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THE EFFECTS OF RADICAL DISCONNEXION OF OCCIPITAL AND TEMPORAL CORTEX ON VISUAL BEHAVIOUR OF MONKEYS[1]

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THE behavioural analysis of primates prepared with experimental brain lesions has flourished during the past twenty years. These studies were initially designed to develop animal "models" of clinical neurological syndromes. The usefulness of such models has been that precise neurobehavioural analysis could be performed, an advantage over the ordinary indefiniteness of clinical material. As a result a good deal is known about the functions in monkey of the part of the cerebral mantle which is usually referred to as "association cortex." One of the major findings has been that the posterior association cortex can be subdivided into areas each of which serves one or another specific sensory modality. Thus cortical loci for taste (Bagshaw and Pribram, 1953), somesthesis (Pribram and Barry, 1956; Wilson, 1957), audition (Weiskrantz and Mishkin, 1958; Dewson *et al.*, in preparation), and for vision (Blum *et al.*, 1951; Chow, 1951; Mishkin and Pribram, 1954) have been delineated. Interestingly, however, these loci are *not* where one would expect them to be, i.e. immediately adjacent to the primary sensory areas of the modality served. Rather, the sensory specific association areas proved to be some distance from the "parent" primary cortex.

Further, a tantalizing result was obtained when the primary sensory systems were circumsected by removals of the cortex intervening between the projection and the sensory specific association areas: little deficit in discrimination behaviour of any sort could be demonstrated. Other forms of disconnexion such as crosshatching the cortex (Pribram *et al.*, 1966) also failed to affect discrimination.

Much of the extensive analysis of the problem has been effected by experiments performed in the visual modality. These experiments provide excellent examples of the issues involved. Visual discriminations are seriously disrupted by removals of the inferior temporal convolution. A similar syndrome is not seen after removals of the prestriate cortex. The question therefore arises as to how the inferotemporal

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cortex exerts its influence over visual processes. The common view is that visual information is relayed via transcortical connexions from the striate to the association cortex and that at each relay, operations are performed on the information. This might be called the "serial processing model" of cortical function. For the monkey this model encounters serious difficulty on the basis of the results of the experiments noted above.

A second possibility suggests that the inferotemporal cortex receives visual information from the striate cortex via U-fibres. Unfortunately such direct U-fibre connexions have not been shown to exist. All known connexions emanating from striate area relay in the peristriate cortex which, as already noted, can be removed without much disruption of visual discrimination.

A third possibility has been proposed (Pribram, 1958, 1960) and tested: viz. that the major connexions between the visual mechanism and the inferotemporal cortex traverse in a cortical-subcortical, rather than a cortico-cortical direction. Special emphasis has been given the possibility that the inferotemporal cortex exerts efferent, corticofugal control over subcortical visual mechanisms (Spinelli and Pribram, 1966, 1967). This view has been called the "parallel processing model."

It is within this background of primate laboratory studies that the present experiment was undertaken. It seemed reasonable on the basis of the experimental evidence in hand to pursue the parallel rather than the serial view of information processing in the brain. Yet, new evidence from other sources continued to be interpreted and interpretable according to the serial model. Most important of this new evidence were (1) the unit analyses of the receptive fields of cells at various stations in the visual system and (2) the reports of clinical syndromes carefully analysed, both behaviourally and anatomically. The micro-electrode studies of visual neural units did not as yet pose a serious problem. They were performed mostly in cats and when monkeys were investigated a somewhat different organization emerged (*see* Discussion). As yet no definitive receptive field experiments on non-primary primate cortex have been reported. The clinical observations, on the other hand, seemed to provide evidence directly opposite to that obtained in the experimental studies.

It became important therefore to examine the experimental results once more. These were based on the assumption that if a chain of transcortical connexions from primary to "association" cortex were crucial then one would expect that cutting the middle steps would disrupt performance as severely as would removal of the "association" cortex itself. When this does not occur it could be due to faulty experimentation—especially to incomplete surgery. This argument is especially effective when addressed to negative findings and can only with difficulty be countered. Yet we felt that an attempt to produce a behaviourally tested preparation with as radical a removal as present surgical skill would allow, should prove an up-to-date and effective experimental test of the serial view of the mechanism of operation of the subhuman primate "association" cortex. Thus, to counter the charge of insufficiently complete surgery to the extent that it can be countered at all, and to

satisfy ourselves as to the actual capacities of monkeys with such radical disconnexions, we prepared three animals with massive bilateral removal of the cortex surrounding the visual sensory projection area and tested them, both for retention and original learning of some difficult pattern discriminations.

