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KARL H. PRIBRAM*
LAWRENCE KRUGER†
FRANKLIN ROBINSON‡
A. J. BERMAN§

Department of Neurophysiology, The Institute of Living, Hartford, Connecticut, and Laboratory of Physiology, Yale University School of Medicine

THE EFFECTS OF PRECENTRAL LESIONS ON THE BEHAVIOR OF MONKEYS¶

Professor Fulton's important contributions to the study of the precentral motor cortex need no reviewing. At the time the studies here presented were undertaken in his laboratory, certain questions stimulated by his work repeatedly entered our discussions. This report is addressed toward some of these questions.

Bucy, in the introduction to the recent monograph on the precentral motor cortex, makes the following statement: "The editor regards the precentral motor cortex as the principal efferent or effector cerebral cortical mechanism by which the brain expresses its activity through the skeletal musculature."[3] Implicit in this statement is the suggestion that all learned behavior finds expression through efferents leaving the precentral region; that afferents relay signals to the "primary projection" cortices whence internuncials lead to the "association areas" which, in turn, play upon the precentral cortical areas. To be validated, this viewpoint must be supported by the following evidence: *anatomical,* showing a preponderance of separation between afferent and efferent tracts in the cerebrum; *electrophysiological,* showing a minimum of overlap between those areas responding (by "evoked potentials") to peripheral stimulation, and those areas which, when electrically excited, give rise to peripheral responses; *behavioral,* showing that learned behavior is impaired by injury to the more-or-less "final" common path. This paper deals with some experiments in the last category. The

* Chairman, Department of Neurophysiology, Institute of Living; Lecturer in Physiology, Yale University School of Medicine.

† Fellow in Public Health, Department of Physiology, Johns Hopkins University School of Medicine, Baltimore, Maryland.

‡ Chief of Neurosurgery, St. Raphael Hospital, New Haven, Connecticut; Clinical Instructor in Neurosurgery, Yale University School of Medicine.

§ Department of Neurosurgery, Brooklyn Jewish Hospital, Brooklyn, New York.

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discussion will be concerned, as well, with experiments in the other categories.

Regarding the specific effects of lesions of the precentral cortex on behavior, three conceptions need clarification. The data giving rise to these conceptions are, in brief, as follows: a lesion of the precentral cortex interferes with patterned muscular contraction in some situations but these same or approximately similar patterns may be observed intact in other situations. Thus, an animal may fail to grasp a nail which holds together a box containing a peanut but grasps an equivalently thin wire composing the side of a cage while climbing; a patient may fail to close his fist on a tennis ball when asked to grasp it but will turn a doorknob in order to escape from a ward during a conflagration. Such data have given rise to two conceptions popular in clinical neurology: that the precentral cortex is implicated in "skilled movement"; that the precentral cortex is implicated in "voluntary movement." A third conception emphasizes the lack of effect of precentral lesions on the solution of problems provided some movement is possible. This conception looks, therefore, for an alternate approach to the specification of the relationship of the precentral areas to behavior and characterizes the impairment as an involvement of "postural mechanisms."[9] In order to estimate the relevance of each of these conceptions, we reinvestigated the effect of precentral lesions on the patterns of response under several conditions. Opening a hasp box to obtain a peanut, discrimination of visually presented patterns, and the delayed reaction were used as quantitative behavioral indices. Neurological examination and observations of the animal's activity in his living quarters and in a relatively free environment provide background material against which the experimental data can be interpreted.

Some questions regarding the nature of the precentral lesions affecting behavior were also asked. Does locus of lesion within the precentral areas make a difference? Is extent of lesion an important factor? Is the decrement in performance of a limb contralateral to a lesion increased when that lesion is made bilateral? Is the effect of lesion dependent on removal of tissue or the result of "irritative" scar at the borders of the removal? The experiments reported suggest answers to these questions.

MATERIALS AND METHODS

Nine immature rhesus monkeys were used in these experiments. Eight of these animals received one or another resection of the precentral agranular cortex, the ninth was given multiple implantations of aluminum hydroxide cream over this cortex.

