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EFFECTS OF LATERAL HYPOTHALAMIC LESIONS ON PLACENTOPHAGIA IN VIRGIN, PRIMIPAROUS, AND MULTIPAROUS RATS[1]

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Lesions of the lateral hypothalamus (LH) were produced in pregnant and nonpregnant female rats through chronically implanted electrodes to investigate the effect of LH damage on placentophagia. Other variables investigated were prior parturitional experience and stimulus properties of the placenta. Lesions were produced under ether anesthesia 24 hr. prior to parturition in pregnant females and 24 hr. prior to placenta presentation in nonpregnant females. The LH lesions produced aphagia to a liquid diet. Pregnancy was not a significant variable in the initiation of placentophagia, but prior parturitional experience was a critical variable. Virgin and primiparous females did not exhibit placentophagia following LH damage, but multiparous females would eat placenta whenever the opportunity arose, independently of LH damage and pregnancy.

The lateral hypothalamus (LH) is known to play an integral role in the initiation of homeostatic ingestive behaviors and to be involved in the selection of foods and fluids for stimulus qualities, i.e., finickiness occurs after the production of LH lesions in rats (Teitelbaum & Epstein, 1962). One infrequent ingestive behavior in females of most mammalian species—placentophagia—represents a major predictable departure from normal feeding and drinking. Placentophagia refers to the eating of the placenta during parturition. Since placentas are accessible to the female rat only under extremely unusual behavioral and endocrine circumstances (parturition), it is not known whether placentophagia is under the same hypothalamic control as homeostatic feeding. The relationship between placentophagia and what is considered to be normal homeostatic feeding has not been explored.

Several studies have been conducted demonstrating the importance of placentophagia in rats to subsequent foster care of the pups (Grota, 1968; Grota, Denenberg, & Zarrow, 1967), but no studies have been designed exclusively to test the mechanisms involved in the initiation of placentophagia.

Avar and Monos (1966, 1967, 1969) investigated the effects of LH lesions during pregnancy on maternal behavior and offspring survival in rats. In all three studies, electrolytic lesions were produced through acutely inserted electrodes on Days 16–18 of pregnancy in females having no prior parturitional experience. The high offspring mortality rate (30% within 3 hr., > 80% within 48 hr.) was attributed to a disruption of the normal pattern of postnatal maternal behaviors. Nursing, nest building, and retrieving behaviors were absent in LH-lesioned mothers, and pups from intact mothers fostered to LH-lesioned mothers fared as poorly as pups born to LH-lesioned mothers (Avar & Monos, 1967). Avar and Monos also mentioned that placentas as well as pups showed evidence of being gnawed. According to the food- and water-intake data presented by Avar and Monos (1966), their LH-lesioned pregnant females were not aphagic or adipsic at parturition.

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This could be attributed to the fact that the lesions had been produced 5 or 6 days earlier.

The goal of the present experiment was to study the role of the lateral hypothalamus in placentophagia and to investigate other variables possibly influencing the behavior, such as prior experience, the internal milieu and behavioral phenomena associated with parturition, and the stimulus properties of the placenta. In order to minimize the physiological effects of surgery on parturition, while producing lesions at a time that would yield a maximum effect on food and water intake in conjunction with the occurrence of parturition, lesions would have to be produced through chronic (indwelling) electrodes inserted prior to the mating of the females.

Preliminary observations led to the conclusion that placentophagia would occur in an experienced pregnant rat made aphagic by the introduction of bilateral LH lesions 1 day prior to parturition. In addition, it was indicated that placentophagia would occur following a day of normal food intake in the intact rat (Kristal, 1971). Thus, placentophagia could not be solely a product of hunger or food deprivation. These observations led to the hypothesis that placentophagia might not be part of homeostatic feeding regulated by the hypothalamus. If not, it was expected that placentophagia would be more affected by prior experience or by behavioral and endocrine changes at parturition (a pregnancy effect) than by destruction of the LH.

Method

Subjects

Fifty Sprague-Dawley female rats, 100–150 days of age, were used. At the outset of the experiment, one half were virgins and one half had given birth to, and nursed, one or more litters. Each female was housed individually in an 8 × 8 × 15 in. wire-mesh cage and maintained on a 7:00 a.m. – 7:00 p.m. daylight cycle.

