INTRODUCTION

In the visual system from retina to cortex the diversity of cell types as judged by their electrophysiological response properties may be explained by the repeated convergence of one group of cells upon another, using both excitatory and inhibitory mechanisms. Such a convergence starts within the layers of the retina itself. It continues from the retinal ganglion cells to the neurones of the lateral geniculate nucleus and thence to layer 4 of the striate cortex. In the striate cortex, interaction occurs within the columns between the cells of the different layers.

Throughout this retinal–geniculate–striate cortical system and then on to the anatomically defined areas 18 and 19 and in spite of the repeated convergence that seemingly occurs, a gross topography is maintained anatomically. This anatomical topography correlates with the physiology in the sense that cells in any particular part of these structures will be excited only if the appropriate stimulus is presented in the appropriate, restricted part of the visual field. This anatomical arrangement appears to dissolve at the posterior bank of the superior temporal sulcus which has been shown to receive afferents from large parts of the primary visual cortex. Such a convergence at first sight appears to represent a step in obtaining cells with very much wider receptive fields, enabling them to respond to wider parts of the visual field—a logical anatomical substratum to the fact that the visual world is seen as one continuous whole and not as a mosaic of discrete parts. But although the striate cortex sends a convergent input to the cortex of the posterior bank of the superior temporal sulcus, it also sends a topographically organised input to areas 18 and 19. This raises the possibility that there might be two independent pathways between the striate cortex with its precise topographical organisation and the cortex of the posterior bank of the superior temporal sulcus with its apparently eroded topographical organisation. One pathway, already demonstrated, is a direct one between striate cortex and the superior temporal sulcus. The other one might be an indirect pathway, through areas 18 and 19 and consisting possibly of more than one step at each of which the topographical organisation becomes less sharply defined. In pursuing this work on the
organisation of the visual cortical areas in the monkey, therefore, the obvious next step was to look at the cortical projections of areas 18 and 19. In particular we were anxious to see how these fields project to the posterior bank of the superior temporal sulcus, if at all, and also to study the possible projections from these two areas to the cortex lying between the anterior border of area 19 and the posterior bank of the superior temporal sulcus. Studies of callosal connections of prestriate cortex\textsuperscript{17} have suggested that the region lying between area 19 and the posterior bank of the superior temporal sulcus may be composed of more than one area in the sense that the interhemispheric degeneration was not continuous along the whole of this region but fell in separate foci within it. Finally, interhemispheric degeneration following punctate lesions in areas 18 and 19 was briefly examined in this study. Cortex anterior to the depth of the superior temporal sulcus was not examined for degeneration.

MATERIAL AND METHODS

Nine rhesus monkeys were used in the present study, the animals weighing about 2.5 kg at the time of sacrifice. For the aseptic operations the animals were given an initial dose of 0.3 ml Sernylan followed by gas (halothane, nitrous oxide and oxygen) anaesthesia. Postoperatively, they were maintained on a single dose of 2 ml bicillin antibiotic.

Six of the lesions were effected by passing a 0.1–0.2 mA positive current for about 5 sec through a steel electrode thrust in the appropriate region. The stereotaxic operations were performed with the monkeys in a Baltimore stereotaxic apparatus. The coordinates were obtained previous to the operation by using animals of roughly the same weight. The electrodes were coated with an epoxy resin and had a tip diameter of about 10 \( \mu \)m. I am much indebted to Dr. K. E. Webster for his help during these operations. Lesions of the striate–prestriate boundary were made by subpial suction. In all cases a small hole was made in the skull with a trephine, the lesion effected and then the dura re-approximated and covered with gelfoam and dental cement (to prevent herniation). Survival times ranged from 9 to 11 days.

The animals were perfused with formol–saline following Nembutal injections. The brains were removed, photographed and kept either in 10% formalin or a solution of 10% formalin in 30% sucrose for periods varying from 1 to 4 weeks. After this the brains were sectioned horizontally at 30 \( \mu \)m and 3 sections in every 30 or in every 15 were saved. These sections were stained by a slight modification of the Fink–Heimer silver impregnation method\textsuperscript{14} and sections passing through the lesion were also stained with cresyl violet for Nissl substance. Alternatively, all the sections were counterstained with cresyl violet.

