A stereotaxic map of brainstem areas critical for locomotor responses in a novel environment

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Latency to locomote from an illuminated compartment into a dark compartment was recorded in brain-damaged and control rats. The distribution of lesions sustained by those rats failing to enter the dark compartment within 10 min was plotted on the Massopust atlas. Clearly represented within the resulting map were those brainstem areas occupied by nigro-striatal and pallidofugal pathways.

In connection with a series of experiments on the rodent's preference for a dark compartment over an illuminated compartment (Thompson & LeDoux, 1974; unpublished report), we observed that some rats with bilateral injuries to a circumscribed region of the brainstem (those areas occupied by nigro-striatal and pallidofugal pathways) failed to show any locomotor movement within a novel environment. This observation is of considerable interest since no other bilateral lesions placed elsewhere within the tel-, di-, mes-, or metencephalon yielded this dramatic effect. By virtue of this striking degree of localization, a separate report of these results seemed warranted.

METHOD

Adult male albino rats of the Wistar strain were used. The majority sustained bilateral cortical or subcortical lesions 6 to 20 days prior to testing. The size of the lesions investigated in this experiment was comparable to those reported in a recent study which also canvassed many different cortical and subcortical structures with lesions (Thompson, 1974). During the postsurgical period, each subject was handled and its behavior periodically observed on a black masonite table. Food and water were available at all times.

The apparatus consisted of a two-compartment box which has been fully described elsewhere (Thompson & LeDoux, 1974). The test consisted of placing the subject into the illuminated compartment and observing the subject for 10 min. The latency to enter the dark compartment (scored when the subject placed all four feet within the threshold), the number of exits from the dark compartment, and the total amount of time spent in the dark compartment were recorded. (Only the results concerning the latency measure will be discussed).

RESULTS

Table 1 presents the mean latency scores for all groups and the percentage of subjects from each group that failed to enter the dark compartment during the 10-min observation period.

The various groups of brain-damaged subjects fall neatly into one of four different categories. The first

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Table 1
Mean Latency Scores for All Groups and Percentage of Rats from Each Group that Failed to Enter the Dark Compartment

Group	N	Mean Latency (sec)	Failures
Control	20	54.9	0
Blind	12	47.3	0
Olfactory bulb	5	3.0*	0
Anterior neocortex	4	12.2	0
Posterior neocortex	5	27.8	0
Decorticate	14	14.9*	0
Caudate nucleus	4	20.5	0
Globus pallidus	7	370.3*	57.1**
Cingulate cortex	9	24.6	0
Hippocampus	9	95.6	11.1
Septal area	7	80.8	0
Amygdala	7	24.6	0
Anterior thalamus	5	24.6	0
Ventromedial thalamus	5	482.6*	80.0**
Dorsomedial thalamus	7	36.3	0
Lateral thalamus	5	130.0	0
Ventral thalamus	5	40.4	0
Posteromedial thalamus	12	351.5*	41.7**
Anteromedial hypothal.	8	96.2	12.5
Anterolateral hypothal.	6	76.2	0
Posteromedial hypothal.	11	286.8*	27.3**
Posterolateral hypothal.	8	474.4*	62.5**
Internal capsule	3	600.0*	100.0**
Subthalamus	10	126.6*	0
Ventral tegmental area	7	441.3*	71.4**
Prerubral area	4	353.2*	50.0**
Red nucleus	4	266.5*	0
Substantia nigra	8	275.4*	25.0**
Central tegmental area	7	66.0	0
Superior colliculus	6	18.0	0
Inferior colliculus	3	22.3	0
Rostral mes. ret. form.	8	317.9*	25.0**
Dorsal mes. ret. form.	5	37.2	0
Lateral mes. ret. form.	6	24.3	0
Caudal mes. ret. form.	5	94.0	0
Pontine ret. form.	6	53.5	0
Cerebellum	4	360.0*	0

* $p < .05$ (Mann-Whitney U test)

** $p < .05$ (Fisher exact probability test)

category would consist of those groups showing significantly longer latency scores than the controls and containing subjects that failed to enter the dark

Figure 1. Critical area (cross-hatched) for initiation of locomotor movement shown at 10 frontal levels derived from the Massopust (1961) atlas. (Abbreviations: ac = anterior commissure; AT = anterior thalamus; cg = central gray; ep = entopeduncular nucleus; fm = habenulopenduncular tract; fx = fornix column; gl = lateral geniculate nucleus; gm = medial geniculate nucleus; H = habenula; IC = internal capsule; ip = interpeduncular nucleus; L = lateral thalamus; Im = medial lemniscus; M = medial dorsal thalamus; mt = mammillothalamic tract; oc = optic chiasma; RF = midbrain reticular formation; rn = red nucleus; sc = superior colliculus; sn = substantia nigra; st = subthalamic nucleus; VM = ventromedial thalamus.)

compartment. The second category would consist of those groups showing significantly longer latency scores than the controls but devoid of subjects failing to enter the dark compartment. The third category would consist of those groups that did not differ significantly from the controls in latency scores and the last category would consist of those groups showing significantly shorter latency scores than the controls.

Mapping the critical area for this deficit in the initiation of locomotor movement within a novel environment was based upon the distribution of lesions sustained by those subjects falling within the first category that failed to enter the dark compartment. Figure 1 depicts this critical area which will be named, for convenience, the "critical locomotor initiating area" (CLIA). It will be noted that the caudal extent of the CLIA embraces mainly the subrubral (ventral tegmental) area and the substantia nigra (Figure 1J). At more rostral levels, the CLIA appears to bifurcate, one branch coursing into the posterior thalamus and the second into Forel's fields and the posterior hypothalamus (Figure 1F). Further rostrally, the CLIA converges in a region surrounding the mammillothalamic tract and fornix column (Figure 1D) and then passes laterally into the internal capsule (Figure 1C) and subsequently into the globus pallidus (Figure 1A).

DISCUSSION

The anatomical structures making up the CLIA are generally in agreement with earlier findings on the rat (Balagura, Wilcox, & Coscina, 1969; Robinson & Whishaw, 1974; Smith, 1970; Vanderwolf, 1971) and closely correspond to pathways originating within the substantia nigra (Hedreen, 1971; Ungerstedt, 1971) and globus pallidus (Morgane, 1961).

It is important to note that lesions of the CLIA not only impair initiation of movement but may impair eating and drinking behaviors (Morgane, 1961; Ungerstedt, 1971), pain-escape performance (Runnels & Thompson, 1969), and retention of various learned tasks (Thompson, 1974). This raises the question concerning the extent to which the foregoing disorders are products of a common disturbance. In discussing the nigro-striatal dopaminergic system (which forms a part of the CLIA), Ungerstedt (1971) has suggested that a reduction in general arousal levels may constitute the common disturbance. We are led to speculate that the entire CLIA may serve arousal functions, especially in view of the conspicuous overlap between the rostral portions of the ascending reticular activating system (Starzl, Taylor, & Magoun, 1951) and the CLIA.

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