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THE THIRD VISUAL COMPLEX OF RHESUS MONKEY PRESTRIATE CORTEX

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SUMMARY

1. Two independent but neighbouring visual areas, V3 and V3A, sharing a common cytoarchitectural plan, but in each one of which the visual fields are separately represented, have been studied anatomically, functionally, and in combined anatomico-physiological experiments.

2. The properties of single cells in the two areas are so similar, judged by the techniques used in this study, that it is often impossible to tell whether any one penetration was sampling from cells in V3 or V3A. This is especially so if the cells have receptive fields in the lower hemi-quadrants, since the vertical meridian of the lower visual fields is represented along the V3–V3A boundary and since a transition from V3 to V3A along this border is not accompanied by a shift in receptive field positions of cells.

3. Since the visual fields, including the vertical meridian, are separately represented in these two areas, and since regions of vertical meridian representation are callosally connected, a simple and certain method of specifying the boundary between V3 and V3A is to examine the degeneration following section of the callosal splenium. A heavy patch of degeneration then marks the V3–V3A boundary. Within this patch, however, is a sub-patch containing fewer callosal fibres, or none at all. The boundary between V3 and V3A was taken to be at this subpatch.

4. Since the horizontal meridian is represented at the V2–V3 boundary, and since V1 projects to both these areas, sending coarse fibres to V3 and fine fibres to V2, it was found that the boundary between V2 and V3 could be precisely drawn by making a lesion in the horizontal meridian representation in V1 and noting where, in the prestriate cortex, fine fibres give way to coarse ones, without an intervening gap.

5. Double tracer anatomical experiments, in which tritiated proline was injected into V1 of animals whose callosal splenium had been sectioned, showed that whereas V3 receives a direct input from V1, V3A does not. V3A, instead, was found to receive an input from V3. Double tracer anatomical experiments were undertaken to study a possible input from V2 to V3A. Although such experiments did not reveal a direct input from V2 to V3A, they were not entirely conclusive.

6. The vast majority of cells in V3 and V3A were binocularly driven, without obvious monocular preferences. Some cells, however, though responding to stimulation of the individual eyes, summated their responses to binocular stimulation.

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Others responded only when both eyes were simultaneously stimulated. In any oblique penetration, cells preferring binocular stimulation only occurred either singly or in groups.

7. In an oblique penetration, the shift from a cell responding to binocular stimulation only to one responding equally well to stimulation of either eye was not necessarily accompanied by a shift in orientational preferences, shifts in the latter being quite independent of shifts in the former. This suggests the presence of two independent, but overlapping, systems within V3 and V3A, one for registering the degree of binocular interaction and another for registering orientations.

8. The great majority of cells in both areas were orientation selective, although in both areas cells responding to all orientations were found. In addition, cells were found in both areas that were orientation selective when tested with slits of light but also responded briskly to spots of light. There were isolated examples of cells with a high maintained discharge which were inhibited by light.

9. There was an orderly change in orientational preferences of successive cells in oblique penetrations. There were, however, remarkable examples of abrupt changes even in perpendicular penetrations.

10. All the cells in both areas were tested for colour preferences. The overwhelming majority of cells responded to the appropriate stimulus regardless of its colour.

INTRODUCTION

The prestriate visual cortex of the rhesus monkey is a very interesting cortical field, rich in surprises and contrasts. Extensive in size and buried mostly within deep fissures, it is strikingly uniform when surveyed in cytoarchitectonic preparations. Yet this drab cytoarchitecture is misleading, for it conceals the presence of a variety of areas which can be distinguished from each other on connexionistic and functional criteria. Often, so remarkable are the functional differences between one region of the prestriate cortex and a contiguous one, of similar cytoarchitectonic design, that one can predict with great accuracy what area of the prestriate cortex one is recording from. The two contiguous areas in the posterior bank of the superior temporal sulcus, a medial one concerned with the analysis of motion in the visual fields (Zeki, 1974) and a lateral one containing heavy concentrations of colour coded cells (Zeki, 1977*b*), provide a striking example.

The differences between two contiguous areas sharing a common cytoarchitectural plan can, however, be more subtle. In this paper, I describe two neighbouring areas, V3 and V3A, in each one of which the visual fields are separately represented. They lie within the depth and anterior bank of the lunate sulcus laterally and the posterior and anterior banks of the parieto-occipital sulcus medially, and share a common cytoarchitectural plan. Although these two independent areas differ in their topographical organization (Van Essen & Zeki, 1978) and anatomical inputs, so similar are the properties of cells in them that the transition from one area to the other frequently goes unnoticed, unless one uses special anatomical markers to demarcate the borders. Because of these similarities, I refer to the entire region as the third visual complex, a term which includes not only the two independent areas V3 and V3A, but possibly other ones as well.

METHODS

The surgical, electrophysiological, histological and autoradiographic methods are described elsewhere (Zeki, 1969, 1970, 1974, 1976, 1978*a*). The method of reconstructing the prestriate cortex with the sulci unfolded to show the distribution of degeneration and label following double tracer anatomical experiments is described in a companion paper (Zeki, 1978*a*).

Terminology. The terms V2 and V3, as used in this paper, are synonymous with areas 18 and 19, as determined from anatomical lesion experiments (Zeki, 1969), but are different to the cytoarchitectonic areas 18 and 19 of Brodmann (1905). Since 1971 (Zeki, 1971), I have used the terms V2 and V3, and the anatomical 18 and 19, interchangeably. I now drop all references to 18 and 19 and use the terms V2 and V3 exclusively.

RESULTS

Text-fig. 1 illustrates two penetrations made in the prestriate cortex of an animal in which the corpus callosum had been sectioned 6 days before the recording experiment. The degeneration in the cortex following callosal section is represented as dots. Penetration A was made in the posterior bank of the parieto-occipital sulcus at the edge of the first band of callosal fibre degeneration after the V1–V2 boundary. It was consequently made, without doubt, in area V3 (Zeki, 1977*a*; Van Essen & Zeki, 1978). Penetration B was made in the anterior bank of the lunate sulcus, lateral to the most medial two bands of degeneration within it. Consequently, penetration B was, without doubt, in area V3A (Van Essen & Zeki, 1978). Disregarding for the moment differences in receptive field positions of the cells in the two penetrations, which would in any case be expected to vary according to the position within each area from which cells are sampled, the properties of the cells in the two penetrations are remarkably similar. In both penetrations, all the cells were orientation selective when tested with slits of light and responded equally well to movement of the slit at right angles to its axis in either direction. They were all binocularly driven, and there was no hint that any of the cells illustrated in either penetration was particularly concerned with colour.

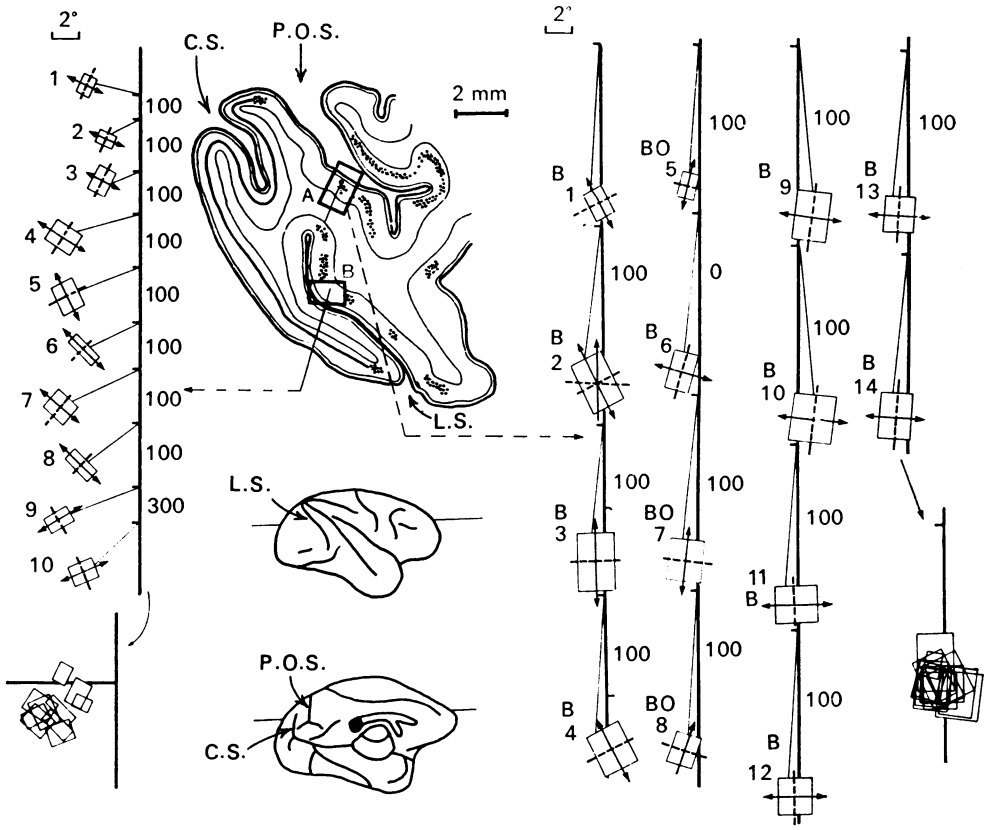
And yet, despite this striking functional similarity, the two components, V3 and V3A, differ in their topographical organization and anatomical inputs. In Part I of this paper the anatomical inputs to the two areas of this third visual complex are described, followed by a more detailed description, in Part II, of the responses of single cells in its various parts.

PART I

The anatomical inputs to V3 and V3A

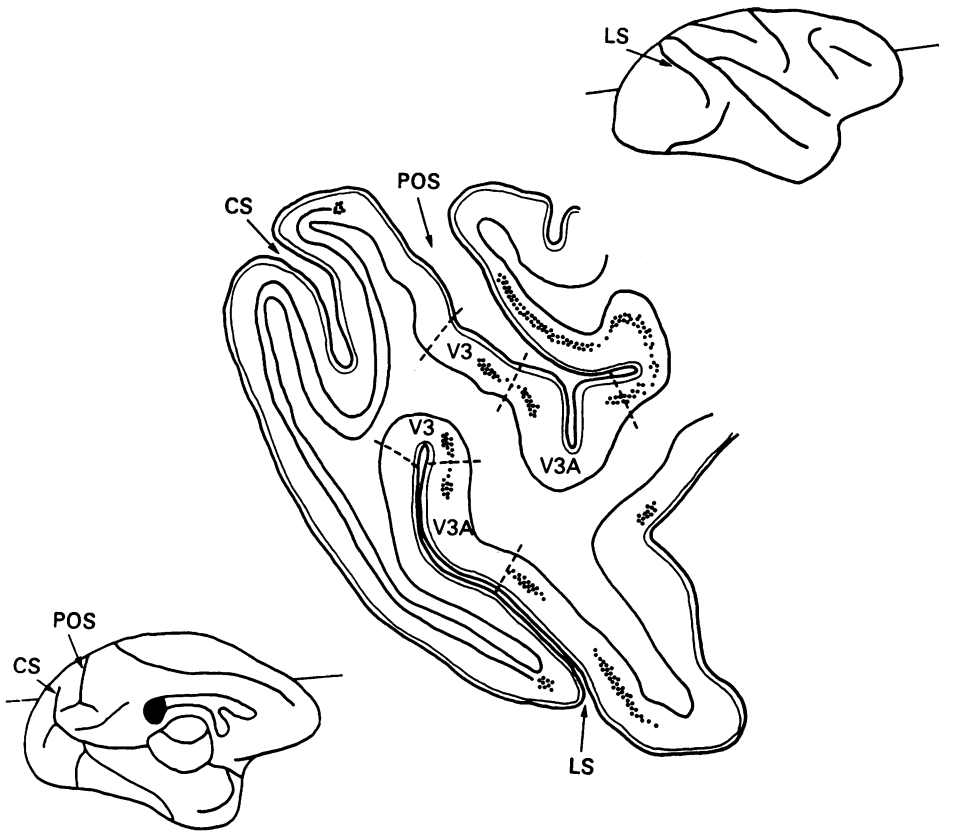
(A) *Inputs from the striate cortex (V1)*

As described in detail elsewhere (Van Essen & Zeki, 1978), areas V3 and V3A lie in the lunate sulcus laterally, V3 being medial and posterior to V3A. They also extend over the annectant gyrus into the parieto-occipital sulcus, where V3 lies posterior to V3A. Text-fig. 2 shows the position of the two areas as seen in a horizontal section of the brain. There is no cytoarchitectonic boundary between V3 and V3A but a border between the two areas can be adequately demonstrated anatomically by sectioning the corpus callosum and noting the distribution of the resulting fibre degeneration.



