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Neocortical and Hippocampal EEG in Normal and Lateral Hypothalamic-damaged Rats

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DE RYCK, M. AND P. TEITELBAUM. *Neocortical and hippocampal EEG in normal and lateral hypothalamic-damaged rats.* PHYSIOL. BEHAV. 20(4) 403–409, 1978. – Cortical and hippocampal EEG were correlated with behavior in rats before and after bilateral lateral hypothalamic (LH) damage. In Stage 1 of recovery (aphagia and adipsia), the neocortex showed continuous large amplitude slow activity. It did not desynchronize during spontaneous acts such as grooming as well as during tail pinch-induced struggling or orienting, even though a slow form of hippocampal theta accompanied these acts. However, during Stage 2 (anorexia), the neocortical EEG did desynchronize when such theta appeared. Therefore, as behavioral recovery progresses after LH damage, there appears to be a concomitant recovery of cortical participation in such behavior. Early in recovery, LH rats, unlike normals, showed slow (3–4.5 Hz) atropine-sensitive hippocampal theta during automatisms such as grooming as well as during immobility. Thus, LH damage, while temporarily abolishing fast, noncholinergic theta, appears to release slow cholinergic theta. Later in recovery, faster atropine-resistant (noncholinergic) theta becomes functional again.

Hippocampus EEG Lateral hypothalamic lesions Recovery

SEVERE bilateral lateral hypothalamic damage causes rats, cats, and other mammals to stop eating and drinking [1]. Unless maintained by tube feeding, such animals will die of inanition [37]. They eventually recover, regaining partial regulatory control of feeding and drinking in a series of stages which constitute the lateral hypothalamic syndrome ([36]; for reviews see [8,32]). Because electrical or chemical stimulation of the lateral hypothalamus elicits not only ingestion [5,10], but also barpressing for food and water [7,11], much attention has focused on the deficits in motivation and regulation of food and water intake and body weight, particularly as evident in the later stages of recovery [9, 25, 31, 33].

However, in Stage 1 (aphagia and adipsia) and in Stage 2 (anorexia and adipsia), the exact nature of the deficit is still poorly understood. This is so, in part, because other behavioral deficits, long considered as side effects, and therefore irrelevant, are prominent during these early stages. For instance, in Stage 1, somnolence, akinesia, catalepsy, and sensory neglect may be present, in addition to aphagia and adipsia. However, some of the variables involved in these side effects also control normal ingestion and regulation [2, 20, 22, 23, 29, 46]. Therefore, much can be learned about motivation and regulation, both normal and abnormal, by studying these apparently irrelevant early symptoms of lateral hypothalamic damage.

The aberrant behavior of an animal with large lateral hypothalamic lesions reflects the correspondingly disrupted activity of its nervous system. An alternate view of its abnormality, and of its course of recovery, should therefore be revealed in its electroencephalogram (EEG). For instance, when somnolence is present, the cortical EEG should show large amplitude slow activity (synchronization). In normal rats, changes in behavior are closely correlated with changes in the electrical activity of the hippocampus [39, 40, 45]. For example, when an animal walks, runs, or swims, rhythmical slow activity (RSA) appears. This EEG rhythm is also known as theta (a near-sinusoidal wave varying in frequency from about 6 to 12 Hz). However, when such an act terminates and the animal pauses in alert immobility, or engages in acts believed to be more automatic (such as grooming, scratching, licking, etc.), theta disappears, and the hippocampal EEG pattern becomes one of large amplitude irregular activity (LIA) [40, 42, 43, 44].

The behavior of lateral hypothalamic rats in the early stages of recovery from large lesions reveals an analogous dissociation between voluntary acts (self-initiated or self-guided patterns, such as those involved in orientation or walking), and more simple stereotyped automatisms [20]. Such rats do not spontaneously walk, rear, swim, or even orient to stimuli. However, it is quite striking to see such an animal, in the course of its maintained semi-stuporous somnolence and catalepsy, suddenly engage in a rather complex bout of grooming. Other reflex automatisms such as chewing, teeth chattering, scratching, and head and body shakes also may interrupt their persistent immobility. Robinson and Whishaw [26] have earlier shown a similar dissociation in posterior hypothalamic-damaged rats. In both preparations, stereotyped reflexive behavioral auto-

matisms remain, although more complex self-initiated exploratory locomotion and orienting are absent.