The electrode penetrations were through the striate cortex (area 17) and this raises the question whether the resulting degeneration was due to a lesion of area 17, created by the electrode track\textsuperscript{15}. Three types of control were available for this, two indirect and one direct. An indirect control is formed by previous studies in which the striate cortex itself was the site of the lesion\textsuperscript{1,18}. The degeneration ensuing after such lesions has been studied and served as a control for the present study. Another
indirect control may be found in the lesions made in monkeys AM 5, AM 8 and AM 20. These animals suffered lesions of prestriate cortex at its boundary with the striate cortex, without involving the latter. The appearance of degeneration following such lesions in the same regions as that following lesions of inaccessible parts of prestriate cortex (made by electrode penetrations through striate cortex and white matter) was further evidence that the degeneration described was due to lesions of prestriate cortex itself. Finally, in one animal a direct control was available. In this animal, an electrode penetration, similar to the one in monkey AM 22, was made but no current was passed through the electrode. The track involved the striate cortex, white matter and prestriate cortex and ended in the anterior bank of the inferior occipital sulcus. This same control animal also sustained a lesion, small in size, in the striate cortex of the opposite hemisphere, not involving prestriate cortex. In this way a control was available for the

Fig. 1. Summary diagram of the location of lesions in this study. Arrows point to the sites of the lesions, most of which were made with electrodes thrust in the appropriate region of the lunate and inferior occipital sulci. The lesions were planned to fall in region of vertical and horizontal meridian re-representation in areas 18 and 19 of prestriate cortex. In this and subsequent figures ls = lunate sulcus; sts = superior temporal sulcus; ios = inferior occipital sulcus. The uninterrupted line in the cortex represents the primary visual cortex (area 17, V1).
control and the two sides of the brain were stained simultaneously, using the same solutions. Such a control for the control was important since in the case of a completely negative staining reaction, it would have been necessary to know whether this was due to the absence of degeneration or whether it was due to the difficulty of staining the degenerated fibres with the particular solutions, as sometimes happens. The degeneration that was observed was extremely heavy in the hemisphere with the lesion in striate cortex but relatively sparse in the opposite hemisphere with an electrode track passing through striate and prestriate cortex. When the degeneration in this hemisphere with an electrode track but without a current was compared to the degeneration obtained in monkey AM 22 with an exactly comparable electrode track but also with a current passed through it, the degeneration was found to be heavier and more extensive in the brain of the experimental monkey compared with the control. In brief, therefore, the degeneration outside areas 18 and 19 was undoubtedly due to lesions of prestriate cortex. Some of the degeneration in the posterior bank of the superior temporal sulcus may also have been due to the track passing through striate cortex. However, the density of degeneration here in the experimental animals compared with the control animal, together with the degeneration in this region to be seen in animals with lesions in prestriate cortex not involving striate cortex or white matter suggests that the posterior bank of the superior temporal sulcus also receives a direct projection from areas 18 and 19.

RESULTS

Previous anatomical studies\textsuperscript{1,16} have shown that anterior to area 17 there are two topographic re-representations of the vertical meridian, defining the posterior border of area 18 and the anterior border of area 19 respectively, with a horizontal meridian re-representation defining the boundary between the areas 18 and 19*. It also appeared from such studies, and also from the studies of Myers\textsuperscript{10}, that the prestriate areas related to the upper and lower visual fields are topographically separate. Lesions were therefore planned to fall in regions of vertical and horizontal meridian re-representations of areas 18 and 19 of both upper and lower prestriate cortex (see Fig. 1).