SURGICAL METHODS

Under intraperitoneally administered barbiturate anesthesia and using aseptic technique, a frontal osteoplastic craniotomy was performed through a coronal incision. The bone flap was hinged on the temporal muscle, the dural flap based on the sagittal sinus. The frontal cortex was exposed from the arcuate to the intraparietal sulcus, from the midline to the Sylvian fissure. Subpial ablation of the appropriate cortex was carried out through a pial incision and with a 19-gauge needle suction tip. The anterior removals included the posterior bank of the arcuate sulcus; the posterior removals included the anterior bank of the central fissure. All removals extended into the superior bank of the calloso-marginal sulcus. The extensive lesions involved all of these "buried" areas. Hemostasis was accomplished by temporary packing with cottonoid pledgets and waiting, rather than by cautery. For the "irritative" lesion, aluminum hydroxide cream was placed in five one-centimeter silver discs and these were distributed over the lateral and medial frontal agranular cortex and the dura closed tightly over them. Interrupted silk closure was routine. Bilateral procedures were carried out in two stages, separated by a 7- to 8-month interval.

BEHAVIORAL METHODS

Experimental subjects were observed in their living cage at least twice a day for 15 minutes and in a large enclosure once a day. They were observed in a relatively free situation; in addition, their reaction to threat and to feeding were noted. Their manipulations of food, of sticks, of the cage and enclosure walls during climbing, and their locomotion were recorded. Neurological examinations were performed on the animals while they were restrained in a neurological chair. At least two observers compared their findings in most instances.

Quantitative behavioral measures, made in a modified Wisconsin general testing apparatus designed for primates,[15] included timing of the opening of a simple latch box. This box, approximately 5 by 8 cm. is closed by a sliding metal lid which is locked by a 2" nail inserted through the lid into the side of the box. To open the box (which contains a peanut during the test trials) the monkey has to grasp the nail, pull it out, push the lid back, and take the peanut from the box. These maneuvers have to be performed in the order stated to accomplish the trial. The mean of the time taken in 30 consecutive trials was used as an index of performance. Thirty trials per day were given until stable performance was reached preoperatively; postoperatively, 30 trials per day were given for five days following surgery and then five days each month. Motion pictures were taken both pre- and postoperatively in this situation, and these were studied when shown at regular and slow motion speed.

Another quantitative behavior measure recorded performance on the classical delayed reaction task. In this situation *two* cups appear before the monkey. Into one of these (*which* one is determined by a random sequence) a peanut is dropped in full view of the animal. Then an opaque screen is lowered, hiding the cups from view for 10 seconds. When the screen is again raised, the animal has to choose between two identical appearing, closed cups. Correct choice depends on the monkey's remembering which of the two cups had been baited 10 seconds before. The subjects were trained beginning two weeks postoperatively until they reached a performance level of 90 per cent on a hundred consecutive trials. The number of trials required to reach this criterion was compared with the mean of the number of trials taken by 35 unoperated monkeys tested in the same manner.

Visual discrimination was also tested. This task utilized the same boxes as those used in the delayed reaction, but in this instance the boxes were closed by covers on one of which a black plus sign was painted, on the other, a black square. The peanut was invariably concealed in the box marked "plus" but the position of the "plus" was varied from box to box in random sequence. Thus, the task for the monkey was to choose the box marked plus; the numbers of trials taken by the operated animals to accomplished this in 90 out of 100 consecutive trials was compared with the mean score taken to reach the same criterion by 44 unoperated control subjects.

ANATOMICAL METHODS

Following the completion of the neurological and behavioral examinations, all the animals were sacrificed, the brains perfused with saline and formalin, dehydrated in alcohols, and imbedded in celloidin. In animals 2400 and 2345 the operated region was exposed under ether anesthesia and excitable points were identified and mapped in cortex and exposed white matter. Serial sections at 25 micra, were made, every tenth section saved and every other saved section stained with thionin. From these sections the extent of lesion was orthogonally reconstructed onto graph paper; reconstructions and representative cross-sections demonstrating depth of resection are presented in Figure 1. Retrograde thalamic degeneration was plotted and is also shown in this figure.

RESULTS

EXPERIMENT 1. UNILATERAL LESIONS

Five monkeys were tested preoperatively, operated upon, and retested. Two of these animals (2428 and 2427) received extensive unilateral resections of the dorsolateral, frontal, agranular cortex; in one animal (2376), the unilateral resection was restricted to the posterior; in another animal (2434), to the anterior portion of the frontal agranular cortex. A fifth animal (2418) received a resection of the greater portion of the frontal agranular cortex, including that of the anterior cingulate gyrus.

