correct responses, nor between CNV's for hits as compared to misses.

In this study, the evoked potential data are reported in terms of peak-to-peak measurements. However, since most of the literature on evoked potentials deals with specific components, the data were reanalyzed with N and P_3 measured separately from the baseline at signal onset. The same relationships were found between P_3 and percent bias as between NP_3 and percent bias, whereas the N component was barely, if at all, related to the criterion measure.

Although the average signal-to-noise ratio was constant in our experimental conditions, it might be argued that increased sensitivity, and not the response criterion, was responsible for the changes in P_3. This was not the case, as evidenced by the sensitivity index A' (*9*), which did not vary significantly in our experimental conditions. Nor could criterion effects be attributed to changes in incentive, since the combined total value of the rewards and penalties was equivalent in all conditions: the overall value of each payoff matrix was the same in all cases, 12 cents gained for correct decisions (hits plus correct rejections) and 12 cents lost for incorrect decisions (misses plus false affirmatives).

An interpretation proposed by Hillyard *et al.* (*4*) for low-detectability signals provides a consistent way of viewing both the sensitivity relation to P_3 and the criterion relation to P_3 reported here. They suggested that for difficult discriminations the increase in the amplitude of P_3 with sensitivity might reflect the degree of confidence felt by the subject in his decisions with respect to the signals. As the intensity is increased, the subject becomes increasingly confident in the accuracy of his decision that the signal was presented. Similarly, in our study, the P_3 amplitude for hit trials may be said to grow with the subject's increased confidence that a signal did occur. However, it is important to point out that the growth of P_3 is not necessarily a result of increased amplitude in individual trials. It may be due to the segregation of trials according to different levels of confidence: for example, in the cautious condition where P_3 is large, the evoked potential wave form for hits is the average for trials in which the subject was highly confident that he heard the signal. On the other hand, in the liberal condition where P_3 is small, the wave form for hits is an average for many trials in which the subject said "yes" in spite of a relatively low degree of confidence in his judgment.

A more general interpretation, of which confidence might be seen as a special case, is that the P_3 amplitude is increased whenever the "salience" of a stimulus is enhanced (*10*). In our experiment, it is reasonable to assume that whenever a cautious criterion is adopted which results in the correct detection of a signal, that particular signal is more salient for the subject—salience varying as a result of changing the probability of signal occurrence or changing the payoff contingencies. However, at this point all interpretations must remain tentative in the absence of a strong theoretical structure capable of encompassing the data accumulated in the last decade (*11*). Nor is it possible to assume that P_3 represents coding by the nervous system of the psychological variables inferred in our study. Nevertheless, the empirical findings reported here support an approach in terms of signal detection theory to the analysis of discrimination behavior and add to the growing evidence that P_3 is remarkably sensitive to subtle psychological changes.

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8. Percent bias is a nonparametric analog of L_x. See H. Hodos, *Psychol. Bull.* **74**, 351 (1970).
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6 April 1972; revised 15 May 1972 ■

Biting Attack Elicited by Stimulation of the Ventral Midbrain Tegmentum of Cats

Abstract. *An area in the ventral midbrain tegmentum has been discovered in which electrical stimulation elicits biting attack. The midbrain sites from which attack was elicited correspond well with the zone in the midbrain tegmentum where degeneration was previously observed after lesions were made in lateral hypothalamic attack sites.*

Although the hypothalamus is a center for the integration of the visceral and somatic components of aggressive behavior (*1–3*), the neural pathways by which the hypothalamus mediates these components have usually been surmised only indirectly through the results of stimulation and ablation (*1*, *4*, *5*). However, Chi and Flynn (*6*, *7*), using modifications of the Nauta method for staining degenerating axons with reduced silver, have visualized directly the neural pathways associated with attack behavior elicited by hypothalamic stimulation in the cat. Most of the degeneration associated with biting attack followed the course of the medial forebrain bundle, the descending component of which could be traced into the ventral midbrain tegmentum. Since a role for the ventral midbrain tegmentum in the mediation of attack behavior had not been suggested before these degeneration experiments (*6*, *7*), we have examined the effects of electrical stimulation of this area. It is important to verify whether the ventral tegmental area is involved in mediation of

attack, because the areas of degeneration observed after lesions of lateral hypothalamic attack sites are not necessarily related to attack but might be related to one of many other functions mediated by the hypothalamus.