However, as locomotion and orienting reappear, the animal begins once again to walk over to food and eat it [20,21]. Eventually, in the later stages, it will even perform an operant act to obtain food [15,36], or water [28]. It has been suggested [35] that the stages of recovery of motivated eating and drinking reflect stages of "re-encephalization" of function, from a decerebrate-like level to a more normal one. During such recovery, therefore, hippocampal and neocortical EEG patterns ought to reflect the varying levels of function. For instance, normal hippocampal theta, reflecting a higher level of nervous control [39,40], should be absent during the early stages of recovery. As normal behavior reappears, so too should normal hippocampal theta.

METHOD

Animals and Surgical Procedure

We implanted 15 male Long-Evans hooded rats with bipolar recording electrodes bilaterally in the dorsal hippocampus. Each electrode-pair consisted of two parallel, Formvar-coated, stainless steel wires of 250 μm. The parallel electrode wires were either in apposition or separated by 1.0 mm in the sagittal plane. The uninsulated electrode tips were separated by 1.0 mm in the vertical plane. The electrode pair was positioned so that it would straddle the pyramidal layer of the CA1 subfield of the dorsal hippocampus. Its optimal location was determined by means of acute recordings during implantation under chloral hydrate anesthesia (600 mg/kg) or under Equithesin anesthesia (0.25 cc/100 g body weight). In this condition, tail pinch elicited a train of hippocampal theta. The stereotaxic coordinates of the deep electrode tip (with the skull level between lambda and bregma) were 4.0 mm posterior to bregma, 2.5 mm lateral to the exposed midline sinus and 3.0 mm ventral to dura of the cortex. In addition, 2 pairs of surface-to-surface electrodes, consisting of 00 by 1/8 in. stainless steel miniature screws were implanted bilaterally over the parietal and occipital neocortex. Five rats also received chronically implanted monopolar electrodes in the lateral hypothalamus. These electrodes were constructed from Formvar-coated stainless steel insect pins (Size 0) with bare tips of 0.5 mm length. The stereotaxic coordinates (leveled skull) were: 2.5 mm posterior to bregma, 2.0 mm lateral to the midline sinus, and 8.0 mm ventral to the dura of the cortex.

EEG Recording

Behavioral observations on freely moving intact rats were begun after a two week recovery period. Concomitant EEG recordings were made with a Grass polygraph (either a Model 5 or 7, using half-amplitude points of 0.3, 1 or 1.5 Hz and 15, 30 or 60 Hz). After several weeks of testing, 5 rats were subjected to bilateral lesions in the lateral hypothalamus by passing an anodal current of 1 ma for 30 sec through each of the chronic lateral hypothalamic electrodes and a rectal reference probe.

Histology

At the end of the experiments, each animal was perfused through the heart with isotonic saline followed by 10% Formalin under an overdose of sodium pentobarbital. The brains were removed and after additional fixation in Formalin, brain sections of 40 μm thick were cut by means of a frozen tissue technique. The sections were stained with cresyl violet for cell bodies.

RESULTS AND DISCUSSION

Histology

The tracks of the hippocampal electrode wires were all found at the level of the CA1 subfield of the dorsal hippocampus. The deep electrode tips typically ended in the dorsal or ventral blade of the dentate gyrus at levels A3990 μ to A3290 μ of König and Klippel [18]. Eight of the deep electrode tips were placed in the hilus of the dentate gyrus. In accordance with Vanderwolf *et al.* [43], we found that these sites produced theta bouts mixed with fast activity. However, none of these hilus electrode sites belonged to the lesioned rats. The dorsal electrode tips of the hippocampal electrode pair were found in stratum oriens of CA1 or in deeper layers of the corpus callosum tangential to it between levels of A2970 μ and A2790 μ.

The lateral hypothalamic lesions were centered at the level of the medial to posterior parts of the ventromedial nucleus of the hypothalamus. The anterior-posterior axis of the lesions extended from the anterior part of the ventromedial nucleus (A5150 μ) to the level of the posterior hypothalamic nucleus (A3180 μ). Medially, the lesions invaded the medial forebrain bundle and laterally they extended to the medial part of the capsula interna. Dorsally, the lesions destroyed in varying degrees the zona incerta, including posteriorly the fields H1 and H2 of Forel. Ventrolaterally, the damage bordered but did not include the optic tract.