A preliminary report of the results of the neurological examinations has been presented elsewhere.[1] Observations of the movements of the animals in the home cage and the large enclosure under "free" conditions and in feeding and threatening conditions are summarized; performance in the formal testing situation is described in detail below.

Subtotal resection of the frontal agranular cortex (2376 & 2434)

In the free situation, a paucity of spontaneous and associated movements of the extremities contralateral to the lesion was noted for two weeks to one month postoperatively. Anterior lesions affected especially the movements of the proximal musculature: movements about the shoulder and hip were

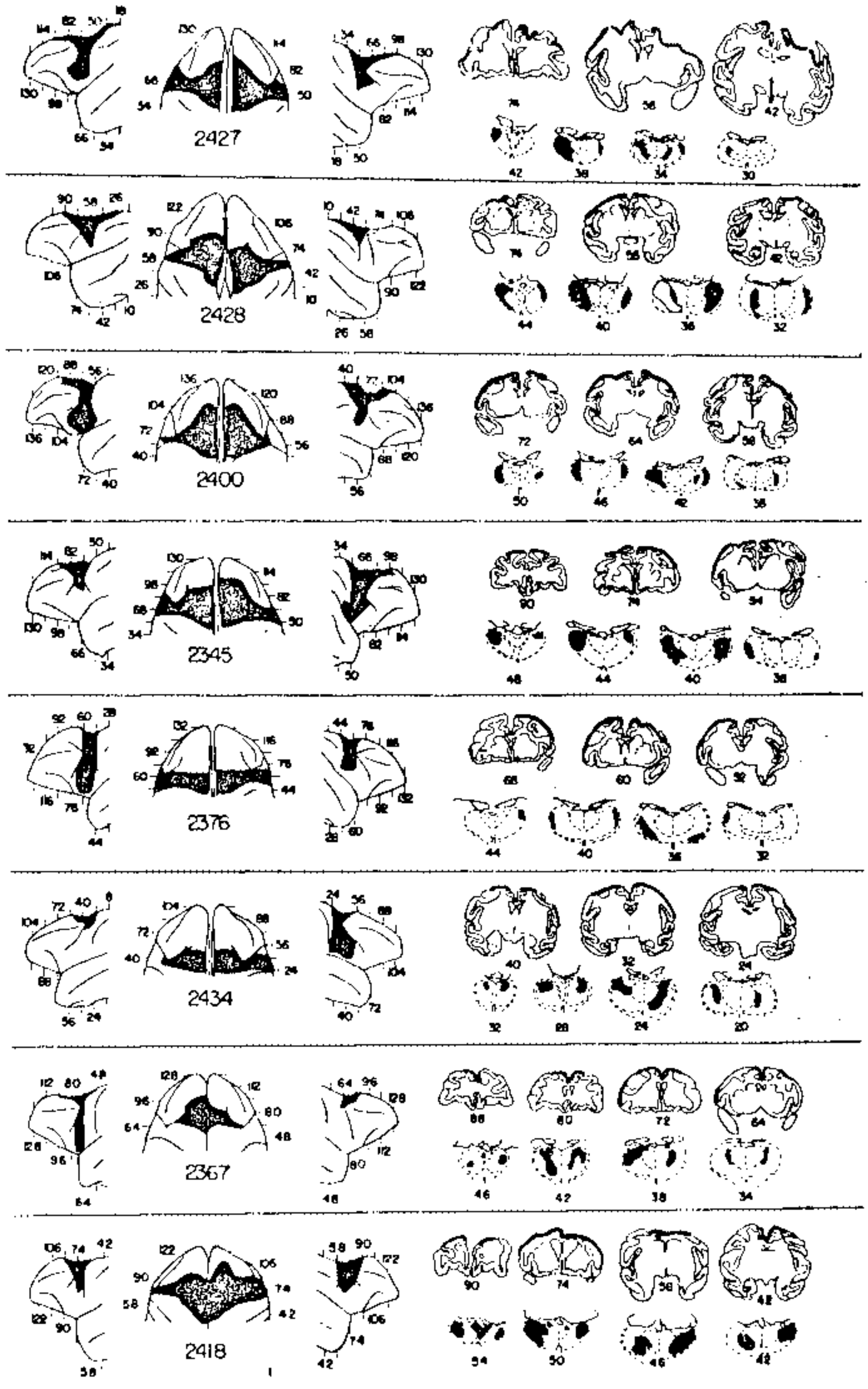

FIG. 1. Reconstructions and representative cross-sections through lesion and thalamus of brains of the animals used in this experiment. Black indicates locus and extent of ablation of cortex (left side of figure) and locus and extent of retrograde thalamic degeneration (right side of figure). Numbers refer to subject and to the actual cross-section from which diagram was made.

