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# Vestibular Versus Tail-Pinch Activation in Cats With Lateral Hypothalamic Lesions

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O'BRIEN, D. P., R. M. CHESIRE AND P. TEITELBAUM. *Vestibular versus tail-pinch activation in cats with lateral hypothalamic lesions.* PHYSIOL BEHAV 34(5) 811–814, 1985.—Mild tail-pinch induces eating in animals which are aphagic following lateral hypothalamic lesions. This study compares the specificity of the behavior produced by tail-pinch to that produced by vestibular stimulation in cats which are akinetic and aphagic following lateral hypothalamic lesions. In these cats, tail-pinch elicited licking and biting at food while vestibular stimulation preferentially elicited forward locomotion. These results suggest that such stimuli activate specific movement subsystems rather than producing general, non-specific arousal.

Vestibular stimulation Locomotion Tail-pinch Eating Lateral hypothalamus Activation Movement subsystems

EATING behavior is controlled by a variety of homeostatic or food related stimuli (e.g., blood glucose, stomach distension, palatability). In addition, however, a variety of manipulations which are not related to homeostasis or to food related stimuli can affect eating. Mild pressure applied to the tail of a rat will induce eating in sated rats [1, 2, 6, 15]. It has been hypothesized that such non-injurious tail-pinch (TP) stimulates a general activational system because, in the presence of appropriate goal objects, TP can induce a variety of behaviors in addition to eating [1, 2, 6, 15], such as digging [20], sexual behavior [19], and maternal behavior [14].

TP can also induce eating in animals which are otherwise aphagic due to lateral hypothalamic (LH) lesions [1, 5, 9, 21, 22] or to 6-hydroxydopamine (6-OHDA) depletion of dopamine [7,10]. This has led to the suggestion that the deficits seen with dopamine-depleting LH lesions or 6-OHDA injections reflect damage to such a general activational system [9,22]. However, although they eat in response to tail-pinch, rats with LH lesions continue to reject water, suggesting some specificity to the arousal generated by tail-pinch [9]. Also, in normal rats there is evidence that differences in the type of activating stimulus (TP vs. electrical stimulation of the tail) can result in different responses (biting vs. licking respectively) [15].

In their study of cats made aphagic and akinetic by lateral hypothalamic lesions, Wolgin and Teitelbaum [21,22] also demonstrated tail-pinch induced eating. In addition, they reported that otherwise akinetic cats would walk after being handled in a manner that appeared to stimulate the vestibular system (e.g., picked up by the scruff of the neck and placed on the floor). However, the role of vestibular stimulation as a form of activation has not been thoroughly investigated, although it is well known that otherwise cataleptic animals may retain vestibular righting [11, 16, 17]. Clinically, it has been noted that shifts in body weight can help overcome the akinesia associated with Parkinson's disease [8].

The purpose of this study was to explore the role of vestibular stimulation as an activating stimulus and to compare the specificity of the response elicited by it with that elicited by tail-pinch in cats following electrolytic lesions of the LH.

## METHOD

Five adult domestic shorthair cats of both sexes were used as subjects. The cats were anesthetized with Na thiobarbital, intubated and maintained on Halothane gaseous anesthesia. Some cats were given Acetylpromazine (0.1 mg/lb) as a preanesthetic sedative. They were mounted in a stereotaxic instrument (Kopf model 1404) and prepared for sterile surgery.

Using a rectal indifferent electrode, LH lesions were made by passing 3.0 mA of anodal current for 90 sec through stainless steel electrodes insulated except for 0.5 mm at the conical tip. The coordinates, taken from Snider and Niemer [13] were 11.0 mmA, 3.0 mmL, and −4.0 mmH. During surgery the cats were given 10 mg/lb of ampicillin and 1 mg/lb dexamethasone. They were allowed to recover in an incubator at 29–30°C. Some cats were kept in the incubator for 1–2 days post-operatively.