EEG and Behavioral Observations in Normal Rats

In normal rats with implanted electrodes, we have replicated Vanderwolf's [39, 40, 41] original findings. Hippocampal theta always occurred during sniffing, walking, running, or rearing. Hippocampal LIA always appeared during scratching of the trunk, head, or neck with the hind leg, licking, gnawing, and teeth-chattering, as well as during alert immobility (standing still, with eyes open). Figure 1A provides an example of cortical and hippocampal activity accompanying locomotion (walking forward with all four legs) in the normal rat. The upper pair of traces illustrates low voltage fast activity in the left and right neocortex (LC, RC, respectively), indicating neocortical EEG arousal. The lower pair of traces, representing EEG activity from left and right hippocampus (LHi and RHi), shows hippocampal theta (varying in frequency from 7.5 to 10 Hz, and in amplitude from 270 μV to 1.44 mV) during quadrupedal locomotion. However, with a sudden arrest in such walking, hippocampal theta disappears and is replaced by LIA (Fig. 1B). These phenomena were seen repeatedly (at least 20 times) in all 15 normal rats.

EEG and Behavioral Observations in Rats After Large Lateral Hypothalamic Lesions

As reported earlier [20], in the first few days after large bilateral lateral hypothalamic lesions, the aphagic-adipsic animal (Stage 1 of recovery) is somnolent (lying with eyes closed, without limb support) or akinetic (crouching or standing immobile, with eyes open, typically in a hunched