The issue raised by these experiments is an important one. If indeed the evidence holds that in the monkey corticocortical disconnexions are ineffective in disrupting behaviour, the clinical evidence supporting the disconnexion syndromes needs to be re-evaluated in terms of the possibility that cortico-*sub*cortical rather than cortico-cortical connexions are interrupted. If it should turn out, as at present it appears likely, that some clinical syndromes (especially those in which verbal coding is involved) cannot be explained by recourse to the cortico-*sub*cortical explanation, a great deal will have been gained from the experimental primate studies. For the first time reliable evidence will be available about the critical difference in brain organization which separates man from monkey.

Method

Subjects

The subjects were three naïve Rhesus macaque monkeys weighing about 6 kg. at the time of sacrifice. They were housed in individual cages and fed about 16 standard lab pellets during the periods when they were being tested and more when they were idle. Supplementary vitamins and fresh fruits were given throughout the experiment.

Apparatus

A computer-controlled testing apparatus similar to the one used in this experiment has been described elsewhere (Pribram *et al.*, 1962). The subject sat in a transport cage facing 16 panels in a 4×4 array. Each panel was $1\frac{3}{8}$ in. in diameter. Patterns were displayed from behind on some number of panels, and presses of the correct panel were rewarded by a banana pellet delivered into a dish beneath the array. Presentation of stimuli and rewards was controlled by a PDP-8 computer located in an adjacent room, which also recorded the stimulus pressed, its location, and the latency of the response measured from the stimulus onset. The testing chamber was constantly illuminated by a dim light, and the noise of a blower masked extraneous sounds.

Training

The monkeys were pretrained to press a single illuminated panel by displaying (and rewarding) a gradually reducing series of numeral 1's. Pretraining was continued until approximately 60 consecutive responses had been made to a single 1. The subjects were then trained to criterion (90 per cent correct responses over two days—100 trials) on a discrimination between the numerals 3 and 8 (3 rewarded). In all phases of testing, 50 trials were given per day unless a subject refused to test. They were tested for retention after two weeks and brought again to criterion. One monkey (283) did not have surgery until three months after retention testing. He was retested for 50 trials just before surgery.

Postoperative retention was tested following a few days of pretraining with 1's displayed (*see* Postoperative Recovery). This was done to minimize the effects of any field defects which might be present. When criterion was reached on the 3–8 problem, the subjects learned a new discrimination between the numerals 4 and 6 (4 rewarded).

Surgery

Surgery was carried out under aseptic conditions using intravenously administered diabutol anæsthetic (36 mg./kg.). An osteoplastic flap was turned over the parietal and anterior occipital regions. Cortex was aspirated subpially along both banks of the lunate sulcus, forward over the convexity of the hemisphere extending anteriorly to include the inferior bank of the interparietal sulcus. Dorsomedially the cuneus and precuneus were removed entirely; ventromedially great caution was exercised because of the proximity of the fovcal radiation fibres. However, in one subject (283) the entire cortex ventral to the inferior occipital sulcus was ablated and the resection extended upward on the medial surface to the borders of the calcarine fissure. At the completion of surgery the dura and bone flap were sutured in place and the skin closed. Bicillin was routinely administered as the subject roused from the anæsthetic. At least two weeks were allowed for recovery before any testing was attempted.

