ACTA NEUROBIOL. EXP. 1976, 36: 129–140

Memorial Paper in Honor of Jerzy Konorski

HYPERTHERMIA AND INHIBITION OF FEEDING PRODUCED BY SELF-STIMULATION IN THE SEPTUM AND PREOPTIC AREA IN DOGS

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Abstract. Dogs bearing electrodes implanted in the anterior part of the basal forebrain were tested for their response to food upon electric stimulation of rewarding sites, and for self-stimulation–produced hyperthermia. Self-stimulation and forced (experimenter–induced) stimulation of 12 out of 16 loci evoked a negative reaction to food the strength of which was determined according to occurrence and persistence of three effects: ignoring food (the dog performing self-stimulation in the presence of readily available meat), food rejection (the dog's failure to take meat offered together with passive stimulation of the rewarding site), and food ejection (throwing meat out of the mouth upon passive stimulation). The rise in body temperature during self-stimulation was positively correlated with the rate of responding. Hyperthermia was significantly higher during self-stimulation in sites where stimulation produced a strong negative reaction to food, as compared with those where stimulation failed to stop the animal from eating. The stimulus–contingent negative reaction to food may reflect a short-term satiety which is supposed to play an essential role in the mechanism of reinforcement. Hyperthermia, and particularly stabilization of hypothalamic temperature on an elevated but fairly constant level, argues for a shift of the set-point for temperature regulation. Occurrence of the two effects supports the claim that self-stimulation produces some complex activation of neural processes controlling energy homeostasis.

INTRODUCTION

There are data showing a mutual relationship between feeding and thermoregulation. Body temperature is known to rise following ingestion of food. Hypothermia is observed together with aphagia due to amygdalar and hypothalamic lesions in dogs (17). Lateral hypothalamic stimulus-bound eating threshold is found to depend on environmental temperature (2). Increasing evidence indicates that the phenomenon of self-stimulation is related to excitation of a neural system controlling food intake. Self-stimulation is also found to produce hyperthermia in rats (7, 10, 26) and dogs (13, 36).

Previously it was demonstrated that self-stimulation in some forebrain areas of dogs elicits a negative response to attractive food, and this effect was interpreted as due to a state of strong short-term satiety produced by the stimulus (35). The purpose of the present investigation was to find out whether there is any relation between the strength of the negative reaction to food and the increase in body temperature during self-stimulation through the same electrode using the same current parameters.

METHODS

Electrodes and stereotaxic procedures. The experiment was performed on 10 male mongrel dogs bearing stainless steel concentric electrodes chronically implanted in the basal forebrain. Two electrodes were inserted on either side. Each electrode consisted of a 0.5 mm diameter stainless steel tube containing a 0.1 mm diameter steel wire protruding 1–1.5 mm beyond the end of the outer part. Both parts of the electrode were covered with a varnish, and the insulation was removed 0.5 mm back from their tips. Extracranial ends of the electrodes were soldered to pins of a plug fastened in a plexiglass socket which was screwed into the parietal bone.

The electrodes were placed with stereotaxic quidance at the following standard coordinates: A, 24–27 mm (anterior placements) and 21–24 mm (posterior placements); L, 3–5 mm; and V, 10–11 mm (the position of the end of the tube). On the basis of previous experience (34, 36) these positions were found optimal for self-stimulation. Due to variability of skull dimensions the standard A coordinates were corrected for each animal using either of the following empirically found coefficients: (i) the APO, infraorbital coefficient which is the ratio of the actual distance between the interaural and the infraorbital planes, and the standard distance equal to 65 mm; (ii) the APO, bregma coefficient calculated by dividing the actual distance between the interaural plane and the bregma by the standard 23 mm taken from the Dua-Sharma's et al. atlas (14). The L and

V coordinates were not corrected. The electrodes were fixed to the bone using acrylic cement. Additionally, in three dogs a glass-insulated thermistor was placed in the posterior hypothalamus.

Self-stimulation procedure. Experimental sessions started after at least 14 days of recovery. The dog was partially restrained on a Pavlov-type stand and the electrodes were connected to a stimulator delivering a 0.2–0.5 s train of 240 Hz sine wave upon each lever press. For forced stimulation of the brain, the stimulator was activated by the experimenter. The amount of current was determined by measuring voltage drop across a resistor connected in series with the electrodes, and expressed as its base-to-peak value. The range of the latter was 0,2–0,6 mA.