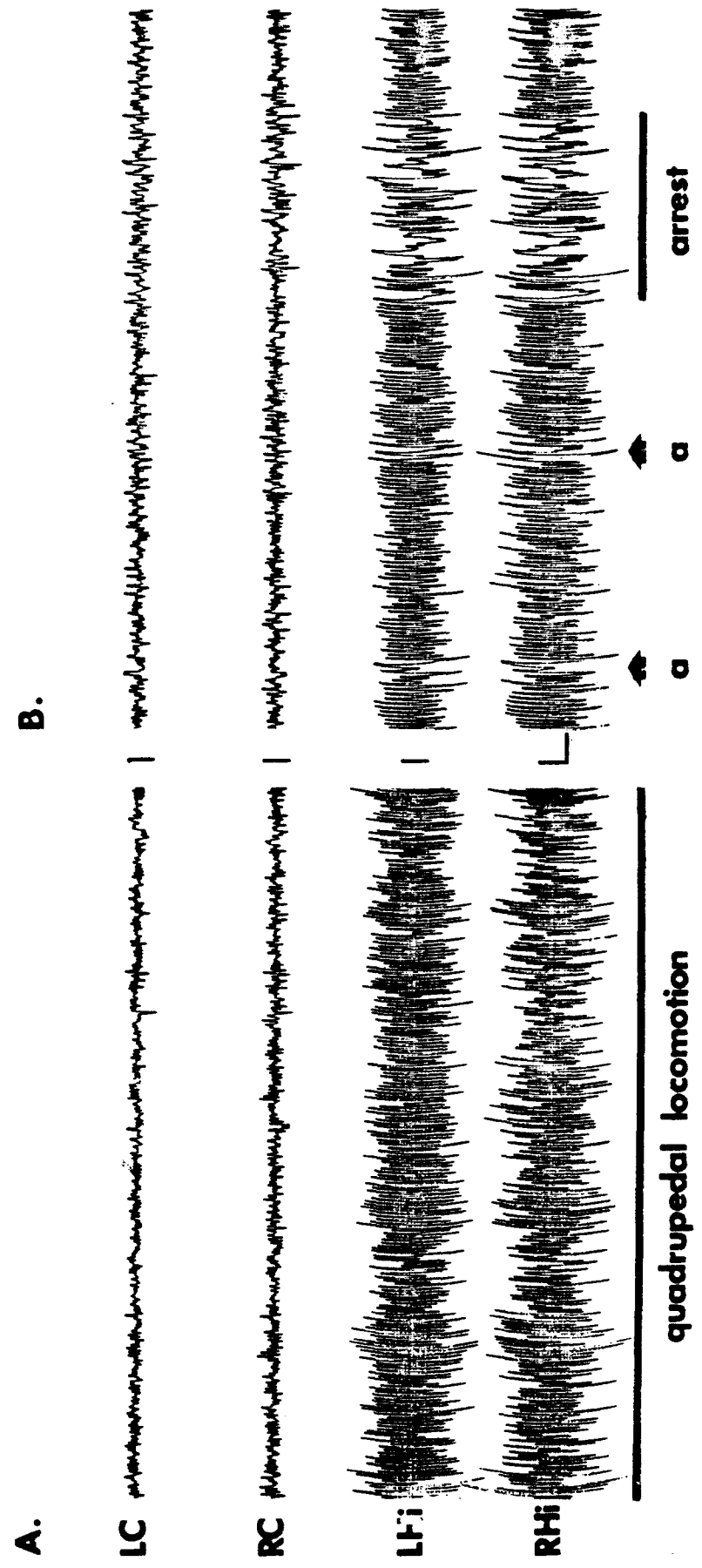

FIG. 1A. Normal rat (HH 8): Hippocampal theta of varying frequency (7.5–10 Hz) and amplitude (270 μV–1.44 mV) during quadrupedal locomotion in the normal awake rat. B. Normal rat (HH 8): short arrests (*a* with arrow) produce irregularities in hippocampal theta accompanying locomotion; prolonged arrests are associated with Large Amplitude Irregular Activity (LIA). Abbreviations: *LC and RC, left and right neocortex, respectively; LHi and RHi, left and right hippocampus, respectively.* Calibration: *1 sec, 300 μV.*

