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SEPARABLE FOREBRAIN SYSTEMS CONTROLLING DIFFERENT MANIFESTATIONS OF SPONTANEOUS ACTIVITY

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Discrete lesions were placed within the dorsal and ventral aspects of the frontal poles of rats and at various levels of two tracts (the medial forebrain bundle and the inferior thalamic peduncle) which relate this area with the diencephalon. The animals were then tested for wheel and stabilimeter activity during ad-lib and food-deprivation conditions. The results indicated that: (a) a discrete system running from the ventral frontal cortex into the medial forebrain bundle inhibits wheel running, and (b) the substrates of stabilimeter activity are more diffuse with the inferior thalamic peduncle playing a central role. From this, it appears that there are at least two overlapping neural systems in frontal cortex of the rat which have very different behavioral functions.

Previous work has shown that bilateral removal of the frontal pole area in the rat produces two types of activity change: (a) an immediate and chronic increase in daily wheel running (Richter & Hawkes, 1939), and (b) a potentiation of treatments (e.g., starvation, amphetamine) which normally increase stabilimeter cage activity (Campbell & Lynch, 1969; Lynch, Ballantine, & Campbell, 1969). This latter result has been interpreted in terms of a reticular loop hypothesis first advanced by Hugelin and Bonvallet (cf. Hugelin, Bonvallet, & Dell, 1959). According to this explanation, the frontal cortex normally reacts to dampen increases in the level of reticular (and behavioral) arousal caused by a wide range of internal changes. Interruption of the reticulo-cortico-reticular loop would be expected to greatly amplify the effects of conditions which act primarily through the reticular formation.

This type of negative feedback model accounts for the effects of frontal lesions on stabilimeter cage activity, but it does not readily explain the chronic increases in wheel running which occur without experimental treatment other than the lesions. One suggestion for resolving this discrepancy is that wheel-running and stabilimeter cage activity have different anatomical substrates both of which have frontal cortical components. This is given credence by recent anatomical and neurobehavioral studies showing that the frontal lobes across a range of species contain heterogeneous subdivisions (cf. Divac, Rosvold, & Swarczbar, 1967; Nauta, 1964) and the extensive behavioral evidence dissociating wheel and stabilimeter activity. The present experiments were designed to test the hypothesis that overlapping neural systems are present in the rat's frontal poles and that simultaneous destruction of these accounts for the divergent activity results reported above.

EXPERIMENT 1

Based on neuroanatomical and behavioral studies, several authors have proposed that the dorsal and ventral ("orbital") surfaces of the frontal lobes of primates may be functionally distinct (Akert, 1964; Divac et al., 1967). This dichotomy, while probably not directly applicable to rodents, is

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made immediately relevant by the finding of Zubek and De Lorenzo (1952) that total removal of the frontal poles causes increased running but dorsal frontal cortical lesions alone do not. In the first experiment, the effects of dorsal and ventral frontal lesions were compared in stabilimeter cages and running wheels during ad-lib and food-deprived conditions.

Method

Subjects. Male rats of the Sprague-Dawley strain purchased from Perfection Breeders, Douglassville, Pa., were used. The animals weighed 275-325 gm. at the beginning of the experiment.

Apparatus. The running wheels used were standard Wahmann activity cages (Wahmann Manufacturing Co., Baltimore, Md.); the stabilimeter cages have been described elsewhere (Campbell, 1964). Briefly, the latter were 15 × 7 × 8 in. wire-mesh cages, mounted on a central axle with a microswitch located at one end of the cage, which opened or closed each time the animal moved from one side of the fulcrum to the other. Food and water were suspended in containers over the axle to avoid unbalancing the cage. The cages were kept in two temperature-controlled soundproof rooms (ambient temperature 20° C.). Servicing of cages, feeding, and weighing of Ss was carried out during a 2-hr. interval from 10-12 A.M. during which activity was not recorded.

The rooms were kept on a 12-hr. day-night cycle (lights on at 6 A.M.) for all experiments. In this situation, Ss were only exposed to sounds indigenous to the rooms—fans, other rats, etc.—and were effectively isolated from the laboratory environment and recording equipment.