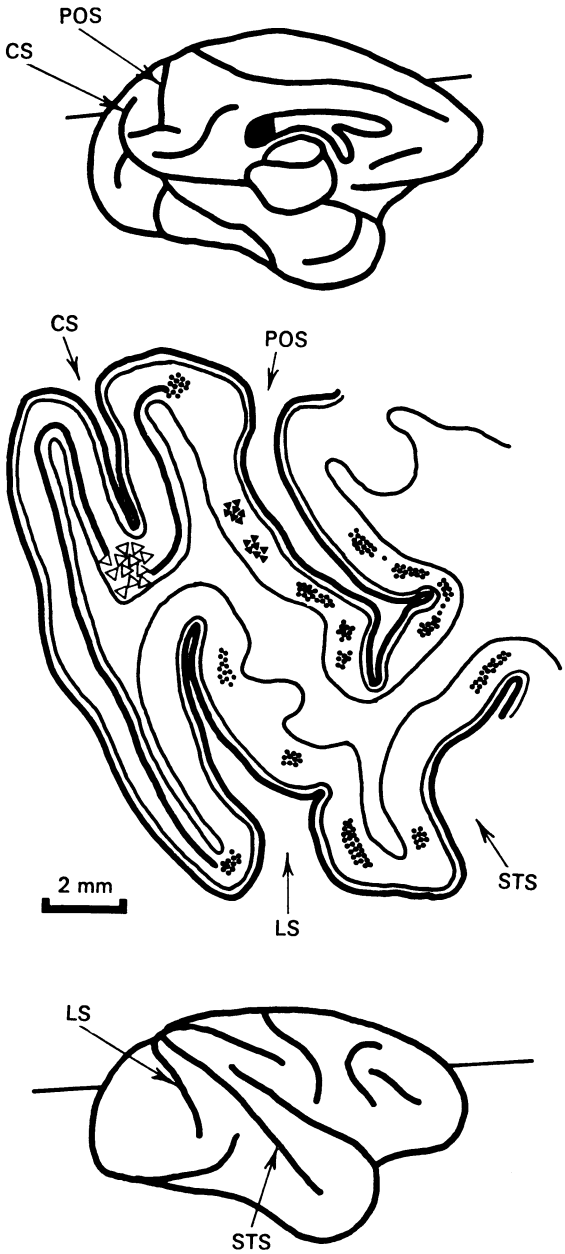
Text-fig. 1. Reconstruction of two penetrations through the third visual complex of the rhesus monkey. The two tracks are reconstructed on a tracing of a horizontal section taken at the level indicated on the surface drawing of the brain. The dots in the cortex represent the fibre degeneration following transection of the splenium of the corpus callosum. The continuous line in the cortex represents area 17 (V1). Penetration A was through the parts of area V3 lying within the parieto-occipital sulcus and its termination was marked with a lesion. Receptive fields of cells encountered in penetration A are shown to the right of the figure and the cells are numbered from the white matter end (cell 1) to the pial surface (cell 14). Penetration B was through the part of V3A lying in the anterior bank of the lunate sulcus. Here the first cell was closest to the pial surface. Receptive fields encountered in this penetration are shown to the left of the figure. In each track, the receptive fields of successive cells are drawn with respect to the centre of gaze. The latter is represented by the short horizontal lines intersecting the long common vertical line. In each track, the number to the left is the cell number, that to the right is the distance, in μm, between one cell and another. Interrupted lines drawn through the receptive fields signify that the cells were orientation selective. Arrows show the direction of motion of the stimulus to which the cell was responsive. The cumulative receptive field plots for each track are given below. LS, lunate sulcus; POS, parietal-occipital sulcus; CS, calcarine sulcus. All the cells illustrated in the track to the left were binocularly driven, without monocular preferences. In the track reconstructed to the right B = binocularly driven cell; BO = cell responding to binocular stimulation only.

Consequently, perhaps the simplest and most convincing way of demonstrating that V3 receives a direct input from V1, whereas V3A does not, is to inject a radioactive tracer into area V1 of an animal whose corpus callosum has been sectioned. One can then note the distribution of the label, in relation to the bands of callosal degeneration that define the boundaries of V3 and V3A.



Text-fig. 2. Tracing of a horizontal section, taken at the level indicated on the surface drawings of the medial and lateral sides of the brain. The dots in the cortex represent the degeneration following transection of the splenium of the corpus callosum. Continuous line indicates area 17 (V1). The positions of V3 and V3A, in both the lunate and parieto-occipital sulci, are shown and their boundaries are drawn in interrupted lines. The two portions of each area are continuous across the annectant gyrus. Conventions as in Text-fig. 1.

In the experiment illustrated in Text-fig. 3, [^{3}H]proline was injected into the region of horizontal meridian representation in V1 at about 30° from the centre of gaze (Zeki, 1977*a*) in an animal in which the corpus callosum had been sectioned 6 days before the injection. The degeneration produced by sectioning the corpus callosum is represented as dots. In both the lunate and parieto-occipital sulci, the second patch of callosal degeneration after the V1–V2 boundary represents the anterior border of V3 (Zeki & Sandeman, 1976). The distribution of the autoradiographic label is shown as filled triangles. Since the injection was into a region of the striate cortex lying in the calcarine cortex and representing the visual fields at 30°



Text-fig. 3. Reconstruction of a double tracer anatomical experiment in which [^{3}H]proline was injected into the horizontal meridian representation of V1, at about 30° from the centre of gaze, in an animal in which the splenium of the corpus callosum had been sectioned. The degeneration is shown as dots, the site of injection as open triangles, and the site of label distribution as filled triangles. Conventions as in previous Figures. Note that there is no label anywhere within the territory of area V3A following such an injection, although label appears both in V2 and in V3. Such an experiment thus shows that whereas V3 receives a direct input from V1, V3A does not.

from the centre of gaze, in the horizontal meridian, the transported label appeared in the posterior bank of the parieto-occipital sulcus where the more peripheral part of V3 is situated (Zeki, 1977*a*). Here, two small patches of label could be seen, lying between two patches of callosal fibre degeneration. Of these, the more posterior patch of label belongs to V2 and the more anterior one to V3 (Zeki, 1977*a*). There is no trace of label in that region of the parieto-occipital sulcus lying anterior to the boundary of V3 (anterior to the second patch of callosal degeneration after the V1–V2 boundary) as defined by the distribution of the callosal degeneration, or anywhere within V3A, as defined by the same criterion. Such an experiment, therefore, shows at a glance that whereas V3 receives a direct input from V1, V3A does not. Similar experiments also show that in the lunate sulcus, as well, V3 receives a direct input from V1 but V3A does not.

The double tracer anatomical experiment described above is really a more convincing way of demonstrating what is already known from single tracer lesion studies (Zeki, 1969; Cragg, 1969). In such studies, when a lesion is made in V1, degeneration appears both in V2 and in V3, but not in regions which are geographically recognizable as being V3A. However, such experiments reveal that the degeneration in V3 following a lesion in V1 is much coarser than that in V2, a fact not hinted at by autoradiographic studies (see Pl. 1). This circumstance can be put to advantage in trying to define the boundary of V2 with V3.

(B) *The boundary of V2 with V3.*

It has been argued elsewhere on the basis of anatomical (Cragg, 1969; Zeki, 1969) and combined anatomical and electrophysiological evidence (Zeki & Sandeman, 1976) that the horizontal meridian is represented at the boundary of V2 and V3. The exact location of this boundary has, however, been difficult to determine from these previous studies, although using the double tracer technique described above, it has been possible to narrow down the zone of uncertainty to 500 μm (see Fig. 2 and Zeki, 1977*a*). One way of eliminating even this uncertainty is to make a lesion in the horizontal meridian representation of V1. The rationale behind this experiment is the following: V1 projects to both V2 and V3 (Cragg, 1969; Zeki, 1969) sending fine fibres to V2 and coarse ones to V3. Since the horizontal meridian is represented at the V2–V3 boundary it should be possible to draw the boundary between V2 and V3 with great accuracy by making a lesion in the horizontal meridian representation of V1 and noting where, in the prestriate cortex, fine degeneration gives way to coarse degeneration.

In the experiment illustrated in Text-fig. 4, two lesions were made in the horizontal meridian in V1 of the same hemisphere, with the same sucker. Starting on the lateral surface, a small lesion was made in the region of representation of the horizontal meridian in V1, at 3° from the fovea, as judged from the map of Daniel &Whitteridge (1961). Then, another lesion was made in the posterior bank of the calcarine sulcus. Recording experiments show that the horizontal meridian at about 30° from the centre of gaze is represented in the region in which the second lesion was made (Zeki, 1977*a*). The two lesions in this animal were therefore separated by about 27° of visual field representation in the horizontal meridian.

It is known from previous work that the lateral surface of V1, in which the central

5–8° of the visual fields are represented, projects to the parts of V2 and V3 which lie in the lunate sulcus (Cragg, 1969; Zeki, 1969). On the other hand, the medial part of V1, buried in the calcarine sulcus, projects to the parts of V2 and V3 lying in the parieto-occipital sulcus (Zeki, 1977*a*). Consequently, from such a double lesion, single-tracer, experiment, degeneration was expected in both the parieto-occipital and lunate sulci. In Text-fig. 4, which is a reconstruction of this experiment, the small dots represent the fine degeneration, and the large dots the coarse one. Both the lunate and parieto-occipital sulci have been reconstructed according to methods described in a companion paper (Van Essen & Zeki, 1978).

Throughout most of the two sulci reconstructed in Text-fig. 4, both coarse and fine fibres could be seen. In some sections (e.g. the yellow one) the coarse and the fine fibres were well separated from each other so that, in these sections, this approach was not of much help in determining the boundary between V2 and V3. Yet in other sections, through both sulci, the fine degeneration gave way to coarse degeneration without an intervening gap and this transition allowed one to determine the boundary between V2 and V3 with great accuracy. In the reconstruction of Text-fig. 4 the boundary between V2 and V3 can be drawn at every level at which the fine degeneration meets the coarse without an intervening gap. It should be noted that at the most ventral levels of the lunate sulcus at which there was degeneration (e.g. green line) the grain of the degeneration could no longer be distinguished into coarse and fine.