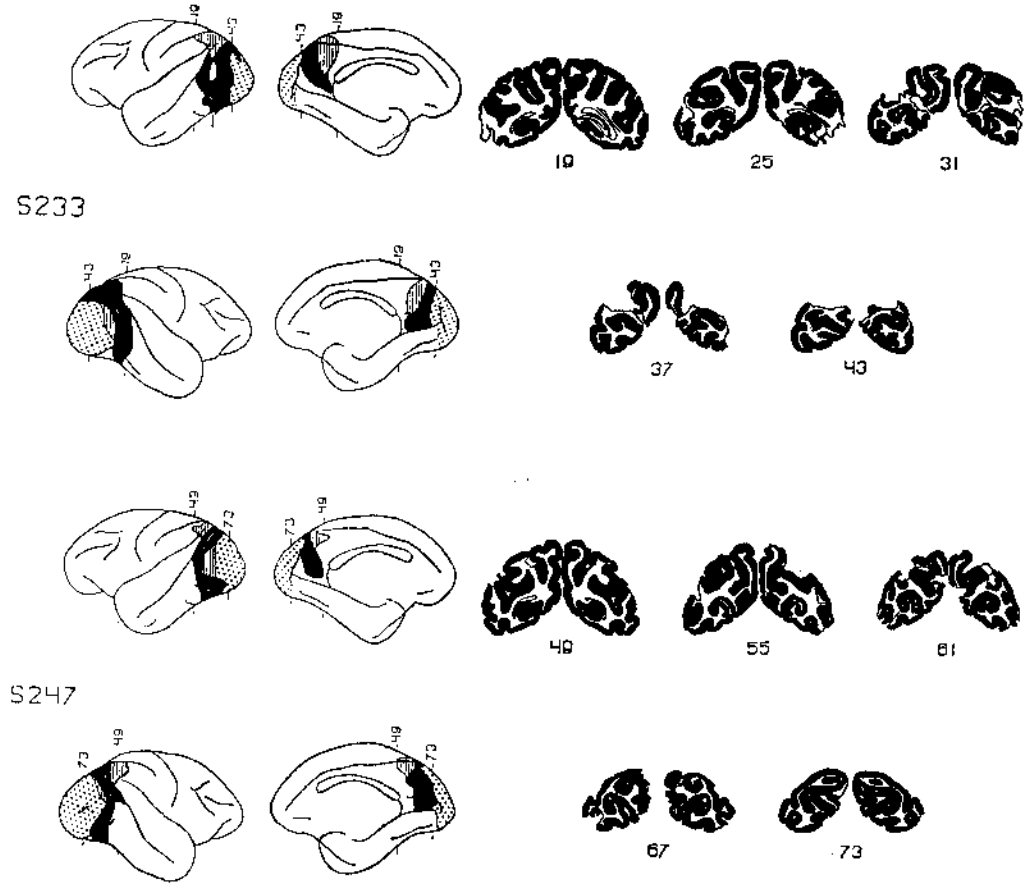

FIG. 1.—Reconstruction on standard brain of prestriate lesions in subjects 233 and 247. Solid black indicates lesion visible from surface; stripes indicate deep lesion only. Dots show undamaged striate cortex. Five representative cross sections are taken at 3 mm. intervals over the region of the lesion.

Postoperative Recovery

Most monkeys recovered from surgery uneventfully. All showed some temporary misreaching and lack of response to objects moved into portions of their visual field. This disappeared within a few days, and when they were placed in the testing apparatus they showed no obvious difficulty in responding to illuminated panels.

Subject 283 showed more severe visual disturbance. For some time no response to visual stimuli (except pupillary responses) could be elicited, and up to three or four weeks after surgery there was little response to stationary stimuli. Later, objects moved into the field from below elicited no response, although they did when moved in from above. About a month after surgery the monkey was presented on three different days with an array of food and non-food objects and by the second day was consistently selecting the food, although at times it misreached and bumped other objects. When finally brought into the testing chamber, the monkey at first failed to distinguish those panels on which cues (numeral 1's) were displayed from the vacant panels. Gradually, responses to blank panels diminished, and when the monkey was consistently responding to 1's, formal discrimination testing was resumed (about two months after surgery).

Histology

At the completion of the experiment the monkeys were given an overdose of barbiturate and perfused intracardially with normal saline followed by 10 per cent formalin. The brains were removed, placed in