Procedure. For the stabilimeter studies, the rats were given one of four operations (dorsal frontal ablations, n = 9; ventral frontal lesions, n = 8; posterior cortical lesions, n = 10; or sham operations, n = 10) and placed for 1 wk. in individual colony cages. During this period weights were taken daily and pellets were scattered about the cage floor. At the end of this time, the animals were transferred to stabilimeter cages and given 3 days of ad-lib food and water followed by 5 days of food deprivation.

Unlike stabilimeter activity, wheel running takes several days to reach a stable level and varies greatly between animals. Therefore, the rats were given 10-14 days of preoperative wheel running and animals which ran more than 2,500 or fewer than 100 revolutions per day were discarded. In this way, a relatively homogeneous group of runners was obtained and it was no problem to match the experimental groups for activity. As with the stabilimeter experiments, the groups were also equated for preoperative body weight.

After this adaptation period, the wheel-housed animals were assigned to one of three groups: dorsal frontal lesions (n = 5); ventral frontal lesions (n = 7); and sham operated (n = 15). Sham-operated animals were run in wheels throughout the course of the three experiments in this report and are grouped together (consequently the comparatively large n) rather than being arbitrarily divided up among the separate studies. Following surgery, the rats were returned immediately to their cages and given a 14-day period of wheel running with ad-lib food and water.

Animals housed in running wheels die after shorter deprivation periods than do those kept in stabilimeters (Campbell & Lynch, 1963) and because of the histological complications consequent to death, starvation was limited to only 2 days for the wheel groups.

At the end of the deprivation period, cardiac perfusions with saline followed by 10% formalin were performed on all subjects. The brains were removed and serial coronal or horizontal slices were made at 50 μ on a freezing microtome. Representative sections were then stained with cresyl-violet, photographed, and reconstructions made with a microprojector.

Surgical Procedure. All surgery was performed under sodium pentobarbital anesthesia and the lesions (bilateral in all cases) were made using an aspirator and fine glass pipettes. The difficulties involved in performing discrete replicable lesions on the rat's lissencephalic cortex are formidable, and these are greatly compounded in attempting to reach relatively inaccessible areas such as the ventral aspect of the frontal poles. For the present studies, skull markings were used as guides for all dorsal lesions (anterior and posterior); the ventral frontal lesions were made by opening the skull at the level of the bregma and angling the pipette forward towards the olfactory bulbs. This procedure necessitated making an opening through the dorsal frontal cortex but the use of small diameter pipettes kept this damage to a minimum.

In doing the dorsal lesions, care was taken not to penetrate through the corpus callosum. Posterior lesions were made midway between lambda and bregma and the midline ("cingulate") cortex was avoided. Frontal lesions were placed in front of the coronal suture up to the juncture of the frontal and nasal bones and extended to the lateral wall of the cranial cavity. For sham operations, the skull overlying the frontal cortical area was removed but the dura was left intact.

Results

Histology revealed that the dorsal frontal lesions were of similar size and shapes; posterior cortical lesions were more irregular but about equivalent in size to the anterior lesions. There was considerable variation between the ventral lesions although the lower portion of the frontal poles was damaged in every case; the dorsal surfaces of the poles and the cortex
overlying the caudate received varying degrees of damage (Figure 1).

The stabilimeter data for these groups are plotted in the right hand panel of Figure 2. Dorsal lesions had no effect on activity during ad-lib feeding conditions but did potentiate the activity increase during starvation. Both the absolute and percentage increases in activity shown by the frontal animals from last ad-lib to fifth food-deprived day were significantly greater than the changes for either sham or posterior-lesioned rats \((p < .02, \text{Mann-Whitney } U \text{ test})\). Damage to the ventral half of the lobes was followed by a similar syndrome but without the consistency seen after dorsal lesions. Of the eight rats tested, three gave no indication of hyperactivity during starvation while the remaining five showed varying degrees of the potentiation effect. Consequently, the differences between ventrally lesioned animals and sham controls, while suggestive, is not statistically significant. These rats were, however, clearly more active than posterior-lesioned rats during starvation. The large variance in the effects of the ventral lesions may reflect the widely different morphological characteristics of these lesions. None of the animals with posterior lesions showed any sign of being abnormally active during ad-lib or food-deprived conditions.

A previous study (Campbell & Lynch, 1969) demonstrated that rats with total ablation of the frontal poles are nearly 10 times as active during the final stages of food deprivation as are sham controls.