In the first session the animal was stimulated successively through all electrodes in order to select sites where stimulation produced clear effects indicating excitation of the "reward" system, such as closing of the mouth, sniffing and exploratory activity. Subsequently the dog's behavior was shaped toward pressing the lever by applying stimulation of these sites as reinforcement of all manipulandum — directed movements. Once the dog learned to press the lever correctly either with his nose or foreleg or both, one or two sessions were devoted to checking the stability of the response and finding the optimal train duration and current intensity.

Temperature measurements. Temperature measurement sessions were made at ambient temperature 19–23°C and relative air humidity 40–60%. A thermosensive probe of a germanium thermometer was placed in the rectum at the depth of 15 cm from the sphincter ani externus. The hypothalamic thermistor was connected to a thermistor bridge. Both rectal (T_{re}) and hypothalamic (T_{hy}) temperatures were registered on a millivoltmeter-recorder. At the end of each session the record from the thermistor was calibrated against a decade resistor, and the resistance values were translated to degrees centigrade. A powdered carbon transducer was fastened around the animal's chest for transforming respiratory movements to voltage oscillations which were recorded on a polygraph. Electromyograms from the gluteal muscle were registered in some sessions.

After about 15 min of control recording the animal was allowed to press a lever for 30 min using the same current parameters as those found optimal for self-stimulation during the training procedure. This was followed by a 30 min period of rest during which the lever was withdrawn. Usually, in several sessions stimulation through each active electrode was applied.

Checking of the dog's response to food. The dog's response to food was tested in the same way as previously described (35). Three forms of negative reaction to food were distinguished: ignoring food, food rejection and food ejection. Ignoring food consisted of a dog, which had been deprived of food for 20 h preceding the session, performing self-stimulation in the presence of readily available meat. Food rejection was defined as the animal's failure to take meat offered together with forced (experimenter–induced) stimulation of the rewarding site. The response was defined as food ejection if the dog threw out a piece of meat he had taken just prior to the onset of the stimulus. Strong negative reaction to food was characterized by the occurrence and persistence of all of the above described forms. With ignoring food unstable, food ejection lacking and food rejection present, the negative reaction was said to be weak.

Histology. After completion of the experiment the brains were subjected to routine histological procedures, which involved embedding them in celloidin, cutting on a microtome and staining the sections according to Weil.

RESULTS

Hyperthermia and thermoregulatory responses during self-stimulation. Temperature measurements were made in 10 dogs, self-stimulating through electrodes out of the total of 40 implanted in all the animals. T_{re} increased during self-stimulation in most of the points, and this rise (ΔT_{re} usually reached its maximum value between the 20th and the 25th min of self-stimulation. ΔT_{re} was positively correlated with the self-stimulation rate ($r = +0.54$, $P < 0.001$). The regression line is shown in Fig. 1.

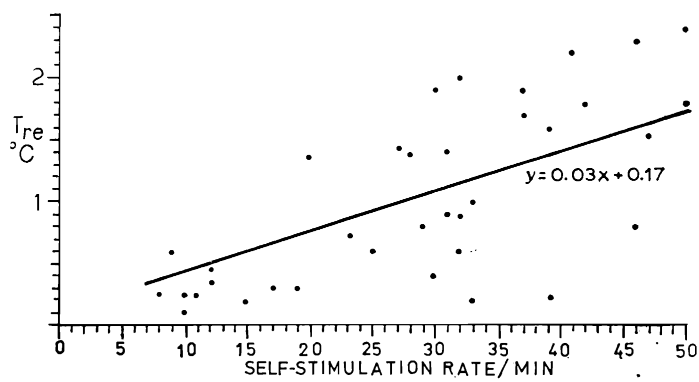

Fig. 1. Maximum increase in rectal temperature ($\triangle T_{re}$) as a function of self-stimulation rate. Each point represents results obtained during one session. Regression line is plotted on the basis of the regression equation.

Self-stimulation was always accompanied by conspicuous changes in respiration. During performance of lever pressing bouts the dog closed its mouth, the respiratory movements were slowed and flattened, and sniffing was superimposed on them. At a certain level of hyperthermia panting developed during intervals between bouts of pressing and consisted of rapid regular respiratory movements with the mouth open. In dogs showing the highest hyperthermia, shivering accompanied each bout and subsided during intervals. T_{hy} rose simultaneously with T_{re}, but reached a lower maximum level and thereafter showed oscillations with waxing phases coinciding with bouts of lever pressing and waning phases with intervals and panting (Fig. 2).

Negative reactions to food. In order to study negative reactions to food, eight dogs were selected which ate voraciously under the experimental conditions. These animals self-stimulated through 16 electrodes.