Observations were made on 11 cats. Each cat was fitted under aseptic conditions with several electrode guides mounted stereotactically on the skull over the midbrain. After each cat had fully recovered from surgery (5 to 7 days) it was placed in a large test cage with a deeply anesthetized rat and a bowl of food. None of the cats spontaneously attacked the rat. Sterile calibrated monopolar electrodes were then advanced in approximately 0.25-mm steps through the guides into the midbrain of each animal. Stimulation was carried out at each step, and the animal's behavior was noted. The stimulation consisted of biphasic square-wave pulses (1-msec half cycle) repeated at a frequency of 62.5 per second (*8*). At least 5 minutes elapsed between trials.

When a biting attack occurred, the site was stimulated at intervals for 1 hour. After this testing for attack was completed, small d-c lesions were made through the electrodes in four cats. These cats were then stimulated through the electrodes at intensities two to three times higher than those previously effective in eliciting attack (*9*). If stimulation still resulted in attack, the lesions were made larger in small steps until attack could no longer be elicited. For the seven other cats, the stimulating electrodes were cemented in place, and these sites were stimulated on subsequent test days (*10*). The positions of the electrodes and the locations of the lesions were verified by studying the brains in sections that were stained alternately for nerve cells and myelinated fibers.

A biting attack similar to that elicited by excitation of the lateral hypothalamus was produced by stimulation of sites in the ventral midbrain tegmentum. As with hypothalamic biting attack, when stimulation began the cat moved swiftly with its nose low to the ground and hair slightly on end and went directly to the rat and bit its head and neck repeatedly, usually killing it with the first few bites. Although the cat would sometimes bite the rat's tail, stomach, or back, biting was ultimately directed to the neck and head. On some trials the cat would circle the test cage several times, ignoring the rat, and then suddenly pounce on the rat and bite it savagely (*11*). This attack was not accompanied by signs of autonomic arousal other than pupillary dilation and piloerection along the tail and midline of the back. The hissing, spitting, growling, baring of teeth, laying back of the ears, and arching of the back—outstanding features of attack elicited from the central gray—were never observed during stimulation of attack sites in the ventral midbrain tegmentum, even at intensities of two to three times the attack threshold. This is in contrast to the quiet form of biting attack elicited from hypothalamic sites, where increased intensity of stimulation usually evokes hissing, growling, flattening of the ears, and full piloerection (*2*). Thus, for the hypothalamus the quiet and affective forms of attack are best considered as two ends of a continuum ranging from little or no display to maximal autonomic activation and display (*12*). In contrast, the quiet biting attack elicited by stimulation of the ventral midbrain tegmentum is always clearly distinguishable from the affective attack and affective defense reaction that result from electrical stimulation of the midbrain central gray and adjacent tegmentum (*1, 4, 13*).

To rule out the possibility that the attack represented a form of eating, there was always a bowl of food as well as an anesthetized rat in the cage. When 13 of the 15 sites were stimulated, the cat ignored the food bowl even when the animal had to pass over the bowl to get to the rat. When the other two sites (1B and 4) were stimulated, the cats came up to the dish, sniffed, and bit at the food, which usually fell out the cat's mouth as it continued to prowl. These data indicate that while there may be some overlap of midbrain sites for the elicitation of eating and attack, the two are by no means inseparable.

At the sites where d-c lesions were made, the attack was eliminated by lesions with diameter of 0.5 to 1.5 mm (Figs. 1 and 2). Cats with lesions no longer approached the rats but showed only a slight degree of alerting, often accompanied by pupillary dilation, in response to stimulation. In some cases the cat would walk about the cage and occasionally sniff at but otherwise ignore the rat and the food. The small size of the lesions capable of eliminating the attack indicates that the spread of