Cats were tested daily, beginning 24 hours post-operatively. The cat was placed on the floor with an inverted Plexiglas box enclosing it for at least five minutes. This elim-

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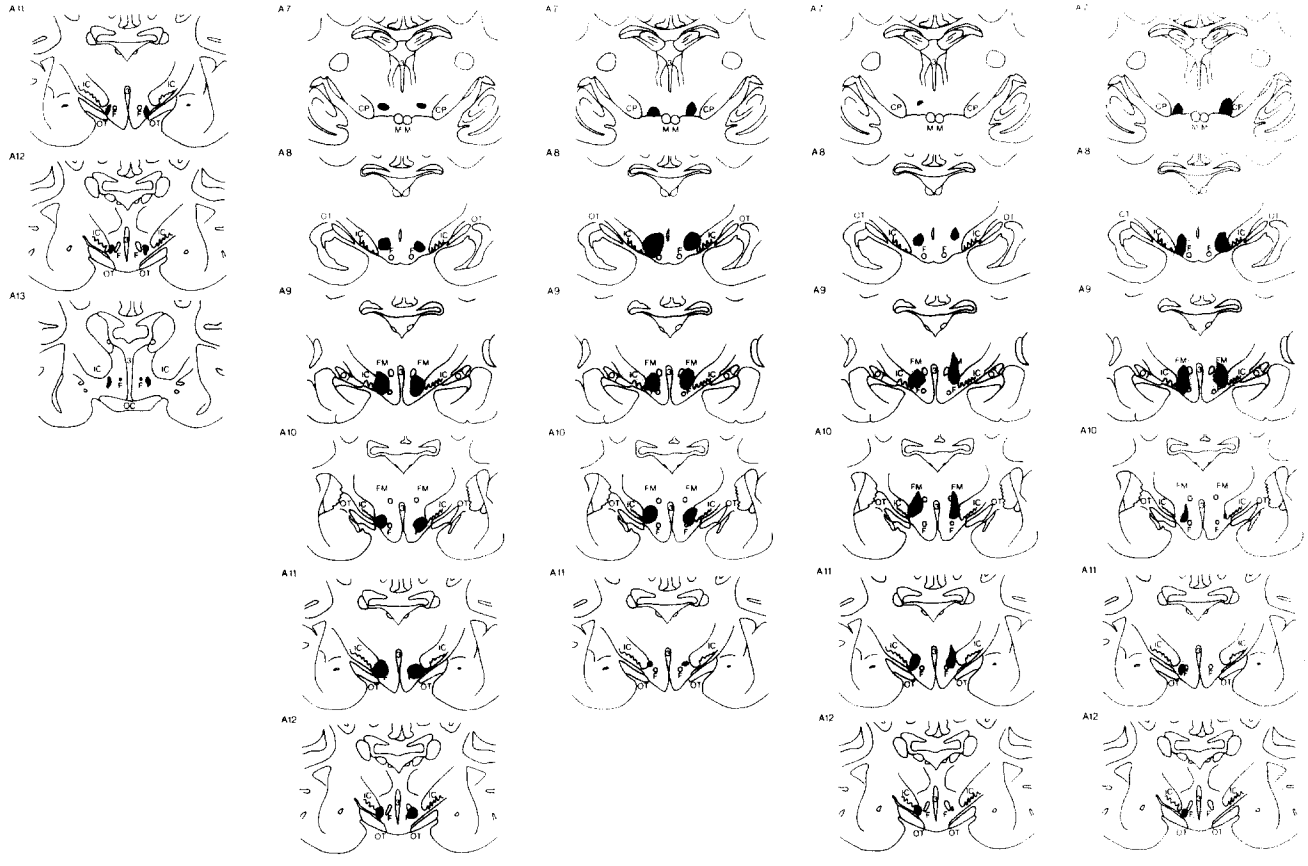

FIG. 1. Tracings of sections of the brain throughout the extent of the lesions in lateral hypothalamic cats. Shaded area represents the lesion. (Abbreviations: AC=Anterior Commissure, CP=Cerebral Peduncle, F=Fornix, FM=Mammillothalamic Fasciculus, IC=Internal Capsule, M=Mammillary body, OC=Optic Chiasm, OT=Optic Tract, 3=Third Ventricle).

inated any residual vestibular stimulation resulting from moving the cat to the experimental room. The Plexiglas box was then removed and the following tests were conducted:

*(1) Spontaneous activity.*The number of steps taken during a five minute period were recorded. A step was defined as a picking up and placing down of the left forepaw which resulted in forward locomotion. The cat was then presented with a highly palatable food (canned tuna, sardines, or liver), and the amount of time spent eating, licking, or investigating the food was recorded for five minutes. The spontaneous walking and investigating, licking or eating food per 30 second period was calculated for comparison with the following test. The order of the remaining test was randomized.

*(2) TP-induced activity.*A pinchcock type spring tubing clamp or large spring steel paperclip (IDL Binderclip No. 100 (large)) was used to apply TP. The clip was applied 6 to 8 cm from the tip of the tail and left in place for 15 seconds. The number of steps and the time spent eating, licking, or investigating the food during the next 30 seconds was recorded.