Such an experiment not only allowed one to draw the boundary between V2 and V3 with great accuracy, at least at some levels, but also showed an additional feature, namely the relative importance of the projections to V2 and V3 from V1. In the lunate sulcus, there was much denser degeneration in V2 than in V3. In the parieto-occipital sulcus, the reverse was the case, V3 now receiving a more powerful projection than V2 (see Pl. 2, Zeki, 1977*a*). Although this has been hinted at by earlier anatomical studies (Zeki, 1977*a*), this study enabled one to compare directly the density of degeneration in V2 and V3 in the lunate and parieto-occipital sulci of

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Text-fig. 4. Reconstruction of a single tracer, double lesion anatomical experiment. In this animal, a lesion (shown in solid black in *B* and on the surface drawing of the brain) was made on the lateral surface of the brain in a region of V1 at which the horizontal meridian at about 3° from the centre of gaze is represented. The sucker was then pushed into the calcarine sulcus, and another lesion was made, this time in that part of V1 at which the horizontal meridian at about 30° from the centre of gaze is represented. Hence the two lesions were separated by about 27° of visual field representation. The degeneration ensuing from the more central lesion appears in the lunate sulcus, that from the more peripheral lesion appears in the pareito-occipital sulcus. The fine degeneration (in V2) is represented as small dots; the coarse degeneration (in V3) is represented as large dots. In the centre, both the lunate and parieto-occipital sulci have been reconstructed. In this reconstruction, the red and the green lines represent the most ventral sections, whereas the blue and yellow lines represent the most dorsal ones. The contour lines of both the lunate and parieto-occipital sulci in successive sections have been straightened out to a greater or lesser degree and placed next to adjacent contour lines so that the continuity in degeneration can be directly traced from one contour line to another on this flat reconstruction. The boundary between V2 and V3 can be drawn with accuracy at every level at which fine degeneration meets coarse degeneration without an intervening gap. For further details see text and Pl. 1. Scale = 1 mm.

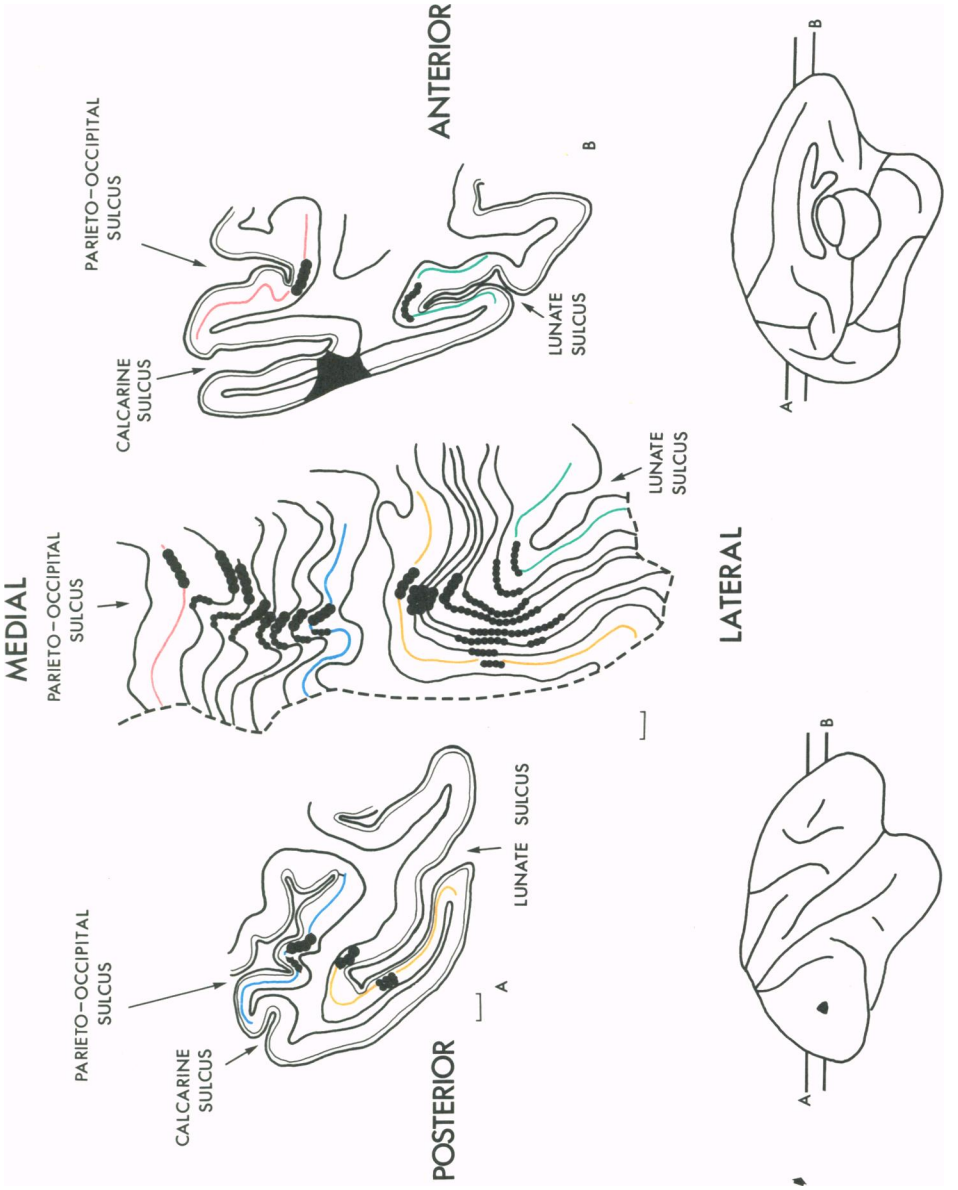


Figure 4. For legend see opposite page.

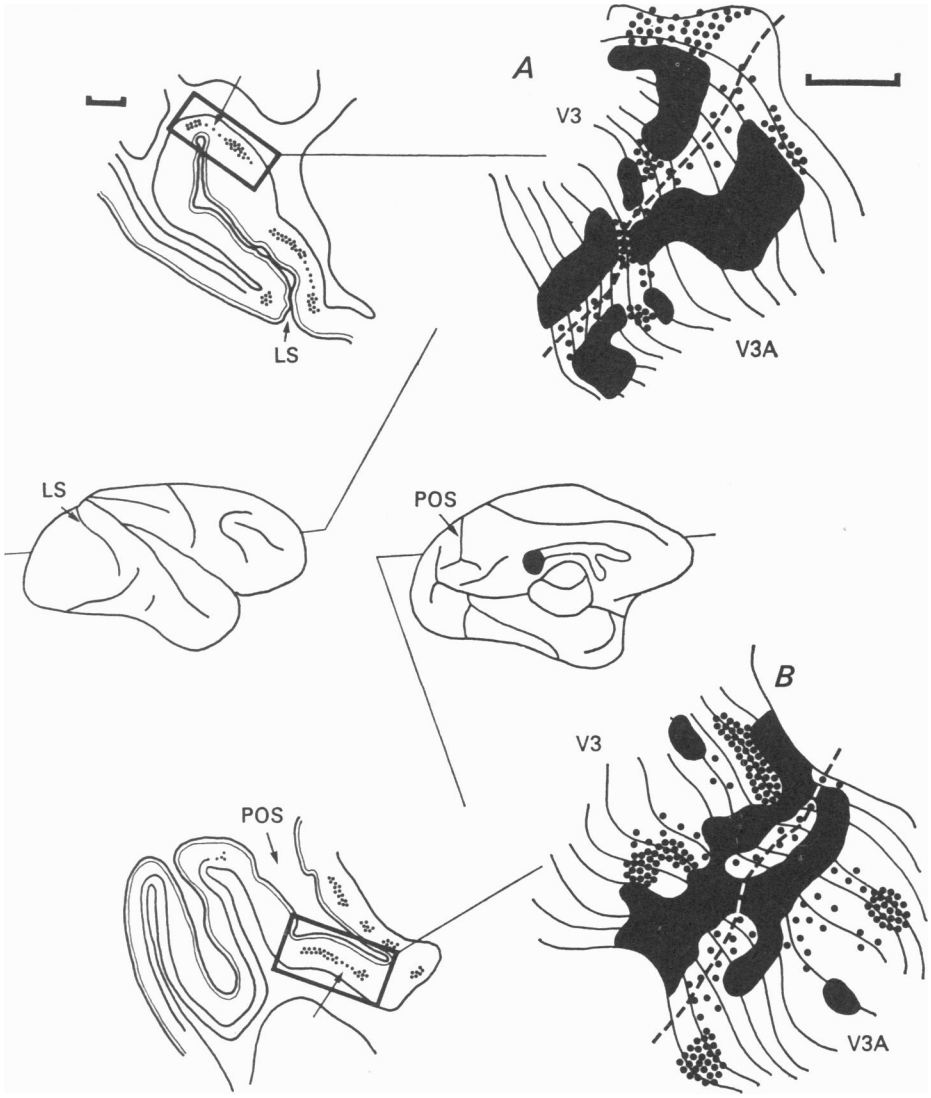
the same hemisphere. The conclusion seems inescapable that peripheral V1 sends a more powerful input to V3 than does central V1, but the reasons for this difference are not clear.

(C) *The boundary of V3 with V3A*

Since the boundary of V2 with V3A can be drawn with such accuracy, at least at some levels, it was interesting to see whether one cannot also draw a sharp boundary between V3 and V3A. In both the lunate and parieto-occipital sulci, the boundary between these two areas has been taken to lie at the level of the second patch of callosal degeneration after the V1–V2 boundary (Van Essen & Zeki, 1978). This patch of degeneration is rarely less than 1 mm in its medio-lateral extent and, at some levels, is in excess of 2 mm. When one examines it, either in the lunate or in the parieto-occipital sulcus in especially well-stained sections, a gap of lower degeneration density appears within it in some, though by no means all, sections. Text-fig. 5 illustrates this. In this Figure, this patch of degeneration is reconstructed in detail for a small region of the lunate (in *A*) and parieto-occipital (in *B*) sulci. Areas of heavy degeneration are shown in solid black and the density of the black dots represents moderate or sparse degeneration. It is quite evident that not only are there regions within this callosal patch in which a central zone of sparse degeneration is surrounded by heavier zones of degeneration but, more strikingly, in some sections the zone between the two heavy patches of degeneration is free of all degeneration. It is not clear just what part of the visual field is represented at this zone (that is, exactly how the change from V3 to V3A occurs). But I suggest, tentatively, that the boundary between V3 and V3A lies near or at this zone between the two heavy patches of callosal degeneration. It must be emphasized that the central zone, where the boundary between V3 and V3A lies, is not evident in all sections. Consequently, the boundary between the two areas cannot be drawn with great accuracy, using this technique, throughout the prestriate cortex. The boundary of V3A with V4 is dealt with in another paper (Van Essen & Zeki, 1978).