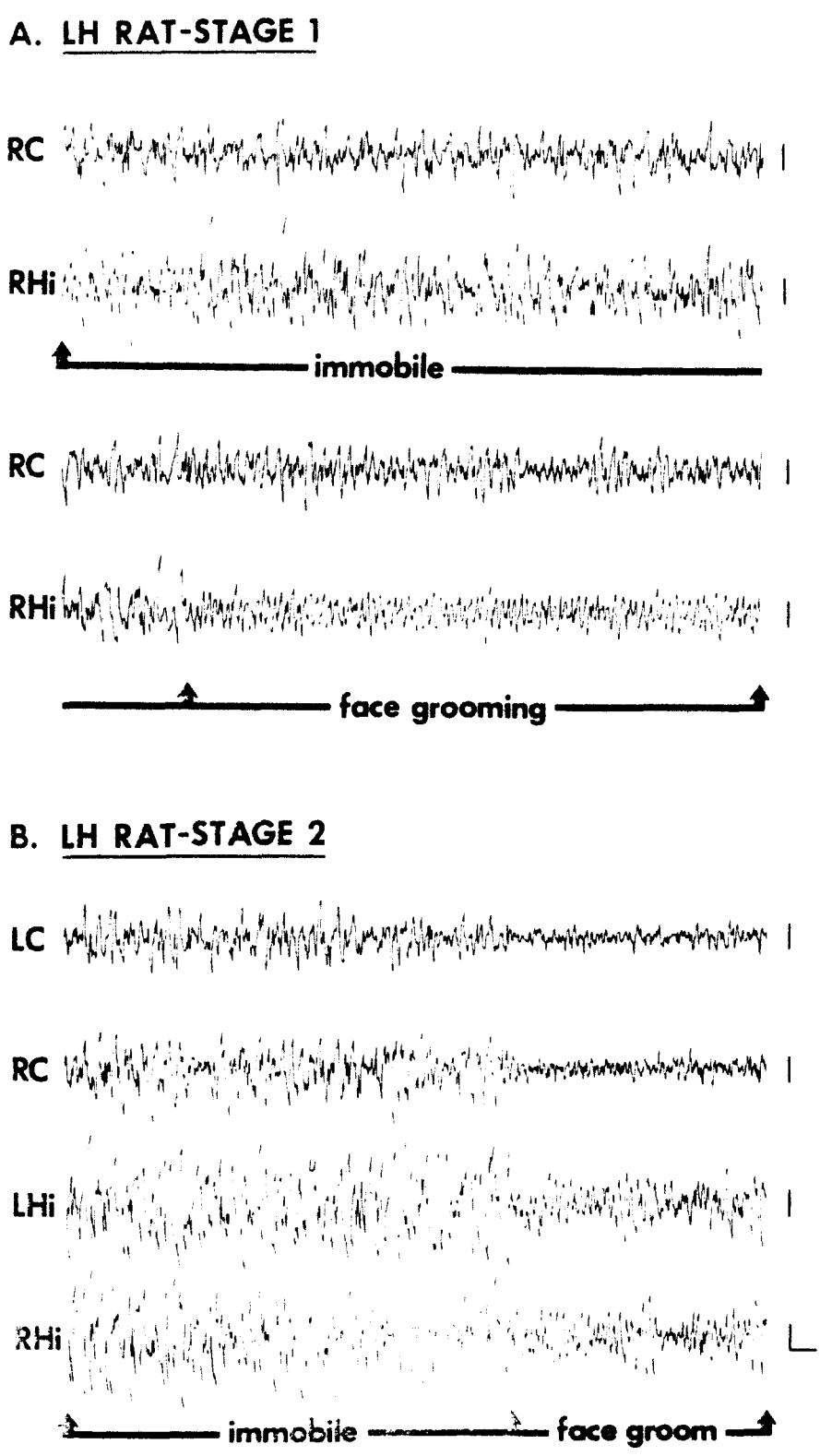

FIG. 2. Neocortical and hippocampal EEG after lateral hypothalamic damage. A. Stage 1 of recovery. Upper pair of traces: Cortical and hippocampal large amplitude irregular slow activity during somnolent immobility in a first stage aphagic-adipsic rat. Lower pair of traces: When face grooming occurs, hippocampal activity shifts to theta without concomitant neocortical low voltage fast activity. B. Stage 2 of recovery. In the second stage lateral hypothalamic rat, neocortical desynchronization accompanies hippocampal theta during face grooming. Both records from Rat HH 2. Calibration: 1 sec, 300 μV.

posture). During both forms of immobility, the neocortical EEG showed persistent large amplitude slow activity (synchronization), suggesting that even with its eyes open, the rat was in a type of somnolent state (Fig. 2A, top trace). During such somnolent-akinetic immobility, the hippocampal EEG typically showed large amplitude irregular activity (Fig. 2A, second trace).

However, when the Stage 1 lateral hypothalamic animal engaged in a stereotyped act, such as a bout of face grooming, a form of hippocampal theta appeared (see Fig. 2A, lowest trace). Stage 1 theta was always slower than normal. Initially, it ranged between 3 to 4 Hz. A similar finding was reported by Robinson and Whishaw [26] in posterior hypothalamic-damaged rats. The reduction of hippocampal theta frequency during Stage 1 of recovery was not a function of a decrease in body temperature [42], because during the first 2 postoperative days, rectal temperature of LH rats in a thermoneutral environment (25°C) was higher than that of normals. For 5 normal and 5 LH rats, mean rectal temperature ± SD was 37.8 ± 0.15°C, 38.6 ± 0.2°C, respectively, on postoperative Day 1 and 37.7 ± 0.3°C, 38.5 ± 0.15°C, respectively, on Day 2. In addition, theta amplitude as well as frequency was reduced, even during strongly activating manipulations such as tail pinch, pick-up from the ground, or righting from an imposed supine position (see also [17]). During Stage 1 (aphagia-adipsia), the amplitude reduction during these behavioral events ranged from 25% to 50%.

During this initial period of somnolence, when grooming occurred or struggling and postural shifts were induced by tail pinch, the neocortex did not desynchronize when hippocampal theta appeared (Fig. 2A, third trace from top), suggesting that these acts can be mediated by lower level neural systems, independent of cortical activity. When the same animal had progressed in its recovery to the anorexic stage (Stage 2), a bout of grooming interrupting a period of immobility produced hippocampal theta of somewhat higher frequency (4–4.5 Hz). In Stage 2, such grooming-associated or tail pinch-induced theta was always accompanied by neocortical desynchronization (see Fig. 2B). This suggests that as behavioral recovery progresses after lateral hypothalamic damage, there is a concomitant recovery of cortical participation in these behaviors.

It is noteworthy that Arduini and Pompeiano [3] have reported the occurrence of hippocampal theta along with a neocortical slow wave sleep pattern in the acute cerveau isolé rabbit. A similar observation has been reported by Kawamura and Domino [13] in the rostral midbrain-transected cat. These authors also noted that electrical stimulation of the posterior hypothalamus induced long trains of hippocampal theta without neocortical desynchronization. Finally, Olmstead and Villablanca [24] have shown that in decerebrate cats hippocampal theta appears prior to neocortical desynchronization. This indicates that in decerebrate cats, as in early stage LH rats, hippocampal theta and cortical desynchronization can be dissociated. The resemblance of these decerebrate preparations to the early stages of recovery from lateral hypothalamic damage is quite remarkable. It lends additonal support to the view that, early in recovery, LH rats display a decerebrate-like level of function [20].

