A BEHAVIORAL STUDY OF THE FUNCTIONS OF THE ROLANDIC CORTEX IN THE MONKEY

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EIGHT FIGURES

INTRODUCTION

The significance of the primate Rolandic fissure remains a subject of controversy despite extensive experimental analyses. Before the turn of the century many investigators regarded the entire Rolandic cortex as a "sensorimotor" unit, but early in this century the dominant viewpoint shifted toward a sharp demarcation of the precentral "motor" and postcentral "sensory" cortex. Recently, as a result of electrophysiological investigations, the emphasis has begun to swing back toward the nineteenth century conception. Electrical stimulation of the cortex with recording of the consequent muscular contractions has revealed that a pattern comparable to that described for the precentral gyrus can be determined for the postcentral gyrus in the monkey (Woolsey et al., '53). Electrical excitation of peripheral nerves or of dorsal roots has been shown to elicit electrical responses which can be evoked in the precentral as well as the postcentral gyrus (Woolsey, Chang and Bard, '47; Ruch, Patton and Amassian, '52; Malis, Pribram and Kruger, '53; Gardner and Morin, '53; Adey, Porter and Carter, '54; and

Kruger, '56). Such precentral responses are obtained under a variety of anesthetic states and in unanesthetized animals; however, potentials evoked in the precentral gyrus by tactile stimulation are difficult to demonstrate.

The present study was undertaken in order to determine the role of the Rolandic afferent projection in *sensation* — a problem which can only be clarified by behavioral experiments. Specifically, an answer was sought to the question: is the cortex implicated in somatic sensory function coextensive with the full extent of the afferent projection as delineated electrophysiologically, or is removal of the more restricted postcentral area sufficient to totally disrupt somesthetic discrimination?

Most previous studies concerned with this problem rely mainly on neurological examinations of animals or patients with partial lesions of the Rolandic cortex and usually contain scanty reports regarding the extent of the lesion and resulting thalamic degeneration. The animal experiments suffer from numerous difficulties inherent in testing sensation by observing a motor response, especially in animals with some degree of paralysis; the clinical material, although easier to interpret, is limited to partial ablations or to tumors or vascular lesions. Quantitative behavioral experiments have been few and these are largely limited to the study of mammals lacking the central fissure. Experiments from Pavlov's laboratory (Pavlov, '27) suggested that tactile conditioned reflexes were transiently lost after lesions of the coronal and anterior ectosylvian gyri in dogs, but permanent defects were found only after complete hemidecortication. Allen ('47), who also used conditioned dogs, found that bilateral lesions of tactile receiving areas I and II led to an abolition of a differential foreleg response, though a positive conditioned reflex to tactile stimulation could still be established. Using rats trained to perform roughness and tactile form discriminations, Zubek ('51 and '52a) demonstrated that lesions of tactile areas I and II did not abolish or prevent learning of either task, although some decrement in perform-

ance did occur. In cats, Zubek ('52b) found that ablation of areas I and II in one case led to a "permanent abolition of the capacity to make roughness discrimination."

In studies on monkey, chimpanzee and man (Ruch and Fulton, '35; and Ruch, Fulton and German, '38) discrimination of small differences in weight, roughness and form (somesthetic) was possible after destruction of the postcentral gyrus or posterior parietal cortex. However, during the initial phases of postoperative retraining the differences between the stimuli had to be made larger than those used preoperatively; only then were the smaller differences again discriminated. Ablation of the entire parietal cortex in one monkey reported by Blum, Chow and Pribram ('50) led to a deficit (i.e., an increase in the number of trials necessary for retraining) in several somesthetic discriminations, but the deficit was not a "complete" loss of somesthesis (i.e., the animal again performed the somesthetic test after prolonged training). Cole and Glees ('54) have also found that small lesions of the postcentral hand area result in a temporary discriminative defect, but that the discriminations can be relearned readily. Since these primate studies do not include ablations of tactile receiving area II, the effect of combined ablation of tactile receiving areas I and II as well as the effect of combined precentral *and* postcentral resection in primates requires clarification. The present study explores this problem, using quantitative behavioral measures, describes deficits revealed by neurological examination, and presents some relevant findings concerning the thalamic connections of the Rolandic cortex.

MATERIALS AND METHODS

Six immature rhesus monkeys (*Macaca mulatta*) were used. After preoperative testing and repeated neurological examination the animals were operated upon under Nembutal anesthesia, which was administered intraperitoneally. Aseptic technique was used throughout. An osteoplastic flap was turned and hinged on the temporal muscle. The dural flap

was based on the midline. Subpial ablation was performed through a small pial incision with a 19-gauge needle suction tip. Hemostasis was secured by gentle packing with cotton pledgets. The pledgets also served to provide retraction in the depths of sulci so that one bank could be ablated with minimal damage to the other. The dura and scalp were closed with interrupted silk sutures and a subcutaneous continuous suture. When a monkey showed hand preference prior to operation, the first operation was performed on the hemisphere opposite to the preferred hand. Bilateral operations were always performed in two stages.

Before and after the operative procedures the animals were routinely observed in their cages and in a large wire enclosure and were examined at least once each week in detail while seated in a neurological chair. Each animal was tested daily in a transport cage which was moved into an air-conditioned, sound-proof and light-proof testing room. The cage was rolled into a dark enclosure which was fitted with an opaque screen that separated the animal from the testing apparatus.

When the screen was lifted the animal was confronted with two boxes, (2½ by 2½ inches, with exactly fitting covers) 18 inches apart, on which the discriminanda were placed. The discriminanda used consisted of a three dimensional figure "L" and its inversion, a discrimination used in studying the effects of removals of the occipito-parietal cortex by H. Pribram and Barry ('56). The figures were 1½ inches high, ½ inch thick and ½ inch wide. For each trial the predetermined "correct" figure was placed over the right or left cup according to a random series and a peanut placed under this figure. The animal then palpated the cover of each box and one trial was scored when either lid was lifted. Correction for error was allowed only during the first 100 trials; thereafter a wrong choice remained unrewarded and the next trial in the series was presented. Thirty or 50 trials were presented daily until the monkey made 90 correct choices in 100 consecutive trials. The scores presented are the number of

trials required to reach this criterion; the total number of trials given is obtained by adding 100 to these scores.