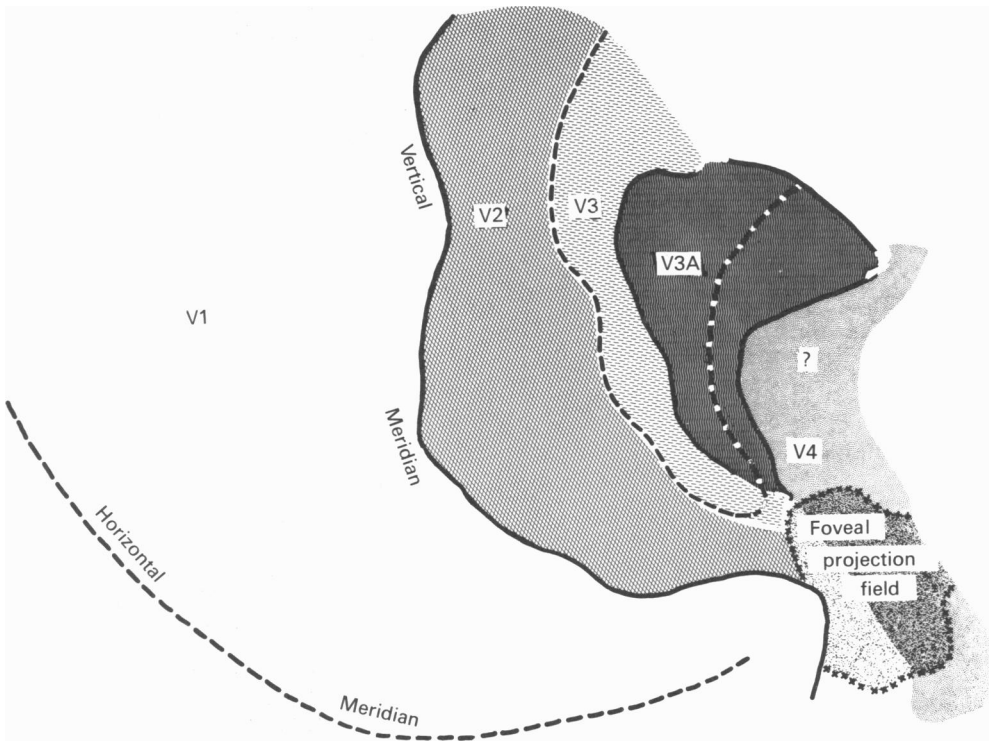
(D) *The ventral extent of V3 and V3A in the lunate sulcus*

Although the experiments described above and elsewhere (Zeki, 1977*a*; Van Essen & Zeki, 1978) allow one to draw the anterior and posterior boundaries of V3 and V3A with considerable accuracy, they are of little help in defining the ventral boundaries of these areas. The ventral boundary of V3 in the lunate sulcus is especially problematic. The callosal band that defines the anterior boundary of V3 in the lunate sulcus terminates abruptly (Van Essen & Zeki, 1978) and its place is taken by the callosal band lying in the anterior bank of the lunate sulcus and defining the anterior boundary of V3A. The separation between the termination of one and the beginning of the other is sometimes of the order of 1 mm or even less (see Text-fig. 2, Van Essen & Zeki, 1978). One interpretation of these anatomical observations is that the upper part of V3 (representing lower visual fields) itself also ends abruptly, together with its callosal band and is totally separate from the lower part of V3 (in which upper visual fields are represented). Indeed, this was my original interpretation (see Fig. 8, Zeki, 1969). If such an interpretation were correct, then one would have to argue that V3 does not receive a direct input from foveal striate cortex, even though a lesion in



Text-fig. 5. Detailed reconstruction, in successive sections, of the first patch of callosal fibre degeneration after the V1–V2 boundary, in both the lunate (above) and parieto-occipital sulci (below). The region of each sulcus reconstructed is boxed in the tracings of the horizontal sections. Within this region, the callosal fibre degeneration is often composed of a central, sparser zone of degeneration (indicated by arrows in the tracings of the horizontal sections to the left), flanked by regions of denser degeneration. In the reconstructions to the right, regions of dense degeneration are shown in solid black and the frequency of the dots indicates whether the remaining degeneration was moderate or sparse. The interrupted lines in both reconstructions runs through the central zone of sparse degeneration within the callosal patch and is interpreted as a possible boundary between V3 and V3A. In the centre are surface drawings of the medial and lateral surfaces of the brain to show the level at which the central section in each reconstruction lies.

foveal striate cortex produces both fine and coarse degeneration in the lunate sulcus (Zeki, 1978*a*), characteristic of the projections from V1 to V2 and V3 respectively. This is because the projections from foveal striate cortex go to regions of the lunate sulcus which lie ventral and lateral to the level at which the callosal band defining the anterior border of V3 terminates. There are, however, other interpretations of these anatomical observations. One that comes readily to mind is summarized diagrammatically in Text-fig. 6. In this diagram, regions of the prestriate cortex in which the

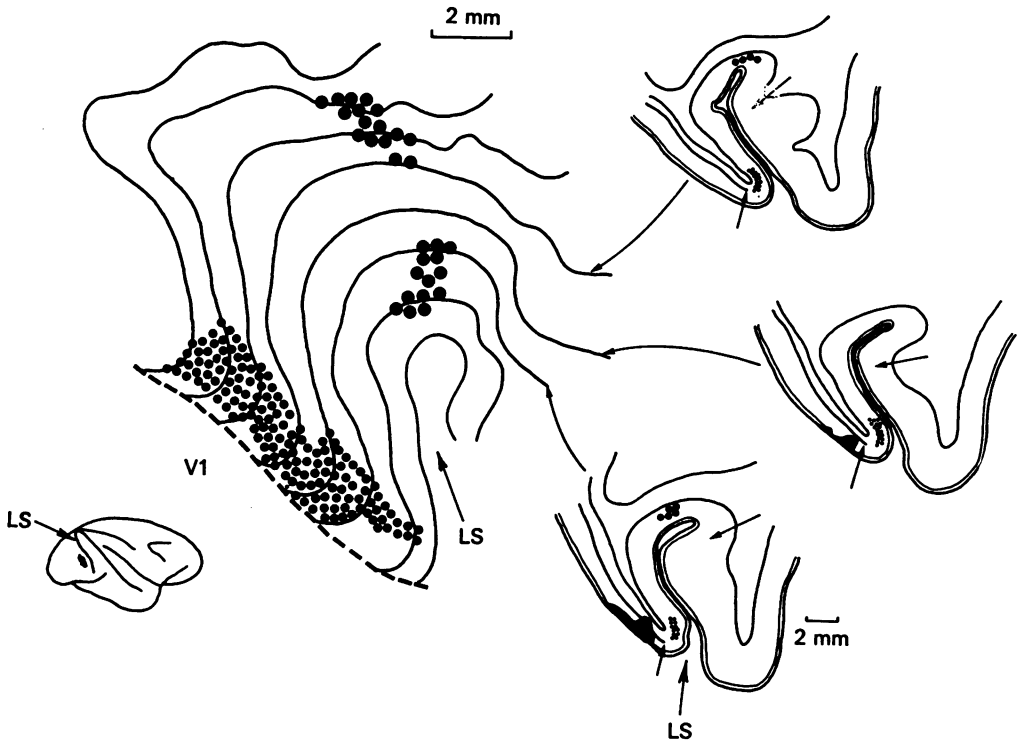


Text-fig. 6. Diagram to illustrate the manner in which the horizontal meridian representation at the V2–V3 boundary and that within V3A come close together above the projection field of foveal striate cortex. Vertical meridian representation is shown as solid lines, horizontal meridian representation as interrupted lines. The manner in which the two horizontal meridian representations may come close together (even if they are not continuous with one another) may explain the discontinuity in the vertical meridian representation of V3. For further details, see text. The reconstruction of this figure is based on the reconstructions shown in the two companion papers (Van Essen & Zeki, 1978; Zeki, 1978*a*).

vertical meridian is represented and which are callosally connected are shown in solid lines and regions of horizontal meridian representation are shown as interrupted lines. In such a schema, the termination of the callosal band defining the anterior border of V3 is envisaged as being due to the manner of forking of the horizontal meridian, as traced outwards from the region of foveal representation in the prestriate cortex (Zeki, 1978*a*). In a sense, this is an interpretation based on direct observation because it is known that the horizontal meridian is represented between the two patches of callosal degeneration (shown as continuous lines) that define the anterior

and posterior borders of V3A (Van Essen & Zeki, 1978) and that the horizontal meridian forms the posterior boundary of V3 (Zeki & Sandeman, 1976). If such an interpretation were correct it would mean that the vertical meridian representation of V3 is discontinuous at this level, which would be something of a curiosity. But this discontinuity would help to explain the following observations:

(*a*) Foveal striate cortex lesions produce fine and coarse degeneration in the lunate sulcus (Zeki, 1978*a*). The coarse degeneration produced by such lesions occurs in



Text-fig. 7. Reconstruction of an anatomical experiment in which a small lesion was made in the region of vertical meridian representation in V1 (shown in solid black on the surface drawing of the brain to the lower left). The brain was sectioned parasagitally. Contour lines for sections spaced by 500 μm are drawn in the centre and the degeneration is shown as small dots (in V2) and large ones (in V3). The contour lines were taken between the regions marked by arrows in the representative sections shown to the right. To obtain a map on a flat sheet, some unbending of the contour lines, to avoid cross overs, was necessary. Note (*a*) the presence of a continuous line of fine degeneration at the V1–V2 boundary and (*b*) the presence of two discontinuous patches of coarse degeneration within the depth of the lunate sulcus.

regions of the lunate sulcus ventral and lateral to the point at which the callosal fibre patch defining most of the anterior border of V3 ends. The characteristic of the degeneration in V3, following a lesion in V1, is that it is coarse. Hence one can assume that V3 receives a direct input from foveal striate cortex.

(*b*) Such an interpretation would help explain an otherwise bizarre pattern of projections from V1 to V3 following lesions in regions of central vertical meridian

representation in V1 that I have occasionally observed. Such a projection is shown in Text-fig. 7. Note that, whereas there is a continuous band of fine degeneration in the posterior bank of the lunate sulcus (V2), the coarse degeneration in its depth (in V3) is discontinuous and occurs in two patches. Such a discontinuity in the projections to V3 has not been observed with horizontal meridian lesions. If the vertical meridian representation in V3 is indeed discontinuous, then the above anatomical observation becomes explicable. Such a schema is offered only tentatively, as a possibility, to explain the anatomical observations. For the moment suffice it to note that, if the above schema is correct, then it would be possible to postulate that V3 is a continuous area to the level at which the lunate sulcus disappears and, possibly, ventrally, into the inferior occipital sulcus as well (see Discussion).

It is still unclear how area V3A terminates in the lunate sulcus. It is very likely that it does terminate at this sulcus, however, since, unlike V3, it contains a representation of both superior and inferior hemiquadrants in the dorsal part of the prestriate cortex (Van Essen & Zeki, 1978).