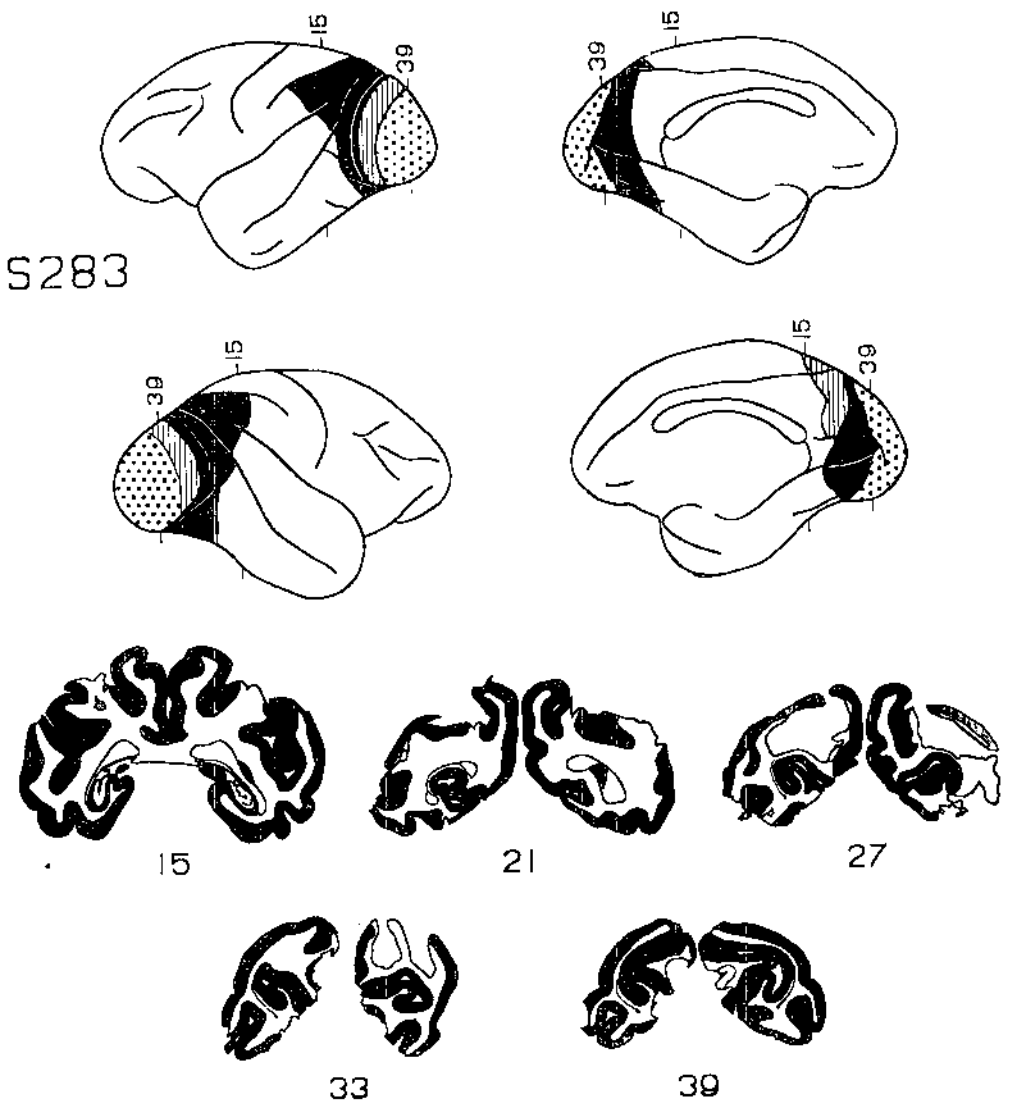

FIG. 2.—Reconstruction of prestriate lesion in subject 283.

formalin and later in 20 per cent alcohol. They were cut in 50 μ frozen sections, saving every tenth. Odd-numbered sections were stained with thionin (*see* procedure in Sherer and Pribram, 1962). These extensive lesions caused considerable distortion in the shape of the posterior end of the brain, and direct reconstruction did not produce an informative view of the areas removed. Hence the measurements of the lesion were transferred as accurately as possible to standard lateral and medial views of the hemispheres. Fig. 1 shows these reconstructions for subjects 233 and 247. Solid black indicates damage visible from the surface; stripes indicate deeper lesion. Damage in the interparietal sulcus appears on both views. The dotted areas indicate undamaged striate cortex. Also shown are five actual cross sections taken at 3 mm. intervals over the extent of the lesions. Comparable data for subject 283 appears in fig. 2. Degeneration in the lateral geniculate nuclei (LGN) of all three subjects is shown in fig. 3.