During training one arm was strapped to the animal's side by means of a waist belt and bracelet. In this way only the unrestrained arm could be used to perform the test.

The animals were first allowed to discriminate visually, i.e., a light was gradually decreased until the animal was performing in total darkness. In this situation the experimenter watched the animal on a television screen upon which the image of the animal was obtained by means of an infra-red scanning device, described in detail elsewhere (Cox and Kruger, '55). During the early trials the monkey palpated both covers several times before making a choice; later the choice was made rapidly and often only one object was touched.

Another task given employed a hasp box. This test utilizes the time taken by the monkey to lift a peg holding the cover of a cup, to slide open the cover and to secure the peanut from the cup as a measure of motor capacity (Pribram et al., '56).

Electroencephalograms were recorded from scalp needle electrodes in two of the animals. As the findings revealed no abnormality, they are not discussed. Cutaneous temperatures recorded from foot and hand pads were measured with a McKesson-Dermalor.

At the end of the experiments the animals were sacrificed and their brains perfused with 0.9 normal saline solution followed with 10% formalin and imbedded in celloidin (one brain, in paraffin). Serial sections were cut at 30 μ throughout the area of the lesion and the thalamus and every 4th section was mounted and stained with thionin. From the sections the extent of the cortical lesions was reconstructed by orthogonal projection and transposed to a standard diagram to minimize the distortion due to shrinkage of tissue in the region of ablation. Drawings of thalamic degeneration were produced as follows: appropriate sections were photographed on direct positive paper at a magnification of 15 x. The

visible neurons were then inked with India ink. The regions of severe degeneration and dense gliosis were inked solid black. The regions which showed a moderate amount of degeneration but in which some normal neurons remained were encircled. Following the inking the photograph was bleached and the resulting drawing rephotographed and reduced for reproduction.

<div style="text-align:center">RESULTS</div>

1. The effect of unilateral and bilateral ablation of the postcentral gyrus and tactile receiving area II

Two animals were used for postcentral removals. Both were trained to perform the visual and somesthetic tasks preoperatively and both required a somewhat greater number of trials to learn the somesthetic than the visual discrimination problem.

Immediately after unilateral ablation of the left postcentral gyrus and the buried fronto-parietal operculum (fig. 1), both contralateral limbs were maintained in the paretic posture described by Kennard and Kessler ('40), Peele ('44), and Cole and Glees ('54). During the first two months both animals often held the affected arm in an awkward position. Cutaneous temperatures were elevated 0.5–1.0° C in the limbs contralateral to the ablation on the first postoperative day. However, after several days the temperature fell below that in the unaffected extremities, becoming approximately equal in both within the first postoperative month. Palpable muscle tone was diminished on the right side for 2–3 weeks. Knee, ankle and elbow tendon jerks showed a transient hyper-reflexia during the first week, which gradually subsided. Though highly variable, resistance to passive manipulation of the right extremities developed within the first few postoperative days. This change subsided gradually in the next two weeks and became difficult to evaluate. After one month, however, resistance on the the affected side was less than on the left ("normal") side; this hypotonia persisted throughout the period of observation (4 and 11 months respectively). By

contrast, the digits of the affected limb appeared to be hypo-
tonic immediately after the operation but showed an increas-
ing resistance to passive manipulation within the first month.
A proximal traction reaction [*] could be crudely elicited in the

PREOP

POST OP

UNILAT LT

203 L

VISUAL	SOMESTH
90	120

VISUAL		SOMESTH	
RT	LT	RT	LT
0	0	70	20

204 R   204 L

PRE OP

VISUAL	SOMESTH
180	200

POST OP
UNILAT LT

VISUAL		SOMESTH	
RT	LT	RT	LT
0	0	210	0

BILAT

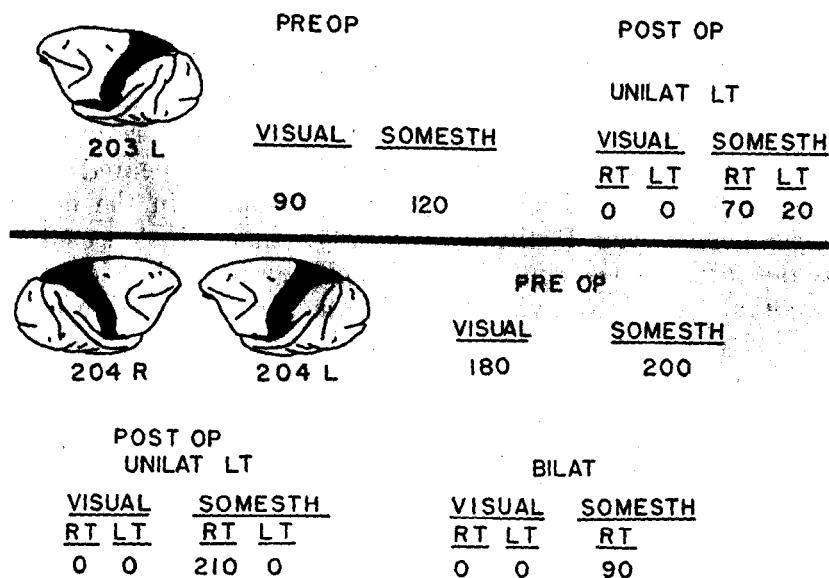| VISUAL		SOMESTH
RT	LT	RT
0	0	90

Fig. 1 The extent of cortical ablations of somatic areas I and II and the
learning scores for these animals in performing visual and somesthetic form
discrimination tasks. In this and subsequent figures, the arm used is indicated
only when one arm was immobilized; otherwise ''right'' and ''left'' indicate the
arm used to perform the task.

