infinite in extent since they do not terminate. Specific layers of such a sphere, if caused to differentially expand or contract sufficiently to cause deformation, will fail polygonally (*1*).

Desiccation of wet, tabular shaped clay bodies at room temperature and field observations of similar phenomena indicate that the polygonal fracture systems apparently caused by negative (tensile) stress (Fig. 3*B*) develop in the clay layers in essentially the same order as the thrust and fold systems of the salt crust. The ratio of layer thickness to cell width varies considerably. Polygonal cells developed in 3-mm-thick clay layers, drying at room temperature, had a thickness-to-width ratio of 1:12 (*2*).

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2. Investigation supported by a grant from the University of Utah Research fund.

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Hypothalamic Temperature in the Cat during Feeding and Sleep

Abstract. *Anterior hypothalamic temperature is reported for the unanesthetized cat resting at an air temperature of 22° to 25°C during the ingestion of cold or warm liquids, and during sleep. Drinking cold (5°C) milk resulted in an immediate depression of hypothalamic temperature and a period of peripheral vasodilation in the ear and forepaw foot and toe pads, followed by a drop in rectal temperature. Drinking warm (body temperature) milk did not bring about these changes. Hypothalamic temperature during sleep is lower by approximately 0.5°C and is characterized by widely varying, slow-frequency oscillations, compared to the higher, more precisely controlled temperature seen when the animal is awake.*

The anterior hypothalamus in several species of homoiotherms has been identified as a central nervous system site intimately involved in body temperature regulation. Not only has this area been shown to play a neural integrating role for the maintenance of biothermal control (*1*), but the temperature level of this portion of the diencephalon has also been shown to be one of the inputs for internal thermal control mechanisms (*2*).

Anterior hypothalamic temperature was measured in the cat by a small thermistor bead (VECO 32A7) housed in the tip of a 23-gauge stainless steel needle chronically implanted in the region of the supraoptic nuclear groups, and recorded on a single-point, calibrated, constantly recording Honeywell potentiometer. Rectal temperature was measured by a thermistor probe inserted 10 cm into the lower colon. Skin and extremity temperatures were measured with 36-gauge copper-constantan thermocouples attached to the skin by a single layer of thin plastic tape and recorded on a multiple-point, continuously recording potentiometer. All measurements were made on unanesthetized cats resting in a plastic wire mesh hammock. None of the animals tested in this portion of the study demonstrated any thermoregulatory consequences of this type of restraint. Ambient temperature during these measurements was between 23° and 25°C.

Figure 1*A* shows the pattern of anterior hypothalamic temperature in an awake, attentive animal through the 12 minutes of measurement. The 0.1°C fluctuations seen in this record have been reported earlier for this species under similar testing conditions (*3*). In the present study, after the animal became accustomed to the restraining procedures it would spontaneously sleep if left unattended for longer than an hour. Hypothalamic temperatures during these sleeping periods are reported in Fig. 1, *B* and *D* and the first half of tracing *C*. Rectal or room temperature during these periods did not change.

During sleep, hypothalamic temperature became more labile (tracings *B* and *D* in Fig. 1) and fell to a lower level than when the animal was awake, as shown in Fig. 1*C*. As the cat was awakened (arrow in Fig. 1*C*), anterior hypothalamic temperature increased and assumed a greater stability than the pattern recorded during sleep (compare the first and last half of tracing *C* in Fig. 1). The changes in this temperature pattern were not attributable simply to the dependent head position, since these altered patterns of hypothalamic temperature did not develop until after the animal had assumed a sleeping position for a number of minutes. Furthermore, head dependency per se associated with feeding (see below) did not result in modified hypothalamic temperature.

Considering the vascularization in this brain area and the rapidity with which hypothalamic temperature changed under these conditions, it would seem more likely that convective (local blood flow) rather than local metabolic thermal influences are responsible for these patterns. More direct measurements of blood flow in these areas would be interesting in relationship to the recently demonstrated changes in neural activity with sleep (*4*) and in view of the possibility that blood flow within the brain may show some degree of local control.

Local temperature changes in the anterior hypothalamus have been reported to bring about peripheral vascular adjustments appropriate to whole body thermal exposure (*5, 6*). Since drinking volumes of cold liquid has been used to readjust internal body temperatures in humans in which more direct local control is unfeasible, it was of interest to examine the thermoregulatory consequences of ingested cold liquids in cats with thermistors chronically implanted in the anterior hypothalamus.

Figure 2 (top) indicates that coincident with the beginning of drinking milk at 5°C, anterior hypothalamic

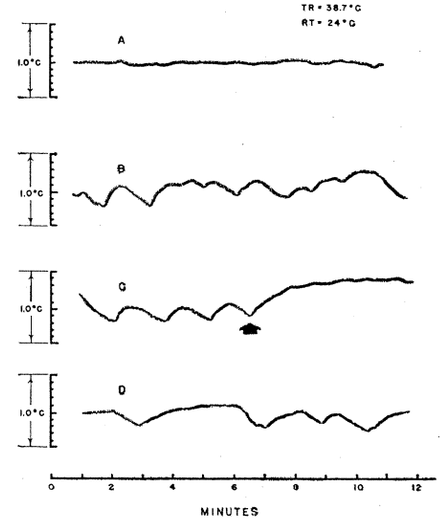

Fig. 1. The hypothalamic temperature shown in tracing *A* was recorded while the animal was awake and attentive to its environment; records *B* and *D* and the first half of tracing *C* were obtained while the same animal was asleep. The arrow in record *C* marks the point at which the sleeping animal was awakened. *TR* shows rectal and *RT* indicates room temperature during these measurements.

temperature fell precipitously, too closely related (in time) to the period of drinking to be accounted for by diencephalic circulation of blood cooled (by the ingested fluid) within the main body mass. Further, rectal temperature was seen to follow, rather than lead, these temperature changes in the hypothalamus. It would appear, therefore, that the reduced diencephalic temperature under these conditions was due to a loss of heat along conductive, rather than convective avenues.

