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Cortico-cortical connexions of the cat visual areas

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INTRODUCTION

The three cytoarchitectonic areas of Otsuka & Hassler (1962), area striata (17), area occipitalis (18) and area praeoccipitalis (19) have been shown to correspond to the three visual areas (visual I, II and III respectively) determined electrophysiologically by Hubel & Wiesel (1965). The topographical organization of these areas has been described in more detail by Bilge, Bingle, Seneviratne & Whitteridge (1967) (see Fig. 1).

Visual areas I and II and the lateral part of the middle suprasylvian gyrus receive

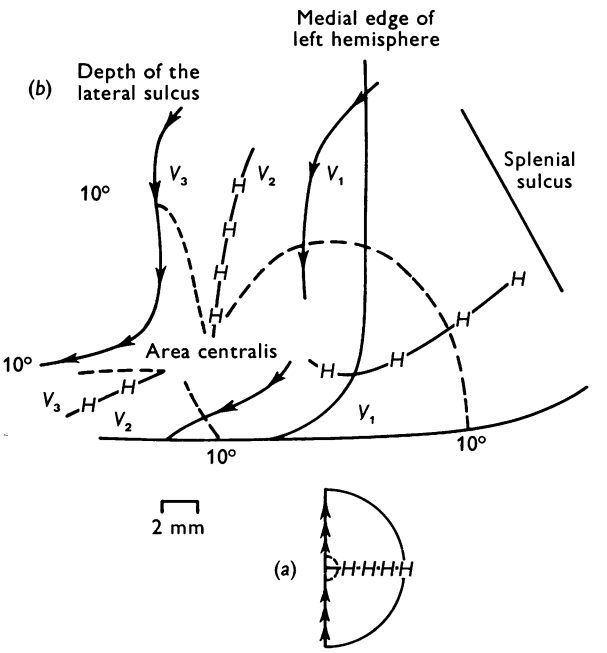

Fig. 1. (*a*) The perimetric representation of the half visual field. (*b*) The representation of this half visual field on the left cerebral cortex of the cat viewed from above. The brain is represented flat in the medio-lateral direction by taking measurements around the perimeter of the lateral gyrus from the medial edge of the hemisphere (i.e. the junction of the supero-lateral and medial surface of the hemisphere). →→→–, vertical meridian; -*H*-*H*-*H*-, horizontal meridian; V_1, V_2, V_3 are the visual areas I, II and III respectively. This figure is modified with permission from Bilge *et al.* (1967).

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a direct projection from the dorsal lateral geniculate nucleus (LGNd). Visual III and the lateral part of the middle suprasylvian gyrus receive a projection from the medial interlaminar nucleus of the LGN (Wilson & Cragg, 1967). Polley & Dirkes (1963) and Hubel & Wiesel (1965) have shown projections to more lateral cortical areas in the ipsilateral and contralateral hemispheres from area 17. Choudhury, Whitteridge & Wilson (1965) recorded from the cat visual cortex and as the ipsilateral optic tract had been cut the activity recorded must have traversed the corpus callosum. They found photically evoked responses only in the region of the representation of the vertical meridian (the visual I/II boundary). This supports the anatomical observations of Ebner & Myers (1965) on the distribution of the corpus callosum using the Nauta method.

The present study was undertaken to provide more information about the cortical projections to both hemispheres, not only from visual I but also from visual II and III. Particular attention was paid to the topographical representation of the visual field on the cortex.

METHODS

The lesions were made under aseptic conditions in twelve cats anaesthetized with sodium pentobarbitone. A small burr hole was made with a dental drill in various predetermined positions in relation to bregma and the sagittal suture. Except in certain cases the bony defect did not exceed 3 mm. The dura was opened under an operating microscope, and to make the lesion about 1 mm^2 of pia mater was torn away using two pairs of watchmakers forceps. In addition, it was usually necessary to disrupt the outer layers of the grey matter with forceps or gentle suction. A larger hole was necessary for the lesions made between the hemispheres. This was drilled over the sagittal sinus and for about 2 mm over the lateral gyrus on the side on which the lesion was to be made. The dura was cut alongside the sagittal sinus. The brain shrinkage caused by the slow intravenous injection of 20 ml of a 30 % solution of urea in 5 % dextrose* given immediately after the anaesthetic was valuable in allowing a good exposure of the splenial gyrus without packing or retraction.