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[*] The stimulus necessary to elicit grasping is different at the several stages of
recovery. We have therefore adopted the clinically used terminology introduced
by Seyffrath and Denny-Brown ('48) and Twitchell ('51). The ''proximal trac-
tion reaction'' is defined as the form of grasping elicited by stretch of the
shoulder musculature—a response which has been called ''forced grasping'' by
some authors. The ''grasp reflex'' as used here is elicited by a deep, distally
moving, cutaneous stimulus and is maintained by stretch. The ''instinctive
grasp'' reaction is the form of grasp elicited by a light, stationary, tactile
stimulus seen in normal monkeys. Normal animals also often withdraw from a
light tactile stimulus, so that we consider tactile avoidance and the instinctive
grasp reaction together as reactions to stationary tactile stimuli. Orienting
reactions may also be considered an integral part of the instinctive grasp re-
action, but these were not studied in sufficient detail to warrant description.
Disturbances in orientation to cutaneous stimuli were evident after all Rolandic
lesions studied, but unfortunately were difficult to analyze with our limited
methods of observation.

right hand during the first month but at this time a "grasp reflex" could not be elicited in either affected extremity. During the second postoperative month a true "grasp reflex" (obtained by a deep, distally moving cutaneous stimulus) clearly appeared in both animals. The "instinctive grasp" and the tactile avoidance reactions were obtained only 4–6 months after operation and never as consistently as on the left side. Hopping and placing reactions were absent on the right side for the first three weeks even in the presence of visual cues; thereafter, hypermetric hopping appeared only when the leg was dragged medially. Hopping in the other directions appeared still more slowly. In all instances the angles of displacement required were greater than on the "normal" side. Non-visual tactile placing could not be convincingly demonstrated with the right arm or leg when the phalanges were touched with absorbent cotton, but such placing was obtained when the phalanges were brought in contact with a broad, hard surface. Whether placing under these circumstances is due to proprioceptive or to tactile stimulation is difficult to ascertain since slight movements of the joints probably do occur. This form of the placing reaction returned in 6–9 weeks postoperatively, by which time hopping had recovered considerably.

Immediately after operation both animals performed the visual discrimination readily without retraining, although they had considerable difficulty in opening the box and acquiring the peanut with the right (contralateral) hand. Somesthetic testing was begun at the end of the second postoperative month (by which time the animals could perform the hasp box test awkwardly and a "grasp reflex" could be elicited readily). Performance with the arm ipsilateral to the lesion showed practically complete retention, whereas some retraining (70 and 210 trials) was necessary when performance with the affected extremity was required. When the animal used the affected arm the cue "L" was never palpated gently with the fingers as before operation or when the unaffected arm was used. Rather, the animal made coarse rapid

strokes across the surfaces of the stimulus cue. After the box had been opened the monkey would reach in for the peanut and close its hand, which was often brought to the mouth empty. The animal would try this maneuver again until the peanut was grasped, though occasionally discouragement had to be overcome by allowing the animal to use visual cues. After several days of testing the defect became less pronounced and the animal usually brought the peanut into the mouth. Normal palpation of the peanut and grasping were never observed. After the first postoperative week testing was begun on the opening of a hasp box. Neither animal accomplished this task with the right (contralateral) arm until the 5th postoperative week. Performance with the affected arm was severely defective; the peg was lifted awkwardly, the cover pushed back slowly, and the peanut grasped poorly. Three months elapsed before the task could be performed equally rapidly with both extremities.

In animal no. 204, ablation of the postcentral area on the right side three and one-half months after the initial operation (on the left side) resulted in a recrudescence of the defect in the right arm which required an additional 90 trials before criterion performance was again reestablished in the somesthetic discrimination. This was accomplished during the first postoperative month. The defect in the left limb (contralateral to the second lesion) was somewhat more severe but showed more rapid recovery than had that of the right extremity after the initial operation.

II.   The effect of unilateral and bilateral ablation of the precentral agranular cortex

Two animals were used for the precentral ablations, which included almost the entire "agranular" field (fig. 2). Immediately following the preoperative training in the somesthetic and visual discrimination tasks, the precentral cortex opposite the preferred hand of each animal was ablated.

Details of the neurological defects and the hasp box performance of monkeys with a variety of total and subtotal pre-

central lesions have been presented elsewhere (Berman, Kruger and Fulton, '54; Pribram, Kruger, Robinson and Berman, '56). Cutaneous temperatures in the limbs contralateral to the lesion showed the same initial rise and subsequent fall seen with postcentral lesions.
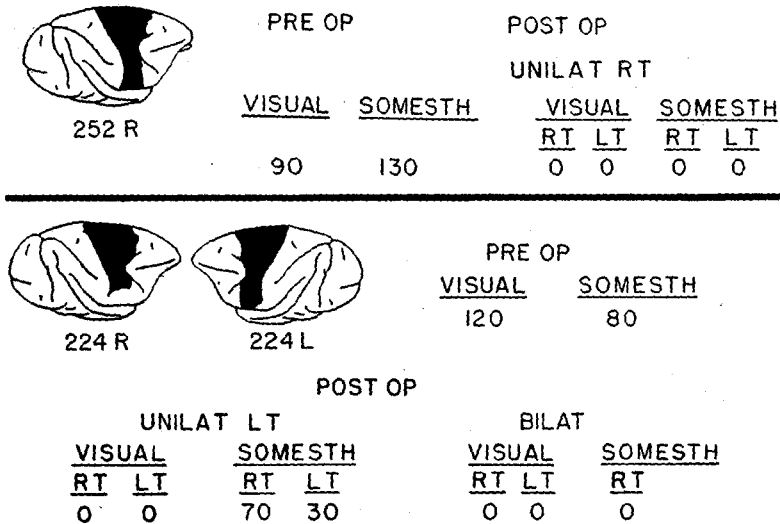

Fig. 2 The extent of cortical ablations of the precentral agranular field and the learning scores for these animals in performing visual and somesthetic form discrimination tasks.