The region between the anterior border of area 19 and the depth of the superior temporal sulcus was examined for degeneration, bearing in mind that studies of inter-hemispheric connections\textsuperscript{17} have shown that this region may be divided into more than

\* In a recent paper, Spatz \textit{et al.}\textsuperscript{11} suggest that in \textit{Saimiri} only one cortical area, 18, receives projections from area 17 (apart from the field in the superior temporal sulcus). This would seem, at first sight, to represent a discrepancy with the pattern of cortical projections from the primary visual cortex in the rhesus monkey\textsuperscript{5,16}. Although the reasons for this discrepancy are not entirely clear, a possible explanation may be found in the siting of the lesions in the squirrel monkey compared to the rhesus monkey. In \textit{Saimiri} the lesions were relatively large and quadrantc rather than meridional as in the studies on the rhesus monkey. We have evidence that lesions falling within a quadrantc area of visual cortex in rhesus monkey would also yield one continuous belt of degeneration in the circumstriate cortex (in addition to the degeneration in the cortex of the superior temporal sulcus). There might be other reasons for this discrepancy but the explanation given above may offer a possible clue.

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one field on the basis of its interhemispheric connections which show discontinuities.

In monkey AM 20 a lesion was made by subpial suction just behind the lunate sulcus, that is within the vertical meridian re-representation in area 18. The lesion did not involve white matter and was about 2.5 mm in diameter (Fig. 2).

Degenerating fibres could be seen streaming from the lesion for a short distance down the posterior bank of the lunate sulcus (Fig. 2A). This field of degeneration was quite local. Another field of degeneration appeared at the medial end of the anterior bank of the lunate sulcus, corresponding to the position at which the vertical meridian is re-represented (Fig. 2B). At still more ventral levels, 3 new areas of degeneration appeared (Fig. 2C). One was halfway up the anterior bank of the lunate sulcus, another at the lateral quarter of the anterior bank of the lunate sulcus and spreading on to the prelunate gyrus. Finally, and somewhat more ventrally, a region of relatively coarse degeneration appeared at the posterior bank of the superior temporal sulcus. A few degenerated fibres could also be seen in the more anterior parts of the prelunate gyrus. This latter degeneration could be seen more clearly in another monkey, AM 5, which also had a lesion at the posterior lip of the lunate sulcus. In other respects the degeneration in AM 5 was similar to the one described for AM 20. Beyond the degeneration in the posterior bank of the superior temporal sulcus an occasional degenerated fibre could be seen more medially in the depth of the superior temporal sulcus. Finally, it should be noted that these fields of degeneration, with the exception of the one bordering the lesion, appeared below the level of the lesion.

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Fig. 3. Diagrams of sections, taken at the levels indicated, from brain AM 8 with a punctate lesion at the lower striate-prestriate boundary. The arrow on the surface view of the brain points to the lesion. Note that the degeneration picture following such a lesion shows a somewhat different pattern compared to the degeneration picture following lesions in upper striate-prestriate boundary; a major part of the degeneration in this instance falls in the posterior bank of the inferior occipital sulcus. This change in the pattern of degeneration may be ascribed to the change in the sulcal and gyral pattern of the brain from dorsal to ventral levels (compare sections 3B and 3C with section 2C). For further details see Discussion. Note also the very limited degeneration at the bottom of the superior temporal sulcus (section A).

In monkey AM 8 a lesion was effected by subpial suction in lower vertical 18, that is at the lower striate-prestriate boundary (see Fig. 3). The lesion did not involve white matter, was well clear of striate cortex and was comparable in size to the lesion in monkey AM 20. Just beyond the lesion, medial to the posterior lip of the inferior occipital sulcus, there was a region of fairly heavy degeneration. This field of degeneration, although continuous, appeared to be concentrated around two foci (Fig. 3C, D). The rest of the posterior bank of the inferior occipital sulcus was free of degeneration. In the anterior bank, a few degenerated fibres appeared at the region of the re-representation of the vertical meridian in area 19 (Fig. 3B). More laterally, there was a restricted area of degeneration, containing small numbers of degenerated fibres (Fig. 3B). This latter field of degeneration is considered to be the homologue, ventrally, of the field of degeneration that appears more dorsally in the anterior part of the prelunate gyrus and evidence for this may be found in the shifting position of the inter-
hemispheric fields of degeneration as these are traced from dorsal to ventral sections (see Discussion). Finally, degeneration was seen in the cortex of the posterior bank of the superior temporal sulcus, as shown (Fig. 3A). In addition, an occasional degenerated fibre was seen in the depth of the superior temporal sulcus. In all monkeys of the present series, this degeneration at the depth of the superior temporal sulcus did not appear in any predictable way and this, together with the paucity of the degeneration in this region when it appeared at all, makes it difficult to assign these few degenerated fibres to any particular field at the present time.

