for the fourth treatment. Intragastric and liquid oral feeding were effected with the aid of an infusion pump set at a rate comparable to that of a rat's normal ingestion rate for liquids. The temperature of all ingested and infused substances was 23°C (room temperature).

Treatments were counterbalanced to control for order of testing and time of day. All Ss were run twice a day for 4 days. On each trial, S was placed in the apparatus and allowed to attain a steady baseline temperature. This baseline was the mean temperature over the final 2 min prior to initiation of feeding. Temperatures were recorded at a sampling rate of once every 7 sec and analyzed in terms of 70-sec intervals. Feeding sessions lasted for 30 min after the initiation of eating. Only the first 6 min of the liquid sessions involved actual ingestion or infusion of substances, but recording continued for another 24 min to complete the session.

RESULTS AND DISCUSSION

Mean temperatures were computed for each successive 70-sec interval. A difference score was obtained by subtracting the baseline temperature from the maximum 70-sec mean temperature. A mean difference was then computed for

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Fig. 1. Mean preoptic temperature change from prefeeding baselines over successive 140-sec intervals.

each S for each treatment. Analysis of variance of these data yielded a significant main effect due to treatments ($p < .001$). Individual comparisons of treatment means, using *t* tests, showed a significant increase in preoptic temperature for only the oral solid condition ($p < .01$). The mean temperature changes over successive 140-sec intervals for each treatment is shown in Fig. 1.

Maximum temperature changes for the oral solid treatment had a range of 1.05°C to 1.23°C with a mean increase of 1.12°C . The maximum temperature change ranged from 0.10°C to 0.38°C , 0.10°C to 0.20°C , and 0°C to 0.20°C for the oral liquid, gastric liquid, and gastric water, respectively. The corresponding mean increases were 0.26°C , 0.16°C , and 0.11°C .

The results of this study indicate that the ingestion of nutrient substances and the increased heat production associated with the metabolism of such substances do not necessarily cause an increase in the temperature of the preoptic area. Such an increase in brain temperature clearly occurred only during the consumption of solid dry food. This effect was noted even though the SDA of the liquid food was higher than that of the solid dry food. These data, therefore, do not support Brobeck's thermoregulatory interpretation of the regulation of food intake since such an approach predicts that both ingested and infused liquid food should also increase preoptic temperature.

Three alternative hypotheses could be used to account for the observed increase

in preoptic temperature following consumption of solid dry food. The first of these is an increased level of neural activity and its associated production of heat as suggested by the work of Serota & Gerard (1938) and Tachibana (1966). These investigators observed localized temperature increases when their Ss were stimulated in the sensory modality associated with these specific brain areas. This interpretation, however, predicts an increase in temperature resulting from increased sensory input for oral liquid as well as oral solid food.

Secondly, as shown by Grossman & Rechtschaffen (1967), the temperature of the food in the mouth when well above or below body temperature will change brain temperature. Although the substances used in this study were approximately 15°C below body temperature, all substances were at the same temperature when infused or ingested. This explanation, therefore, cannot account for the differential effects obtained in this study.

Lastly, Abrams & Hammel (1964), Hamilton (1963a), and Grossman & Rechtschaffen (1967) suggest that temperature increases in the brain may be the result of heat produced by muscular activity associated with feeding. The results of this study are consistent with this interpretation. Ingestion of a solid dry food, which required handling and chewing, produced an increase in preoptic temperature. Ingestion of an oral liquid, requiring significantly less muscular effort, and infusion of liquids, requiring almost no effort on the part of the organism, failed to

produce any increase in brain temperature.

It should be noted that in this study, the oral ingestion of a liquid diet did not significantly increase brain temperature whereas Grossman and Rechtschaffen do report an increase with this treatment. The liquid diet used by these authors was at body temperature while all substances in this study were at 23°C (room temperature) to facilitate maintenance of a constant temperature for all substances. Since lapping does involve muscular activity, it is probable that heat is produced by this behavior although less than that produced by chewing. Apparently, the lower liquid temperature used in this study was enough to offset the effects of the heat produced by the muscular activity required for lapping. Since no changes in preoptic temperature were observed following infusion of nutrients directly into the stomach, wherein oral factors are eliminated, the concept of SDA cannot be used to explain the increase in brain temperature associated with the oral consumption of nutrient substances.

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