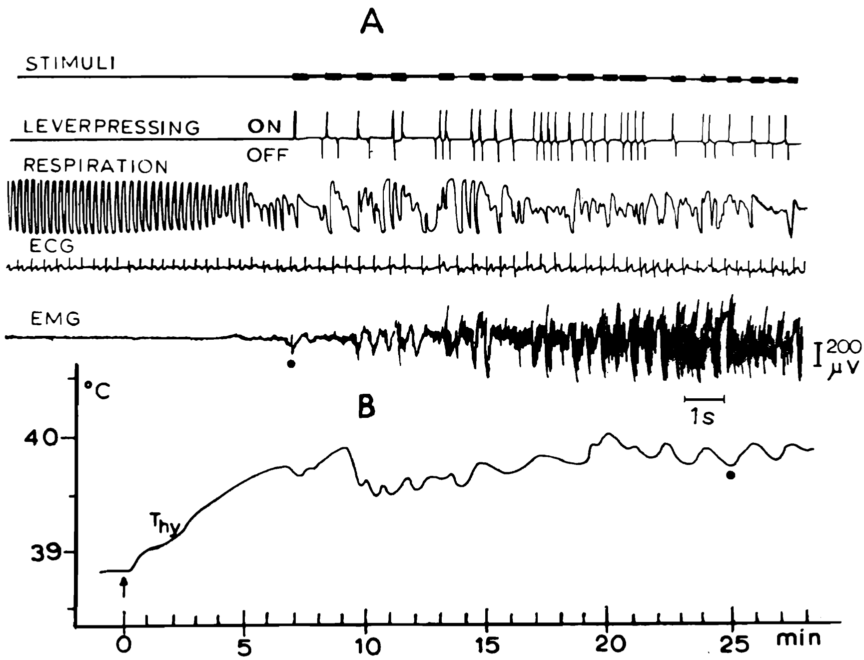

Fig. 2. Changes in respiration, electrocardiogram (ECG), electromyographic activity (EMG) and hypothalamic temperature (T_{hy}) during self-stimulation producing a high-level hyperthermia. *A*, sample of a record showing transition from interval to bout of lever pressing. Each press on the lever (ON) triggers a train of pulses (stimuli). Releasing the lever (OFF) is not effective. Note inhibition of panting immediately before and during the bout, and shivering (inrease in EMG activity recorded from the gluteal muscle) in the course of lever pressing. *B*, increase in T_{hy} after the onset of self-stimulation marked by an arrow and its oscillations during the performance. Black circles mark the same moment of recording.

Strong negative reactions were revealed in 8 sites, weak one — in 4. Self-stimulation and forced stimulation in the other 4 sites failed to affect the animals normal response to food.

TABLE I

Coincidence of negativism to food and hyperthermia produced by self-stimulation. Each mark (x) represents data obtained from one rewarding site. The data were collected by stimulating 16 sites in eight dogs

Negative reaction to food	Mean increase in rectal temperature (°C)												
	0.0	0.25	0.35	0.4	0.5	0.6	0.7	0.8	1.4	1.7	1.8	1.9	2.1
strong						x	x	x		x x	x	x	x
weak	x			x					x	x			
none		x x	x		x								

Comparison of negative reaction to food	Whitney–Mann statistics (two-tailed)
strong vs. weak	$U = 7$ ($n_1 = 4, n_2 = 8$); $P = 0.144$ (nonsignificant)
strong vs. none	$U = 0$ ($n_1 = 4, n_2 = 8$); $P = 0.004$ (significant)
weak vs. none	$U = 5$ ($n_1 = n_2 = 4$); $P = 0.868$ (nonsignificant)

Table I shows the coincidence of the negative reactions to food with the rise in T_{re}. Self-stimulation in sites yielding a strong negative reaction produced a significantly higher ΔT_{re} as compared to those where stimulation did not inhibit food intake. Weak negative reactions coincided with a variable level of hyperthermia.

Histology. Histology, available for 16 active electrodes in nine dogs, shows that self-stimulation points were distributed within the preoptic area, ventral part of the septum and the anterior hypothalamus (Fig. 3). In a previous study this area was shown to be the most rewarding (34, 36).

DISCUSSION

The principal point of this study is the finding of a coincidence of a strong negative reaction to food and a high level of hyperthermia produced by self-stimulation and/or forced stimulation in the same forebrain areas using the same current parameters. A question arises as to whether the two phenomena are mutually related and are due to the action of one common mechanism, or whether their coincidence is, instead, unrelated, resulting from simultaneous activation of independent neural circuits regulating food intake and body temperature.