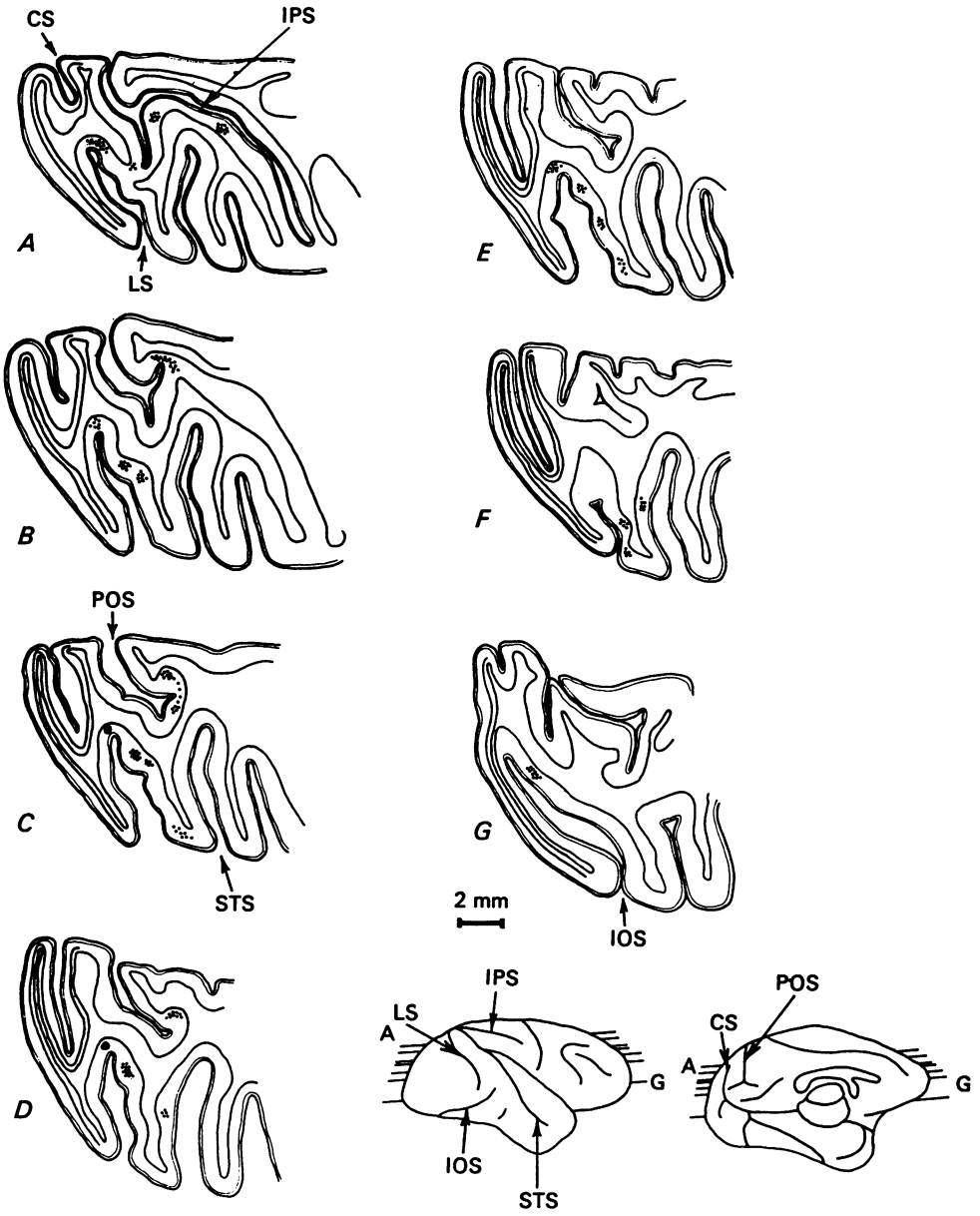
(E) *The anatomical inputs to V3A*

The above experiments show that V3A does not receive a direct input from the central 30° of the striate cortex. If, on the other hand, a lesion is made in V3, a direct projection to V3A can be observed. Such an experiment is reconstructed in Text-fig. 8. In studying the pattern of projections from V3 to V3A, it is as well to recall that much of V3A lies within the parieto-occipital sulcus and that, within it, central visual fields may be represented not only in that part of it lying within the lunate sulcus, but also in that part that lies within the parieto-occipital sulcus (Van Essen & Zeki, 1978).

In the experiment illustrated in Text-fig. 8, a small electrolytic lesion was made in V3 in the depth of the lunate sulcus. The lesion was made in that part of V3 that represents central visual fields at about 5° below the centre of gaze (Zeki & Sandeman, 1976). In the most dorsal section to show degenerating fibres, these appeared in the depth of the lunate sulcus (V3), in the anterior bank of the parieto-occipital sulcus (V3A) and in the posterior bank of the intraparietal sulcus. The latter field of degeneration probably belongs to a new, and distinct, visual area which will not be further discussed in this paper. More ventrally (sections *B*, *C*, *D*) the degeneration in the depth and anterior bank of the intra-parietal sulcus was maintained but degeneration also appeared in V3A in the anterior bank of the lunate sulcus. In addition, in section *C*, a small patch of degeneration appeared on the surface of the prelunate gyrus only to disappear again at the level of section *D*. At still more ventral levels (section *E*), not only was there degeneration in V3A but degeneration also appeared more laterally in the anterior bank of the lunate sulcus, in the territory of the fourth visual complex, and this degeneration was maintained at the level of section *F*.

In addition to these projections, there was also a discontinuous projection to the posterior bank of the superior temporal sulcus (sections *D* and *F*) and a projection to the anterior bank of the inferior occipital sulcus in what is almost certainly lower V3 (Cragg, 1969; Zeki, 1969).

Because there was no additional anatomical landmark in this experiment, such as



Text-fig. 8. The distribution of the cortical degeneration following a small lesion in area V3 (shown as solid black in sections *C* and *D*). Conventions as in previous Figures. CS, calcarine sulcus; LS, lunate sulcus; POS, parieto-occipital sulcus; STS, superior temporal sulcus; IOS, inferior occipital sulcus. Below are surface drawings of the medial and lateral surfaces of the brain to show the levels from which the horizontal sections were taken.

that provided by callosal fibre degeneration, it is not quite certain whether all the degeneration in the parieto-occipital sulcus was in the territory of V3A; the more anterior part of the degeneration in this sulcus may have been in a cortical field situated outside V3A (see below).

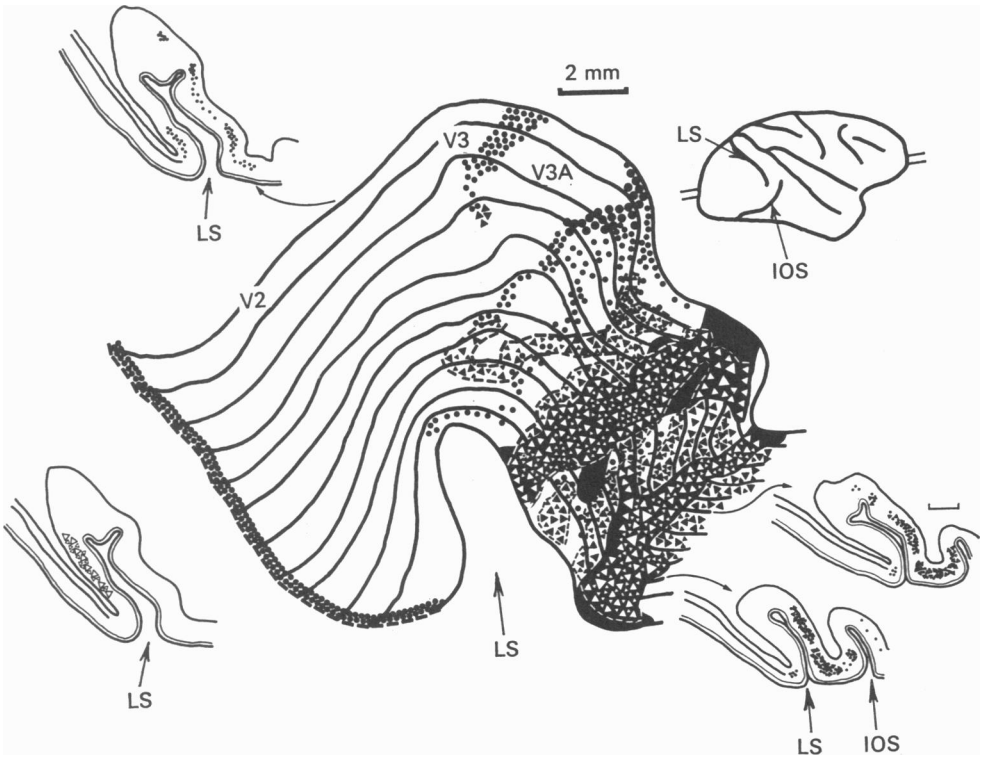
(F) *Does V3A receive an input from V2?*

In the lunate sulcus, V3A is sandwiched between V3 and the fourth visual complex. Both V3 and V4 receive a direct input from V2 (Zeki, 1971) but, because there is no cytoarchitectural boundary between V3A and its neighbouring areas, it is not possible to determine from such single tracer anatomical experiments whether V3A might not, also, receive a direct input from V2. It seemed worth while to examine this problem using a double tracer technique and injecting labelled proline into V2 in an animal in which the splenium of the corpus callosum had been previously sectioned. The degeneration resulting from such a procedure permits one to draw the boundaries between the prestriate areas more reliably. There was a hope, therefore, that such a procedure might allow one to determine with greater certainty whether V3A receives a direct input from V2.

Two such experiments were done. In one the label was injected into the region of vertical meridian representation in V2. This experiment is reconstructed in the previous paper (Zeki, 1978*a*, Text-fig. 8*A*). On the face of it, the results of that experiment were negative from the viewpoint of a projection from V2 to V3A. The label appeared within the band of callosal degeneration defining the anterior boundary of V3, and there was also a generous distribution of label in the region of the callosal bands defining V4, thus confirming earlier anatomical results (Zeki, 1971). But no label appeared anywhere within the territory of V3A in the lunate sulcus, nor was there any label within that part of V3A lying within the parieto-occipital sulcus, not even within regions in which central visual fields may be represented (Van Essen & Zeki, 1978). Nevertheless, the results were not as conclusive as one might wish, since label appeared in the callosal bands that define the anterior and posterior boundaries of V3A. It would be possible to argue, for example, that some of this label may have been in V3A.

In order to overcome this difficulty, [^{3}H]proline was injected into V2 in another brain in which the splenium of the corpus callosum had been sectioned. But, in this animal, the label injection was more extensive and included the vertical, as well as the horizontal, meridian representation in V2. It was hoped that, if there was a direct input from V2 to V3A, then label would appear in the part of V3A where the horizontal meridian is represented and which is free of callosal degeneration. Text-fig. 9 is a reconstruction of this experiment. Only the lower part of the lunate sulcus has been reconstructed in this experiment, since there was no label anywhere within the parieto-occipital sulcus. In the lunate sulcus, the label (which is indicated by filled triangles) was distributed heavily in the region of the callosal bands defining V4 and there was no label anywhere within the callosal free zone of V3A, at which the horizontal meridian is represented. However, label did appear in a callosal free zone at the depth of the lunate sulcus, but this patch was continuous, when traced dorsally, with the label in the V4 complex. Whether this patch of label lies within V3A would depend largely on how far ventrally V3A extends in the lunate sulcus, something that is still uncertain. Consequently, neither of these two experiments was conclusive in giving an answer to the question of whether the part of V3A lying in the lunate sulcus receives a direct input from V2, and the possibility remains open. To settle the question, lesions would have to be made in parts of the horizontal meridian repre-

sentation of V2 at greater eccentricities than the ones done for this study. What can be stated with greater certainty is that those parts of V3A lying within the parieto-occipital sulcus and in which central visual fields may be represented (Van Essen & Zeki, 1978) do not receive a direct input from corresponding parts of V2.