Surgery

After normal estrous cyclicity was determined by vaginal smears, each female (except those in Group CI) underwent surgical implantation of chronic LH electrodes. Implantation was performed under Equithesin anesthesia (.3 ml/100 gm body weight, ip) preceded by .05 mg. of atropine sulfate (ip) to suppress the secretion of mucus. A precast electrode assembly consisting of a pair of Formvar-insulated .012-in. stainless-steel electrodes (with the tips clipped off) was inserted stereotaxically according to the following coordinates: 2.5 mm. posterior to bregma, ±1.8 mm. lateral to the midline sinus, and either −7.5 or −8.0 mm. from the dural surface. The skull was positioned with the frontal bones parallel to the plane of the platform of the stereotaxic apparatus. The electrode assembly was anchored with dental cement to four stainless-steel screws (0-80 × 3/16 in.) imbedded in the skull. Prior to the insertion of the electrode assembly, the hole in the skull beneath the assembly was packed with powdered Gelfoam and sprinkled lightly with crystalline sulfathiozole. The edges of the incision were also sprinkled with sulfathiozole. Following surgery, each animal received 45,000 U (im) of Bicillin (Wyeth Laboratories, Inc., Philadelphia).

Procedure

Each animal was allowed to recover for 1 wk. following surgery before the taking of vaginal smears was resumed. If the normal estrous cycle was not reestablished, the female was excluded from the experiment at this point. The remaining females were placed in the following groups prior to the introduction of lesions: (*a*) Group CI (primiparous/multiparous, pregnant, nonlesioned) —intact control group of pregnant females; (*b*) Group PPL (primiparous, pregnant, lesioned)—females with no prior parturitional experience, receiving electrode implants, bred, placed on liquid diet on Day 14 of pregnancy, and lesioned on the day prior to parturition; (*c*) Group MPL (multiparous, pregnant, lesioned)—females with prior parturitional experience, treated identically to females in Group PPL; (*d*) Group VL (virgin, lesioned)—virgin females receiving electrode implants, placed on liquid diet 4 wk. later, lesioned after 7 days on liquid diet, and presented with diced placentas 24 hr. after lesioning; and (*e*) Group MNL (multiparous, nonpregnant, lesioned) —females with prior parturitional experience treated identically to females in Group VL.

Placentophagia pretest. After recovery from surgery and resumption of estrous cycling, but before mating, six virgin and six nonpregnant multiparous rats were presented with 10 ml. of diced placentas. The placentas for the pretest and for presentation to nonpregnant females after lesioning were obtained from donor females (sacrificed by cervical dislocation on Day 21 of pregnancy) and frozen with a few drops of physiological saline in small vials. For use in testing, the vials containing placentas were placed in 37° C. water until the placentas were thawed and slightly warmer than room temperature. The placentas were then diced finely and the placentas and the fluids associated with them were immediately presented to the experimental females. It was as-

sumed that diced placentas treated in this manner were an acceptable substitute to nonpregnant females for placentas expelled at parturition. Each of the females in the placentophagia pretest was allowed exposure to the diced placentas for 15 min., during which time behavior toward the substance was noted.

Mating. Vaginal smears were taken daily to determine the course of the estrous cycle. When a female entered late proestrus, she was placed with a breeder male which had previously fathered at least one litter. Vaginal smears were then taken each morning and evening until sperm in the vagina or a vaginal plug was detected or until the female passed the estrus stage. When copulation was evident, the female was considered to be in Day 1 of pregnancy. She was then returned to her home cage.

Food intake. Pregnant females were continued on their ad-lib diet of tap water and food pellets (Purina Lab Chow) until Day 14 of pregnancy. At that time ad-lib water was continued and the food pellets were replaced by a modified form of the eggnog developed by Teitelbaum and Epstein (1962), consisting of 240 ml. of Similac (Ross), 9 gm. of dry ground baby cereal (Gerber Mixed Cereals), 50 ml. of reconstituted whole egg, 100 ml. of 50% (w/v) sucrose, and a 1:100 solution of 10% Formalin. The addition of cereal and Similac increased the palatability of the diet for the rat (Teitelbaum. 1971). The liquid diet was presented in 100-ml. calibrated glass founts (Wahmann Mfg. Co., Baltimore). The founts were cleaned and filled each morning at the onset of the daylight segment of the day–night cycle. Nonpregnant females were switched to liquid diet at approximately the same number of days following surgery as the pregnant females.