awkward when they were present at all. Posterior lesions, on the other hand, affected movements of the distal musculature: especially flexion of the digits in climbing was impaired. When in a threatening or feeding situation, the affected movements would be present initially but faded as the situation continued. However, these effects were not detected beyond the first postoperative month.

In the latch box test situation these animals showed a decrement in performance only during the first postoperative week. Whereas their performance with the hand ipsilateral to the lesion continued at the preoperative rate of under 2 sec. to accomplish a trial, performance with the hand contralateral to the lesion took between six and seven seconds during this period. The performance difficulty differed for the two subjects, however. The animal with the posterior resection was clumsy in manipulating the nail and the box cover; the animal with the anterior resection showed clumsiness in bringing the arm to the latch box and occasionally in "letting go" the nail or box cover preparatory to making the next movement in the sequence. After the first postoperative week, these difficulties were no longer observed and performance time became equal for both extremities.

Extensive resection of the frontal agranular cortex (2428 and 2427)

In the free situation a hemiparesis of the limbs contralateral to the lesion was consistently noted during the first postoperative month—i.e., the animals rarely used the affected extremities; when movements did occur, they were gross, clumsy, and of short duration. During the second postoperative month the affected extremities were moved in association with climbing and running movements of the other side; however, when only one extremity was required for the performance of an act, the extremity ipsilateral to the lesion was consistently employed. This preference persisted for three months after which it receded gradually and disappeared, first in the threatening and feeding situations. In these situations the affected extremities were used after the second postoperative week to accomplish prehension of, or escape from, the stimulus object.

In performing the latch box task no effect of the resection was observed when the animal used the arm ipsilateral to the lesion. However, when only the arm contralateral to the lesion was available for the task performance, *no trial was accomplished within six months following surgery.* Only occasionally would the subject make an attempt to dislodge the nail and open the box; on each occasion the movements were executed in a gross manner precluding accomplishment of the trial. For the most part, the animal just sat quietly or began to play in the testing cage rather than manipulate the latch. As soon as the arm ipsilateral to the resection was

TABLE 1.

TWO-STAGE BILATERAL RESECTIONS OF FRONTAL AGRANULAR CORTEX									
Animal	Lesion	Pre-2nd op.		1 Week	1 Month	2 Months	3 Months	4 Months	5 Months
2400	Extensive Dorsolateral	L. 1.4 R. 1.4	RIGHT SIDE OPERATED	L. - R. 6.7	L. 11.4 R. 11.0	L. 4.1 R. 3.5	L. 3.2 R. 3.4	L. 2.2 R. 4.7	L. 2.0 R. 2.1
2345	Extensive Dorsolateral	L. 1.9 R. 2.7		L. - R. 4.3	L. - R. 13.5	L. 16.9 R. 4.3	L. 6.9 R. 4.1	L. 5.1 R. 3.3	L. 3.4 R. 3.2
2376	Precentral	L. 1.9 R. 3.9		L. 3.7 R. 5.1	L. 1.7 R. 2.1	L. 1.4 R. 3.4	L. 1.8 R. 2.4	L. 1.7 R. 2.0	
2434	Anterior	L. 2.6 R. 2.1		L. 4.2 R. 2.3	L. 3.2 R. -	L. 2.8 R. 1.9	L. 2.3 R. 2.2	L. 2.3 R. 2.5	
2367	Anterior plus Cingulate	L. 1.4 R. 1.4		L. 4.4 R. 1.6	L. 3.0 R. 2.2	L. 2.0 R. 1.6	L. 1.7 R. 1.7	L. 1.6 R. 1.7	