While using the arm ipsilateral to the lesion, both animals performed the visual and somesthetic tasks without observable deficit. Testing with the affected extremity was initially impossible because of paresis and mild spasticity. The phalanges were most markedly affected, making it difficult for the animal to pick up a peanut from a flat surface. After several weeks, the visual task was performed readily, although awkwardly. However, the animals refused to work in the dark despite prolonged food deprivation. Only after 4 (no. 252) and 8 (no. 224) months did the animals perform, without deficit, the somesthetic discrimination with the affected extremity. The cues were gently and slowly palpated with the thumb and index finger. In this respect performance differed

markedly from the coarse palpation that followed postcentral
ablation. Of particular interest is the finding that the ani-
mals performed the somesthetic task only after the return of
the "instinctive grasp" reaction.

In animal no. 224 ablation of the precentral gyrus of the
second side (sparing part of the face area to facilitate feed-
ing), renewed the motor defect in the limbs ipsilateral to
the lesion. A slightly more marked defect resulted in the
limbs contralateral to this second lesion, but this defect
was less severe than that observed after the initial ablation.
Hopping and placing reactions, although initially lost, re-
turned rapidly. During the first postoperative month the
animal was able to perform the visual and somesthetic dis-
crimination tasks without deficit. Performance in the dark
after unilateral or bilateral precentral ablation differed from
that seen preoperatively only in an increased degree of clum-
siness in opening the box and securing the peanut.

III. The effect of unilateral ablation of the precentral and postcentral arm area

In this experiment a comparison was made between the
limbs ipsilateral to and contralateral to an extensive lesion
made to conform to a generous estimate of the Rolandic
"arm" area (fig. 3). This area was determined by the extent
of the cortical region activated by median and ulnar nerve
volleys. Two animals had been trained preoperatively in the
visual and somesthetic discrimination tasks but they did not
survive for a sufficient time to complete the study. The two
animals described had no preoperative training.

The defect following this extensive lesion is far more
marked than that seen after separate precentral or post-
central lesions, in spite of the fact that the total extent of
cortex removed is roughly equal in all three types of lesions
described. Initially, both extremities contralateral to the les-
ion were dragged while the animal ran and climbed. Both arm
and leg showed an increased resistance to passive manipula-

tion; brisk, irradiating tendon reflexes without clonus and movements restricted to those associated with movement of the limbs ipsilateral to the lesion. These abnormalities gradually disappeared in the leg during the second postoperative week but subsided more gradually in the arm. "Spontaneous" use appeared in the affected arm during the second postoperative month. Throughout the period of observation this arm was often maintained in awkward positions, e.g., remaining behind the animal's back after having been placed there by the experimenter. These awkward positions were usually corrected with the aid of vision. The affected arm showed a loss of palpable tone and diminished resistance to passive manipulation; these reactions appeared in the third to fifth postoperative weeks, in spite of slightly hyperactive tendon reflexes. A "grasp reflex" was irregularly elicited at the end of the first postoperative month. An instinctive grasp reaction could never be elicited in the affected arm. Hopping and placing reactions were absent during the first postoperative month. Sluggish hopping returned during the second postoperative month but required large limb displacements; no form of cutaneous placing was demonstrable in no. 174 (observed for four months), but no. 159, after six months, inconstantly showed some placing when the affected arm was brought into contact with a broad surface.

Both animals were trained in a visual discrimination and, using their unaffected (right) arm, they rapidly reached adequate performance in this task. During the second postoperative month the right arm was tied to the belt at the animal's waist. The task was performed without additional training with the affected arm, although the animals showed some awkwardness when using this limb. Subsequently, each animal learned the somesthetic discrimination with the unaffected (right) arm, but when this arm was immobilized, forcing the use of the affected limb, the task could *not* be performed. The animal's arm could be seen reaching out in the dark, often coming into contact with test objects, but no changes in behavior that could be attributed to contact were

observed, nor were the discriminanda or the box manipulated. When they were sufficiently starved, the animals tried hard to find the box containing the food, but were unsuccessful. The animals were observed and tested weekly for 4 (no. 174) and 11 (no. 159) months respectively but failed completely to perform the somesthetic task during the period of observa-

POST OP

VISUAL   SOMESTH

RT   LT   RT   LT

159 R   150   0   50   F

POST OP

VISUAL   SOMESTH

RT   LT   RT   LT

174 R   120   0   180   F

Fig. 3 The extent of cortical ablations of the combined precentral and postcentral arm area and the learning scores for these animals in performing visual and somesthetic form discrimination tasks. ''F'' denotes failure to perform the task rather than a failure to learn after a given number of trials.

tion, despite the ability to perform the same task easily when vision was permitted. These results suggest that the cortical representation of somatic sensation for the arm had been extensively interfered with in this experiment.

IV. Anatomical results

Lesions of the *postcentral* gyrus (fig. 1) include the posterior bank of the central sulcus, the anterior bank of the interparietal sulcus, part of the superior bank of the cingulate

sulcus and the cortex lying between these landmarks. Ventrally the lesions extend into the Sylvian fissure and include most of the parietal and frontal operculum extending to the island of Reil. In all cases, there is some cortex remaining at the base of the central sulcus which possesses a distinct granular layer and in the leg area there is a region of granular cortex extending into the anterior wall of the central sulcus. The resultant retrograde degeneration in the thalamus in the three hemispheres with the lesion was most marked in the ventro-basal complex (VB), (as defined by Mountcastle and Henneman, '52, using both cytoarchitectonic and evoked potential criteria). The only ventro-basal cells clearly unaffected are in the cluster of cells in the arcuate nucleus lying ventrolateral and adjacent to the wing of the nucleus centrum medianum (fig. 8). The parvocellular division of the arcuate nucleus was intact. The nucleus ventralis posterior inferior (VPI) of Olszewski ('52) is largely intact. The oral and lateral sectors of the external ventro-basal nucleus are difficult to define in the region where they merge with ventralis lateralis (VL) or Olszewski's nucleus ventralis posterior lateralis, pars oralis (VPL$_0$). This region of transition extending into ventralis (VL) is filled with glia and there is a sparse scattering of cells here which might belong to the ventro-basal complex. All other affected regions of the ventro-basal complex are filled with glia and display almost complete cell loss. Because of the failure to recognize any portion of the thalamic tactile region (ventro-basal complex) as undegenerated, (with the exception of a small region of the arcuate nucleus discussed below), we interpret the lesion of the postcentral gyrus to be essentially complete although it is recognized that the granular cortex which remained intact at the base of the central sulcus may still have possessed connections with the ventro-basal nucleus. Our interpretation of this material is that the tactile receiving area of the postcentral gyrus appears to represent the essential projection area of the ventro-basal nucleus in its external and large-celled arcuate portions, with the exception of a group of ar-