The results in the 3 monkeys described above show that, apart from the post-
rior bank of the superior temporal sulcus, which appears to receive projections from all these areas, regions of vertical meridian representation in area 18 project to two strips outside (anterior to) area 19 and that a third, separate, field receives a small projection (anterior part of prelunate gyrus in upper area 18 lesions, anterior bank of inferior occipital sulcus in lower area 18 lesions). A small projection also appears to exist from the region of representation of the vertical meridian in area 18 to the corresponding region in area 19 (Fig. 2B). It was naturally interesting to see whether regions of horizontal meridian representation in areas 18 (and 19) would project to the same or different regions. The placing of lesions in this instance is somewhat more difficult, for the exact boundary between the two areas is not very clear, although it appears to fall in the posterior bank of the lunate sulcus for upper prestriate cortex and the posterior bank of the inferior occipital sulcus for lower prestriate cortex\textsuperscript{1,16}. In 3 monkeys, lesions were placed in these areas and they were made slightly more lateral in the hope that they would fall within area 18 (see Fig. 1).

In monkeys AM 1 and AM 4 electrolytic lesions were made in the posterior bank of the lunate sulcus and the resulting degeneration in the two animals was similar so that only one need be described. In monkey AM 4 the lesion did not involve white matter and was about 2.0 mm × 1.5 mm in total extent (see Fig. 4). From this lesion, degenerated fibres streamed more medially and, several sections below the lesion, an area of degeneration appeared at the bottom of the lunate sulcus (Fig. 4). Such fibres presumably represent local connections between areas 18 and 19 and the appearance of the degeneration well below the lesion, as in monkey AM 20 (Fig. 2), suggests an interquadrantic interconnection. In the anterior bank of the lunate sulcus two separate areas of degeneration appeared, one halfway up the anterior bank of the lunate sulcus and another more laterally in the posterior part of the crown of the prelunate gyrus (Fig. 4E). Further ventrally, sparse degeneration could be found in the anterior bank of the prelunate gyrus (Fig. 4F) and, finally, relatively coarse degeneration appeared in the cortex of the posterior bank of the superior temporal sulcus (Fig. 4E, F) in addition to a few scattered fibres at the bottom of this sulcus. It can be seen, therefore, that the picture of degeneration ensuing after a lesion in the region of horizontal meridian re-representation in upper prestriate cortex was similar to the degeneration picture obtained after lesions in regions of vertical meridian re-representation, suggesting a gradual breakdown in topography.

In the light of the foregoing results, it was expected that a lesion in the region of the horizontal meridian re-representation in lower prestriate cortex would lead to degeneration in the same regions as the ones appearing after lesions of the vertical meridian re-representation of lower area 18. That is, degeneration was expected in the posterior bank of the inferior occipital sulcus just beyond the opercular lip, in the anterior bank of the inferior occipital sulcus (lateral to the boundary of lower area 19) and also in the posterior bank of the superior temporal sulcus. The results of monkey AM 15 (see Fig. 5) showed that this is indeed so. This animal had a small electrolytic lesion involving white matter only minimally and comparable in size to the lesion in monkey AM 4 (Fig. 4). The lesion was placed in the posterior bank of the inferior occipital sulcus. Because of the difficulties stated earlier, it was not possible to determine

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Fig. 5. Degeneration in the brain of monkey AM 15 following a punctate lesion placed in the posterior bank of the inferior occipital sulcus by means of an electrode. The lesion appears in section D (arrow). To the right are two photomicrographs of the lesion (× 25) showing the extent of the lesion from surface to white matter as this appears in cresyl violet stained sections. This shows the maximum involvement of white matter and in the other monkeys reported here there was no visible involvement of white matter. Otherwise this lesion is representative of the other lesions reported in this study. Comparison of the degeneration in this brain (sections C and D) with the degeneration in brain AM 8 (Fig. 3, sections B and C) will reveal the similarity between the two patterns of degeneration despite the different lesions.