After survival periods of 7–8 d (but 14 and 22 d for cats W4 and W8) the animals were anaesthetized with sodium pentobarbitone and perfused with 10 % formolsaline. The brains were stored for 2 days to 2 weeks in 10 % formolsaline and during this time were photographed or drawn to scale to help with the reconstructions. The brains were divided by two coronal cuts, the position of which varied according to the site of the lesion. Degeneration in the rostral 15 mm of the cerebral cortex will not be reported in this paper; the rest of the brain was examined. Coronal sections were cut 30 μ thick on a freezing microtome. Every tenth section through the lesion was stained with cresyl violet or chrome alum gallocyanin and every 20th section by the Nauta method. A multi-compartmented sieve, previously described by Wilson & Cragg (1967) was used to facilitate the handling of sections and to maintain serial order for the Nauta method.

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RESULTS

The cytoarchitectonic boundaries described by Otsuka & Hassler (1962) could not be reliably recognized, perhaps because frozen sections were used. Consequently, the position of the lesions and the degeneration has been described in terms of the gyral pattern and by comparison with the map of the representation of the visual field on the cortex (Bilge *et al.*, 1967).

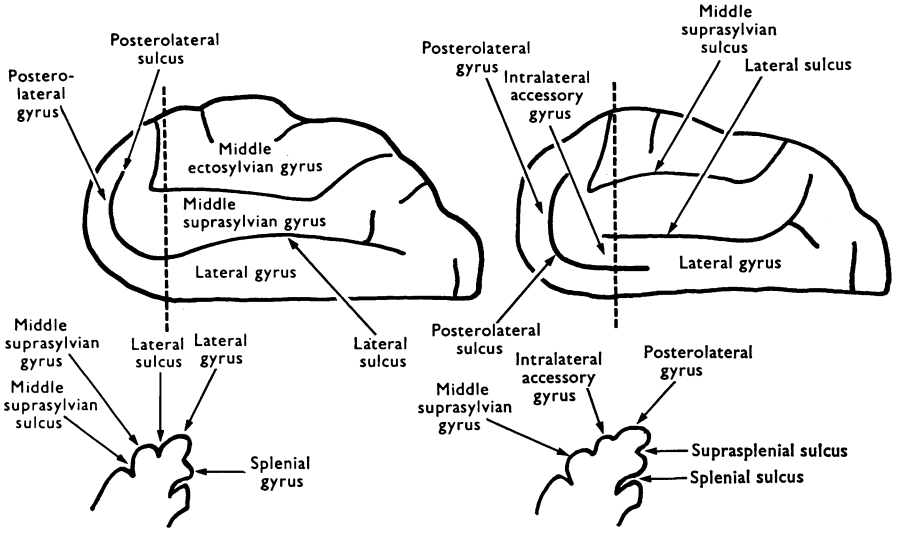

Fig. 2. The two most common variations in the gyral pattern of the cat brain (left hemispheres, dorsal aspect). For each brain a coronal section taken at the level of the dotted line is reproduced.