cuate cells lying at the ventral surface of the nucleus centrum medianum. Parts of the nucleus lateralis posterior and the pulvinar lying dorsal to the ventro-basal nucleus are also degenerated (fig. 4). This degeneration is inferred to be due to ablation of the posterior portion of the postcentral gyrus (Brodmann's area 5) from the work of Le Gros Clark and Powell ('53). Portions of the ventral nucleus anterior to the
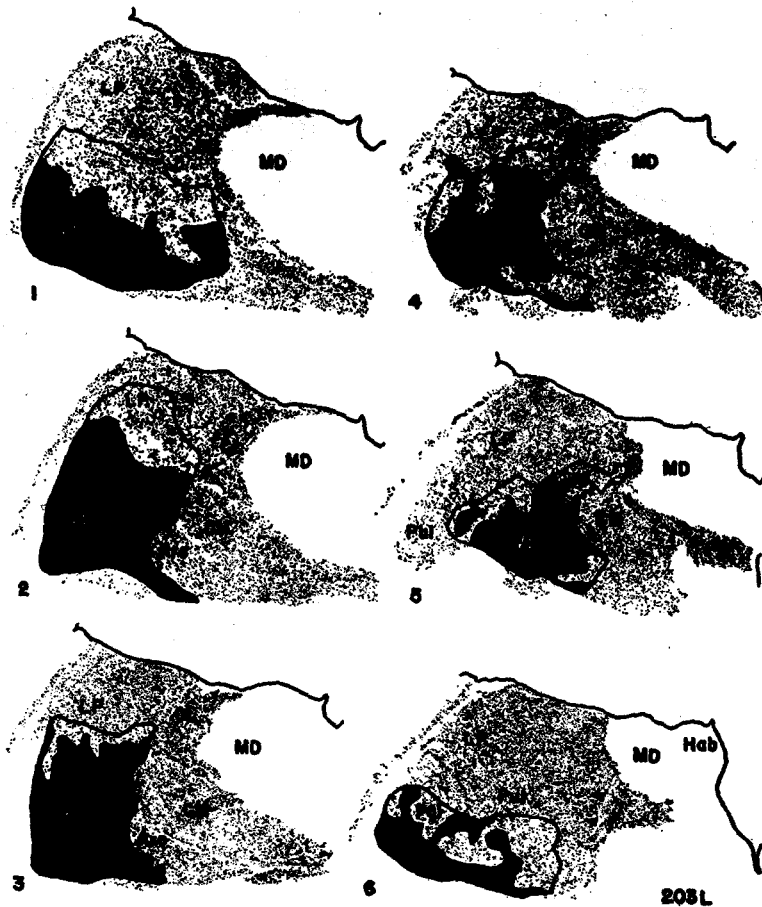

Fig. 4 Representative sections to show the extent of retrograde thalamic degeneration after ablation of somatic areas I and II. The regions of degeneration are enclosed: regions of severe gliosis and cell loss are in black. Magnification 5 ×.

ventro-basal complex do not show distinct degeneration of
neurons but some gliosis is present in the adjacent portions
of the nucleus ventralis lateralis or Olszewski's ventralis
posterolateralis, pars oralis (VPL$_0$).

Ablations of the precentral gyrus include the anterior bank
of the central sulcus and extend anteriorly to include the cor-
tex of the posterior bank of the arcuate sulcus. Medially, the
lesions in all three hemispheres spare some of the cortex of
the "leg area" in the depths of the cingulate sulcus. Poster-
iorly, there is agranular cortex present at the base of the
central fissure in all brains. Ventrally, the lesion continued
beyond the end of the central sulcus, extending to, but not
into, the Sylvian fissure, except for the right hemisphere of
no. 224, where the face area was largely spared. The poster-
ior boundary of the removal in the face area is defined by an
extension of the ventral course of the central sulcus.

The resultant retrograde degeneration (fig. 5) begins in the
anterior portion of the ventral nucleus in ventralis lateralis
(pars oralis, VL$_0$, of Olszewski), sparing ventralis anterior.
The nucleus ventralis lateralis in its oral and caudal portions
is heavily degenerated but area X of Olszewski is largely
spared. As one proceeds posteriorly only the medial portion of
the ventral nucleus is degenerated, irregularly sparing a por-
tion of ventralis lateralis corresponding to ventralis postero-
lateralis, pars oralis (VPL$_0$) of Olszewski, which we do not
recognize as a portion of the ventro-basal nucleus. However,
it appears from a study of other material (Pribram, Kruger,
Robinson and Berman, '56), that preservation of cells in this
region is due to a failure to remove the entire extent of
agranular cortex in the depths of the central fissure. Degen-
eration in the nucleus ventralis posterolateralis, pars oralis
(VPL$_0$) of Olszewski or parts of the posterior portion of ven-
tralis intermedius of Crouch ('34) appears in lesions which
include most of the buried cortex in the central sulcus, but the
present material is not adequate to prove this point. Poster-
iorly, the degeneration clearly extends into the ventro-basal
arcuate nucleus, sparing the medial parvocellular division,

but resulting in complete degeneration of the cells remaining after postcentral lesions (fig. 7). These cells are spared only in the right hemisphere of no. 224, in which the face area was not removed. Other portions of the ventro-basal complex show a questionable mild shrinkage of cells and slight gliosis. The essential projection of the precentral agranular field appears to include a portion of the nucleus ventralis lateralis, possibly the nucleus ventralis posterolateralis, pars oralis (VPL$_o$) of Olszewski or the posterior portion of Crouch's nucleus intermedius. The precentral projection of the arcuate nucleus previously described by Le Gros Clark ('37) and Walker ('38) is limited to the face area and includes only the small region shown in figures 5 and 7. The tactile receiving area for the mouth nearly extends into the precentral face area (Benjamin and Welker, '57) of the squirrel monkey and reconstruction of the cortex at the base of the central sulcus reveals a strip of granular cortex extending into the "precentral" region in most primates (Bucy, '35). Degeneration of a portion of nucleus centralis lateralis can also be seen in figures 5 and 7. A similar result was also noted by Walker ('38). Although according to some authors this nucleus projects upon the striatum it is difficult to conceive that striatal fibers would be destroyed by lesions as superficial as those in the material presented here. The possibility must be considered, therefore, that nucleus centralis lateralis has connections with the frontal cortex.