The lesions in the first two subjects (233 and 247) are very large, and are complete except for some prestriate cortex on the medial surface above and below as well as inside the calcarine fissure. A small

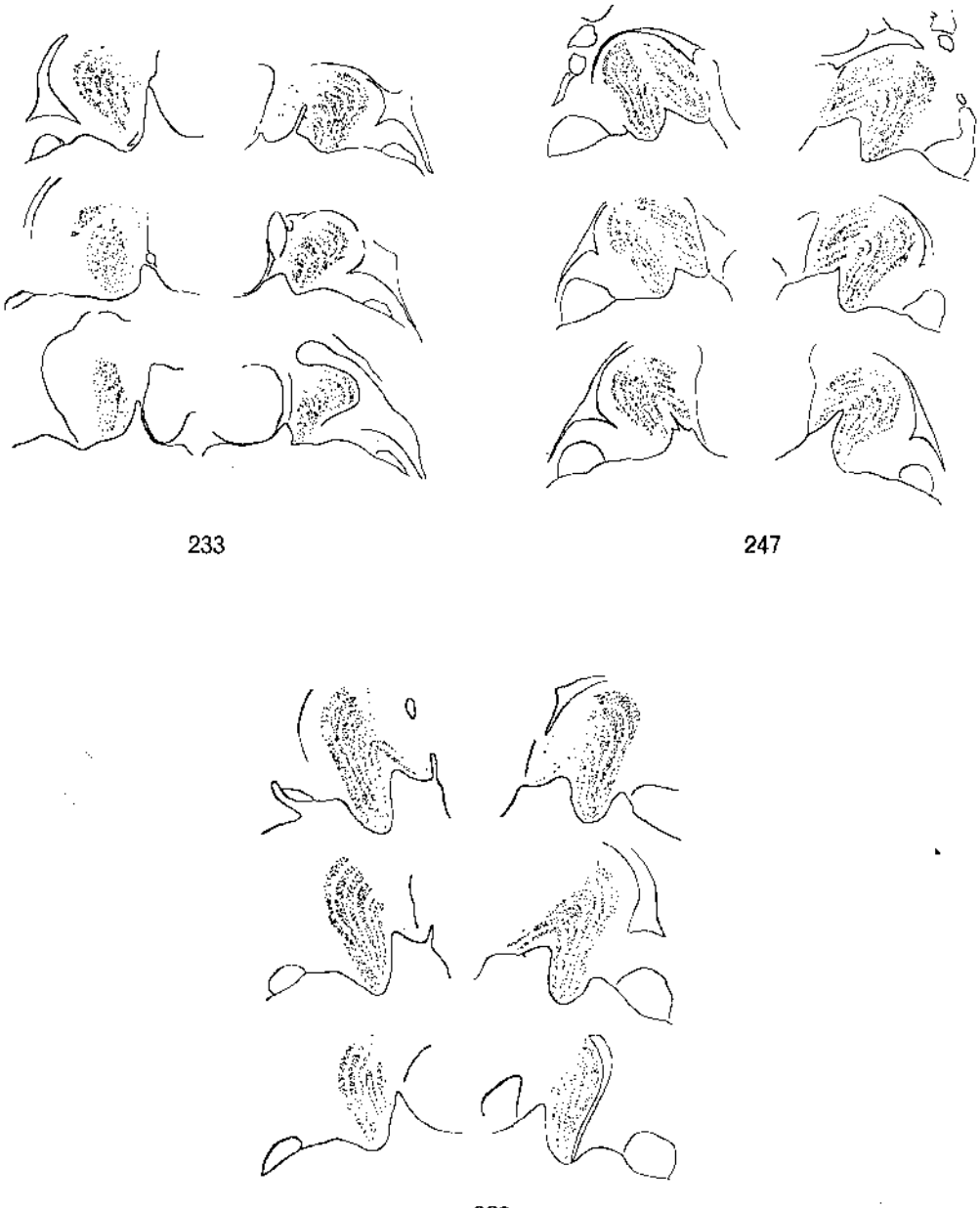

FIG. 3.—Three sections at 1 mm. separation through the lateral geniculate bodies of each of the prestriate subjects. Degenerated areas are shown by lighter stippling.

amount also remains at the posterior end of the gyrus which lies beneath the inferior occipital sulcus. The lateral surface of the interparietal sulcus and the posterior bank of the lunate are destroyed, including some cortex from areas PE and PG (von Economo), and striate cortex along the lunate sulcus. Degeneration was found in the medial and dorsal LGN in subject 233 and in a central wedge and a more medial segment in subject 247.