*(3) Vestibular stimulation.*The cat was held with both hands around the thorax and swung up and down in about a 2 foot arc for 15 seconds (generally about 12 swings). The cat was then placed on the ground, and the locomotion and eating induced by the procedure were similarly recorded for the next 30 seconds.

*(4) Tactile stimulation.*To insure that any arousal was the result of vestibular stimulation and not just handling, the cat was held and its sides were rubbed for 15 seconds with minimal movement of the head. The locomotion and eating were then recorded as above.

The data was analysed using the Friedman's two way analysis of variance and the Wilcoxon signed-ranks test [12].

### *Histology*

At the completion of the experiment the cats were injected with an overdose of sodium pentobarbitol. They were perfused through the thoracic aorta with isotonic saline followed by 10% formalin. The brains were removed and stored in 10% formalin. Frozen sections 40 $\mu$m thick were cut through the extent of the lesion. Every fifth section was mounted on a glass slide and stained with cresyl violet. Selected slides were projected through a microprojector (Bausch and Lomb) and tracings were made of the brain sections throughout the extent of the lesion. Affected structures were identified with the aid of a stereotaxic atlas [13].

## RESULTS

### *Histology*

The brains of all five cats were analyzed. Representive sections are present in Fig. 1.

In all cats the lesions were centered at the level of the

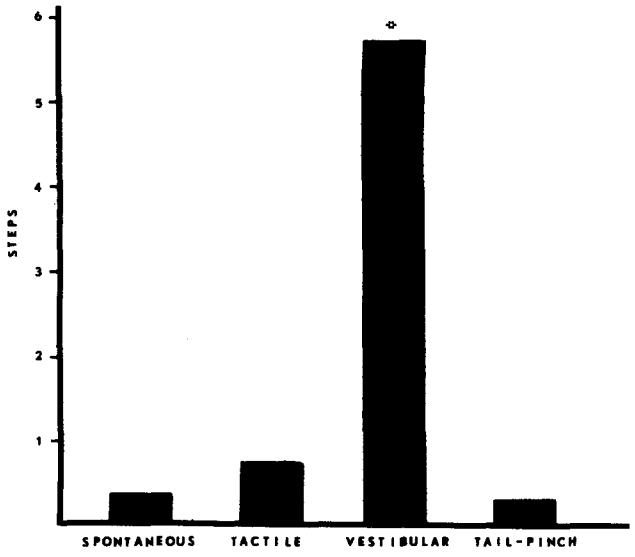


FIG. 2. Mean number of steps taken by each cat per 30 seconds over the first four days after lateral hypothalamic lesions(*$p<0.05$).

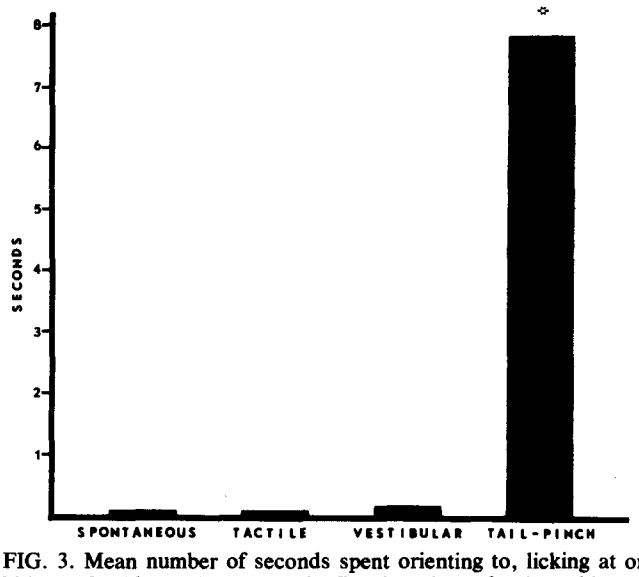


FIG. 3. Mean number of seconds spent orienting to, licking at or biting at food by each cat over the first four days after lateral hypothalamic lesions (*$p<0.05$).

ventromedial hypothalamus. The lesions were located in the far lateral hypothalamus at the tip of the internal capsule and extended ventrally to the base of the brain.