"IRRITATIVE" UNILATERAL, LEFT 2398									
	1 Month	2 Months	3 Months	4 Months	5 Months	6 Months	7 Months	8 Months	9 Months
L.	2.1		2.0	1.6	1.8	2.1	1.8	2.0	1.8
R.	2.6	2.8	2.5	2.4	4.2	11.0	10.0	22.8	11.1

released from restraint, however, testing with this extremity proceeded at speeds indistinguishable from those obtained preoperatively. In the seventh to ninth postoperative month, clumsy attempts were made by the animals to open the latch with the affected extremities. Performance scores of 16 to 30 sec. were recorded, but difficulty in maintaining the animal's attempts to perform a series of trials with the affected hand attended each session. Consistent performance with this extremity was thus never re-established in these animals. The grave deficit shown by all animals made it difficult to evaluate any difference between them (differences which might be attributed to the additional resection of the cingulate cortex in one of them).

Aluminum hydroxide cream implantations of the precentral agranular cortex

During the fifth postoperative week, this animal (2398) began to have Jacksonian convulsions in the right side of the face, contralateral to the lesion and spreading quickly to involve the entire right side. Occasionally the seizures would become generalized. Seizures occurred most frequently during the third and fourth postoperative months; at this time, from four to a dozen seizures per day were noted. The feeding and threatening situations almost invariably induced convulsions. As reported for other "irritative" frontal lesions,[8] the monkey soon became "conditioned" so that when a stick was shown him, the seizure activity would be initiated. Beginning in the fifth postoperative month these generalized seizures became less frequent; instead, an almost continual 3-4 per second rhythmic activity of the right arm and hand was present whether or not the arm was engaged in some activity. This "tremor" persisted but became less noticeable toward the end of the first postoperative year.

As indicated in Table 1 the scores of performance of the latch box task reflect the appearance of the "tremor" but do not correlate with the onset of Jacksonian convulsions. Time scores taken for the affected extremity are markedly increased after the fifth postoperative month and continue to reflect the awkwardness of performance for the remainder of the time the animal was tested.

TABLE 1. *Upper:* Results of the two-stage bilateral procedure. Numbers indicate the mean of time scores of 150 consecutive trials. Dash indicates that no score was obtained, usually because the subject failed to manipulate the latch box.

Lower: Results of the irritative scarring procedure. Time scores indicated as in upper portion of table.

Electroencephalographic abnormalities (marked slowing and spike activity) began to appear in the left central scalp leads during the second postoperative month and persisted thereafter, with gradual decline, for the entire postoperative year. The appearance of the electrical abnormality correlated with the appearance of convulsive episodes, not with change in test performance.

EXPERIMENT 2. BILATERAL LESIONS

Seven monkeys served as subjects for this experiment. Four of these (2427, 2428, 2434, 2367) had been used in Experiment 1. Since consistent latch box performance with the affected extremity had not been re-established by monkeys 2427 and 2428, they were used following the second surgical procedure only to study performance of the delayed reaction and visual discrimination tasks. Two other subjects (2400 and 2345), with somewhat more superficial but equally extensive resections of the precentral agranular cortex, were trained in the latch box situation beginning six months after the initial unilateral procedure. When consistent performance of this task was established with both extremities, the second surgical procedure was undertaken. A similar program of testing was pursued with another monkey (2367) with an ablation of the cingulate cortex in addition to an anterior precentral lesion. In this animal the cortex surrounding the central fissure was spared in order to assure the possibility of some performance in the test situation. Subject 2376 with a posterior and subject 2434 with an anterior lesion were used to compare the effects of bilateral subtotal resections. Before the second surgical procedure was undertaken, these animals had re-established consistent latch box performance following the unilateral procedure as described in Experiment 1.

Protocols of the neurological examinations of these animals will be presented elsewhere. Observations of the movements of the animals in the home cage and the large enclosure under "free" conditions and in feeding and threatening conditions are summarized; performance in formal testing situations is described below.

Subtotal resections of the frontal agranular cortex (2376, 2434, 2367)

In the free situation a paucity of movement was observed in the extremities contralateral to the second lesion for one, or at the most two, weeks. When these extremities were used, paresis was noted; e.g., the animal did not suspend itself from the cage wall for as long a period with the affected extremity. After the first week or two no difference between the two sides could be distinguished. However, the animals continued to tire easily when running (in the threatening situation) for about six months after the second operation; no difference between the two sides could be seen. Nor was a difference between the effects of anterior and posterior subtotal resection as marked as that observed after unilateral ablation. Nevertheless, posterior resection affected primarily the movements of the hands and feet as in climbing; anterior resection affected primarily running and hanging from cage walls and ceiling.