The two hemispheres with *combined precentral and postcentral lesions* show degeneration in the same regions described above for partial lesions, except for the preservation of a band of cells laterally and medially throughout the ventral nucleus from ventralis lateralis to the posterior end of the ventro-basal nucleus.

The important anatomical fact noted in this experiment is that the ventro-basal nucleus as defined cytoarchitectonically and by tactile evoked potentials (Mountcastle and Henneman, '52) projects for the most part to the postcentral gyrus and, except for one portion, remains intact after lesions of the

anterior bank of the central sulcus. The portion of the ventro-
basal arcuate nucleus which remains intact after postcentral
lesions has been shown to project to the precentral face area,
which receives a tactile projection from the mouth, and which
possesses a distinct granular layer, thereby providing evi-
dence that the essential projection of the ventro-basal nucleus
is to the granular cortex of the tactile receiving area. De-
generation of the thalamus attributable to ablation of the
second tactile receiving area in the frontoparietal operculum
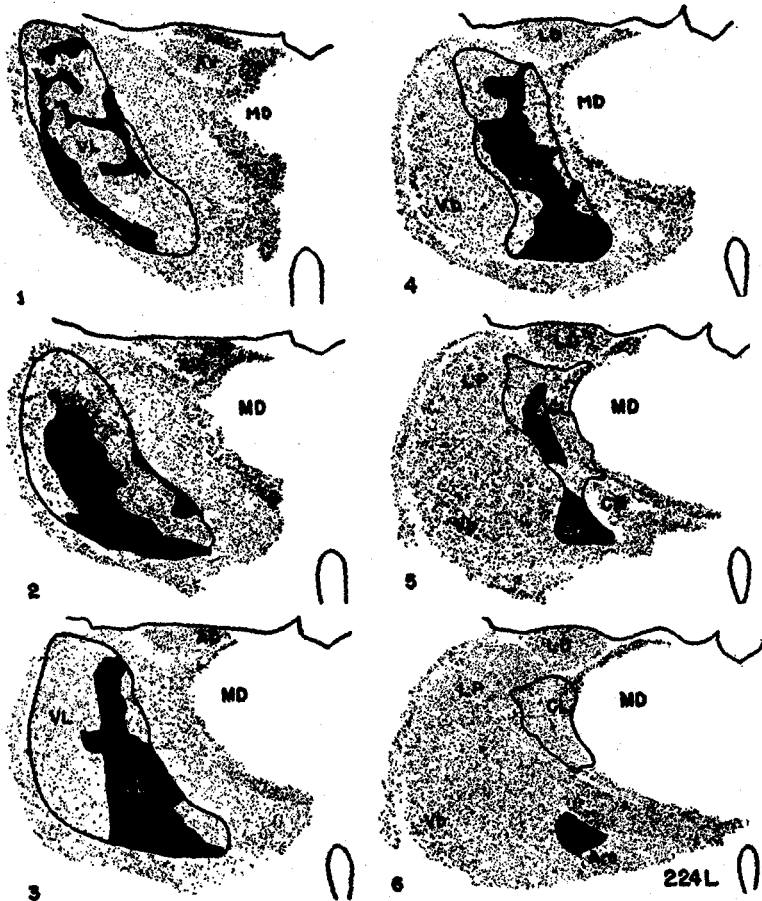has not been identified.

Fig. 5 Representative sections to show the etxent of retrograde degeneration
after ablation of the precentral agranular cortex. Magnification 5 ×.

DISCUSSION

The present study was undertaken to investigate the possible role of the precentral gyrus in somatic sensory processes. Because of the limitations of neurological tests and the crude simplicity of the somesthetic form discrimination used, only a partial answer can be given to the question raised.

The most striking finding is that after ablation of the postcentral gyrus and somatic area II, despite a severe deficit in *tactile* sensitivity as revealed by neurological examination, monkeys discriminate the form of the test objects and secure food in the absence of visual cues. The discrimination is permanently impaired only if the precentral gyrus as well as the postcentral gyrus is included in the lesion. No deficit is demonstrable if the ablation is restricted to the precentral gyrus alone. This finding is in keeping with the recent experimental demonstration of precentral responses to cutaneous nerve volleys after postcentral ablation (Malis, Pribram and Kruger, '53; Gardner and Morin, '53; and Kruger, '56) and with Dusser de Barenne's observation ('24) that sensory symptoms can be produced by strychninization of the precentral gyrus following ablation of the postcentral gyrus.