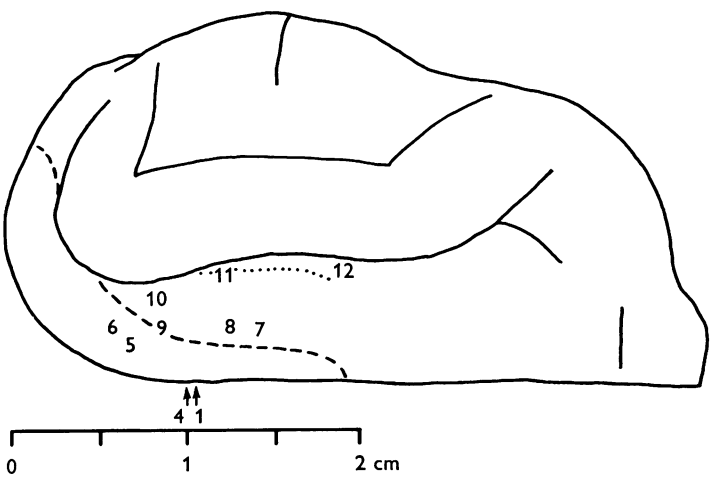

Fig. 3. Composite dorsal view of a cat left cerebral hemisphere showing the positions of the various lesions in the different brains. The two arrows indicate the rostrocaudal positions of the medial wall lesions in cats W1 and W4. The dashed line is the area 17/18 boundary and the dotted line is part of the area 18/19 boundary of Otsuka & Hassler (1962).

Figure 2 shows the two most common variations in the gyral pattern of the cat brain. The relevant sulci and gyri are labelled. For convenience the sites of most of the lesions have been marked on a single dorsal view of a cat brain (Fig. 3). The dashed line marks the approximate position of the area 17/18 boundary and the

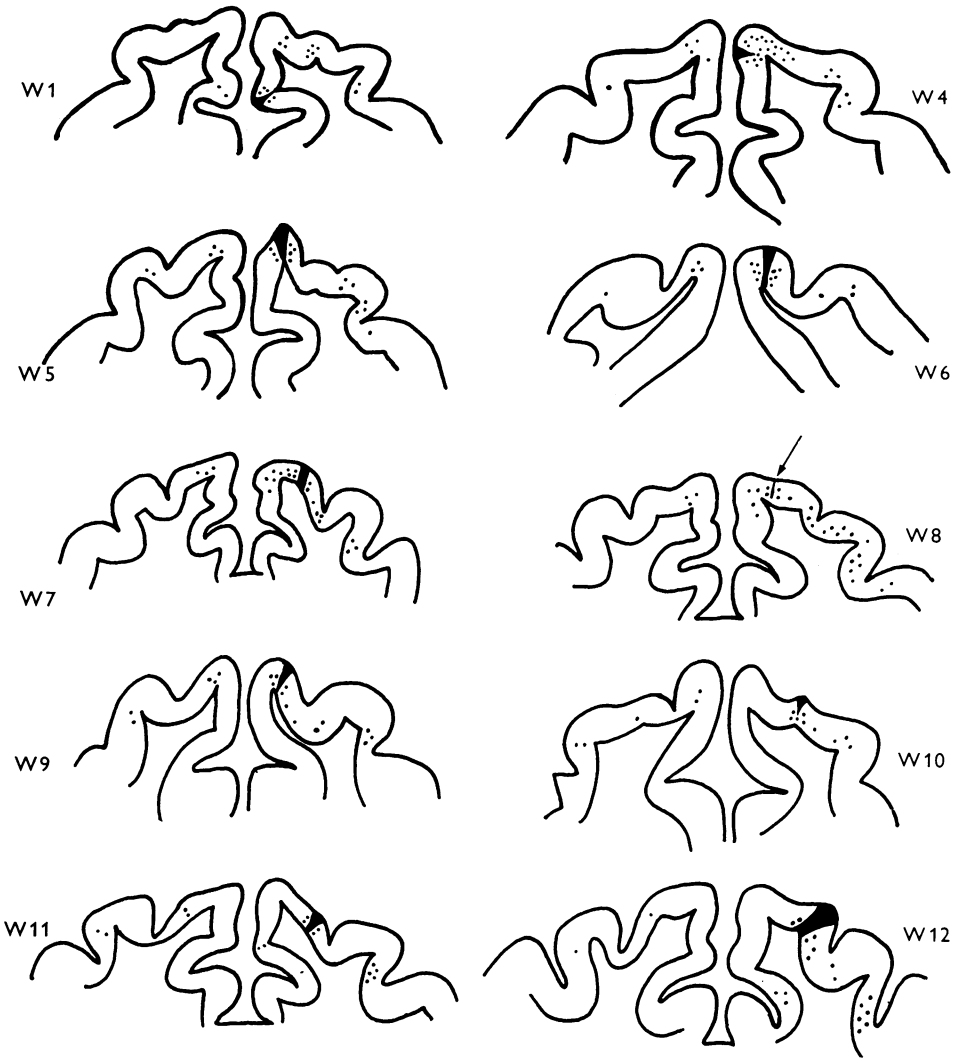

Fig. 4. Representative coronal sections through the lesions shown in Fig. 3. The lesions are marked in black. See text for details.