Lesion production. Lesions in the pregnant females (Groups PPL and MPL) were produced at the beginning of the daylight cycle on the day prior to expected parturition. The range of actual intervals between lesioning and parturition was 20–26 hr. In the nonpregnant females (Groups VL and MNL) lesions were produced after 6 days of continuous liquid-diet presentation. Group CI received no lesions.

Lesions were produced under light ether anesthesia. A rectal cathode was inserted and 1.5 ma. dc was passed through each of the indwelling LH electrodes for 10 sec.

Observations of placentophagia after LH damage. Pregnant females were observed as parturition occurred. To prevent the loss of pups and placentas through the floor of the wire-mesh cage, a floor of ⅛-in.-mesh hardware cloth was placed in the cage 24 hr. prior to parturition. The presence or absence of placentophagia was noted at parturition, as were other oral maternal behaviors such as licking and retrieving. Bedding was excluded from the cage, thus eliminating the opportunity for the females to build nests. It was found that nesting material interfered with the quantification of food intake. If placentophagia occurred, every other mother was then presented with three or four placenta-sized pieces of rat liver to control for the presence of meat rather than of placenta per se. Alternate mothers received pieces of ground beef as a meat control. Rat liver was chosen because liver and placenta, although quite different, perform many of the same enzymatic functions and contain many of the same biochemical constituents. In addition, liver and placenta are slightly similar in appearance. Ground beef was chosen arbitrarily, because of its dissimilarity to placenta.

Nonpregnant females were presented with 10 ml. of diced placentas 24 hr. after the production of lesions. Each female received a 1-hr. exposure to the placentas. As with the pregnant females, when placentophagia occurred, alternate females were presented with pieces of liver and beef as a meat control.

Any lesioned female (pregnant or nonpregnant) exhibiting a marked increase in food intake following the production of lesions was assumed to have damage in an area other than the LH and was removed from the experiment. The remaining lesioned females were assigned to the appropriate lesioned or lesioned-control group after histological examinations.

Histology

Three to 14 days after lesioning, the animals were injected with 1.3 ml. of Equithesin (ip) and perfused with physiological saline followed by 10% Formalin. The brains were removed, stored in acid Formalin for 1 wk., and returned to 10% Formalin. The brains were then dehydrated, imbedded in celloidin, cut coronally at 35 μ, and stained with cresylecht violet.

RESULTS

Placentophagia Pretest

All six of the multiparous females in the pretest ate diced placentas. Of the six virgins in the pretest, only one ate diced placentas. The difference was significant when tested by the Fisher exact probability test ($p = .01$).

Placentophagia following LH Damage

Histology. Without knowledge of behavioral results, histological examination of the brains of animals exhibiting a decrease in food intake following LH lesions was performed independently by two experienced observers, and their findings were compared. There was perfect agreement on what constituted good lesions in terms of size and placement. All animals without good LH lesions (as judged by histological examination) were placed in a lesioned-con-

trol group. It was found that there was a perfect correlation between rejection of an animal on the basis of histological evidence and the absence of aphagia in that animal's behavioral data, i.e., in every case an animal rejected for lack of aphagia was found to have poor lesions, and vice versa.

The effective lesions were comparable to those of Avar and Monos (1966). In most cases, almost the entire LH was ablated at the region of electrode insertion, with relatively little anterior and posterior spread of damage. All of the brains containing effective lesions had little or no damage to the internal capsule, fornix, optic tract, or ventromedial hypothalamus. Figure 1 is a diagrammatic representation of the effective lesions, in each of the four groups, at the anterior–posterior section containing the maximum damage. Figure 2 contains photomicrographs of representative effective lesions for comparison with the diagrams in Figure 1. Four additional females exhibited the effects of well-placed lesions, but their brains were not available for histological examination. Because of the high reliability of prediction of well-placed lesions from the behavioral observations, these four animals were included in the data on placentophagia following effective lesions.