with certainty whether the lesion actually fell in area 18 or crossed the boundary into area 19. The lesion did, however, correspond to the position at which the horizontal meridian is re-represented\(^1\)\(^-\)\(^6\). As may be seen by reference to Fig. 5, the degeneration obtained was similar to that following lesions in regions of vertical meridian re-representation in lower area 18 (monkey AM 8, Fig. 3). There was, however, in this instance some degeneration in the posterior bank of the lunate sulcus, that is, in the

Fig. 6. Degeneration in the brain of monkey AM 22, with a lesion in the anterior bank of the inferior occipital sulcus. The lesion appears in section C (arrow). To the right is a photomicrograph of the degeneration as this appeared at a magnification of × 250, representative of this and other brains. The lesion fell approximately in the region of re-representation of the vertical meridian in area 19. Compare sections B and C of this figure with section C of Fig. 5. Such a comparison will show that the degeneration in the two brains was similar, despite the different lesions. In addition, however, clear degeneration was seen, in monkey AM 22, in more dorsal sections at the level of re-representation of the vertical meridian in upper area 19 (section A, this figure).

region of re-representation of the horizontal meridian in upper prestriate cortex. It is possible, therefore, that the lesion in this animal had involved area 19 since lesions which had been deliberately placed in area 19 did show reciprocal connections between upper and lower parts of area 19, thus possibly further eroding topography. This the next two animals illustrate.

Monkey AM 22 had a lesion situated in the anterior bank of the inferior occipital
sulcus, corresponding to the region which previous anatomical studies\(^1\),\(^{16}\) have suggested would be the region of re-representation of the vertical meridian, forming the anterior boundary of area 19 (Figs. 1 and 6). The degeneration that resulted was largely similar to the degeneration picture described for monkeys AM 8 (Fig. 3) and AM 15 (Fig. 5). Thus, there was degeneration in the posterior bank of the inferior occipital sulcus, just beyond the opercular lip (Fig. 6). This field gave hints of being composed of two fields. The rest of the posterior bank of the inferior occipital sulcus was free of degeneration. In the anterior bank, an area of degeneration could be seen just beyond the lesion. There was the usual degeneration in the posterior bank of the superior temporal sulcus although in this animal the degeneration here was more extensive than in the previously described animals and encompassed more than just the field defined as receiving a convergent input from the striate cortex\(^{18}\) (see Fig. 6B). In addition to these fields of degeneration another, distinct, field of degeneration appeared more dorsally (Fig. 6A). This latter field fell in the region of re-representation of the vertical meridian in upper prestriate 19, thus suggesting that the two quadrantic representations, hitherto separate, may be interconnected at this level.

Another monkey, AM 21, had a lesion in upper area 19, falling at the depth of the lunate sulcus, that is to say at some point between the re-representations of the vertical and horizontal meridians in this cortical area (see Fig. 7). The lesion was placed electrolytically and did not involve white matter. It was of the same dimensions as the other electrolytic lesions reported in this study. The degeneration in this animal was very similar to the degeneration seen following lesions in regions of vertical

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**Fig. 7.** Diagrammatic representation of the degeneration in monkey AM 21 with a lesion in the depth of the lunate sulcus, that is within area 19. Notice that the degeneration is similar to the one in animals with lesions in prestriate cortex at its boundary with striate cortex (compare sections B and C of this figure with section C of Fig. 2) or in the posterior bank of the lunate sulcus (compare sections B and C of this figure with sections E and F of Fig. 4). But there is additional degeneration in this animal in the inferior occipital sulcus (section C) suggesting connections between upper and lower parts of area 19.

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meridian re-representation (AM 20, Fig. 2) and horizontal meridian re-representation (AM 4, Fig. 4) of upper area 18. Thus, there was degeneration in the anterior bank of the lunate sulcus just beyond the anterior boundary of area 19. Another field of degeneration appeared in the posterior part of the prelunate gyrus. A few degenerated fibres appeared in the anterior part of the prelunate gyrus. The degeneration in the posterior part of the superior temporal sulcus was present although somewhat more widespread than in the other monkeys of the present series. More ventrally, in lower area 19, there was another field of degeneration (Fig. 7C), thus giving further evidence of a dorsoventral connection, possibly of homologous points, between the upper and lower parts of area 19.