As previously reported, all cats with lateral hypothalamic lesions showed varying degrees of catalepsy, akinesia, and aphagia (Wolgin and Teitelbaum, 1978). For the first few days after the lesion, the cats took very few spontaneous steps and appeared somnolent. During this period tactile stimulation did not activate locomotion. With vestibular stimulation, however, the cats took 2 to 22 steps forward. Initially, if the cats encountered a wall or corner, they would rear and briefly paw at the wall with their forepaws. Later, they would turn away from the wall or corner and continue walking. By the fifth day, spontaneous locomotion recovered in most cats and the vestibular stimulation had no detectable effects. Figure 2 shows the mean number of steps taken by each cat per 30 seconds over the first four days after the lesion.

Tail-pinch produced a different type of activation. While the vestibular stimulation produced no obvious "emotional" reaction, tail-pinch often elicited growling and piloerection. The cats oriented to food and occasionally took a few steps toward the food. They then licked and bit at the food or at the food container. Some individual cats ingested food, but frequently only licking and mouthing appeared. Some cats extended the hindlimbs during the tail-pinch and made stepping movements in place with the hind limbs. However, the cats' forelimbs remained stationary as it licked and bit at the food and thus this was not scored as locomotion. In contrast, vestibular stimulation did not result in any orientation or mouthing, and if food was placed in front of a cat, it simply walked through the food. Tactile stimulation again had no effect. By the fourth or fifth day most cats ate spontaneously, but responded similarly to the tail-pinch. Figure 3 shows the mean number of seconds spent orienting to food or licking/biting at it.

## DISCUSSION

Bilateral electrolytic lesions of the lateral hypothalamic area (LH) produce a syndrome of aphagia, adipsia, catalepsy, akinesia, and sensory neglect [7, 9, 16, 22]. Such animals are believed to suffer from a deficit in arousal because, although they appear somnolent and akinetic, non-specific activation by tail-pinch or amphetamines can induce eating [3, 9, 22]. TP has been considered a non-specific activation or stress because it can induce a number of seemingly unrelated behaviors such as eating [1], digging [20], sexual behavior [19], and maternal behavior [14]. Szechtman [15] has recently argued that such TP-induced behavior does not represent a non-specific arousal, but rather a redirection of behavior that would otherwise be directed at the stimulus on the tail. Thus the oral behaviors of orienting, licking, and biting, which would normally be directed at removing the offending stimulus from the tail, are directed toward an alternative goal object depending on what is present in the environment.

Our results support this concept. If TP induced a general increase in arousal, it should be as likely to induce locomotion as it would be to induce orienting, licking, and biting. However, our results indicate that TP was specific in eliciting orienting, licking, and biting if food was present. If no food was present, locomotion was still not produced by TP.

Vestibular stimulation has been observed to elicit righting in otherwise cataleptic, akinetic animals [11,22]. In the present experiment we demonstrated that vestibular stimulation can also activate forward locomotion. Such activation also appeared to be relatively specific because it did not induce orienting, mouthing, and biting, although TP did so. Vestibular activation of locomotion has not, to our knowledge, been demonstrated in animal models of akinesia and catalepsy. In human Parkinsonian patients, however, it has been shown that vestibular stimulation, in the form of gentle rocking of the patient from side to side, can briefly induce more normal locomotion in otherwise akinetic patients [8].

The concept of a non-specific reticular activating system which controls cortical synchronization and arousal has recently been criticized [18]. Our results suggest that there is some specificity to the type of behavior that is elicited by different activating stimuli.

In the analysis of the recovery from LH lesions, we have found it useful to view the animals' behavior as consisting of a number of well-defined motor subsystems such as orienting, mouthing (licking and biting), and forward locomotion [16,17]. For example, in catalepsy produced by lateral hypothalamic lesions or haloperidol administration, postural support systems are still functional while forward locomotion, orienting, and mouthing are not functioning. Different types of activating stimuli are able to activate different subsystems. Thus, in the present experiment vestibular stimulation activated forward locomotion. On the other hand, tail-pinch preferentially activated orienting, licking, and biting at food. Some forward locomotion occurred, particularly if the food was not immediately in front of the cat. However, orienting, licking and biting were the predominant response to tail-pinch. This was especially evident in some cats where a disconnection between the behavior of the hindquarters and that of the head and forequarters occurred. Tail-pinch elicited stepping in place in the hindlimbs, but the forepaws remained stationary while the cat licked or bit at the food, and thus no forward locomotion occurred. The demonstration that movement subsystems can be differentially activated by tail-pinch or vestibular stimulation further supports the concept that such activation operates on particular movement subsystems rather than generally on all components of behavior.