Seven females showing some decrease in food intake following the production of lesions were considered to have ineffective or only partially effective lesions. Upon histological examination, it was found that the ineffective lesions were either too dorsal and/or too lateral (Subjects MPL 7, MNL 6, VL 4, and MNL 7), too small (Subjects PPL 16 and MPL 4), or unilateral (Subject MNL 9). Figure 3 is a diagrammatic representation of the ineffective lesions at the anterior–posterior region of maximum damage. One female exhibiting behavioral indices of ineffective lesions was included in this lesioned-control group, although this brain was not available for histological examination.

Aphagia. Sixteen females were found to have effective lesions, i.e., exhibited aphagia in the 24 hr. following the production of lesions. The base-level liquid-diet intake for Days 2–5 prepartum (Days 1–4 prelesion) did not differ significantly from the base-level liquid-diet intake of the intact pregnant females of Group CI ($t = .62$, $df = 20$, $p > .1$). The drop in liquid-diet intake following LH damage, however, differed significantly from base level ($t = 10.03$, $df = 15$, $p < .001$). Table 1 summarizes the data on liquid-diet intake in unlesioned and lesioned females. In the aphagic animals, 24-hr. intake dropped to zero following LH damage in only a few cases because the effect of the anesthetic lasted only a few minutes, but it took several hours for the lesions to become fully effective (P. Teitelbaum, personal communication, February 1971). Hence, animals which were completely aphagic 24 hr. after lesioning ate small amounts in the hours immediately following lesioning.

Eight females exhibited a drop in food intake but were not classified as aphagic (lesioned-control group). The base-level intake of these females did not differ significantly from that of Group CI ($t = .52$, $df = 12$, $p > .1$). The decrease in food intake manifested by nonaphagic females following the production of lesions was significant ($t = 3.55$, $df = 7$, $p < .01$). Although it would appear that these animals were hypophagic, it should be noted that the difference between this group (lesioned-control group) and the intact control (Group CI) was that on the last day of pregnancy, the intakes of all females in Group CI were included in the group data; in the lesioned groups, females showing an increase in intake following lesioning were excluded from the experiment. Although for this reason the postlesion intake level in the lesioned-control group was significantly different from base level for that group, the postlesion intake of the lesioned-control group was still not significantly different from the intake on the last day of pregnancy for Group CI ($t = .65, df = 12, p > .1$).

Placentophagia. Table 2 summarizes the group data on placentophagia. When pregnant aphagic rats (Groups PPL and MPL) were compared with nonpregnant aphagic rats (Groups VL and MNL) for existence of placentophagia by means of a Fisher exact probability test, it was found that pregnancy per se was not a significant fac-