As described above, grooming in the Stage 1 lateral hypothalamic rat can occur without neocortical desynchronization. This supports Vanderwolf's [40] view that grooming may be an automatism under low level nervous control. In the normal rat, however, large amplitude irregular hippocampal activity, not theta, typically accompanies grooming and other such automatisms [39]. Why then does hippocampal theta accompany grooming in the lateral hypothalamic rat, capable only of simple automatisms? This problem can be resolved by distinguishing between two types of hippocampal theta activity [19, 41, 43]. As we have seen, hippocampal theta in LH rats is slow (less than 7 Hz). Furthermore, in early stage LH rats, as in posterior hypothalamic rats [26], long trains of slow theta may appear spontaneously during total immobility, without any visible postural displacement (movement-unrelated, immobility-concurrent theta). In contrast, hippocampal theta of normal rats is fast (7–12 Hz), but does not occur during immobility, and only appears in such acts as walking, jumping, rearing, and orienting (movement-related theta). It has been shown that these two types of theta can be pharmacologically dissociated in rabbits and rats [19]. Movement-related theta is abolished by anesthetic dosages of ether or urethane, but is resistant to high dosages of anticholinergic agents such as atropine or scopolamine. Since movement-related theta is atropine-resistant, it is considered to be noncholinergic ([19,27], but see [34]). Movement-unrelated, immobility-concurrent theta is resistant to anesthesia, but is abolished by anticholinergics. Since movement-unrelated theta is atropine-sensitive, it is considered to be cholinergic [41].

Whereas slow, immobility-concurrent theta has been demonstrated in the normal waking rabbit [12, 16, 19], and cat [6,14], it does not appear in the normal, undrugged and unstressed rat [41]. Indeed, when normal rats are immobile, only hippocampal LIA is present. However, slow, immobility-concurrent theta, similar to that observed in LH rats, can be seen in normal rats anesthetized with chloral hydrate (observed by us in 8 rats with 600 mg/kg chloral hydrate), ether (observed in 5 rats), or urethane (see also [19]) in which cases it appears either spontaneously or in response to tail pinch without any visible movement. Interestingly, our own preliminary observation on 3 rats, as well as findings by Kolb and Whishaw [17] have shown that early after lateral hypothalamic damage, the slow hippocampal theta rhythm may be abolished by large doses of atropine sulfate (50 mg/kg) or scopolamine hydrobromide (10 mg/kg).

It appears therefore that the early postoperative effect of lateral hypothalamic damage on hippocampal and cortical EEG activity is functionally equivalent to that of anesthesia in normal rats: LH lesions, like anesthetic agents, produce continuous slow waves in the neocortex. In addition, they abolish fast, movement-related, atropine-resistant theta while releasing slow, movement-unrelated, atropine-sensitive theta activity. In effect, this is a decencephalized EEG pattern. Later in recovery, cortical desynchronization reappears and hippocampal theta becomes resistant to atropine or scopolamine [17]. This suggests that the akinetic, cataleptic, somnolent rat with large lateral hypothalamic lesions retains, during the early stages of recovery, only one type of theta – i.e., slow cholinergic theta. Such theta appears to be released when it is never seen in normal rats – i.e., during immobility and during behavioral automatisms (e.g., grooming). In this respect, early stage LH rats apparently resemble decerebrate cats with the high mesencephalic transections, in which exaggerated amounts of 3–5 Hz atropine-sensitive theta

have been reported [24]. Later in recovery from LH damage, as locomotion returns, theta frequency increases, but it is still mediated by cholinergic mechanisms. As recovery proceeds still further, the noncholinergic theta system becomes functional again [17].