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## REFERENCES

1. Antelman, S. M., H. Szechtman, P. Chin and A. E. Fisher. Tail-pinch-induced eating, gnawing and licking behavior in rats: dependance on the nigrostriatal dopamine system. *Brain Res* **99:** 319–337, 1975.
2. Antelman, S. M. and H. Szechtman. Tail pinch induces eating in sated rats which appears to depend on nigrostriatal dopamine. *Science* **189:** 731–733, 1975.
3. Antelman, S. M., N. E. Rowland and A. E. Fisher. Stress related recovery from lateral hypothalamic aphagia. *Brain Res* **102:** 346–350, 1976.
4. Antelman, S. M., N. E. Rowland and A. E. Fisher. Stimulation bound ingestive behavior: A view from the tail. *Physiol Behav* **17:** 743–748, 1976.
5. Ellison, G. D., C. A. Sorenson and B. J. Jacobs. Two feeding syndromes following surgical isolation of the hypothalamus in rats. *J Comp Physiol Psychol* **70:** 173–188, 1970.
6. Fass, B., H. Strub, J. M. Greenspon, D. A. Stevens and D. G. Stein. A descriptive analysis of tail-pinch elicited eating-behavior of rats. *Physiol Behav* **26:** 355–359, 1981.
7. Marshall, J. F., J. S. Richardson and P. Teitelbaum. Nigrostriatal bundle damage and the lateral hypothalamic syndrome. *J Comp Physiol Psychol* **87:** 808–830, 1974.
8. Martin, P. J. *The Basel Ganglia and Posture*. Philadelphia: JB Lippincott, 1967.
9. Mufson, E. J., S. Balagura and W. Riss. Tail pinch-induced arousal and stimulus-bound behavior in rats with lateral hypothalamic lesions. *Brain Behav Evol* **13:** 154–164, 1976.
10. Rowland, N., D. M. Marques and A. E. Fisher. Comparison of the effects of brain dopamine-depleting lesions upon oral behavior elicited by tail pinch and electrical stimulation. *Physiol Behav* **24:** 273–281, 1980.
11. Schallert, T. and P. Teitelbaum. Haloperidol, catalepsy, and equilibrating functions in the rat: Antagonistic interaction of clinging and labyrinthine righting reactions. *Physiol Behav* **27:** 1077–1083, 1981.
12. Siegel, S. *Nonparametric Statistics for the Behavioral Sciences*. New York: McGraw-Hill Book Co., 1956.
13. Snider, R. S. and W. T. Niemer. *A Stereotaxic Atlas of the Cat Brain*. Chicago: University of Chicago Press, 1961.
14. Szechtman, H., H. I. Siegel, J. S. Rosenblatt and B. R. Komisaruk. Tail-pinch facilitates the onset of maternal behavior in rats. *Physiol Behav* **19:** 807–809, 1977.
15. Szechtman, H. Redirected oral behavior in rats induced by tail-pinch and electrical stimulation of the tail. *Physiol Behav* **24:** 57–64, 1980.
16. Teitelbaum, P., T. Schallert, M. De Ryck, I. Q. Whishaw and I. Golani. Motor subsystems in motivated behavior. In: *Neural Mechanisms of Goal Directed Behavior*, edited by R. F. Thompson, L. H. Hicks and V. B. Shryrkov. New York: Academic Press, 1980, pp. 23–85.
17. Teitelbaum, P., T. Schallert and I. Q. Whishaw. Sources of "spontaneity" in motivated behavior. In: *Motivation: Handbook of Behavioral Neurobiology*, vol 6, edited by E. Satinoff and P. Teitelbaum. New York: Plenum Press, 1983, pp. 23–85.
18. Vanderwolf, C. H. and T. E. Robinson. Reticulo-cortical activity and behavior: A critique of the arousal theory and a new synthesis. *Behav Brain Sci* **4:** 459–514, 1981.
19. Wang, L. and E. M. Hull. Tail pinch induces sexual behavior in olfactory bulbectomized male rats. *Physiol Behav* **24:** 211–215, 1980.
20. Watson, P. J., M. A. Short, D. H. Monroe and D. R. Warfield. Digging and biting induced by pinch stimuli and lateral hypothalamic electrical stimulation. *Physiol Behav* **24:** 267–271, 1980.
21. Wolgin, D. L. and P. Teitelbaum. The role of activation and sensory stimuli in the recovery of feeding following lateral hypothalamic lesions in the cat. Paper presented at the Eastern Psychological Association Meeting, Philadelphia, 1974.
22. Wolgin, D. L. and P. Teitelbaum. Role of activation and sensory stimuli in recovery from lateral hypothalamic damage in the cat. *J Comp Physiol Psychol* **92:** 474–500, 1978.