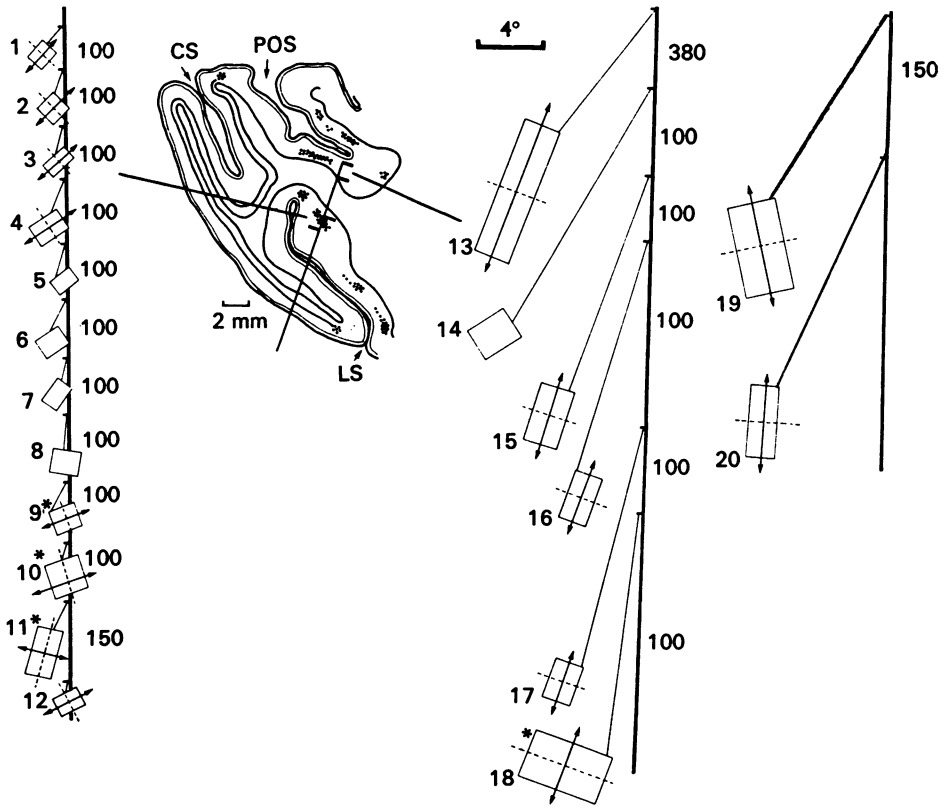
Text-fig. 9. Reconstruction of a double tracer anatomical experiment in which [^{3}H]proline was injected into area V2 in an animal in which the corpus callosum had been sectioned. The site of the label injection is shown as outline triangles on the tracing of a horizontal section to the lower left. In the centre, the lunate sulcus has been reconstructed between the two levels shown on the surface drawing of the brain to the upper right. In this reconstruction, regions of heavy callosal degeneration are shown in solid black and moderate and sparse degeneration is shown by the size and frequency of the black dots. The white and black triangles indicate label distribution following [^{3}H]proline injection into V2. In the anterior bank of the lunate sulcus, the boundaries of regions showing label distribution are enclosed by interrupted lines. Note that, in the anterior bank of the lunate sulcus, label appears both in regions showing callosal fibre degeneration and in regions free of such degeneration. There are two broad bands of label, joined together dorsally. There is also a protrusion from the more medial of the two bands. The ventral part of this protrusion may invade the territory of V3A, depending upon the ventral extent of V3A.

PART II

Electrophysiological recordings

In recordings through the third visual complex, it is often difficult to know, in the absence of detailed anatomical landmarks, such as the ones provided by the callosal fibre degeneration, just which area, V3 or V3A, one has been recording from. In

many experiments undertaken to study the properties of the cells in these two areas, recordings were made from V3 and V3A in the same animal. This was fortunate because it allowed a direct comparison of the properties of the cells in the two areas. Since these properties were so similar, I describe them under one heading, identifying throughout the position of the cells, that is whether they belonged to V3 or V3A.



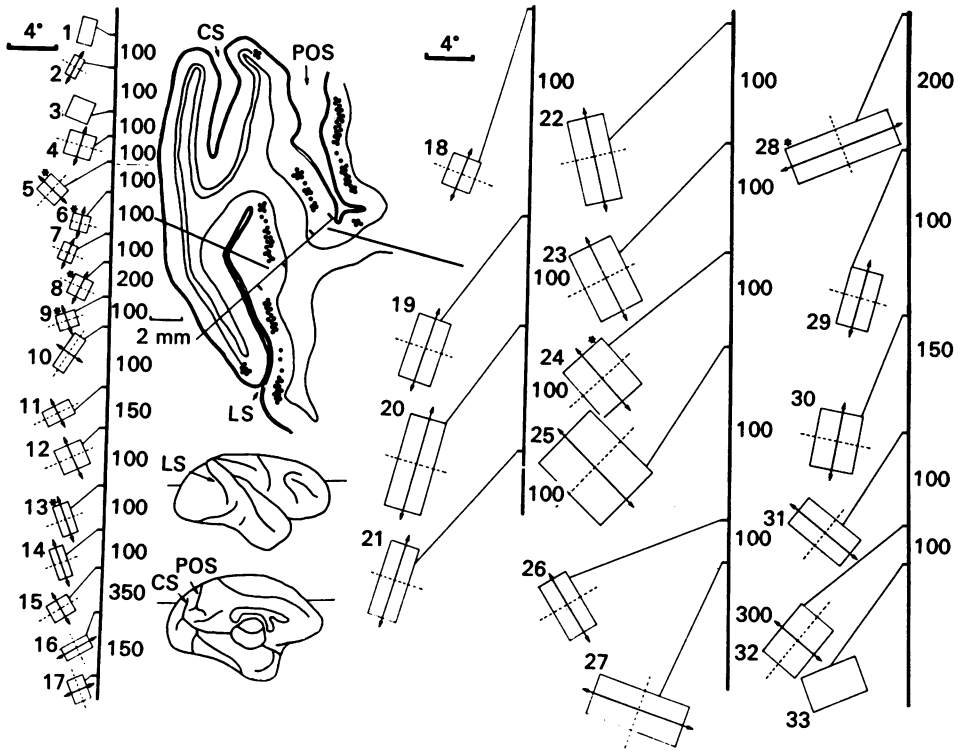
Text-fig. 10. Reconstruction of a long penetration made through area V3A, in the lunate and parieto-occipital sulci. The part of the electrode track from which recordings were made is shown in short bars intersecting the long common electrode track. Recordings from V3A in the lunate sulcus are shown to the left of the Figure; recordings from V3A in the parieto-occipital sulcus are shown to the right. Conventions as in Text-fig. 1. Where there are no interrupted lines within a receptive field, no orientational selectivity was detectable, but the fields were drawn as squares or circles for convenience. Note that such cells are grouped together (5–8). Asterisks next to the receptive fields indicate that, although the cell was orientation selective when tested with slits, it responded briskly to spots. Note that such cells occur either singly (cell 18) or in groups (cells 9–11).

Types of cells within V3 and V3A and their groupings

The most commonly encountered type of cell in both areas was orientation selective, as shown in the histogram given in a companion paper (Zeki, 1978*b*) and most of these behaved like complex cells. Hypercomplex cells, though present, were much less common. Some of these orientation selective cells were also directionally selective but, for both areas, directional selectivity was uncommon. The cells were also

indifferent to the colour of the stimulus. In recordings from over 250 cells in each area, I was unable to find any evidence for colour opponency, although there was a small percentage of cells in both areas that had colour biases (see Zeki, 1978*b*).

Some of the orientation selective cells had an altogether more complicated response pattern. Such cells, when tested with slits of light, showed every sign of being orientation selective and responded to motion in either direction perpendicular to the receptive field axis of the cells. Their response to spots of light was, however, not



Text-fig. 11. Reconstruction of a long penetration made through V3A in both the lunate and parieto-occipital sulci. Conventions as in previous Figures, but the asterisks in this one indicate that, although the cell was drivable through the individual eyes, the response of the cell was powerfully summated by binocular stimulation. Note that such cells occur either singly (e.g. cells 13, 24 and 28) or in groups (cells 5–9). Note also the abrupt change in orientation between cell 28 and 29, separated from each other by 200 μm. In the first part of the penetration, in the lunate sulcus, cells are numbered from the pial end to the white matter end. In the second part of the penetration (in the parieto-occipital sulcus) the first cell (cell 18) was closest to the white matter end whereas the last cell (33) was closest to the pial surface.

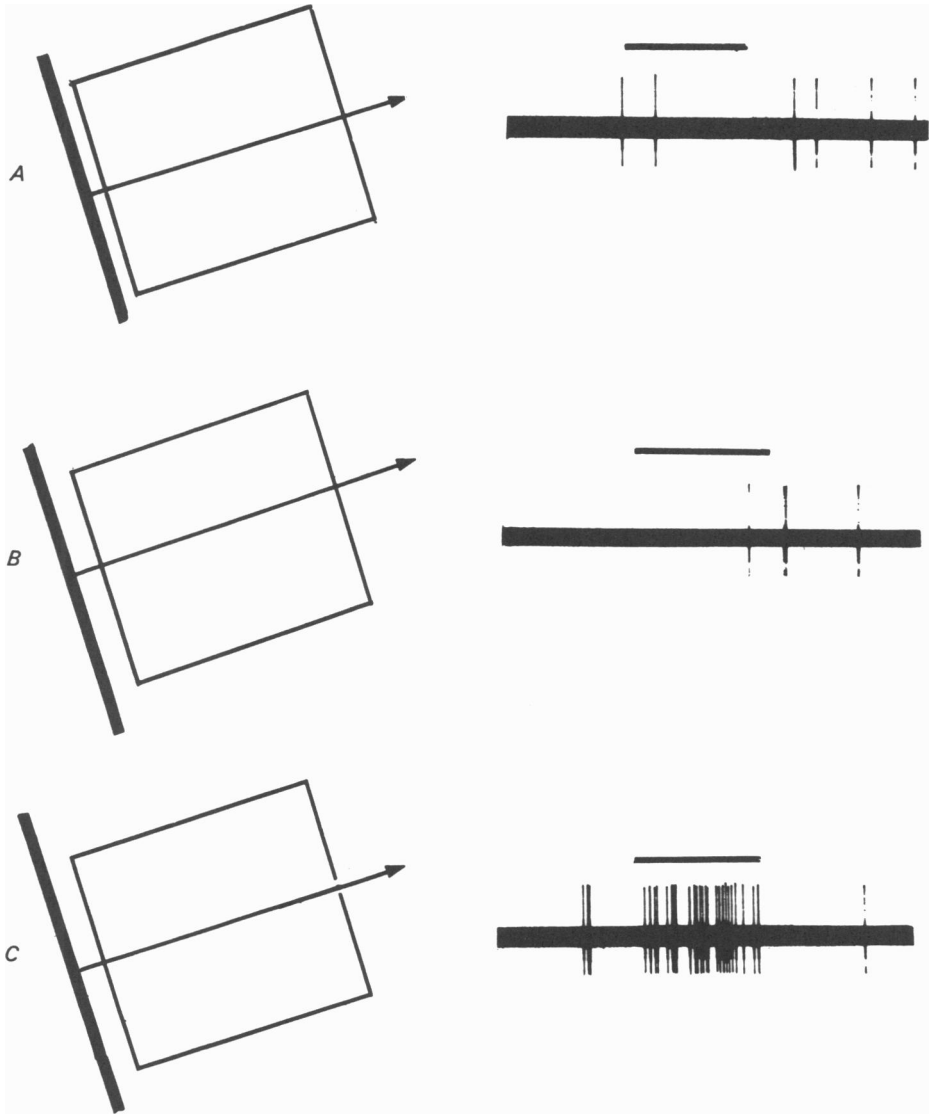
predictable from their response to slits. Unlike most orientation selective cells, which either do not give a response to spots or do so only very reluctantly, these gave a powerful response to spots of light and were reminiscent of the complex cells in layer V of cat visual cortex described by Palmer & Rosenquist (1974). At the other extreme, there were also cells which were responsive to stimuli moving in any direction and did not appear to have any orientational or directional selectivity. Some of these cells had

antagonistic surrounds. Usually, but not always, such cells were grouped together. Text-fig. 10 illustrates a penetration made through V3A in both the lunate and parieto-occipital sulci. In this penetration cells 5–8 had no orientational preferences and were grouped together. Cells 9–11, marked by asterisks, responded briskly to spots of light moved in any direction but showed clear orientational selectivities when tested with slits. Cell 18 behaved in a similar way to cells 9–11 but appeared to occur in isolation among cells which were strictly orientation selective.