Monkey 283 has an essentially complete lesion. All of the areas mentioned above are gone as well as all of the cortex above and below the calcarine fissure and beneath the inferior occipital sulcus. Only a small piece of prestriate cortex within the rostral extreme of the calcarine fissure itself is spared, but this tag is isolated from the remainder of the non-striate cortex. There is considerable damage to striate cortex, especially laterally, and the entire medial half of each LGN has degenerated.

RESULTS

All monkeys including subject 283 performed well on the initial formal post-operative tests. The discrimination performance (trials prior to the criterion run) of each of the subjects is shown in Table I. The postoperative learning scores on the

TABLE I.—DISCRIMINATION PERFORMANCE OF PRESTRIATE *Ss* AND NORMAL *Ss*. ENTRIES ARE TRIALS PRIOR TO THE CRITERION RUN

Ss	*3–8 Learning*	*Preoperative Retention*	*Postoperative Retention*	*4–6 Learning*
NORMALS (N=10)				
Average	285	—	—	—
PRESTRIATES				
S 233	250	0	450	150
S 247	200	100	150	275
S 283	250	0	300	475
Average	233	33	300	300

3–8 discriminations are in the middle of the range (100 to 625 trials) set by the pool of 10 normal subjects from two other studies, who were trained in a comparable manner on this same discrimination (Pribram *et al.*, 1966; Reitz, 1968). The average number of trials is 233 for the prestriates and 285 for the normal pool.

Following the prestriate lesions all of the monkeys began with more than 75 per cent correct responses on the first day of postoperative testing and consistently improved their performance over the succeeding days (the scores of the subjects on the first day are, in order: 76 per cent, 86 per cent, and 84 per cent as compared with 94 per cent, 94 per cent, and 96 per cent on the last preoperative day). The animals required an average of 300 trials to meet criterion, with only one subject showing savings over original learning. Yet their performance is markedly better than that of the monkeys in an earlier experiment (Pribram *et al.*, 1966) whose inferotemporal cortex has been undercut (severed from its vertical input and output). In that experiment the two subjects tested for postoperative retention did not reach criterion in 1,000 trials and remained around the chance level of correct responses over this entire period. Further, all of the monkeys with prestriate lesions readily learned the 4–6 discrimination postoperatively, with a group average of 300 trials.

There is good evidence that the postoperative performance difficulty in the present group of monkeys with prestriate resections was in large part due to the massive field defects they sustained. For instance, an analysis of the location of erroneous presses made by subject 283 on the initial postoperative test days shows that *all* were committed in the blind portion of the subject's visual field, as determined by informal testing and LGN degeneration. The left side of Table II shows the percentage of

TABLE II.—PROPORTION OF TRIALS ON WHICH THE THREE APPEARED IN EACH LOCATION (LEFT HALF), AND PROPORTION OF ERRORS MADE IN EACH LOCATION MINUS THE CORRESPONDING PROPORTION OF APPEARANCES OF THE THREE (RIGHT HALF) FOR EACH *S*.

	·16	·04	·04	·02	·32	−·04	·02	·08
	·07	·00	·02	·08	·03	·00	−·02	−·05
S 233	·00	·00	·09	·06	·00	·00	−·03	−·03
	·08	·05	·10	·16	−·05	−·05	·00	−·16
	·20	·03	·04	·02	−·11	−·01	−·02	·02
	·07	·00	·02	·09	−·06	·00	·00	−·06
S 247	·00	·00	·07	·07	·00	·00	·00	−·04
	·07	·05	·10	·16	·05	·03	·17	·02
	·18	·02	·04	·04	−·03	−·02	−·04	−·01
	·08	·00	·02	·13	·00	·00	−·01	−·04
S 283	·00	·00	·06	·08	·00	·00	·02	−·03
	·06	·04	·12	·12	·08	·01	·09	−·01

times the three appeared in each of the panel locations for the subjects during postoperative testing (this is not a uniform distribution over the panels and varies from subject to subject depending on the order in which they were tested and the number of times they failed to complete an entire 50-trial block). The right side of the table shows the difference between these percentages and the percentage of times an error was made when the three appeared in each location. Thus a positive value indicates that more errors were made when the three appeared in some location than would have been expected on the basis of the probability of its appearing there. It is apparent that subject 283 made most of its errors when the three appeared on the lower panels, that is, in its blind field. Thus these initial errors committed appear to be due to the field defect produced by cutting optic radiations and not to the prestriate lesion directly. Behaviourally this is to be expected: when the three is not immediately visible to the operated subject because of the field problem, the problem is transformed into a successive discrimination until the subject learns to withhold his responses and to search for the "missing" stimulus.