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REFERENCES

1. Anand, B. K. and J. R. Brobeck. Hypothalamic control of food intake. *Yale J. Biol. Med.* **24**: 123–140, 1951.
2. Antelman, S. M., H. Szechtman, P. Chin and A. E. Fisher. Tail-pinch induced eating, gnawing, and licking behavior in rats: Dependence on the nigrostriatal dopamine system. *Brain Res.* **99**: 319–337, 1975.
3. Arduini, A. A. and O. Pompeiano. Attivita elettrica di singole unità dell' ippocampo e sue modificazioni con stimoli afferenti. *Archs Sci. biol. (Bologna)* **39**: 397–406, 1955.
4. Brown, B. B. Frequency and phase of hippocampal theta activity in the spontaneously behaving cat. *Electroenceph. clin. Neurophysiol.* **24**: 53–62, 1968.
5. Brügger, M. Fresstrieb als hypothalamisches Symptom. *Helv. Physiol. Acta.* **1**: 183–198, 1943.
6. Coleman, J. R. and D. B. Lindsley. Hippocampal electrical correlates of free behavior and behavior induced by stimulation of two hypothalamic-hippocampal systems in the cat. *Expl Neurol.* **49**: 506–528, 1975.
7. Coons, E. E., M. Levak and N. E. Miller. Lateral hypothalamus: Learning of food-seeking response mediated by electrical stimulation. *Science* **150**: 1320–1321, 1965.
8. Epstein, A. N. The lateral hypothalamic syndrome: Its implications for the physiological psychology of hunger and thirst. In: *Progress in Physiological Psychology*, edited by E. Stellar and J. M. Sprague. New York: Academic Press, 1971.
9. Epstein, A. N. and P. Teitelbaum. Specific loss of the hypoglycemic control of feeding in recovered lateral rats. *Am. J. Physiol.* **213**: 1159–1167, 1967.
10. Grossman, S. P. Eating or drinking elicited by direct adrenergic or cholinergic stimulation of hypothalamus. *Science* **132**: 301–302, 1960.
11. Grossman, S. P. Direct adrenergic and cholinergic stimulation of hypothalamic mechanisms. *Am. J. Physiol.* **202**: 872–882, 1962.
12. Harper, R. M. Frequency changes in hippocampal electrical activity during movement and tonic immobility. *Physiol. Behav.* **7**: 55–58, 1971.
13. Kawamura, H. and E. E. Domino. Hippocampal slow ("arousal") wave activation in the rostral midbrain transected cat. *Electroenceph. clin. Neurophysiol.* **25**: 471–480, 1968.
14. Kemp, I. R. and B. R. Kaada. The relation of hippocampal theta activity to arousal, attentive behavior and somato-motor movements in unrestrained cats. *Brain Res.* **95**: 323–342, 1975.
15. Kissileff, H. R. Food-associated drinking in the rat. *J. comp. physiol. Psychol.* **67**: 309–319, 1969.
16. Klemm, W. R. EEG and multiple-unit activity in limbic and motor systems during movement and immobility. *Physiol. Behav.* **7**: 337–343, 1971.
17. Kolb, B. and I. Q. Whishaw. Effects of brain lesions and atropine on hippocampal and neocortical EEG in the rat. *Expl Neurol.* **56**: 1–22, 1977.
18. König, J. F. R. and R. A. Klippel. *The Rat Brain. A Stereotaxic Atlas of the Forebrain and Lower Parts of the Brain Stem.* Baltimore: Williams and Wilkins, 1963.
19. Kramis, R., C. H. Vanderwolf and B. H. Bland. Two types of hippocampal rhythmical slow activity in both the rabbit and the rat: Relations to behavior and effects of atropine, diethyl ether, urethane, and pentobarbital. *Expl Neurol.* **49**: 58–85, 1975.
20. Levitt, D. R. and P. Teitelbaum. Somnolence, akinesia, and sensory activation of motivated behavior in the lateral hypothalamic syndrome. *Proc. natn Acad. Sci. U.S.A.* **72**: 2819–2823, 1975.
21. Marshall, J. F. and P. Teitelbaum. Further analysis of sensory inattention following lateral hypothalamic damage in rats. *J. comp. physiol. Psychol.* **86**: 375–395, 1974.
22. Marshall, J. F., B. H. Turner and P. Teitelbaum. Sensory neglect produced by lateral hypothalamic damage. *Science* **174**: 523–525, 1971.
23. Marshall, J. F., D. Levitan and E. M. Stricker. Activation-induced restoration of sensorimotor functions in rats with dopamine-depleting brain lesions. *J. comp. physiol. Psychol.* **90**: 536–546, 1976.
24. Olmstead, C. E. and J. R. Villablanca. Hippocampal theta rhythm persists in the permanently isolated forebrain of the cat. *Brain Res. Bull.* **2**: 93–100, 1977.
25. Powley, T. L. and R. E. Keesey. Relationship of body weight to the lateral hypothalamic feeding syndrome. *J. comp. physiol. Psychol.* **70**: 25–36, 1970.
26. Robinson, T. E. and I. Q. Whishaw. Effects of posterior hypothalamic lesions on voluntary behavior and hippocampal electroencephalograms in the rat. *J. comp. physiol. Psychol.* **86**: 768–786, 1974.
27. Robinson, T. E., B. A. Pappas and C. H. Vanderwolf. Noradrenalin and behavior related EEG activation of the hippocampus and neocortex. Paper read at the Society for Neuroscience, New York City, 1975.
28. Rowland, N. and S. Nicolaidis. Self-intravenous drinking and regulation of hydromineral balance in rats recovering from lesions of the lateral hypothalamus. *Physiol. Behav.* **14**: 457–464, 1975.
29. Schallert, T. and I. Q. Whishaw. Two types of aphagia and two types of sensorimotor impairment after lateral hypothalamic lesions: Observations in normal-weight, dieted, and fattened rats. *J. comp. physiol. Psychol.* **55**: 1978, in press.
30. Schapiro, S., M. Salas and R. Vukovich. Hormonal effects on ontogeny of swimming ability in the rat: Assessment of central nervous system development. *Science* **168**: 147–151, 1970.
31. Stricker, E. M. Drinking by rats after lateral hypothalamic lesions: A new look at the lateral hypothalamic syndrome. *J. comp. physiol. Psychol.* **90**: 127–143, 1976.
32. Stricker, E. M. and M. J. Zigmond. Recovery of function after damage to central catecholamine-containing neurons: A neurochemical model for the lateral hypothalamic syndrome. In: *Progress in Psychobiology and Physiological Psychology*, edited by J. M. Sprague and A. N. Epstein. New York: Academic Press, 1976, Vol. 6, pp. 121–188.
33. Stricker, E. M., M. I. Friedman and M. J. Zigmond. Glucoregulatory feeding by rats after intraventricular 6-hydroxydopamine or lateral hypothalamic lesions. *Science* **189**: 895–897, 1975.
34. Teitelbaum, H., J. F. Lee and J. N. Johannessen. Behaviorally evoked hippocampal theta waves: A cholinergic response. *Science* **188**: 1114–1116, 1975.
35. Teitelbaum, P. The encephalization of hunger. In: *Progress in Physiological Psychology*, edited by E. Stellar and J. M. Sprague. New York: Academic Press, 1971, Vol. 4, pp. 319–350.
36. Teitelbaum, P. and A. N. Epstein. The lateral hypothalamic syndrome: Recovery of feeding and drinking after lateral hypothalamic lesions. *Psychol. Rev.* **69**: 74–90, 1962.
37. Teitelbaum, P. and E. Stellar. Recovery from the failure to eat produced by hypothalamic lesions. *Science* **120**: 894–895, 1954.