An approach to the analysis of the sensory defect following Rolandic lesions can be found in a study of reflex grasping (Seyffarth and Denny-Brown, '48). In all normal limbs the animal grasps in response to a light, stationary tactile stimulus ("the instinctive grasp reaction"), or occasionally withdraws the extremity ("tactile avoidance"). In addition, a "grasp reflex" can be elicited—a response to a deep, distally moving cutaneous stimulus which is maintained by stretch. These reactions are lost immediately after precentral or postcentral lesions and for a short time postoperatively the only form of grasping that can be elicited is a grasp in response to shoulder muscle stretch, the "proximal traction reaction" (Twitchell, '51). The sequence of recurrence of the reactions, after precentral lesions, to various types of stimuli that elicit grasping has been described elsewhere (Berman, Kruger and Fulton, '54). Essentially, after precentral lesions, the proxi-

mal traction reaction, grasp reflex, and instinctive grasp reaction or tactile avoidance all reappear eventually in the sequence named. Following postcentral ablation a "grasp reflex" can be elicited in about one month, while an instinctive grasp reaction or tactile avoidance could be elicited only erratically after 4 to 6 months. After *combined* pre- and postcentral lesions a grasp reflex eventually can be irregularly elicited with heavy stroking of the volar surface of the hand; "instinctive grasp" and tactile avoidance are not seen at any time. This analysis of the recovery of grasping suggests that all Rolandic lesions produce some defect in cutaneous sensation, that precentral lesions are followed by the most complete recovery, that postcentral lesions produce a defect which permanently affects tactile sensation, and that combined lesions result in a more severe defect—only a crude form of grasping elicited by deep stimulation remains.

The non-visual placing and hopping reactions (Bard, '38) also show differences when the effects of different Rolandic lesions are compared. As has been described, both hopping and tactile placing reactions are lost immediately after either precentral or postcentral lesions, but tactile placing recovers more rapidly after precentral lesions, whereas hopping returns rapidly after postcentral ablation (Woolsey and Bard, '36). In these and previously reported experiments, hopping was elicited after precentral lesions, but only with large angular displacements of the limb, and the rapid series of short steps seen in normal hopping has never been seen. The rapid recovery of hopping after postcentral ablation is in accord with the finding that weight discrimination ability is not lost (Ruch, Fulton and German, '38). It is not yet clear, however, whether this test involves that form of sensory stimulation required for "position sense," which appears to be more seriously affected. The recognition of limb position is likely to be dependent upon information conveyed from receptors in the tendons and joints since muscle spindles respond to tension alterations as well as to position changes. In this connection it is interesting to note that the deep

somatic projection to the thalamus and cortex overlaps the central cutaneous representation and that potentials can be evoked by stimulation of fibers from peritendinous and periosteal receptors but apparently not by activation of muscle spindle afferents (Mountcastle, Covian and Harrison, '54).

Tactile placing is lost immediately after precentral or postcentral lesions, but within the first month after precentral ablation a tactile crossed-placing reaction can be elicited, and with the recovery of motor power the non-crossed tactile placing reaction appears. After postcentral ablation placing cannot be elicited by bringing the dorsum of the hand or foot into contact with an absorbent cotton surface, but if the limb is brought into contact with a hard surface, especially when a large area is contacted, a placing reaction can readily be elicited. This may have been the form of placing noted in some previous experiments (Ferraro and Barrera, '35). This form of placing is not necessarily comparable to the tactile placing described by Bard ('38); perhaps slight joint displacement results from such coarse contact, thus constituting a "proprioceptive" placing reaction. On the other hand, since this reaction can be elicited without obvious joint displacement at a time when considerable angular displacements are necessary to elicit hopping, a crude form of cutaneous placing cannot be ruled out. The former interpretation is in better accord with the finding that the "grasp reflex" returns rapidly after operation and with the evidence of the animal's ability to discriminate among objects in darkness.

Neurological observations on the motor effects of Rolandic lesions in animals have led to numerous conflicting reports, few of which even include histological examination of the lesions. Although lesions of the individual architectonic fields of the Rolandic cortex do result in different symptomatology, these differences cannot be simply designated in terms of the difference between paralysis, "loathness to move," the presence of spasticity or flaccidity, or a defect in cutaneous or proprioceptive sensation. To some extent all of the symptoms are present after all Rolandic lesions, but differences in

degree and duration are usually evident (Hamuy, '56; Travis, '55; Travis and Woolsey, '56; Pribram et al., '56).

A defect in locomotion follows precentral, postcentral or combined lesions, but complete paralysis is not observed. During the first postoperative month postcentral lesions produce as severe a deficit in hasp box performance as any total or subtotal precentral lesion, (Pribram et al., '56; Hamuy, '56) but recovery is more rapid after postcentral lesions. Animals with combined pre- and postcentral lesions, though they show severe paralysis, are able to secure food with their affected extremity within the first postoperative month. Thus, though the most severe and prolonged deficit follows precentral ablation, as previously noted by Welch and Kennard ('44), the entire Rolandic region seems to be concerned with movement. It is also clear that Rolandic beta activity is blocked by movement of the contralateral limb, and most markedly in the precentral gyrus (Kruger and Henry, '57).

Differences between the motor deficit following precentral ablations and those following postcentral ablations can be specified. Total precentral ablations result in a more marked and prolonged spasticity which is readily observed in the more proximal portions of the affected limbs. Except for a transient spasticity during the first postoperative week, the postcentral animals show a marked hypotonia, as described earlier by Minkowski ('17) and Peele ('44). The initial spastic phase is similar to that observed with restricted precentral removals (Denny-Brown and Botterell, '48, and Berman, Kruger and Fulton, '54). The marked increased resistance to passive manipulation seen with precentral lesions also contrasts with the prolonged decrease seen after postcentral lesions. But combined lesions of the precentral and postcentral regions produce greater and more prolonged spasticity than occurs in animals with precentral lesions only, as noted previously by Welch and Kennard ('44). This finding suggests that ablation of the postcentral gyrus can also contribute to spasticity. The specific form of the motor de-

fect produced by Rolandic lesions appears to be dependent on the area, size, and order in which the lesions occur.