DISCUSSION

The results for monkey 283, whose lesion was complete except for an isolated tag of prestriate cortex within the anterior portion of the calcarine fissure, are the most crucial in interpreting the effects of these disconnexion experiments. This is the most extensive prestriate ablation yet reported. The visual cortex has been completely circumscribed and yet this subject's performance was at 76 per cent on the first postoperative day in formal testing and continued to improve thereafter. This is in spite of a large scotoma, which from behavioural observation and the locus and extent of geniculate degeneration appears to involve the entire lower visual field including the macular region. As noted, the fact that the spatial location of the errors made by this monkey correlate with the field defect strongly suggests that the

difficulties in visual behaviour experienced by the subjects in this experiment were due largely to the surgical invasion of the optic radiations, not to the removal of prestriate cortex *per se*.

The other monkeys showed even better performance on the first day of formal postoperative testing. Their lesions were less extensive than that of subject 283, but were still larger than most of those previously reported, particularly on the medial surface and at the inferior lateral border. The lesions did disconnect inferotemporal and striate cortex radically, isolating especially the known cortical representation of the fovea. The cortex of the interparietal sulcus which Mishkin (1967) has claimed must be removed in order to produce a deficit, is also gone. Further, the lack of effect of disconnexion is not limited to retention of the preoperatively learned discrimination, but applies to new learning as well. Thus an additional experimental result has been obtained which calls into question the commonly held view that the essential mechanism of operation of the primate "association" areas is through a system of corticocortical connexions from primary projection cortex.

Why then the persistence of Flechsig's 1896 pronouncement on the functions of the primate cerebral cortex? Flechsig based his conclusions on the data available to him: viz. that only certain parts of the brain cortex received projections from the thalamus, which in turn was known to be the head ganglion of the sensory systems. He reasoned, therefore, that the ubiquitous corticocortical connexions served as a substrate for function in the remainder of the cerebral mantle and that the most likely functions served by such connexions would be associative. Since Flechsig's time much of the rest of the cortex has been shown to receive projection fibres from the thalamus, albeit not from the input pathways *per se*. Further, crosshatchings (Pribram *et al.*, 1966) and circumsections which disconnect the "association" cortex from the primary sensory and motor regions have failed to affect behaviour. Yet, as noted in the introduction, the argument can be made and is made that these are only negative findings which reflect a failure of surgery to sever all connexions completely and of psychology to furnish sufficiently sensitive measures of behaviour, an argument difficult to counter. But perhaps the most persuasive support for the century-old view comes from clinical evidence obtained on man and from electrophysiological evidence obtained on cats.

As noted in the introduction, electrophysiological studies have been performed for the most part on the cat. None the less these experiments have lent their weight to the view that intersensory associations take place in cortical regions adjacent to the primary projection systems (Thompson *et al.*, 1963). Polysensory systems are described adjacent to the primary; they are, however, polysensory on the basis of their subcortical connexions. Further, comparative analysis shows that these polysensory systems are not homologous to the primate sensory specific "association" cortex. It is worth emphasizing once again that neurobehavioural experiments in the monkey have repeatedly shown the functions of the primate areas under study to be *sensory specific* (Bagshaw and Pribram, 1953; Dewson *et al.*, in preparation; Wilson, 1957).

An additional electrophysiological source of support for the idea of transcortical source of input to the association cortex has come from unit analysis of the visually receptive cortex of cats. Hubel and Wiesel (1965) have demonstrated a hierarchy of complexity in stimulus shapes to which cells in the marginal gyrus (*gyrus lateralis*, Winkler and Potter, 1914) will respond maximally. This hierarchy is arranged from posteromedial to anterolateral in the gyrus: simple cells (which respond to lines displayed at specific angles) are found posteriorly and medially; as one proceeds anterolaterally, complex cells (which respond best to lines displayed at specific angles over larger areas) and hypercomplex cells (which respond to corners) become the more frequent. Here again, however, the arrangement in the monkey differs (Hubel, 1967). The hierarchy here ranges from concentric to complex and hypercomplex cells and is related in the primate to the layers of the receptive cortex: the simpler cells are found in layer IV which forms the terminus for thalamic afferents; higher (and lower) layers progressively contain a greater number of units with more complex sensitivities.