38. Teitelbaum, P. and D. L. Wolgin. Neurotransmitters and the regulation of food intake. In: *Hormones, Homeostasis, and the Brain*, edited by W. H. Gispen, Tj. B. van Wiemersma Greidanus, B. Bohus, and D. de Wied. *Progr. Brain Res.* 42: 235–249, 1975.
39. Vanderwolf, C. H. Hippocampal electrical activity and voluntary movement in the rat. *Electroenceph. clin. Neurophysiol.* 26: 407–418, 1969.
40. Vanderwolf, C. H. Limbic-diencephalic mechanisms of voluntary movement. *Psychol. Rev.* 78: 83–113, 1971.
41. Vanderwolf, C. H. Neocortical and hippocampal activation in relation to behavior: Effects of atropine, eserine, phenothiazines, and amphetamine. *J. comp. physiol. Psychol.* 88: 300–323, 1975.
42. Vanderwolf, C. H., B. H. Bland and I. Q. Whishaw. Diencephalic, hippocampal and neocortical mechanisms in voluntary movement. In: *Efferent Organization and the Integration of Behavior*, edited by J. D. Maser. New York: Academic Press, 1973, pp. 229–262.
43. Vanderwolf, C. H., R. Kramis, L. A. Gillespie and B. H. Bland. Hippocampal rhythmical slow activity and neocortical low voltage fast activity: Relations to behavior. In: *The Hippocampus: A Comprehensive Treatise*, edited by R. L. Isaacson and K. H. Pribram. New York: Plenum, 1975, pp. 101–128.
44. Whishaw, I. Q. and T. Schallert. Hippocampal RSA (Theta), apnea, bradycardia, and effects of atropine during underwater swimming in the rat. *Electroenceph. clin. Neurophysiol.* 42: 383–396, 1977.
45. Whishaw, I. Q. and C. H. Vanderwolf. Hippocampal EEG and behavior: Changes in amplitude and frequency of RSA (theta rhythm) associated with spontaneous and learned movement patterns in rats and cats. *Behav. Biol.* 8: 461–484, 1973.
46. Wolgin, D. L., J. Cytawa and P. Teitelbaum. The role of activation in the regulation of food intake. In: *Hunger: Basic Mechanisms and Clinical Implications*, edited by D. Novin, W. Wyrwicka and G. Bray. New York: Raven Press, 1976, pp. 179–191.