dotted line part of the 18/19 boundary of Otsuka & Hassler (1962). Representative coronal sections have been reproduced in Fig. 4. The dots give some idea of the density of preterminal degeneration but are not intended to give information about the depth within the cortex. Degeneration usually extended for 1 or 2 mm anterior and

posterior to the reproduced sections. If the degeneration differed in more anterior or posterior planes it is mentioned in the text.

In four cats the skull and dura were opened and either no lesions were made or only the pia was disturbed. No Nauta degeneration was found in these cats, either at the operative site or elsewhere. This then strongly suggests that in the other cats the degeneration seen is attributable to the lesion.

Cat W 1. The first two cats to be fully described reveal the projections from regions well within visual I. In cat W 1 the lesion extended a short distance along the splenial gyrus (from 9 to 13 mm anterior to the occipital pole) and penetrated the deepest layer of the cortex, but not into the white matter. The lesion, then, was in the representation of the peripheral field near the horizontal meridian. There was no sign of other cortical damage in either hemisphere.

A moderate amount of Nauta degeneration was found in the cortex around the lesion but most of the degenerating fibres entered the white matter. Ipsilateral fibre degeneration was found in three other areas. The first patch of degeneration was found in the lateral gyrus between 2–3 mm lateral to the mid-line. The second patch was dense in the medial wall of the accessory intralateral gyrus. There was a scant amount of degeneration on the lateral half of the middle suprasylvian gyrus.

Contralateral degeneration: a few degenerated fibres were found opposite the lesion at the topographically corresponding point (Fig. 5). Although degeneration was sparse it was nevertheless definite and could be found regularly just on the crown of the splenial gyrus on adjacent sections. This finding of a scant projection to the medial wall of the contralateral hemisphere receives support from two other cats (W 2, W 3). These had additional damage; in one, the lesion in the splenial gyrus penetrated deeply into the white matter and in the other the surface of the lateral gyrus was damaged on the side of the lesion. In both cases a few degenerated fibres were seen down the medial wall of the undamaged hemisphere. Although these two cats (W 2, W 3) gave no information about the origin, they confirm a finding in cat W 1 that part of visual area I distant from the region in which the vertical meridian of the visual field is represented receives a few callosal fibres.

Cat W 4 also had a lesion in visual I in almost the same coronal plane as cat W 1 but it was less peripherally placed in the visual field, i.e. only 2 mm down the medial wall of the hemisphere. The lesion did not reach the grey-white boundary.

Ipsilateral fibre degeneration was dense around the lesion. Degenerating fibres could be seen to enter the white matter and also to run through the grey matter parallel to its surface in two bands, one at the upper and the other in the lower boundary of cortical layer IV. Amongst these fibres of passage, degenerating preterminals could be distinguished by their random distribution. This pattern of degeneration extended only a short distance inferior to the lesion but extended about 4 mm across the surface of the lateral gyrus. In this particular cat, at the coronal plane depicted in Fig. 4, there was a break between the postero-lateral sulcus and the lateral sulcus so that for a few millimetres the lateral and middle suprasylvian gyri were continuous. A patch of degeneration was found in the region corresponding to the medial wall of the middle suprasylvian gyrus. Finally, there was a slight amount of fibre degeneration in the lateral wall of the middle suprasylvian gyrus.

Contralateral degeneration was slight over the medial quarter of the lateral gyrus

and occasional degenerated fibres were found opposite the lesion. A few degenerated fibres were found in the lateral wall of the middle suprasylvian gyrus.