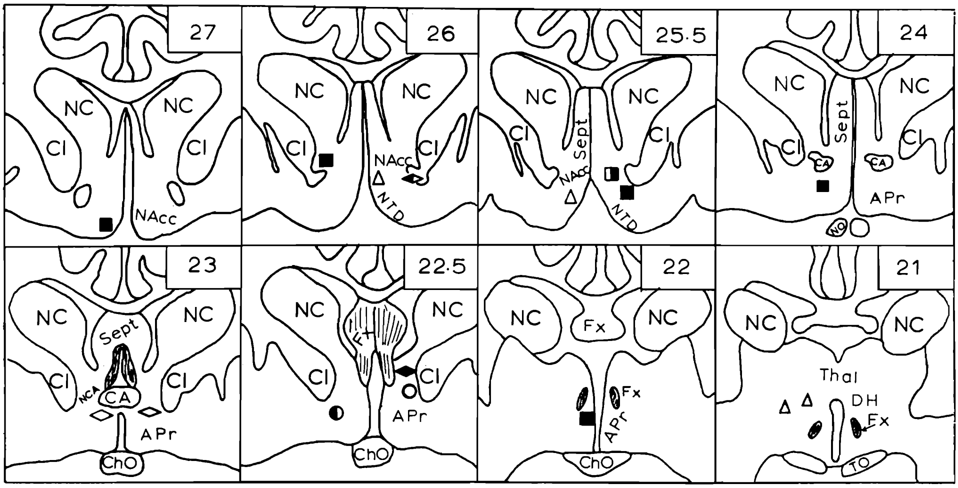

Fig. 3. Frontal sections through the dog's brain. The A coordinates according to Dua--Sharma et al. (10) appear in upper right corners. Each character denotes position of the tip of one electrode sustaining self-stimulation. The level of hyperthermia (mean increase in rectal temperature, ΔT_{re}) produced by self-stimulation in these sites is indicated by the color of the characters: black, $\Delta T_{re} \geqslant 1.5°C$; black-white, $1.5 > \Delta T_{re} \geqslant 1.0°C$; white-black, $1.0 > \Delta T_{re} \geqslant 0.5°C$; white, $\Delta T_{re} < 0.5°C$. Sites where self-stimulation and/or forced stimulation produced strong negative reaction to food are indicated by squares, weak one — by rhomboids, no negative reaction — by triangles. With sites denoted by circles the dog's response to food was not tested. Abbreviations: APr, preoptic area; CA, anterior commissure; ChO, optic chiasma; CI, internal capsule; DH, dorsal hypothalamus; Fx, fornix; NAcc, nucleus accumbens; NC, caudate nucleus; NCA, nucleus commissurae anterioris; NTD, nucleus taeniae diagonalis; Sept, septum; Thal, thalamus; TO, optic tract.

Several years after the discovery of self-stimulation it became obvious that it was due to excitation of the nervous substrate controlling consummatory behaviors, particularly feeding. From the very beginning, however, a controversy arose around the crucial problem as to whether self-stimulation increases or decreases the level of hunger. Data favoring either possibility was available. Procedures known to increase food intake (food deprivation, ventromedial hypothalamic lesions, injection of insulin) were found to enhance self-stimulation whereas those suppressing hunger (satiation, VMH stimulation) produced an opposite effect (3, 6, 22). Rats were found to self-stimulate in the same hypothalamic areas where forced stimulation elicited feeding (27). Instrumental food rewarded reflexes in dogs could be successfully reinforced by electric stimulation of the lateral hypothalamus where stimulation in another situation produced stimulus-bound eating (15, 16). In opposition to these data, inhibition of food intake during self-stimulation was described in other papers, but interpretation of this effect was unequivocal. Some authors explained it in terms of a temporary satiety symptom (33, 38), whereas others were rather inclined to consider it as due to action of another drive concurrent with hunger (40, 42).

The whole problem was recently reexamined using through control of stimulus parameters. Huston (23) and Cruce and Coons (12) demonstrated that eating produced by forced electric stimulation of the lateral hypothalamus is elicited at lower currents as compared to those sustaining self-stimulation, but, in their turn, failing to elicit feeding. Moreover, lateral hypothalamic stimulation at eating threshold in a rat trained to self-stimulate will motivate the animal to self-stimulate rather than to eat (24). Ball (4) showed that self-stimulation may be dissociated from stimulus-bound eating by manipulating with train durations.