The comparison of these results for the primate with experiments using other mammals is difficult because of the less precise demarcation of "sensory" and "motor" fields in subprimates. Zubek's studies on the rat ('51 and '52a) and Allen's study on the dog ('47) are in agreement with the present study, but Zubek ('52b) reported that one cat with ablation of areas I and II showed a "permanent" loss of tactile discrimination ability. Ablation of areas I and II in the monkey might also result in a permanent defect in a difficult tactile discrimination, but this problem requires further study. The results reported here provide suggestive evidence that sufficient somatic sensation remains to perform the simpler somatosensory tasks. These results are analogous in some respects to the finding of Meyer and Woolsey ('52) that ablation of auditory areas I and II and degeneration of the parvocellular medial geniculate nucleus do not produce a complete loss of auditory discrimination capacity in the cat, but that such deficits can be produced by ablation of appropriate adjacent cortical fields (Rose and Woolsey, in press). The results of the present experiments suggest that in addition to the tactile receiving areas, the precentral field must be removed to produce a prolonged and severe somatosensory deficit. The slight degeneration of the nucleus lateralis posterior indicates that some posterior parietal cortex has also been damaged in the animals with postcentral involvement, but it would seem unlikely that a minimal *unilateral* posterior parietal lesion would be contributory to the defect found after combined precentral and postcentral ablation.

Within the limitations of the retrograde degeneration method the material presented here supports the conception that the ventro-basal nucleus, as defined by cytoarchitectonic or evoked potential criteria (Mountcastle and Henneman, '52), projects to the tactile receiving area of the cerebral cortex. Most of this projection is to the postcentral gyrus,

but a small portion of the arcuate division of the nucleus projects to the precentral granular face area—the tactile area for the mouth (Benjamin and Welker, '57). The portions of the ventral group rostral to the ventro-basal complex appear to project upon the precentral gyrus.

The results of these experiments suggest that although the precentral and postcentral cortical fields can be functionally differentiated and have different anatomical connections, they also share several functional properties. General acceptance of the doctrine of a Rolandic sensorimotor unit has been based primarily on the anatomical contiguity of these fields and it is generally assumed that the cortical interconnection of the precentral and postcentral gyri is essential to its functional unity. There is also a possibility that there is a superimposition of sensory and motor function rather than a simple overlap and interconnection of precentral and postcentral fields. Sperry's ('47) demonstration that extensive interruption of intracortical connections in the Rolandic region does not produce any evident defect in sensorimotor function leads one to suspect that the "unity" of this region is not solely dependent on cortico-cortical connections. The reports of sensation produced in patients during electrical stimulation of the cortex (Penfield and Rasmussen, '50) also indicate that both gyri share some common function. However, the afferent projection to the precentral gyrus is clearly different from that to the postcentral gyrus. The tactile projection to the precentral region recently reported by Benjamin and Welker ('57) lacks a separate somatotopic pattern. The potentials evoked in this region by peripheral nerve stimulation appear to require spatial summation of many fibers, display longer latencies than occur postcentrally at points on the free surface of the agranular field, and they appear to be independent of the ventro-basal complex (Kruger, '56).

It is likely, therefore, that the organization of precentral afferents differs from that of the postcentral gyrus in that it represents a crude form of non-localized cutaneous sensation

lacking a precise somatotopic pattern. Its fibers are probably not received from the tactile thalamic region. It is also clear that these fibers are not conveyed via the cerebellum. Recently, Towe and Patton ('57) have presented evidence that "Betz" cell unitary discharge can be driven by both antidromic pyramidal discharge and stimulation of the medical lemniscus. The interaction of antidromic and orthodromic excitation of "Betz" cell units also lends support to the concept of a superimposed afferent and efferent pattern in the motor cortex.

The functional role of the precentral gyrus in sensation is thus still not clear and certainly not equivalent in importance to that of the postcentral field. It is possible that the most severe defect in sensation after postcentral injury is concerned with the spatial aspects of sensation rather than a simple modality loss as has been suggested by Denny-Brown, Meyer and Horenstein ('52). The ability of a monkey with a postcentral gyrus ablation to show a placing reaction only when the extremity is in contact with a broad surface suggests that spatial summation may be necessary for discrimination and that the precentral gyrus may be functionally involved in handling a more crude, less localized form of information. A more complete analysis based on the study of a variety of discrimination problems after Rolandic lesions would be a helpful step in clarifying the functional properties of the precentral and postcentral gyri.

SUMMARY

The experiments reported were designed to study the functional role of the precentral and postcentral gyri with special reference to somatic sensation. Six monkeys were studied neurologically and while they performed visual and somesthetic discrimination tasks. The somesthetic tasks were performed in darkness and the animals were observed with an infra-red television device.

Ablation of somatic receiving areas I and II resulted in a severe tactile defect in the limbs contralateral to the lesion,

but a simple somesthetic form discrimination task was re-learned after unilateral and bilateral ablation. The ability of the animals to perform this task together with the presence of a "grasp reflex" and a form of cutaneous placing reaction suggests that some form of cutaneous sensation remains after ablation.

Ablations of the precentral agranular cortex result in a temporary sensory defect detected by the initial loss of the "instinctive grasp" and tactile placing reactions, but the somesthetic form discrimination was performed without deficit.

Unilateral ablations of the combined precentral and post-central arm area resulted in a severe sensory loss in the contralateral arm and the two animals studied were unable to perform the somesthetic discrimination task with the affected limb although a visual discrimination task was readily performed.

These findings suggest that the precentral gyrus subserves somesthesis in the absence of the postcentral gyrus. The relation of these findings to the character of the precentral afferent projection and to the functional differences between the precentral and postcentral gyri are discussed.

Analysis of the retrograde thalamic degeneration in the brains of the animals reported here, suggests that the granular tactile receiving area is the essential projection field of the ventro-basal (tactile) complex of the thalamus. Observations on the effect of Rolandic lesions on motor function are also presented.

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PLATE 1

EXPLANATION OF FIGURES

6 Photomicrograph of the retrograde degeneration in nucleus ventralis lateralis resulting from ablation of the precentral cortex in the left hemisphere of no. 224. Magnification 10 ×.

7 Photomicrograph of the retrograde degeneration of nucleus centralis lateralis and a portion of the arcuate nucleus in the same hemisphere as shown in plate 1 (no. 224). The regions of degeneration are enclosed in broken lines. Magnification 10 ×.

8 Photomicrograph of the retrograde degeneration of the ventrobasal nucleus after ablation of somatic areas I and II (no. 203 L). The *intact* cells of the arcuate are enclosed in a broken line and are surrounded by a region of severe degeneration. Magnification 10 ×.