Records obtained in the latch box situation appear in Figure 1 (2376, 2434, 2367). Note that only during the first postoperative week was performance with the hand contralateral to the second operation defective. Even during this period, the problem was solved within twice the time taken preoperatively. Note also that the posterior subtotal resection and only this one affected performance of the ipsilateral hand as well as that of the contralateral hand. Such exacerbation of a defect which had subsided during the months following the first resection is still more noticeable with extensive resections as will be seen below. All in all, there is remarkably little effect of these lesions on performance of the latch box task; and this finding is not altered when the cingulate cortex is added to the resection.

Extensive resections of the precentral agranular cortex (2400, 2345, 2427, 2428)

Both with regard to general observations and test behavior, the effects of extensive precentral resections were more drastic and more lasting. In the free situation, the animals with these resections fell frequently to the side contralateral to the second operation. For a month to six weeks they used the extremities contralateral to the lesion sparingly. Instead of running in the threatening situation, they would "lope" somewhat awkwardly. They were never observed to jump from wall to wall following the second surgical procedure. Nevertheless, in both the feeding and threatening situations, movements were executed in appropriate sequences to accomplish getting food into the mouth or in fending off a stick. These movements were carried out slowly, however, and the animals tired easily; they could also be caught easily even six months after the second operation.

Performance of the latch box task with the hand contralateral to the lesion could not be obtained at all for a month in one subject, for over six weeks in another. Scores obtained with the hand ipsilateral to the resection also suffered markedly, as can be seen from Table 1. Performance did not return to preoperative levels during the five months of testing but stabilized for one animal during the second month and for the other animal during the fifth postoperative month at slightly longer values than the preoperative. Performance was thus impaired but not abolished; impairment consisted in awkwardness of movement resulting in longer time scores. The sequence of acts was unimpaired.

The monkeys with extensive bilateral precentral resections performed the delayed reaction and visual discrimination as well as did the unoperated controls. Thirty-five unoperated monkeys tested in the delayed reaction procedure as outlined took a mean of 380 trials to reach criterion. The operated subjects of the present experiments took 210 and 270 trials to reach criterion. Forty-four unoperated monkeys tested in the visual pattern discrimination procedure as outlined took a mean of 375 trials to reach criterion. The operated subjects of the present experiment took 150 and 310 trials to reach criterion. Clearly, there is no deficit in learning of the delayed reaction or of the visual pattern discrimination following extensive resection of the precentral agranular cortex.

DISCUSSION

It is clear to us from our experiments that extreme views of the rôle of the precentral gyrus cannot be maintained. Neither the idea that the "motor" cortex is "more concerned with the maintenance of excitability and the regulation of postural reflexes than with the excitation and control of finely integrated adaptive movements,"[10] nor the conception that this cortex serves as the sole or even "principal cerebral efferent or effector mechanism by which the brain expresses its activity,"[8] accounts satisfactorily for our observations. Nevertheless, both statements, especially when taken in the context of the discussions from which they were excerpted, have considerable usefulness in pointing out problems that must be faced in any evaluation of the effects of precentral lesions on behavior.

Observations of the movements (such as manipulating food) of the monkeys with extensive precentral resections in the free and the more structured situations cannot all be classified as impairments of postural mechanism. The increased time scores made by these animals in the latch box problem, confirming Lashley's own earlier results, make such an interpretation inadequate. Furthermore, according to Lashley's view "impulses descending from the precentral gyrus do not initiate the finer adaptive movements through the lower motor neurons, but only 'prime' these cells so that they may be excited by impulses from other sources."[10] Although Lloyd's work on the pyramidal system of cats[11] does indicate an indirect pyramidal influence on the motoneurons of the ventral horn, the recent work of Bernhard and his co-workers[2] shows that impulses from the motor cortex do indeed directly impinge upon and excite motoneurons *in the monkey*. Lashley's conception was originally derived from experiments on rodents,[8] animals which show little motor defect with cortical ablation. Perhaps assigning the motor cortex a predominant rôle of reflex regulation of postural mechanisms is more easily envisaged in the more primitive mammals, but this view is unlikely to apply to primates.