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**Fig. 1.** Examples of the various types of frontal cortical lesions used in the present experiments. (The left-hand panels show successive anterior to posterior coronal slices through a representative brain with a dorsal lesion; the middle panels are dorsal to ventral horizontal sections through a brain with ventral frontal lesions. The panels to the right are coronal sections from three different brains with electrolytic ventral lesions.)
The animals in the present experiments with subtotal frontal lesions were slightly more than twice as active as sham operatives in the same period. Together with the finding that posterior lesions had little effect on this form of activity, these results suggest that: (a) The cortical system regulating stabilimeter activity during starvation is diffusely organized but localized within the frontal poles, or (b) the tracts relating this system to subcortical regions transverse both dorsal and ventral segments of the frontal pole area.

To summarize the stabilimeter cage data, rats with dorsal cortical lesions were more active during severe starvation than sham-operated or posterior-lesioned animals; ventral frontal lesions gave results that were in the same direction though less consistent than dorsal lesions. Neither lesion produced effects that were comparable in magnitude to those observed earlier following total removal of the frontal poles.

The left-hand panel of Figure 2 shows the influence of partial frontal lesions on daily running activity. Dorsal frontal lesions did not affect wheel activity in any of the five animals tested, (thus replicating Zubek & De Lorenzo, 1952) while large ventral lesions (which again varied considerably from rat to rat) increased activity in six of seven animals. Mean running increased by more than a thousand revolutions per day in the ventrally lesioned rats (last 3 preoperative days compared with the last 5 postoperative ad-lib days) while the average activity of the sham animals remained unchanged ($p < .002$, Mann-Whitney $U$ test; Table 1). Unlike the stabilimeter results then, these data indicate that the dorsal and ventral aspects of the rat's frontal poles are functionally distinct.

The large variance between ventral lesions and the damage to dorsal cortical fiber projections suggested that more discrete lesions would be required before definitive statements on the role of the ventral surface in wheel activity would be possible. Consequently, 11 additional rats were tested under conditions identical to those described above for the wheel-housed rats except that they received discrete electrolytic lesions of the ventral cortical area. These were made with a Lehigh Valley dc lesion maker attached to an anodal .009-diam. electrode, which was Teflon-coated except for the tip, and large rectal cathode (further details are given in the next experiment). Current was 1 ma. for 30 sec. and the lesioning electrode was
TABLE 1

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Preop-running</th>
<th>Postop-running</th>
<th>M change</th>
<th>M % change</th>
</tr>
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<tbody>
<tr>
<td>Shams</td>
<td>15</td>
<td>1165</td>
<td>1158</td>
<td>-7</td>
<td>13</td>
</tr>
<tr>
<td>Ventral</td>
<td>7</td>
<td>939</td>
<td>2085</td>
<td>+1146**</td>
<td>99**</td>
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<tr>
<td>(aspirated)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dorsal</td>
<td>5</td>
<td>972</td>
<td>977</td>
<td>+5</td>
<td>12</td>
</tr>
<tr>
<td>frontal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventral</td>
<td>7</td>
<td>1161</td>
<td>2145</td>
<td>-984*</td>
<td>76*</td>
</tr>
<tr>
<td>(electrolytic)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior MFB</td>
<td>11</td>
<td>1165</td>
<td>1856</td>
<td>+691*</td>
<td>95*</td>
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<tr>
<td>Posterior MFB</td>
<td>11</td>
<td>1010</td>
<td>2842</td>
<td>1832**</td>
<td>228**</td>
</tr>
<tr>
<td>ITP</td>
<td>6</td>
<td>897</td>
<td>905</td>
<td>+8</td>
<td>17</td>
</tr>
</tbody>
</table>

* Mean of last three preoperative days.

** Mean of last 5 postoperative ad-lib days.

* p < .02 compared with sham data; Mann-Whitney U test.

** p < .002.

lowered three times bilaterally at .5-mm. intervals along an anterior-posterior plane 1.0 mm. behind the junction of the frontal and nasal skull bones.

Figure 1 shows examples of the lesions made with this technique. Damage was largely confined to the ventral frontal pole area leaving dorsal cortex and attendant projections intact. Seven of the rats received bilateral lesions; four other sustained unilateral or grossly asymmetrical lesions. All animals showed increases in wheel running following surgery although in the case of two of the unilaterally lesioned rats these were transitory. The mean activity of the rats who had sustained bilateral lesions increased by 1,100 revolutions per day (using the preoperative/postoperative comparison described for the aspirated lesion groups), a change that was clearly different from the effect of the sham operation (p < .01).