The lowered hypothalamic temperature is accompanied by a period of peripheral vasodilation as seen in Fig. 2 (top). Only those body areas shown demonstrated these influences; there were no changes in the temperature levels of the tail, back, or distal portion of the hind leg. That this peripheral vascular response is more related to the reduced hypothalamic temperature and not to the dependent head position during drinking, or to the act of drinking, itself, is shown in Fig. 2 (bottom), which shows the result of ingesting milk at approximately body temperature. No predictable change in rectal or hypothalamic temperature and only a slight response in one extremity was relatable to the period of drinking in the latter test.

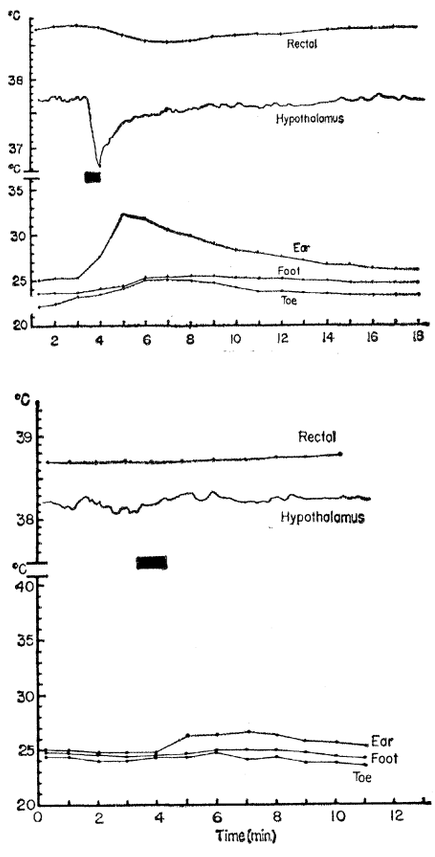

Fig. 2. Rectal, anterior hypothalamic, and extremity temperatures are shown during the ingestion of milk at 5°C (top) or at body temperature (bottom). The period of drinking is indicated by the shaded block under the hypothalamic temperature tracing.

The observation that peripheral vasodilation was consequent to lowering hypothalamic temperature in this manner is particularly surprising, since lowered anterior hypothalamic temperature by the use of thermodes in the cat and other animals has been reported to result in the more appropriate thermoregulatory peripheral vascular response of vasoconstriction (*5*).

The results of changing diencephalic temperature with water-perfused thermodes in the same animals used in the present study confirm the more predictable thermoregulatory responses of peripheral vasoconstriction with hypothalamic cooling (*7*); peripheral vasodilation only followed local hypothalamic heating with this technique. It should be noted, however, that the manner of inducing these thermal effects in the hypothalamus, either locally with thermodes or by feeding cold liquids, is quite different, and certainly more neural tissue is brought under the experimental thermal influence with the latter technique. Nonetheless, these two methods of inducing central nervous system temperature changes would not appear to be comparable physiologic testing procedures (*8*).

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Alteration in Learning Ability Caused by Changes in Cerebral Serotonin and Catechol Amines

Abstract. *Excess of cerebral serotonin decreased maze-learning ability of adult mice; deficiency of serotonin and catechol amines increased it slightly.*

Although there is now a substantial body of evidence to show that malfunctioning of serotonin in the brain can lead to profound changes in behavior of men and laboratory animals (*1*), very little information exists on the relation of this hormone to learning ability. The situation is similar with respect to the catechol amines. The behavioral effects have been found during the exploration of the relationship of serotonin and catechol amines to mental diseases such as schizophrenia, as first described by Woolley and Shaw (*2*). We have wanted to find out whether serotonin likewise might be causally related to some of the inherited idiocies. Consequently, we have wanted to determine whether it has a connection with various aspects of intelligence, such as learning ability, in addition to its relation to behavior.

To measure learning ability of mice, a simple maze was used, consisting of a T-shaped brass channel 3.3 cm wide, with a transparent top so that the mouse always saw the observer. An adult mouse was placed in the maze at the bottom of the stem of the T. As it moved up to the union of the stem with the crossarms it reached the point of decision. If it turned one way and ran along the arm, it received a reward in the serif of the arm of the T. If it turned the other way and ran along that arm, it found no reward. The reward was to escape from the view of the experimenter into a dark cubicle in the serif. In the other arm of the T there also was such a dark hiding hole, but the mouse was prevented from entering it by a screen not visible from the point of decision. The animal was allowed exactly 2 minutes to explore the maze, and to learn that the reward was to be obtained only by making the correct turn. The mouse so trained was then tested in ten tries to determine how well it had learned its lesson. If it had learned nothing in the 2 minutes, chance alone would dictate that it would make the correct turn five times in ten tries. If it had learned perfectly, it would make a score