On the other hand, the evidence that primates with extensive precentral resections learn and perform delayed reaction and visual discrimination tasks without impairment suggests the necessity for a revision of the conception that the precentral pyramidal system need be the principal cerebral efferent mechanism or even the "internuncial" between cerebral mechanisms involved *in problem-solving behavior* and the organism's effectors. The origin of pyramidal tract fibers in regions other than the precentral cortex, the evidence of efferents from the optic cortex and from the supratemporal auditory cortex,[4] the efferents from Ammon's formation (the fornix) as well as the efferents from the anterior frontal (Arnold's bundle) and the temporal cortex (Turck's tract), must be assigned equal importance to precentral efferents when *selective* effects on behavior follow the disruption of each of these systems.[11] Nonetheless, one cannot dodge the issue that the selective effects resulting from the resection of the precentral gyrus is particularly devastating to an organism's *effector* mechanisms. Thus, extensive precentral lesions can be seen to interfere with performance of almost any task given to the primate provided the appropriate measure—e.g., time required for the manipulation—is made. However, in an analysis of the meaning of our measurements and observations, we must take into account the things undisturbed as well as those disturbed. When this is done, it becomes obvious that those actions involving discrete movements or "skills" are the ones most susceptible to disruption by precentral lesions. However, the disruption is limited to the effectors controlled by the portion of precentral cortex involved in the lesion—no general "forgetting" of the problem solution ensues. Thus, skills are interfered with by precentral lesions, not lost through excision of the locus of the "habit." These results are analogous to those following restricted resections of the optic cortex of the occipital lobe. Whereas the latter produces scotomata in the visual field, lesions of the precentral cortex may be said to produce scotomata of action. In vision, no decrement in the retention of learned discriminations follows subtotal occipital resections; in motor activity, no decrement in the retention of problems solution can be determined following precentral resections.

But what of the idea that "will" as well as skill is affected by the precentral lesions? Experimental resections restricted to the precentral cortex of man[5, 6, 12] suggest that such lesions do *not* interfere with what is commonly called "voluntary" activity; in fact, the clinical syndrome differs little from that described here for the monkey. It is likely that the more severe disturbance found in man which is characterized as a "loss of voluntary movement" is due to deep frontal lesions involving the internal capsule. Such lesions disturb not only precentral but a variety of other cerebral mechanisms.

Regarding the effects of different precentral lesions, some reconciliation of divergent views may also be suggested. Woolsey's[48] precise maps of the responses obtained from electrical stimulation of the precentral cortex show that the posterior portions of this area, in and adjacent to the central fissure, are concerned with the control of appendicular musculature, while the anterior portions are more concerned with axial musculature. Our observations show that posterior lesions do, indeed, affect the precision of the animal's hand movements in the latch box more than do anterior lesions. The latter, on the other hand, affect primarily the movements around the shoulder and hip muscles. Former conceptualizations of the difference between the effects of anterior and posterior lesions in terms of "discrete" vs. "coordinated" movements may thus be understood since movements carried out primarily with the distal portions of the extremities *are* more discrete than those involving primarily the larger axial muscle groups. The other differences which are presumed to exist between anterior and posterior lesions have been discussed elsewhere.[1] However, coordination is involved whether discrete or more massive movements are or are not concerned—thus, the conceptualization in terms of somatotopy, which has been presented here, is preferred.

The extent of precentral cortex resected is important as shown by the relatively small and transient effect of subtotal lesions as compared with the greater and more lasting effect of the extensive ablations. However, recourse to a simple notion of "mass action" is ruled out by the apparent lack of increased decrement in performance of the latch box problem when anterior cingulate ablation is added, either to a subtotal or to a total precentral resection. Within the precentral agranular isocortex, when a unilateral lesion is made bilateral, there is an effect on the extremity ipsilateral to the second lesion—so effective "mass" is distributed bilaterally within the cortex. Lesions of comparable extent in other cortical fields studied produce either no observable motor defects (frontal granular, temporal, striate, peristriate, and limbic fields) or minimal transitory defects (postcentral and parietal fields). The recent study of Semmes and Chow[38] in which the effects of lesions of all the cortex except the precentral agranular were found to be similar to those of precentral motor cortex ablation—except in the duration of effect—implies a lack of specificity in the function of different cortical areas in motor behavior. Although other regions of the cortex can be implicated in motor behavior, this may also be said of total de-afferentation of a limb. By applying a diversity of appropriate measures, the specificity of any such deficit may be elucidated. Such techniques used by Fulton and his co-workers[6] in analyzing motor function have led to the demonstration of defects in posture, the various forms of tactile and proprioceptive placing, hopping, resistance to passive limb manipulation, palpable muscle tone, the degree and *form* of tendon reflexes, influence of body and head position, the alterations in stimulation conditions required to elicit grasping, and other detailed neurological tests related to motor function which in our experience clearly differentiate the motor defect following frontal agranular lesions from that seen with any other cortical lesion.