Finally, the degeneration in the animals reported here was confined to the lower three cortical layers and more particularly layers 5 and 6. Only very occasionally could a fibre be seen in the upper cortical layers.

**Interhemispheric degeneration**

Four monkeys brains were stained for degeneration in the contralateral hemisphere. It proved very difficult to stain the interhemispheric degeneration following punctate cortical lesions, thus making these studies time consuming and frustrating. This difficulty may have been partly due to the relatively long survival times used here. The results obtained are given here because they indicate that the interhemispheric connections are wider than we believed.

In monkey AM 8 the degeneration in the opposite hemisphere was very similar to the degeneration in the ipsilateral hemisphere (Fig. 3) thus suggesting that a point in areas 18 and 19 may establish the same connections contralaterally as it does ipsilaterally, as Mettler⁹ suggested many years ago. In monkey AM 22, once more, the degeneration to be observed in the opposite hemisphere was similar to the degeneration seen in the hemisphere ipsilateral to the lesion. But in this animal the degeneration in the contralateral hemisphere was, with the exception of the degeneration in the posterior bank of the superior temporal sulcus, extremely sparse compared to the ipsilateral degeneration. In monkeys AM 15 and AM 20 no degeneration could be found in the opposite hemisphere even though the brains were stained several times over, with various modifications. Whereas the lack of contralateral degeneration in monkey AM 15, with a lesion in the region of representation of the horizontal meridian may reflect a genuine lack of connection between this region and the opposite hemisphere, the lack of such degeneration in monkey AM 20 with a lesion at the striate–prestriate boundary is almost certainly due to the difficulty of staining interhemispheric degeneration following punctate cortical lesions. It would seem, in brief, that the contralateral connections of prestriate cortex may be very extensive, as monkeys AM 8 and AM 22 have shown.