**EXPERIMENT 2**

The previous experiment demonstrated that the cortical neuronal pools which govern wheel running overlap but are not coincident with those involved with stabilitometer activity. In this experiment and the one which follows, we examined the possibility that these subdivisions of the frontal poles exert control over these behaviors by different neurological pathways. This experiment investigated the effects of severing the medial forebrain bundle (MFB) which has been shown to be a link between frontal cortex and hypothalamus in primates (Wall, 1951), cats (Nauta, 1964), mice (Valverde, 1963), and rats (Leonard, 1969). Research using the recently developed fluorescent histochmistry has suggested that the medial forebrain bundle may also be a major source of frontal afferents (Fuxe, 1965).

**Method**

The subjects, apparatus, and testing procedures were identical to those used in the first experiment.

Surgical procedure. Electrolytic lesions were made with a Lehigh Valley Model 1644 dc lesion maker connected to a stereotaxically placed anodal electrode and rectal cathode. The current used was 1 ma. for 30 sec. applied to electrodes made of Teflon-coated platinum-iridium wire of .009 in. outside diameter.

The intent of this experiment was to place discrete lesions at two levels of the medial forebrain bundle; rostral and caudal to the crossing of the anterior commissure. This strategy encompassed a number of advantages: (a) Cortico-hypothalamic fibers apparently enter the MFB at several levels (see Valverde, 1963), (b) inputs from forebrain structures do not always penetrate the MFB at the same rostrocaudal points—e.g., the stria terminalis enters behind the crossing of the anterior commissure while the precommissural fornix travels in Broca's bands in front of this point and (c) destroying the bundle at different points would establish more firmly its unique role in the behaviors in question.

Eleven rats with caudal and eleven with rostral MFB lesions were tested in the wheels; the controls for these animals were described in Experiment 1. Three groups were run in the stabilitometer cages: caudal MFB (n = 10), anterior MFB (n = 6), and sham (n = 9).

**Results**

Examples of the types of MFB lesions used are shown in Figure 3. Although it is not evident from these figures, the electrode tracts for many of the lesions passed through the medial edge of the internal capsule causing some damage to these fibers. The anterior commissure also received occasional slight damage. The posterior group of lesions all fell within
Fig. 3. Reconstruction of a series of transverse sections through representative large and small anterior (left-hand panels) and posterior (right-hand panels) medial forebrain bundle lesions used in the present studies. (Note that the sections are numbered [1-8] in an anterior-to-posterior sequence and that four different lesions are pictured.)

the MFB from the level of the paraventricular hypothalamic nuclei (caudally) to beneath the crossing of the anterior commissure immediately rostral to the descending columns of the fornix. Individual lesions covered a much more limited rostrocaudal extent and it was possible to subdivide this group (posterior MFB) into rats with lesions which were completely behind the commissure and those which were under the commissure and extended back only to the level of the descending limb of the stria medullaris. The anterior MFB group lesions were located between the crossing of the anterior commissure and the level of the diagonal bands.

Lesions at both levels of the MFB caused increased wheel running during ad-lib conditions (Figure 4) but the caudal lesion was the more effective. Comparing pre- and postoperative activity (as described in Experiment 1) emphasizes the extent to which the caudal MFB lesions were the more effective in increasing wheel activity. The anterior MFB group increased their running by 685 revolutions per day while the posterior group showed a change of 1,832 revolutions. Both increases were significantly different from the changes shown by the sham group ($p < .02, p < .002$, respectively; Table 1).

There are a number of possible explanations which could account for the greater increase in activity shown by the rats with posterior MFB lesions. The medial forebrain bundle becomes increasingly diffuse as it proceeds rostrally and it is possible that the more anterior lesions were destroying a smaller proportion of the tract than the posterior lesions. Another hypothesis might be that the posterior lesions were destroying an afferent hypothalamic connection which was involved in running and which entered the MFB behind the anterior lesions. The stria terminalis and ventral amygdalo-fugal pathway, for example, have significant effects on hypothalamic nuclei (Murphy, Driefuss, & Gloor, 1968) and both enter the MFB at a point caudal to most of the anterior lesions used in this study. Subdividing the posterior MFB group as described above
into animals with lesions completely below the commissure ($n = 5$) and those with lesions which fall beneath and behind the commissure ($n = 6$) indicated that damage at either level caused marked wheel running increases.