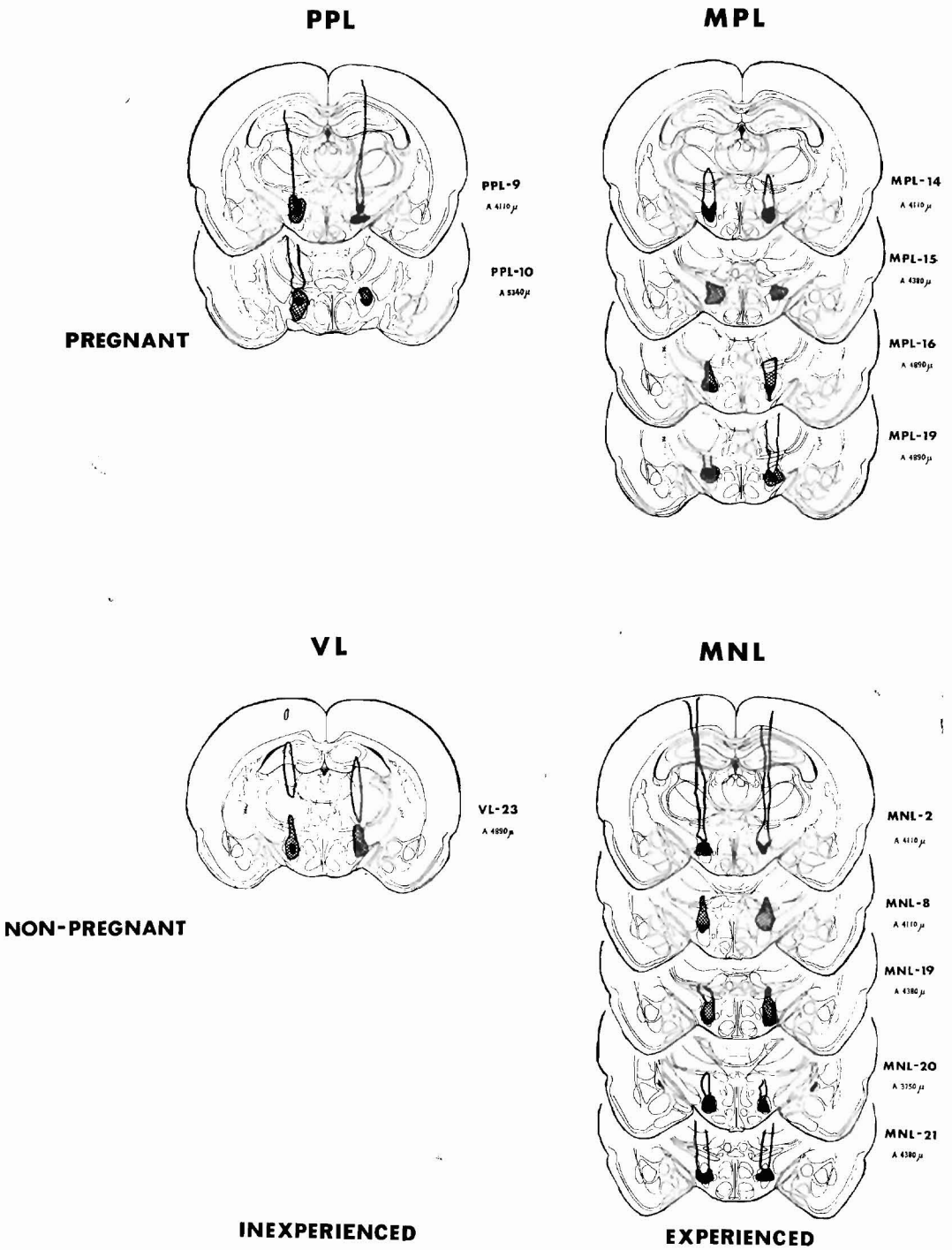

FIG. 1. The effective lesions at the anterior–posterior section of maximum damage. (Diagrams are taken from König & Klippel, 1963. Black area represents encapsulation; cross-hatching indicates gliosis.)