**DISCUSSION**

Together with previous anatomical studies of the organisation of prestriate cortex in the monkey¹¹,¹⁶, the present study makes it possible to begin to understand
Fig. 8. Summary diagram of the projections from areas 18 (V2) and 19 (V3) as revealed in the present study. The projection pattern from upper prestriate V2 and V3 is shown in A and that from lower prestriate V2 and V3 is shown in B. All parts of areas V2 and V3 reported here appear to project to Visual 4 (V4) and Visual 4a (V4a). V4 and V4a of lower prestriate cortex appear as invaginations into lower V2, just beyond the lip of the operculum. Sections C and D show the probable steps by which such a transformation in the position of V4 and V4a occurs, based upon evidence presented in this and previous studies. The obliteration of the lunate sulcus and the opening up of the inferior occipital sulcus bring about this change. The dotted lines in section D show the position along which the inferior occipital sulcus will open up. With such an opening and with the obliteration of the lunate sulcus, V4 and V4a appear in the posterior bank of the inferior occipital sulcus, as may be seen in section B. Beyond the projection to V4 and V4a, areas V2 and V3 send a weak projection to the anterior part of the prelunate gyrus (from upper V2 and V3 — section A) and the anterior bank of the inferior occipital sulcus (from lower V2 and V3 — section B). The change in the position of this field from the anterior part of the prelunate gyrus to the anterior part of the inferior occipital sulcus occurs because of the changes in the sulcal and gyral patterns from dorsal to ventral (see text for further details). The regions of areas V2 and V3 studied also project to the cortex of the posterior bank of the superior temporal sulcus. Not shown are the following connections: local connections between V2 and V3 and within V2 and V3 and the connections between upper and lower parts of V3.
the anatomical organisation of this cortical area. Anterior to the primary visual cortex, area 17, which is topographically mapped\textsuperscript{2,10}, two anatomically defined areas may be found, areas 18 and 19. In these areas topographical organisation is maintained in the sense that there are separate regions of vertical and horizontal meridian re-representations\textsuperscript{1,16} although the anatomy suggests that the topographical organisation at this level may be less sharply defined. Moreover, in areas 18 and 19 a quadratic topography is maintained\textsuperscript{1,16,18} in the sense that projections from ventral area 17 (representing upper visual fields) go to lower areas 18 and 19 and projections from dorsal area 17 (representing lower visual fields) go to upper areas 18 and 19, without a significant degree of overlap between the two quadratic re-representations. In regions anterior to areas 18 and 19 this topography appears to be gradually eroded. In this study, we have examined the projections from areas 18 and 19 of upper and lower prestriate cortex. While the projections from upper areas 18 and 19 are fairly simple, the projections of the lower areas 18 and 19 appear at first sight to be more complex. The upper areas 18 and 19 project to at least 4 fields, of which one receives only a very sparse projection. The first of these fields lies just outside area 19 in the anterior bank of the lunate sulcus (see Fig. 8A) and the second lies more laterally in the lateral one-fifth of the anterior bank of the lunate sulcus and spreading onto the prelunate gyrus. In the study of the interhemispheric connections of prestriate cortex\textsuperscript{17}, this region was found to be interhemispherically connected. But already in that study there was a strong hint that this area may comprise more than one field since the degeneration following splenial sectioning was most dense along foci corresponding to the areas of degeneration described here. In the present study, there tended to exist two patches of degeneration in the anterior bank of the lunate sulcus, beyond area 19, following lesions in areas 18 and 19. In some sections, the degeneration was continuous from one patch to the next making the boundary difficult to define and only a difference in the density of degeneration hinted that these are two different fields. On the whole, however, the two patches were distinct as may be clearly seen, for example, in monkey AM 4 (Fig. 4) and in monkey AM 20 (Fig. 2). This evidence would reinforce our previous conclusion\textsuperscript{17} that the anterior bank of the lunate sulcus, beyond area 19, is made up of more than one area. It could be argued, therefore, that there are two further areas in the anterior bank of the lunate sulcus. These two areas will be referred to as Visual 4 (V4) and Visual 4a (V4a). This terminology, although somewhat cumbersome and prosaic, has the advantage of being an extension of the already described visual areas. To refer to these areas as 20 and 21 would be very confusing. Arabic, rather than Roman numerals are used here, but if these anatomically defined areas correspond to physiologically defined areas, then Roman numerals may be used instead, thereby bringing the terminology of these areas in line with the established terminology for visual cortical areas. Until such physiological studies are reported, however, the use of Arabic numerals would distinguish these areas as anatomically, and not physiologically, defined regions*. The projection from lower areas 18 and 19 appear at first sight to show a different and somewhat more complex pattern. But when studied in conjunc-
tion with the interhemispheric projection fields, some of these difficulties are resolved. Thus, the projection from all parts of lower 18 and 19 to a restricted portion of the posterior bank of the inferior occipital sulcus, just beyond the opercular lip, makes sense when one recalls that the interhemispheric degeneration in the anterior bank of the lunate sulcus, when traced ventrally, appears in the posterior bank of the inferior occipital sulcus, seemingly invaginating into lower area 18 (Fig. 8C, D and ref. 17, Fig. 4). The degeneration in this region showed hints of being composed of two patches although this was clearer in some animals than in others and never as clear as the sharp separation frequently obtained between the two patches of degeneration in the anterior bank of the lunate sulcus, beyond area 19. It is concluded therefore, that Visual 4 and Visual 4a, when traced ventrally, will fall in the posterior bank of the inferior occipital sulcus (Fig. 8D). Again, the appearance of degeneration in the anterior bank of the inferior occipital sulcus would seem at first sight to represent a departure from the pattern of degeneration observed following lesions of upper areas 18 and 19. But when it is recalled that with the change in the sulcal and gyral pattern from dorsal to ventral, the area of interhemispheric degeneration in the anterior part of the prelunate gyrus moves to the posterior part of the inferior occipital gyrus (or anterior part of the inferior occipital sulcus), the projection to this area following lesions of lower areas 18 and 19 becomes more comprehensible.