Stabilimeter activity was unaffected by lesions in the anterior MFB while MFB destruction below and behind the commissure had ambiguous effects (Figure 4). The caudal lesions produced marked enhancements of activity in three of the rats during ad-lib and deprived conditions, the remaining eight animals appeared largely unaffected. The median data for the posterior MFB group are plotted in Figure 4 to emphasize the degree to which these lesions were without effect on the majority of the rats. When the posterior MFB group was subdivided as described above, only those lesions that were beneath the anterior commissure ($n = 4$) produced stabilimeter activity effects.

**Experiment 3**

The previous study investigated the effects of severing the frontal cortical connection with the hypothalamus, the MFB; in this experiment the consequences of destroying a second diencephalic link, the inferior thalamic peduncle (ITP), are examined. There are two acknowledged thalamic origins of fibers to the frontal cortex in rats, the dorso-medial nucleus and the thalamic nonspecific system. The frontal projection of the latter has only recently been convincingly demonstrated neuroanatomically (Scheibel & Scheibel, 1967) although electrophysiological data (Jasper, 1960) had earlier indicated its presence. In view of the massive literature relating the brainstem reticular formation to the nonspecific thalamic system, the ITP must be a critical component in any proposed reticulocortical system.

**Method**

Subjects, apparatus, and testing procedures were identical to those used in the previous two studies.

*Surgical procedure.* Inferior thalamic peduncle lesions were made by the technique used in Experiment 2. These were oriented so as to fall immediately lateral to the lateral ventricle at about the level of the crossing of the anterior commissure, the pathway of the ITP for rats as described by the Scheibels (1967). A slight variation in coordinates was made in order to provide some deviation between animals in the anterior-posterior dimension. Caudate lesions were made 2 mm. lateral to the ventricle, using the same current and anterior-posterior coordinates that were employed for the ITP group.

Eleven rats with ITP lesions were tested in
stabilimeter cages and seven others formed the wheel group. As controls for the stabilimeter-group, six caudate-lesioned and four sham 8s were tested. The wheel control group was described in Experiment 1.

**Results**

Histological analysis revealed that the lesions destroyed the intended areas. Figure 5 shows examples of ITP lesions. Note that the damaged area is extensive in the dorso-ventral direction but is relatively restricted medio-laterally. The effects of these lesions on spontaneous activity is summarized in Figure 6 (the sham- and caudate-lesioned animals were nearly identical in their activity scores throughout the experiment and are consolidated in the graph). The ITP lesions greatly augmented deprivation-induced stabilimeter activity in every animal but did not affect any aspect (ad lib or food deprived) of spontaneous wheel running. The potentiation effect was notable for its magnitude and consistency; the results were more striking, for example, than those obtained with the much larger cortical lesions. The consistency of this effect is indicated by the plot of the median data for the stabilimeter activity of the ITP-lesioned rats in Figure 6. Note, however, that these lesions did not augment the effects of starvation on daily wheel activity.

**Discussion**

From these experiments, we are able to draw some conclusions about the forebrain mechanisms controlling spontaneous ac-
activity. Most striking, perhaps, is the finding that the two forms of activity measured are regulated by different telencephalic systems. This is evident from an examination of Figure 7 which summarizes the results of the three experiments. Large increments in wheel running are obtained following lesions in the ventral aspect of the forebrain from the basal frontal area back into the medial forebrain bundle and anterior hypothalamus. Destruction anywhere else was without conspicuous effect.

Fig. 6. The effects of lesions in the inferior thalamic peduncle on stabilimeter and running wheel activity during ad-lib and food-deprived conditions. (For each group the mean data were used, the median data for the ITP group in stabilimeters is also plotted.)

Fig. 7. Summary of the effects of various forebrain lesions on two forms of spontaneous activity during ad-lib and food-deprived conditions. (Shaded areas are locations where increases in ad-lib wheel running [left side] or potentiation of deprivation-induced stabilimeter activity [right side] were obtained; vertical stripes denote inconsistent results.)
Together with studies showing that lesions of the lateral hypothalamus abolish running (Gladfelter & Brobeck, 1962) while electrical stimulation there elicits it (Rosenquist & Hoebel, 1968), these data indicate a cortico-hypothalamic route via the MFB by which the frontal cortex suppresses wheel running.