Most of the cells (over 90%), whether orientation selective or not, were binocularly driven, without any obvious monocular preferences. Among these were cells which gave a far more powerful response to simultaneous stimulation of the two eyes than would be predicted from their responses to the individual eyes. There was, in other words, a greater degree of binocular facilitation than is commonly seen with binocularly driven cells. Such cells were also usually grouped together, although they sometimes occurred in isolation. Figure 11 illustrates a penetration, made through V3A in both the lunate and parieto-occipital sulci. In this penetration, cells with a marked degree of binocular facilitation are marked with an asterisk. In the first part of the penetration, made within V3A in the lunate sulcus, there is a sequence of such cells grouped together (cells 5–9) and another one (cell 13) found in isolation. Such cells may be involved in detecting binocular disparity and, since I tested at one disparity only, the penetration illustrated in Text-fig. 11 may give a misleading impression of the proportion of such cells in V3A (or in V3). It is possible that, had one tested for different disparities, a far clearer picture of the range of binocular facilitation would have been obtained. That such a range almost certainly exists is clear from examples of cells, found both in V3 and V3A, which respond only when both eyes are simultaneously stimulated and for which monocular stimulation is ineffective (Text-fig. 12). Such cells, too, occurred either singly or in groups. In the penetration illustrated in Text-fig. 13, receptive fields of cells which could only be influenced by simultaneous binocular stimulation are drawn in interrupted lines. It is evident from the penetrations shown in this figure that such cells are grouped together and flanked on both sides by cells driven equally well by either eye. The change from one type of cell to another can apparently occur independently of orientational changes. In the penetration illustrated to the right of Text-fig. 13, for example, the change from cell 2 to cell 3, 80 μm apart, was not in orientation selectivity, which was identical for the two cells, but in the degree of binocular facilitation, cell 2 being driven equally well by either eye whereas cell 3 responded only when both eyes were simultaneously stimulated. This change in degree of binocular facilitation, independently of changes in orientation preferences, can also be seen between cells 8 and 9, 160 μm apart. Both these cells responded to the identical orientation, but cell 8 could only be driven by binocular stimulation whereas cell 9 responded to stimulation of the contralateral eye only. Penetrations such as those illustrated in Text-fig. 13 would tend to suggest that there are two independent systems of cell groupings, one registering the degree of binocular interaction and another registering orientational selectivities, and that the change in one may occur independently of changes in the other.

In penetrations perpendicular to the cortical surface, successive cells tended to have the same orientational preferences whereas, in oblique penetrations, the orientational preferences changed and these changes were usually gradual rather than abrupt

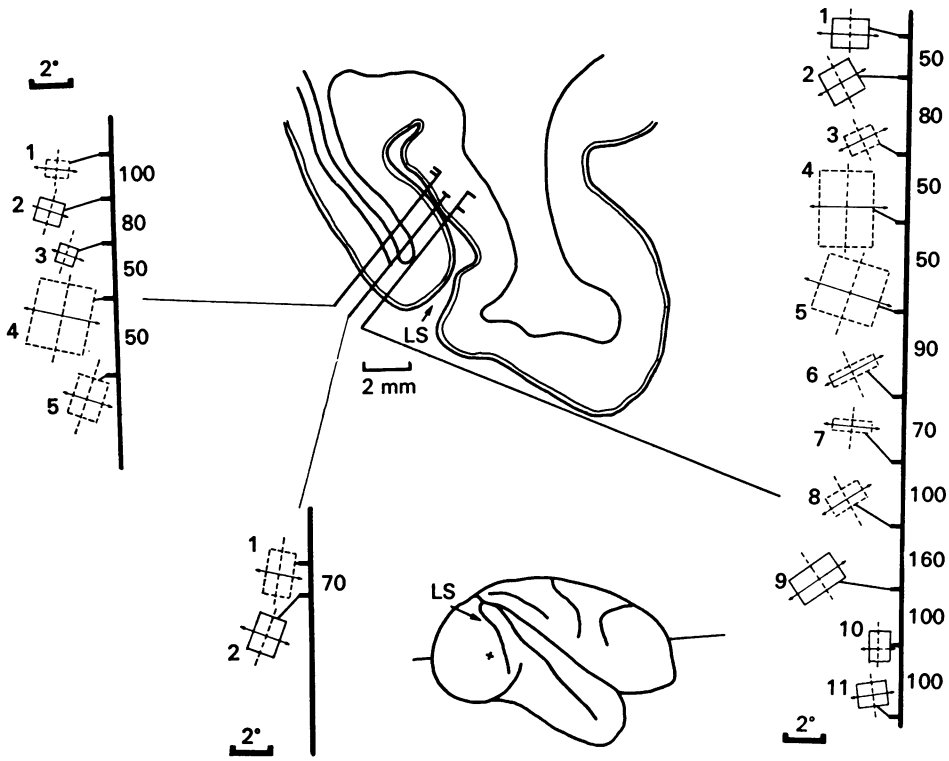
(see Text-figs. 1, 10 and 13). There were, however, instances of abrupt changes, for example that between cells 4 and 5 of Text-fig. 11. More striking examples of abrupt changes are shown in Text-fig. 1. The penetration reconstructed to the right of the



Text-fig. 12. The responses of a cell in area V3A to stimulation of the ipsilateral eye (*A*), the contralateral eye (*B*), and to binocular stimulation (*C*). The cell was 4° × 4½° in size, and situated in the contralateral visual half field. Each sweep about 3 sec with the line above each trace indicating when the stimulus crossed the receptive field.

figure was in V3 and was very nearly normal to the cortical surface. The first five cells (from the white matter end) all preferred a nearly horizontally orientated slit. Yet cell 6, encountered at the same depth as cell 5, responded to an orientation which was exactly orthogonal to the orientation that the first five cells responded to. The next

cell, 7, was removed from cell 6 by 100 μm. But it responded to the same horizontal orientation as the first five cells. A similar abrupt change occurred between cell 8 and 9, 100 μm apart. It is interesting to note that changes in the degree of binocular facilitation of these cells were not necessarily accompanied by changes in orientational preference. For example, cells 4 and 5 were both responsive to a horizontal slit, yet cell 4 was equally well driven by either eye whereas cell 5 responded only



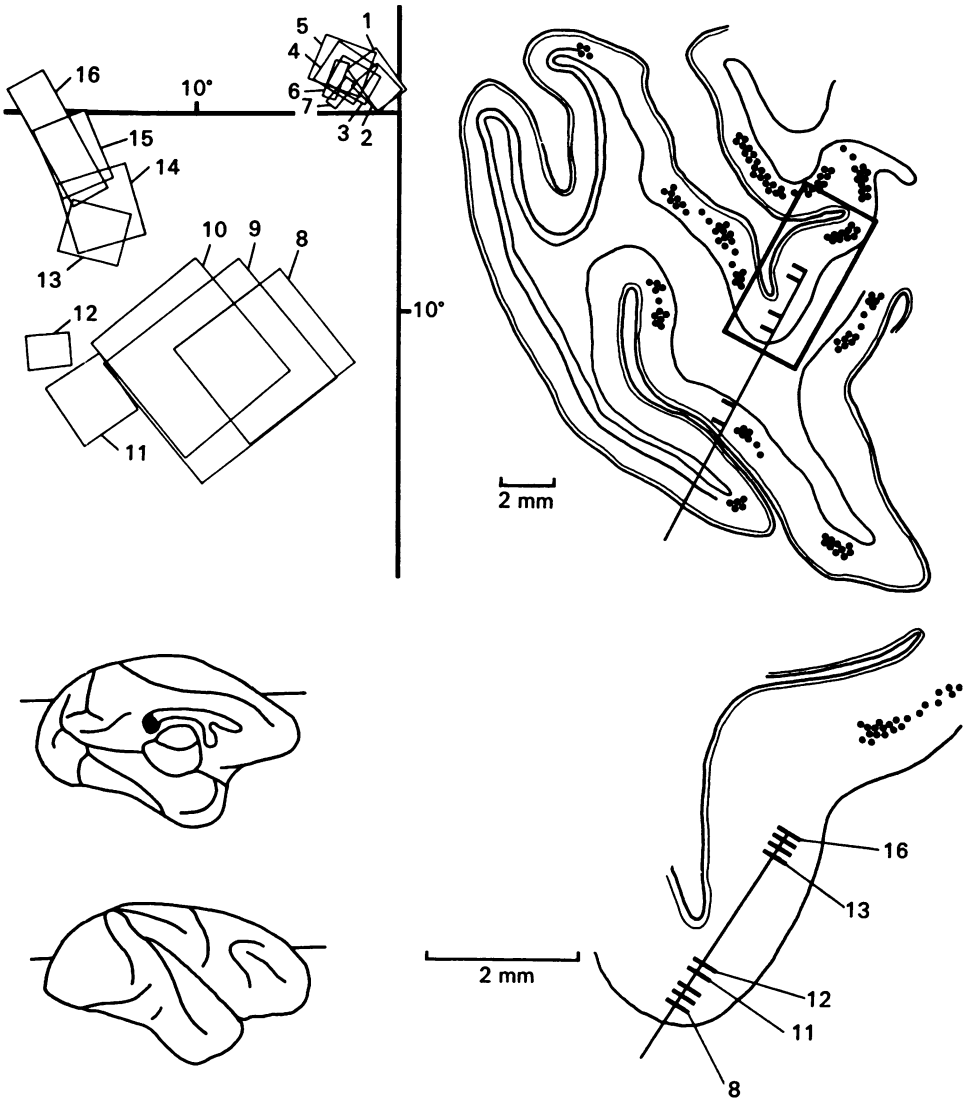
Text-fig. 13. Three parallel penetrations in the anterior bank of the lunate sulcus. Although callosal degeneration landmarks were not available in this animal, the penetrations are interpreted as having been made in area V3A, from the properties of the cells and the position of each track within the anterior bank of the lunate sulcus. In this Figure, the receptive fields of cells that could only be driven by binocular stimulation are shown in interrupted lines. The × on the surface drawing of the brain marks the position of entry of the electrodes. Other conventions as in previous Figures. Note that the changes in binocular facilitation (e.g. between cells 1 and 2 and 3 and 4 to the left, and between 2 and 3, and 8 and 9 to the right) are not accompanied by any shifts in orientational preferences and where changes in orientational preferences occur (e.g. between cells 1 and 2, and between cells 5, 6, 7 and 8 to the right) they do not necessarily entail changes in binocular facilitation.

when binocularly stimulated. Between cells 5 and 6, however, there is a change both in binocular facilitation and in orientation and the same is true of cells 6 and 7, and 8 and 9.

Such results, in perpendicular penetrations, were a little surprising but they are not restricted to V3. Examination of Text-fig. 11, for example, shows that there was

a very substantial shift in orientational preference between cells 28 and 29, 100 μm apart, in a penetration nearly normal to the cortical surface of V3A. This change is also accompanied by a shift from a cell (28) summating its response to binocular stimulation to one (29) driven equally well by either eye.

Hence despite the grouping of cells with common orientational preferences, there



Text-fig. 14. Reconstruction of a long penetration through area V3A, in both the lunate and parieto-occipital sulci. Conventions as in previous Figures. Cells 1–7 were recorded from the part of V3A lying within the anterior bank of the lunate sulcus. The tract was nearly normal to the cortical surface in this part of V3A. Note that there is no detectable shift in receptive field positions. Cells 8–16 were recorded from in that part of V3A lying within the parieto-occipital sulcus. This part of the track was oblique, and is shown at higher magnification to the lower right. Note the net shifts in receptive field positions of successive cells. To the lower right, the short horizontal lines intersecting the long common electrode track indicate the positions of successively recorded cells.

are occasions when abrupt changes in these preferences occurred even with cells separated from each other by no more than 100 μm in perpendicular penetrations. Why there should be instances of such abrupt changes in any one property, for example orientation or degree of binocular facilitation even in perpendicular penetrations, would require a study of many more cell sequences in both areas than presented here. But the results given here suggest, at least tentatively, that a more detailed study of the encoding of information relating to the eyes and to orientation, and the inter-relationship of these two variables may provide an important clue to the functions of the third visual complex.