Whatever the details of these connections, it is clear that beyond areas 18 and 19 (V2 and V3) the organised topographic projection, as anatomically determined, gradually breaks down. In area 17 (V1) there are separate vertical and horizontal meridian representations. Regions of horizontal and vertical meridian representation in area 17 project to areas 18 (V2) and 19 (V3) in a topographic manner thus maintaining, in these areas, the topographic separation between vertical and horizontal meridians. In addition, upper and lower quadrant representations are topographically separate in 18 and 19. In V4 and V4a, this type of topographical organisation is no longer the case. On the contrary, both these areas receive projections from areas of representation of vertical and horizontal meridians in V2 and V3 and also from regions in between (see monkey AM 21, Fig. 7). Moreover, the appearance of degeneration several millimetres below the lesion, as in monkeys AM 4, AM 20 and AM 21, also suggests quadrantic interconnections within those areas, leading one to suspect that cells in V4 and V4a may have wide receptive fields. The receptive fields of cells in these two areas could probably also extend into the ipsilateral visual field for these areas are heavily interconnected interhemispherically. Moreover, lesions that were fully in area 19 (AM 21 and AM 22) showed that there is a projection from lower area 19 to upper area 19 and vice versa. If every point in lower or upper area 19 is connected with its homologue in upper or lower area 19, as the anatomy would suggest, and if area 19 is to project to V4 and V4a in the manner described, then this could mean a possible breakdown in interquadrantic topography. Such anatomical organisation suggests that the receptive fields of cells in V4 and V4a may well be larger than those in V1, V2 and V3, but this may be an unwarranted extrapolation from observations of the connectivity of the system at a relatively crude level. Again, the callosal connections of V4 and V4a suggest that for the first time in visual cortical areas the interhemispheric
connections are no longer restricted to areas of midline representation. This is because V4 and V4a appear to have all parts of central visual fields represented in them. It is clearly unwise to make predictions concerning the function of these areas, but the present findings do suggest that an evoked potential study of this cortex may not be unrewarding.

Not the least interesting feature of the organisation of prestriate areas is the projection field in the cortex of the posterior bank of the superior temporal sulcus\cite{1,8,16}. The striate cortex sends a convergent projection to this area\cite{18} in addition to the more topographically organised projections to areas 18 and 19 (V2 and V3)\cite{1,16}. The latter areas themselves then also send projections to the cortex of the posterior bank of the superior temporal sulcus. It is as if there are two routes from striate cortex to the cortex of the posterior bank of the superior temporal sulcus. One route leads directly to this region, resulting in a staggering of the topographical organisation or perhaps even its abolition in a single step\cite{18}. In the other system the path to the cortex of the superior temporal sulcus is more intricate, taking several steps at every one of which the topographical arrangement appears to become less sharply defined. The reasons for having these two systems will no doubt become clearer with further anatomical and electrophysiological work.

How the prestriate cortex is organised in regions beyond (central to) V4 and V4a remains to be seen. It is perhaps sufficient to point out at present that the organisation of the prestriate areas would seem to be far more complicated than previously envisaged and that the simplistic wiring diagram from area 17 to area 18, from area 18 to area 19 and from area 19 to the so-called ‘inferior temporal’ area will have to be abandoned. At any rate, we were not able in this study to find any projections to the ‘inferior temporal’ areas from areas 18 and 19 (V2 and V3).

**SUMMARY**

Cortical projections from two prestriate areas, 18 and 19, have been studied by using silver impregnation staining methods following punctate cortical lesions in these two areas.

Areas 18 and 19 have been found to project to 4 fields. These are, from posterior to anterior: Visual 4 (V4), Visual 4a (V4a), a region in the anterior part of the prelunate gyrus (from upper 18 and 19) and a region in the anterior bank of the inferior occipital sulcus (from lower 18 and 19) and the cortex of the posterior bank of the superior temporal sulcus.

Regions of horizontal and vertical meridian re-representation of areas 18 and 19 project to these same areas. In addition, lesions of upper area 19 lead to degeneration in lower area 19 and vice versa. All these results are taken to mean an erosion of punctate topographical representation of visual fields in cortical areas central to areas 18 and 19.

Animals studied for interhemispheric connections following punctate lesions in areas 18 and 19 suggest that where such connections exist they may go to the same contralateral points as the ipsilateral ones.

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No direct projections from areas 18 and 19, as here defined, to the so-called 'inferior temporal' areas have been found.

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