The above data permit the conclusion that it is not the increase of hunger which reinforces the animal's lever pressing for electrical stimulation of the brain. On the contrary, as demonstrated in the present study, each stimulus at an intensity sustaining self-stimulation produced a short-term decrease of hunger in most of the dogs. Thus, the rewarding action of electrically elicited "satiety" in self-stimulation may be similar to that of a temporary reduction of hunger occurring intermittently during eating and contingent with each presence of food in the mouth. This last effect, when preceded by a particular goal-directed movement, is believed to play a crucial role in instrumental alimentary conditioning (25).

Since loci where self-stimulation and forced stimulation produce a negative reaction to food are widely dispersed in the basal forebrain, and are also remote from the classical satiety center in the ventromedial hypothalamus, one must conceive of the existence of a widespread neural system regulating short-term satiety. This conclusion may be easily reconciled with the present views on the organization of the alimentary system. According to Panksepp (30, 31) the ventromedial hypothalamus is involved in the control of long-term effects of feeding whereas its role in short-term regulation is questionable. Robinson and Mishkin (32) found that inhibition of feeding in monkeys was produced by stimulating loci widely distributed throughout the basal forebrain. Ejection of food was prominent during stimulation in some sites, and the authors considered it as the most reliable symptom of satiety.

The present result did not confirm Crawshaw and Carlisle's (11) statement that the self-stimulation rate and the increase in body temperature are variables independent of each other. Such was the case in only some particular sessions when a low lever pressing rate produced a high hyperthermia, and in spite of a high rate of self-stimulation ΔT_{re} was relatively low (see Fig. 1). In most cases, however, hyperthermia was positively correlated with the rate of responding which may indicate a relationship between the level of motivation and the activity of the central thermoregulatory mechanism. It is worth noting that even the highest hyperthermia, with ΔT_{re} close to or exceeding 2°C failed to inhibit the response, rather, the animals adopted a strategy which permitted them to self-stimulate while maintaining T_{hy} at fairly constant elevated level. This was achieved by a particular arrangement of bouts of lever pressing during which the hypothalamic temperature increased, and intervals when panting developed and T_{hy} fell to the pre-bout level.

Many, though not all, point where self-stimulation produced hyperthermia were localized within the area of maximum agglomeration of thermosenstivie units (21) and considered as the heat loss center. In contrast to that which might have been expected, electrical stimulation in this region activated not the heat-loss but the heat-gain mechanisms. This finding enables the claim that self-stimulation leads to excitation of some neural system involved in the control of thermoregulation but different from the thermosensitive neurons. Speaking in terms of Hammel's set-point hypothesis (19, 20) hyperthermia during self-stimulation may be regarded as due to the elevation of the set temperature for the animal's thermoregulatory responses. This is the reason for the setting of the hypothalamic temperature at a higher level and for adjustment of the animal's performance so as to prevent further overheating of the brain.

It is worth stressing that the thermogenic effects of self-stimulation and feeding are very similar. Ingestion of food was found to produce an elevation of body temperature (1, 5, 18) whereas food deprived animals under some experimental conditions become hypothermic (39). On the basis of the relationship between feeding and thermoregulation, Brobeck (8) proposed his thermostatic theory of food intake control according to which and increase in body temperature due to the specific dynamic action (SDA) of food inhibits the hunger center. However, internal body temperature was found to rise in the course of eating itself before any SDA action can develop, and also when the caloric content of food was low (1). One is tempted to assume that this postingestional increase in body temperature is due to an elevation of the set-point of thermoregulation by the action of signals arising soon after the consumption of food. As recently suggested, the postingestional cues, together with signals coming from tissue depôts, participate also in a long-term regulation of caloric intake and body weight, and the feeding-produced elevation of the set-point of thermoregulation is considered as an important factor in this mechanism (9).

The „satiety" produced by self-stimulation is probably much stronger than the short-term satiety occurring during normal eating, because there is good reason for believing that the action of the current not only mimics oropharyngeal but also humoral and even metabolic effects of feeding. If, simultaneously with successive satiety-like states, self-stimulation produces elevation of the set-point for temperature regulation, this may be due to a complex activation of central processes controlling energy homeostasis. In other words, during self-stimulation a condition is created not only for stopping the animal from eating, but also favoring energy expenditure. This last conclusion is additionally supported by endocrinological studies showing enough of an increase in blood catecholamines and corticosteroid synthesis during self-stimulation to influence metabolic processes in the tissues (28, 29, 37, 41).

The author thanks very much Mrs Wanda Radziszewska for histological work. This investigation was supported by Project 09.4.1 of the Polish Academy of Sciences.

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Received 7 April 1975

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