There have been suggestions[7] that the effects of lesions are due solely to irritative scars at their borders. Comparison of aluminum hydroxide cream lesions with those of ablations in the present experiments suggests that the two types have different effects—that both these effects might be conceived as a disequilibrium of function, but that a distinction can be made. Resections impair performance, i.e., in a particular situation, the repertory of action available to an animal with precentral resection is more limited than the repertory available to an intact animal. However, within that repertory, a fairly stable, albeit limited, performance of tasks results. The animal with aluminum hydroxide cream implantation, on the other hand, shows impairment in a larger spectrum of behavior by the pervasiveness of the repetitive muscular contractions which persist whether he is active or inactive, whether he is performing a task requiring skill or one where only grosser movements are concerned. Thus, the animal's limitation in a particular task may or may not be as severe as that of the subjects with resection, and often fluctuates to a considerable extent for reasons that have not as yet been determined.

In conclusion: we feel that certain tentative answers can be given to the questions which initiated these experiments and were posed in the introduction. Extent of ablation is an important consideration in evaluating the effect of a precentral lesion. Effects of unilateral ablations are enhanced when the lesion is made bilateral. Effects of ablation cannot be attributed to "irritative" scarring at the border of such lesions since the effect of "irritative" lesions per se can be distinguished from those of ablation.

Precentral lesions interfere especially with skilled acts; the acts are not lost, however, through excision of the locus of a "habit": rather, a scotoma of action may be said to result. Thus, the extreme conception that the precentral cortex is the principal efferent system by which the brain expresses its activity is modified in favor of the conception that the precentral cortex modulates a variety of reflex mechanisms basic to action. The numerous anatomical studies on the afferent and efferent relations of the somatic "sensory" and "motor" areas of the monkey cerebral cortex have recently been supported by electrophysiological demonstrations of efferents[17] and afferents[5] to both of these regions. The "unity" of the "sensorimotor" cortex is further emphasized by the remarkable constancy of proximity of somatic "sensory" and "motor" fields and the absence of intercalated "association" areas in all mammals. Although it is recognized that the preponderance of afferent and efferent fibers differ in the precentral and postcentral regions, we believe that their close anatomical relationship should be emphasized rather than any conceptualization based on analogy with the dorsal and ventral roots of the spinal cord.

Experiments in both monkey and man indicate that resections limited to the precentral cortex do not interfere with behavior of the type usually described as "willed." Rather, we think it possible that deep lesions involving the internal capsule are responsible for such grosser changes in behavior by interrupting a variety of cerebral mechanisms. Thus, the clinically derived conception that the precentral motor cortex serves "skilled action" is corroborated; it is clear, however, that this cortex is not the locus of a "habit" or of a mechanism basic to any performance of the skill, but rather that precentral lesions result in a scotoma of action which interferes with the skill. On the other hand, the clinically derived conception that the precentral cortex is involved in "willed action" is considerably modified: deep lesions involving not only precentral, but also additional, cerebral mechanisms are thought to be responsible for the grosser defects in behavior which led to this conception.

SUMMARY

Ablation of the precentral agranular cortex was found to interfere especially with skilled acts as measured by time scores in performing a latch box task. Lesions restricted to the posterior portion of this cortex caused a greater defect in digital dexterity than anterior lesions which more markedly affected the more proximal musculature. Total ablation caused the most marked defects. Visual discrimination and delayed response performance were unaffected by precentral ablations. Bilateral ablations exacerbated the effects of unilateral ablation and caused a renewed defect on the side ipsilateral to the second operation. The effects of ablation cannot be attributed to "irritative" scarring at the border of such lesions since the effects of "irritative" lesions per se can be readily distinguished from those of ablation. It is concluded that the motor defect in precentral ablations is not due to acts lost through excision of the locus of a "habit," but rather that scotoma of action result.

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