Confusion is abetted by an attempt to homologize uncritically between the anterolateral portion of the marginal gyrus of cat and the cortex of the banks of the lunate sulcus of the monkey. Both are characterized by the presence of large, more or less pyramidal looking cells in layer III. This has led to the labelling (Otsuka and Hassler, 1962) of both as extrastriate (although in the cat the stripes in this "extrastriate" are more marked than in what is usually labelled "striate"). In other respects the cat and monkey areas in question differ markedly: cat anterolateral marginal gyrus receives an essential projection from the lateral geniculate nucleus of the thalamus; monkey lunate cortex receives no such projection. The physiologically obtained topographic map of the cortical projection of the retina includes without break the cat cortex under consideration; in the monkey this map stops sharply short of the perilunate cortex. (Cytoarchitecturally, also, the demarcation in the monkey is abrupt; in the cat, difficult.)

The anatomical question that remains to be discussed is that of the existence of long U-fibres connecting the striate with the inferotemporal cortex, fibres untouched by the resections made in the present study. As far as is known, U-fibres to and from visual receptive cortex (area 17 of Brodman; area O.C. of von Economo) are all short and course only as far beyond its own confines as the immediately adjacent peristriate tissue (area 18; O.B.). The possibility exists that this is not the case for the medially located striate cortex buried in the anterior portion of the calcarine fissure, for this has not been studied, but there is no reason, at this time, to think that in this respect the organization of this portion of the projection cortex differs.

The clinical evidence in support of the cortico-cortical mechanism is more complex and also more difficult to refute because of ambiguities in patho-anatomy and in the descriptions of behavioural observation. None the less, two major categories of evidence can be made out: (1) brain-injured patients show disturbances which appear to be limited to functions which integrate across sense modalities—e.g. disturbances of a visuo-somatic spatial frame of reference by parietal lesions (Semmes *et al.*,

1960); and (2) brain-injured patients show disturbances which "disconnect" previously integrated psychological operations—e.g. the verbal from the nonverbal identification of objects (Geshwind, 1965). The intersensory disturbances are open to other interpretations. There is the possibility that impairment in one sensory category (e.g. the somatic) underlies an apparent integrative disturbance: e.g. visuosomatic-misreaching, for instance, has been shown in monkey to occur together with deficits in discrimination in the somatic mode while leaving intact those in the visual (Wilson, 1957), yet proper reaching has ordinarily been used as a critical test for estimating the integrity of "visuosomatic space." In a similar fashion an impairment in the verbal process *per se* could leave the nonverbal intact (Miller *et al.*, 1960; Pribram, 1965; Rosenberger *et al.*, 1968) yet appear to dissociate the one from the other. Should this prove to be the case, the possibility arises that cortico-cortical disconnexions are linked primarily to disruptions of verbal functions and therefore their effects would be manifest only in man.

In short, it is our view that a critical analysis calls into question the evidence usually adduced to support the common cortico-cortical view of the mechanism of operation of the subhuman primate "association" cortex. The experiment reported here, added to the facts already in the literature and the others in this series (Pribram *et al.*, 1966; Reitz, 1968), makes unwarranted the neglect of alternative explanations which rely on cortico-subcortical influences and which are more consistent with the body of experimentally produced evidence. Should it prove to be the case, as now seems likely, that certain clinical neurological syndromes cannot be explained except by recourse to an explanation in terms of cortico-cortical disconnexions, reliable evidence will for the first time be available as to the critical difference in brain organization which separates man from monkey.

Summary

An experimental study was made in which the striate areas of monkeys were bilaterally and completely circumsected by extensive removals of peristriate cortex. Such cortico-cortical disconnexions failed to disrupt the discrimination of visual patterns. This result, added to others of a similar nature, calls into question the importance for behaviour of cortico-cortical connexions in subhuman primates. The effects of lesions of the sensory specific association areas of monkeys are more readily explained by recourse to their influence on subcortical mechanisms. If some clinical syndromes fail to yield to similar explanation, a critical difference in the organization of the brain of monkey and that of man will have been uncovered.

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