In addition to the types of cells described above, isolated examples of other types of cells were encountered. One such type of cell had a high maintained discharge in the dark, but was inhibited by light of any wave-length. Another type of cell was excited maximally when its entire receptive field was flooded with light. For some of these cells, going beyond the receptive field diminished the response whereas for others invading the surrounds did not alter the response. Even switching on the room light gave a powerful discharge. Both types of cell were rarely encountered and it is still unclear how they may be grouped together in relation to the more common type of cell described here.

Magnification factors. This term refers to mm of cortex per degree of visual angle at different eccentricities. As discussed elsewhere (Zeki & Sandeman, 1976; Van Essen & Zeki, 1978) the cortical magnification factor is much smaller in V3 than in V1 or in V2. This also appears to be the case for V3A where, despite receptive field size and scatter, net changes in receptive field position could still be detected for points separated by as little as 500 μm (see Text-fig. 14). Comparison of this Figure with that of Text-fig. 8 of Van Essen & Zeki (1978) shows that in V3A, just as in V3, the magnification factor is less than a mm per degree even for central visual fields. Because of the distribution of receptive field size and scatter in both areas, a much more detailed analysis of many more cells would be required to determine whether there are any substantial differences between V3 and V3A in this respect.

Preliminary recordings from the medial bank of the parieto-occipital sulcus

As described in Part I, the medial bank of the parieto-occipital sulcus receives a direct input from V3 and V3A. I have made preliminary recordings in that region and, once again, most of the cells appeared to be orientation selective. It is still too early to tell but, if this result is substantiated, there may be a case for including other regions of the prestriate cortex in the third visual complex of areas.

DISCUSSION

Although V3 and V3A are two independent areas, in each one of which the visual fields are separately represented (Van Essen & Zeki, 1978) and although they differ from each other in their anatomical inputs and callosal connexions, it is difficult to make a distinction between the two areas on functional criteria alone, using the techniques described here. Even topography can be misleading. The vertical meridian of the lower visual fields is represented at the V3–V3A boundary and the eccentricities appear to be in precise register. The consequence of such an arrangement is that the

same parts of the vertical meridian are represented along any point of their common border. Therefore, in the absence of additional anatomical landmarks, it is often impossible to tell whether one is recording from V3 or V3A, unless cells have receptive fields in the superior hemifields, in which case the recordings would be in V3A. This stands in marked contrast to other parts of the prestriate cortex, such as the medial and lateral parts of the posterior bank of the superior temporal sulcus, where the distinction can be made on functional criteria alone (Zeki, 1977*b*).

Fortunately, as discussed in this paper and elsewhere, a boundary between these two areas can be drawn by using the callosal connexions of the prestriate cortex as a reference system. Using this as a guide, it has been possible to establish the autonomy of these two areas on topographic (Van Essen & Zeki, 1978) as well as on anatomical grounds.

The boundaries of V3 and V3A

In many ways, this study represents a preliminary excursion into both these areas, with many details still left unsettled. One of these concerns the boundaries of V3 and V3A. The boundary of V3 with V2 can be drawn quite accurately because of the sudden change in receptive field size of cells and the reversal of their position from horizontal back to vertical (Zeki & Sandeman, 1976; Van Essen & Zeki, 1978). The boundary of V3A with V4 can also be drawn quite accurately through the pattern of callosal degeneration and because of the change to higher concentrations of cells with non-orientated receptive fields as well as colour coded properties as one enters V4. But the ventral limit of V3 is still uncertain. The schema given earlier, of a discontinuity in the vertical meridian representation of V3, is only one alternative among others. Another possibility is that V3, as well as V2, may end laterally in the region of the projection of foveal striate cortex, making of the latter a specialized region (see Text-fig. 6). Although this is a possibility, it is not one that is appealing because the pattern of degeneration following foveal striate lesions is very much like the pattern of degeneration following extra-foveal lesions, the only difference being the inclusion of a further area, V4, in the projection field (Zeki, 1978*a*). It is also not clear whether, assuming that V2 is a continuous belt, V3A meets it at any point or whether V3A is cushioned from V2 by V3 all along. A much more detailed anatomical study would be required to unravel the manner in which these areas come together laterally.

Orientation selectivity in the visual cortex of the rhesus monkey

When taken in conjunction with other studies, on other visual areas, such as V1 and V2 (Hubel & Wiesel, 1968, 1970; Zeki, 1978*b*), it becomes obvious that orientation selective cells form the major part of the population in a large part of the visual cortex, extending from V1 to V3A, even though these areas differ from each other in other ways. Obviously, once orientation selective cells are built up in V1 by inputs from the lateral geniculate nucleus, the information on orientation incorporated into the receptive field structure of the cells is not lost. Rather, it is passed along by direct (V2, V3) or indirect (V3A) projections to cells in more central visual areas and is generalized over wider parts of the visual fields, since cells in V2 have larger receptive fields than those in V1 and those in V3 and V3A have larger fields still. The implications of this from the viewpoint of specificity of connexions in the prestriate cortex

are of great interest especially if, as seems almost certain, the orientation selectivity in both V2 and V3 are generated by direct inputs from V1. It implies that a set of cells registering the same orientation in several different hypercolumns in V1 sends two projections, one to V2 and another one to V3, thereby maintaining the information on orientation, but extending it over wider parts of the visual fields. Adjacent cells in the same hypercolumns, registering different orientations, would project to adjacent sets of cells in V2 and V3 thereby maintaining the grouping of cells of common orientational preference in both these areas. The same type of projection can be postulated from V3 to V3A. What changes in these areas, compared with V1, is the relationship of the groupings of cells with common orientational preferences to the information coming from the two eyes. Unlike V1 (Hubel & Wiesel, 1974), most of the cells in these three areas are binocularly driven (Zeki, 1978*b*) and it is apparent from this study that there are changes, in oblique penetrations, in the degree of binocular facilitation of successive cells which are independent of orientational changes. Hubel & Wiesel (1970) recording in the annectant gyrus (and therefore probably in all three areas) also found such changes. The relationship of ocular dominance columns in V1 to the generation of cells with different degrees of binocular facilitation in these areas, the manner in which cells in different layers of V1 project to V2 and V3 and the relationship of orientational groupings to groupings based on degrees of binocular facilitation in these three areas, and whether they differ between one area and another, are but some of the problems which provide an exciting field for future research.

Comparison with other primates

Over the past few years, Allman & Kaas (1975) have studied the topographic organization of the visual areas in a New World primate, the owl monkey, in considerable detail. A comparison of rhesus and owl monkeys indicates that there is no equivalent of a V3 or a V3A in the owl monkey. In this sense, the organization of the prestriate cortex in the two species is quite different. But the differences may be apparent rather than real. Both species contain multiple, independent, representations of the visual fields in the prestriate cortex and it is possible that some of the variety of areas that Allman & Kaas have described in the owl monkey may be functionally homologous to V3 and V3A. When the results of single cell functional studies for the different areas in the owl monkey, as well as their connectivities, become known, it may be possible to decide to what extent the two species differ from each other.

Another New World primate that has been studied is the squirrel monkey. However, whether this primate has an area V3 or not remains a contentious point, Spatz, Tigges & Tigges (1970) claiming that it does not, whereas Cowey (1973) and Martinez-Milán and Holländer (1975) claiming that it does. Until this controversy is resolved and until more is known of the topographic organization of the prestriate cortex in the squirrel monkey, it would be idle to speculate on any similarities or differences between it and owl and rhesus monkeys.

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The experiments reported in this study stretch back over 6 years. During that time I have had excellent histological assistance from Mr George Barrett, Ms Pamela Jacobs and Ms Brenda Crane. I am also greatly indebted to Mr David Sandeman and Mr William Taylor for their help during the recording sessions.

I would like to record my thanks to Professor J. Z. Young for his critical reading of this manuscript.

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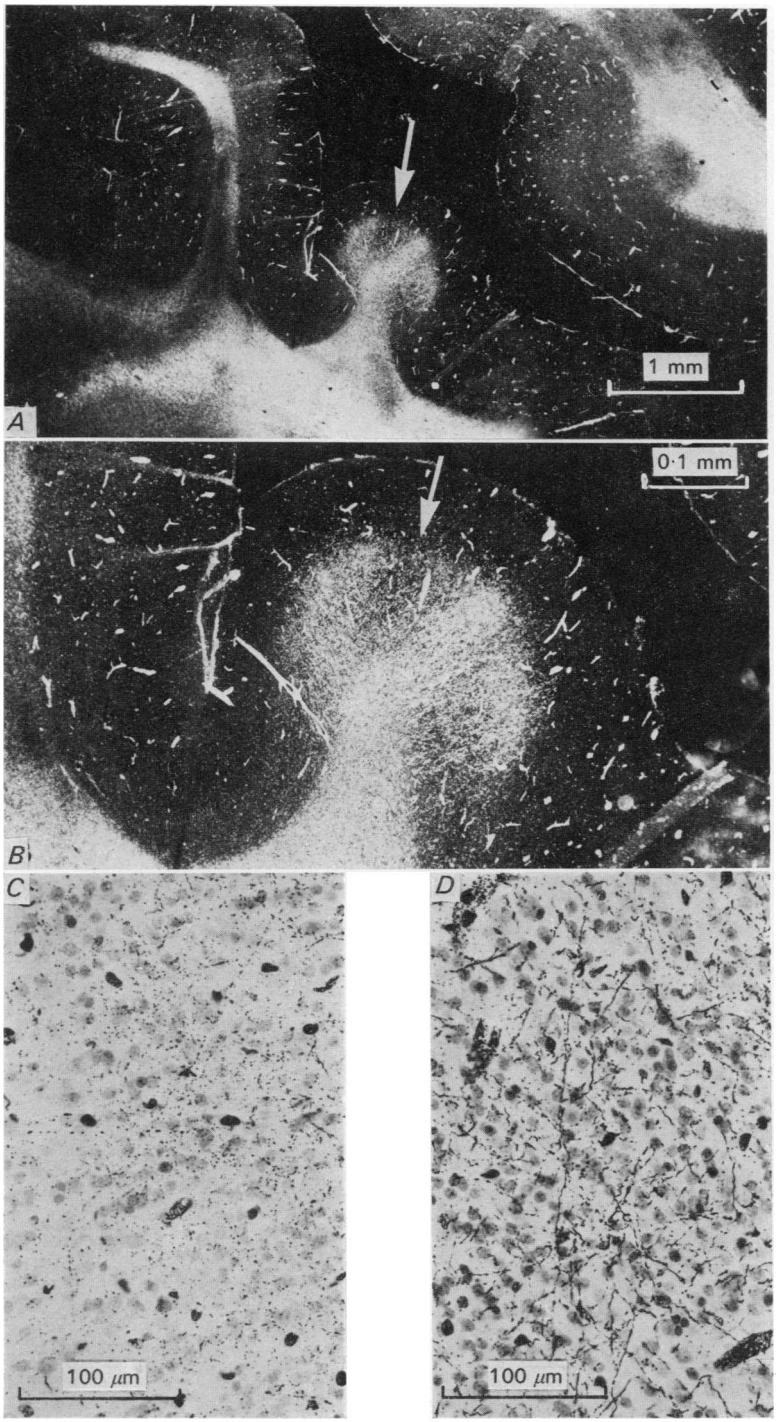
EXPLANATION OF PLATE

The degeneration in the parieto-occipital sulcus, following a lesion in the horizontal meridian representation of V1, at an eccentricity of about 30°. *B* is a higher-power photomicrograph of *A*. In both *A* and *B*, the boundary between V2 (to the left) and V3 (to the right) can be distinguished and is indicated by arrows. In bright field, the degeneration in V2, shown in *C*, is fine whereas that in V3, shown in *D*, is coarse.



(*Facing p.* 272)