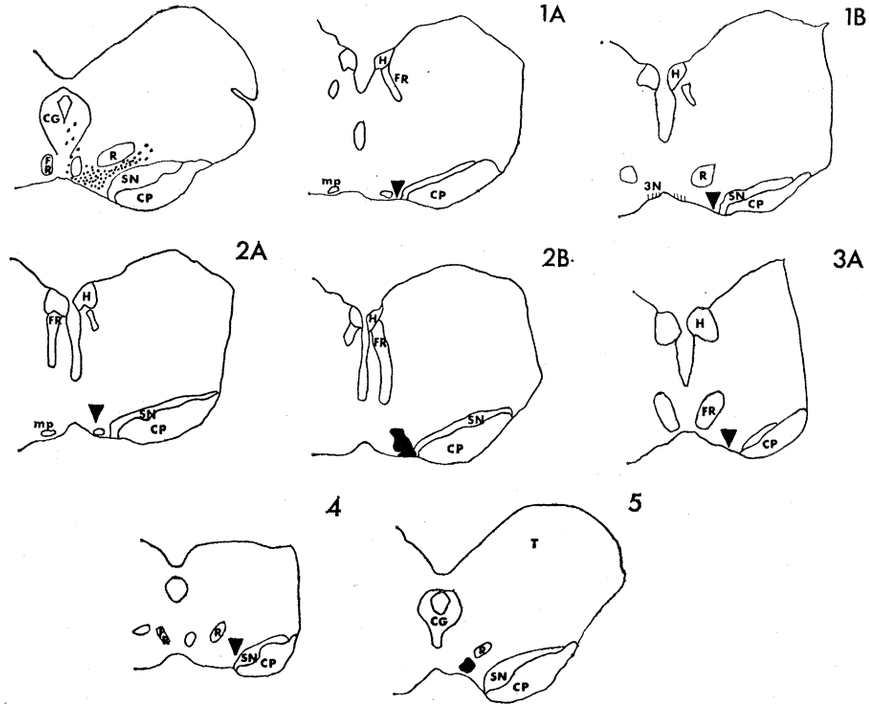

Fig. 1. Midbrain electrode tip locations (triangles) and maximum extents of lesions at attack sites (2B, 5). The animal identification number is to the upper right of each brain section. The pattern of degeneration in the midbrain (upper left section) after a lesion of a lateral preoptic attack site [figure 2A, cat 12099, in (*7*)] corresponds closely with the clustering of midbrain electrodes from which attack was elicited by electrical stimulation; *CG*, central gray; *CP*, cerebral peduncle; *FR*, fasciculus retroflexus; *H*, habenula; *R*, nucleus ruber; *SN*, substantia nigra; *T*, thalamus; and *mp*, mammillary peduncle.

the effective stimulating current was slight.

The ventrolateral zone in the midbrain from which biting attack was elicited extended from the mesodiencephalic junction to the level of the decussation of the brachium conjunctivum (Figs. 1 and 2). The attack sites in the ventral midbrain tegmentum correspond well with the zone (Figs. 1 and 2) where degeneration was observed after lesions were made in hypothalamic attack sites. This indicates that the degeneration descending to the ventral midbrain tegmentum is involved in the mediation of attack behavior (*14*).

Ever since Bard (*15*) showed that spontaneously occurring decorticate rage could not be observed after the brain was sectioned at the caudal end of the posterior hypothalamic area, the notion has persisted that the final common system for the control of all forms of aggressive behavior is at the level of the hypothalamus. However, Ellison and Flynn (*16*) found that neural isolation of the hypothalamus from the rest of the brain caused little change in the pattern of biting attack of cats toward rats and mice. The extensive attack zone that we found in the ventral midbrain tegmentum may account for the ability of cats with a neurally disconnected hypothalamus to make a directed attack. Our results suggest that this midbrain zone, as well as that in the lateral hypothalamus, can function as a center for the integration of the visceral and somatic components of attack behavior.

We have discovered an extensive zone for the elicitation of agressive behavior in that part of the midbrain shown by anatomic techniques to be associated with biting attack. This indicates, at least in the case of aggressive behavior, that the combination of brain stimulation to determine the behavioral function of an area and anatomic methods to trace out associated pathways provides an effective means for delineating the anatomic pathways and structures that mediate the behavior.