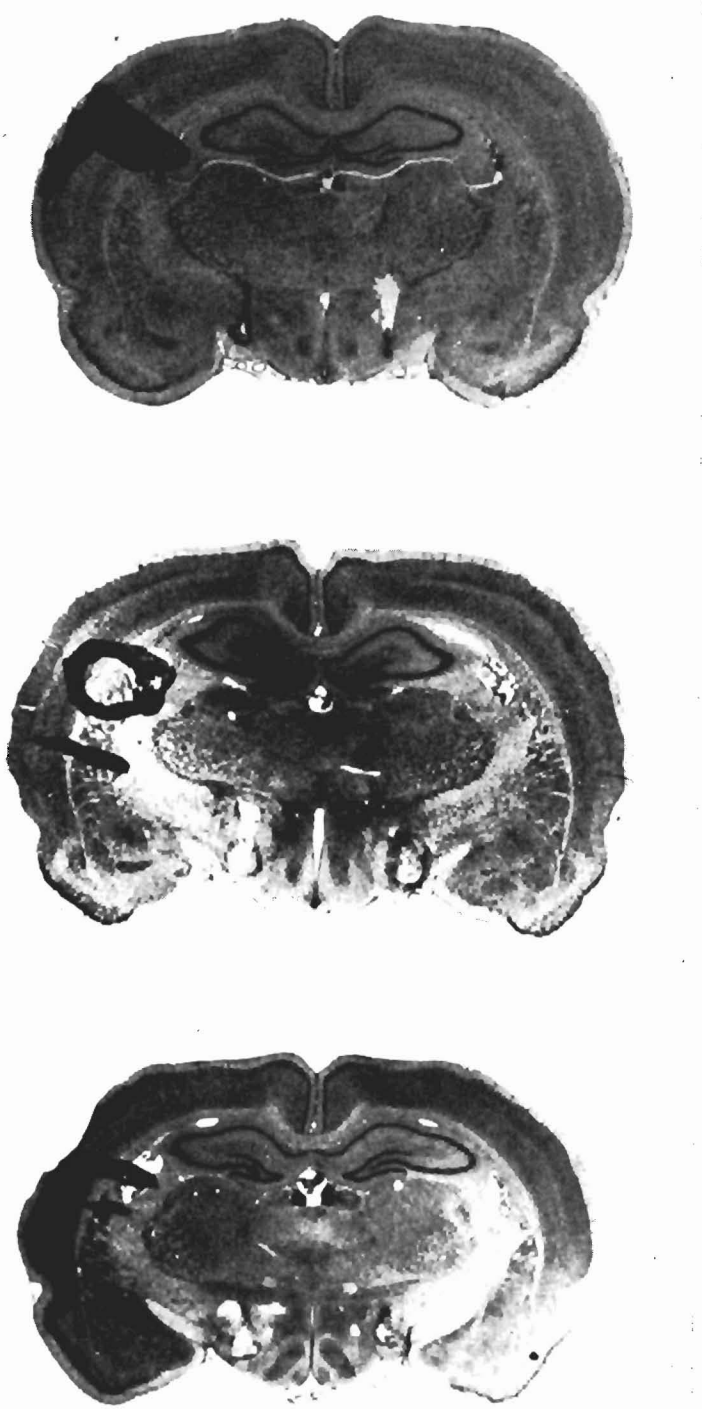

FIG. 2. Photomicrographs of three representative effective lesions at the anterior–posterior section of maximum damage. (Markings: 7 = Subject MPL 16, 10 = Subject MPL 19, 11 = Subject MNL 21.