The differences found in wheel activity following dorsal and ventral frontal lesions suggest that the rat's frontal poles are differentially organized, a finding that is compatible with neuroanatomical evidence that dorsal and ventral distinctions can be made for dog, cat, and primate frontal lobes (Akert, 1964). Recently, a similar anatomical dichotomy has been drawn for the more primitive rat brain. In that research, the anterograde degeneration method revealed a large projection from the ventral area of the poles (dorsal to the rhinal sulcus and tubercle) into the MFB and hypothalamus. It was also found that the dorsal surface had a very different projection pattern (Leonard, 1969).

The physiological processes which regulate stabilimeter activity do not appear to be nearly so discrete as those governing running. As the right hand panel of Figure 7 shows, lesions at a number of sites influenced daily activity in this device particularly during food deprivation. However, by far the greatest effect was obtained within the inferior thalamic peduncle. The hippocampus is also involved in this form of activity (but not in wheel running) during both ad-lib and food-deprived conditions (Lynch & Campbell, 1968). Subtotal frontal lesions potentiated the deprivation-induced increases in stabilimeter activity but to a lesser extent than total lobectomy. This suggests that the whole of the frontal pole area may be involved in the control of deprivation-linked activity; relevant to this is the evidence that the ITP terminates diffusely throughout the frontal area (Scheibel & Scheibel, 1967). Lesions within the system controlling running, with the possible exception of the region beneath the anterior commissure, have ambiguous effects on stabilimeter cage activity. And even this exception requires considerable qualification: (a) The electrode tracts of these lesions penetrated the ITP causing varying degrees of damage and, in view of experiments severing the ITP, this could have caused much of the observed effect; (b) the area below the anterior commissure is a tangle of fiber tracts and, considering that lesions in the MFB above this point had only inconsistent stabilimeter effects, it is possible that one of these systems was central to the obtained result. At any rate, the failure to obtain stabilimeter effects in the anterior MFB group combined with the uncertainty of the ventral frontal results indicates that the system controlling wheel running is not central to the regulation of stabilimeter activity.

A further distinction between these types of activity is suggested by the fact that lesions which potentiate the effects of starvation on stabilimeter activity (ITP, dorsal frontal cortex, and hippocampus) do not augment its action on daily wheel running. The most plausible explanation for this is that food deprivation increases wheel activity by one route and stabilimeter activity by another.

The finding that the telencephalic systems regulating wheel and stabilimeter activity are distinct is not surprising in view of the extended research showing that these behaviors respond differentially to the same experimental manipulations (Strong, 1957). Campbell (1964), for example, reports that running increases as a function of days of water deprivation but the number of stabilimeter crossings actually decreases. In general running is more sensitive to internal variables (e.g., body temperature, starvation, estrus); stabilimeter activity, on the other hand, appears to be more responsive to environmental events (e.g., Teghtsoonian & Campbell, 1960).

Attempts to link these behavioral findings with the present results can only be tentative but certain experimental hypotheses seem warranted. The neural substrates of running are part of the brain regions involved in the production of responses, both physiological and behavioral, to changes in internal homeostatic balance (temperature, heart rate, etc.).
The hypothalamus has long been assigned this function and there is considerable evidence, at least in primates, that orbito-frontal cortex participates in this role via the MFB (cf. Kaada, 1960). Whether the ventral frontal system described in our results acts on the hypothalamic mechanisms controlling biological balance (which running would respond to) or more directly on the response-organizing properties of this area (cf. Glickman & Schiff, 1967), is unknown.

The forebrain system controlling stabilimeter activity (ITP, frontal pole area, and possibly MFB) has been shown by the work of Lindsley and his associates (Skinner & Lindsley, 1967; Velasco & Lindsley, 1965) to be central to at least one electrophysiological manifestation of behavioral suppression, cortical spindles. Other workers have linked the ITP-frontal cortical system with inhibition of reticular facilitation of motor responses (Sauerland, Nakamura, & Clemente, 1967). It thus seems reasonable that the stabilimeter changes following ITP and frontal lesions reflect the disruption of a system regulating changes in the nonspecific arousal formation, the earlier described reticulo-cortical “loop.”

REFERENCES


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