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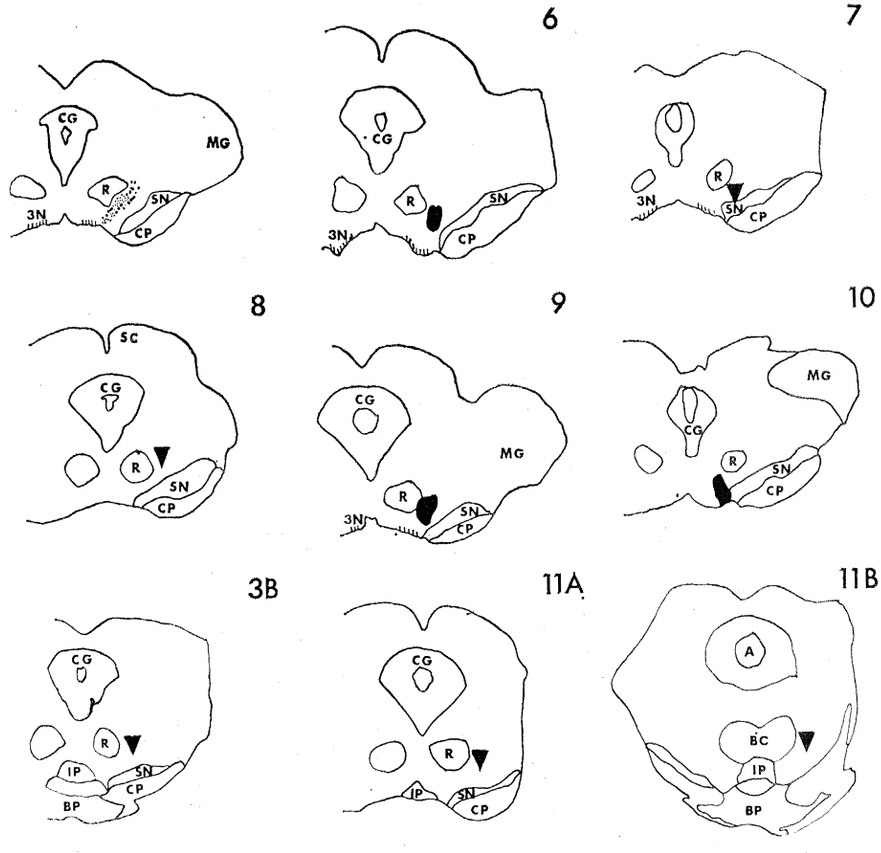

Fig. 2. Midbrain electrode tip locations (triangles) and maximum extents of lesions at attack sites (6, 9, 10). These brain sections are caudal to those of Fig. 1. The animal identification number is to the upper right of each brain section. The pattern of degeneration in the midbrain (upper left section) after a lesion of a lateral hypothalamic attack site [figure 1D, cat brain 01130, in (*6*)] corresponds closely with the clustering of midbrain electrodes from which attack was elicited by electrical stimulation; *A*, aqueduct of Sylvius; *BC*, brachium conjunctivum; *BP*, brachium pontis; *IP*, interpeduncular nucleus; *MG*, medial geniculate nucleus; *SC*, superior colliculus; and *3N*, third nerve.

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8. The peak-to-peak threshold necessary to elicit attack was between 0.1 and 0.6 ma.
9. The lesions were made with a constant-current d-c lesion maker set to deliver either 0.5 ma for 30 seconds or 1.0 ma for 20 seconds.
10. On completion of testing at one of these sites (2B), a d-c lesion was made through the stimulating electrode, and the effects on attack elicited at hypothalamic sites anterior to the lesion were observed.
11. The circling behavior did constitute a stereotyped motor response in that it always was to the side contralateral to the one stimulated. For stimulation at several sites (1B, 3B, 7, 8, and 11B), circling was so fast and vigorous that intensities of stimulation two to three times greater than attack threshold rarely elicited attack.
12. Recent anatomical data support this conclusion. Degeneration in the medial forebrain bundle is most marked in the case of quiet biting attack, whereas degeneration is heaviest in the periventricular system in affective striking attack. However, significant degeneration in the periventricular system is also often observed after lesions at quiet biting attack sites in the lateral hypothalamus [see figure 3, cat 04030, in (*7*)].
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14. Additional data from one cat are in agreement with this conclusion. For cat 2, site B (Fig. 1), the small lesion not only eliminated the attack elicited from that site, but also blocked the attack elicited from ipsilateral hypothalamic attack sites 4 and 6 mm anterior to the lesion. The attack elicited from corresponding hypothalamic attack sites on the side contralateral to the lesion was unaffected. In other cats, control lesions in the substantia nigra and cerebral peduncle did not affect attack elicited from the hypothalamus. While data from only one cat are available, they are consistent with the finding that the degeneration descending to the midbrain after lesions are made at lateral hypothalamic attack sites is primarily on the ipsilateral side (*6, 7*).
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1 March 1972