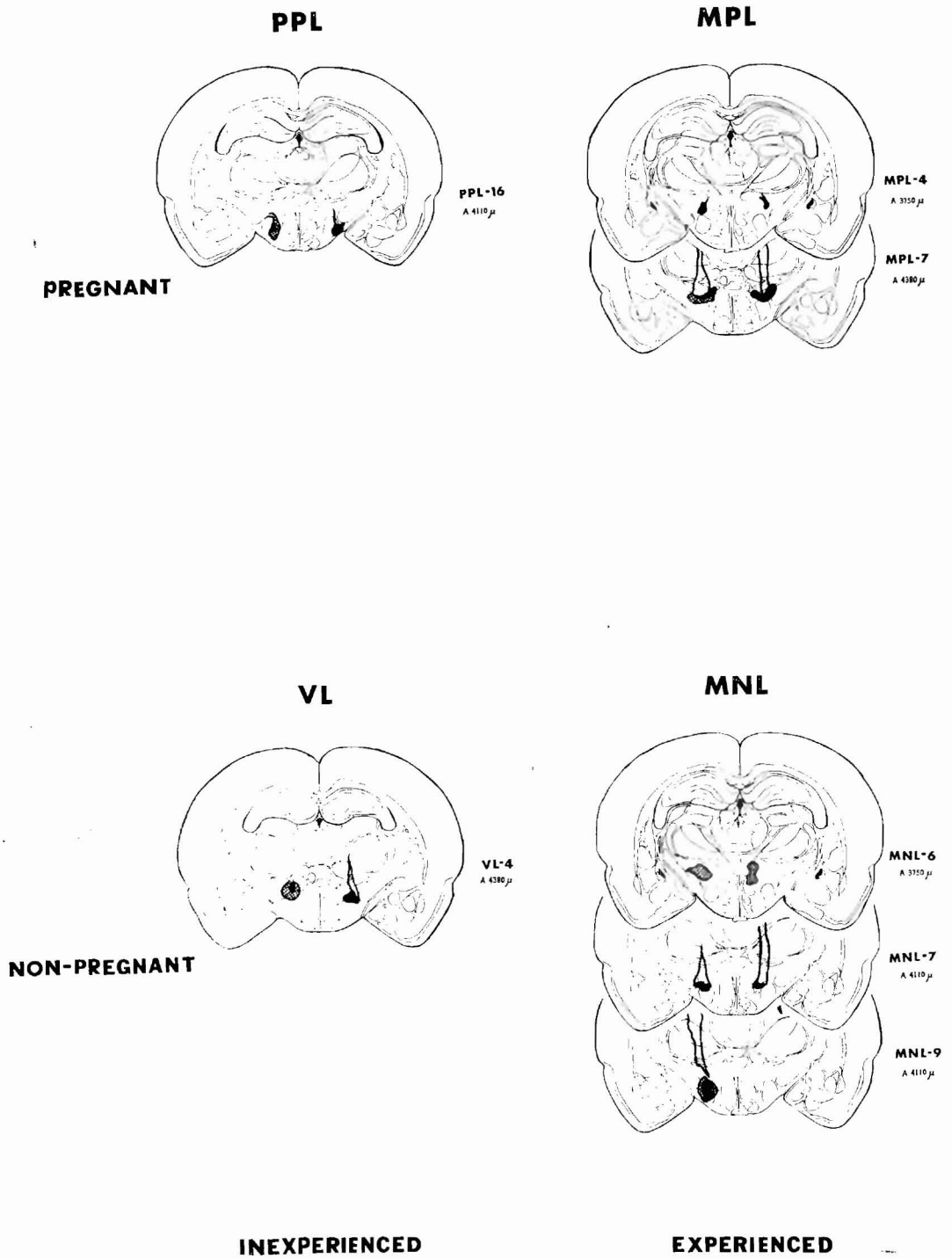

FIG. 3. The ineffective lesions at the anterior–posterior section of maximum damage. (Diagrams are taken from König & Klippel, 1963. Black area represents encapsulation; cross-hatching represents gliosis.)

TABLE 1

LIQUID-DIET INTAKE IN INTACT, LH-LESIONED, AND LESIONED-CONTROL GROUPS OF RATS

Group	*n*	Base level (*M* in ml., Days 2-5)	*SE*	*M* intake (in ml., last day)[a]	*SE*	% change
Intact (Group CI)	5	72.6	4.22	59.2	9.58	−18.4
LH lesioned	16	67.5	7.09	7.0	2.48	−90.0**
Lesioned control	8	68.3	6.49	51.6	7.94	−24.0*

[a] For pregnant females, last day prior to birth; for nonpregnant females, last day prior to placenta test.

* $p < .01$, two-tailed test.

** $p < .001$, two-tailed test.

tor in the initiation of placentophagia ($p = .34$).

When aphagic females with prior parturition experience (Groups MPL and MNL) were compared with females having no prior experience (Groups VL and PPL) by means of a Fisher exact probability test, it was found that prior experience was a major factor in the initiation of placentophagia ($p < .005$).

The eight females not showing aphagia were treated as a lesioned-control group (see Table 2). When pregnant and nonpregnant females within the lesioned-control group were compared in regard to placentophagia by means of a Fisher exact probability test, no significant effect of pregnancy was found ($p = .50$). When experienced and inexperienced females were compared, no significant effect of experience was found ($p = .25$), but this was attributed to the fact that the inexperienced group consisted of only two females, one of which exhibited placentophagia.

Meat control. Of the nine aphagic females showing placentophagia, only two ate liver following placenta. When tested by the Fisher exact probability test, the difference between the frequency of occurrence of placentophagia and of liver eating was significant ($p < .005$). None of the aphagic females ate beef. When compared to the frequency of occurrence of placentophagia, this difference was also significant ($p < .005$).

Other Observations

Maternal care of the young. The primiparous females showing aphagia following LH damage did not exhibit placentophagia (see Table 2). No pups in any of these litters survived. Of the multiparous aphagic pregnant females (all showing placentophagia), two had litters which survived for 14 days, at which time they were sacrified with the mother. The MPL females whose litters did not survive were never seen licking or retrieving their young. Typically, the pups were scattered over the floor of the cage and the mother ignored them.

DISCUSSION

It was demonstrated by Kristal (1971) that in the rat, food deprivation is not a factor in the initiation of placentophagia, since food intake does not decrease significantly on the last day prior to parturition. In regard to other factors involved in the initiation of placentophagia, it was demonstrated in the present experiment that prior parturitional experience, but not concomitant pregnancy, was a key variable. During the first parturition the LH is important: Placentophagia can be eliminated at this time by destroying the LH (Group PPL). During the first parturition the birth experience (endocrine changes and/or a sequence of behavior events) and, possibly, stimulus properties of the placenta act

TABLE 2

INCIDENCE OF PLACENTOPHAGIA IN INTACT, LESIONED, AND LESIONED-CONTROL FEMALE RATS

Group	*n*	No. exhibiting placentophagia
Intact		
CI	5	5
Lesioned (aphagic)		
PPL	4	0
MPL	4	4
VL	3	0
MNL	5	5
Lesioned control (non-aphagic)		
PPL	1	1
MPL	3	3
VL	1	0
MNL	3	3

somehow to alter the control of placentophagia in such a manner that the female will subsequently eat placenta whenever she is exposed to it (multiparous females in the placenta pretest). This predictable placentophagia following the experience of only one parturition becomes independent of the state of pregnancy (Group MNL) and independent of LH control (Groups MNL and MPL).

The effectiveness of prior parturitional experience in removing certain aspects of maternal behavior from physiological (particularly, hormonal) control has been previously demonstrated in rats by Moltz and his coworkers. Although removal of pups by caesarian section from primiparous and multiparous mothers did not effect differentially the acceptance of foster young by these same mothers (Moltz, Robbins, & Parks, 1966), removal of the ovaries at the time of caesarian section eliminated effective maternal behavior to foster pups in 50% of the primiparous females. The multiparous females were virtually unaffected by the ovariectomy (Moltz & Wiener, 1966). More recently, injections of progesterone administered over the last few days of pregnancy have been shown to interfere with the initiation of effective maternal behavior toward foster pups by primiparous, but not multiparous, female rats (Moltz, Levin, & Leon, 1969). Clearly, the mechanisms or processes involved in the initiation and regulation of maternal behavior during the first parturition are not those operating during subsequent parturitions.

The removal of LH control over placentophagia after the first parturitional experience may be due to the corticalization of a previously diencephalic behavior. Teitelbaum and Cytawa (1965), Cytawa and Teitelbaum (1967), and Balinská, Burešová, and Fifková (1967) have demonstrated that particular behaviors, including feeding and drinking, come under control of the cortex in recovery from damage to certain hypothalamic and limbic structures. Teitelbaum has also demonstrated (Teitelbaum, 1971; Teitelbaum, Cheng, & Rozin, 1969) that recovery from LH damage reproduces the sequence of behavioral events existing during ontogenetic development of feeding behavior and that recovery of feeding is produced by the same types of changes in neural organization (reencephalization) that occur in infancy (encephalization). It is possible that loss of LH control of placentophagia following the first parturition represents a continuation of the functional reorganization between the cortex and LH (encephalization) that was begun in infancy.

There are indications in the present experiment that LH control of placentophagia may be independent of control over other oral maternal behaviors such as licking and retrieving. Two of the four aphagic placentophagic multiparous females (Group MPL) did not manifest licking or retrieving. Obviously these results are inconclusive. The design of the present experiment also did not allow for testing the possibility that nest building is a necessary prerequisite to subsequent oral maternal behaviors. It was not possible to eliminate the possibility that nest building would have prevented the failure to manifest placentophagia after LH lesions in the primiparous females (Group PPL) by providing a priming effect for subsequent maternal behavior.

The method employed in the present experiment of producing lesions through chronic indwelling electrodes proved to be a satisfactory method of eliminating the physiological trauma produced by introducing lesions at the time of stereotaxic insertion of the electrodes (acute preparation). Furthermore, the lesions produced by permanent indwelling electrodes did not produce the marked behavioral changes observed after lesions produced by acutely inserted electrodes. The females in the present study did not exhibit the cringing, squealing, sloppiness, and somnolence usually observed after LH lesions in an acute preparation. It should be noted that intracranial self-stimulation—a technique frequently used to check the placement of permanently implanted LH electrodes prior to lesioning (Margules & Olds, 1962)—could not be used in the present experiment. It was found that following intracranial stimulation in the LH, the estrous cycle of the female was severely disrupted in that vaginal smears showed a condition of constant dies-

trus for at least several weeks. This is consonant with a report by Lieblich and Olds (1971) in which they state that females receiving intracranial stimulation would not breed subsequently.

In summary, it was demonstrated in the present experiment that placentophagia is under the control of the LH only during the first parturition. Following the first parturition, placentophagia will occur whenever placenta is available, independently of the state of pregnancy or of the normal function of the LH.

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