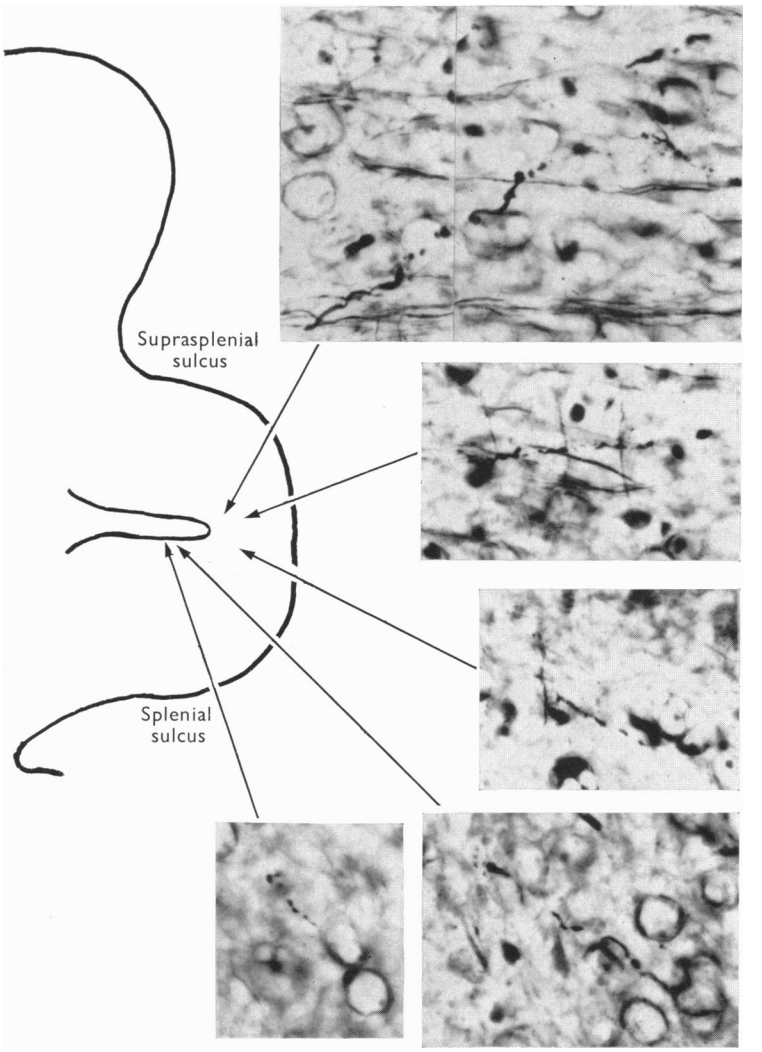

Fig. 5. The splenial gyrus of cat W1 contralateral to a lesion in the splenial gyrus showing some of the Nauta degeneration. (× 500).

These two animals (cats W1 and W4) show a triple ipsilateral projection from the lesion. The contralateral feature of note is the scant projections between homotopic points in area 17.

The next two lesions (W5 and W6 cats) were placed near the representation of the vertical meridian 6 mm from the occipital pole in visual I. Both are near the

representation of the area centralis (which is situated 4–6 mm from the occipital pole).

Cat W5. Ipsilateral fibre degeneration was dense around the lesion. A moderate amount of degeneration was found on the medial wall of the middle suprasylvian gyrus in the same coronal plane as the lesion. One or two mm rostral to this, degeneration was seen on the lateral half of the middle suprasylvian gyrus. There was also slight degeneration on the medial wall of the middle ectosylvian gyrus which merged with the lateral suprasylvian degeneration about 2 mm anterior to the lesion.

Contralateral degeneration was moderate in amount on the medial half of the lateral gyrus and slight on the posterior end of the medial half of the middle suprasylvian gyrus.

A second cat W6 had a lesion placed about 1 mm posterior and lateral to that in cat W5 and showed a similar pattern of degeneration.

Cat W7 also had a lesion close to the vertical meridian but it was more anteriorly placed (14 mm from the occipital pole) than in the last two cats. The lesion could have been in either visual I or II.

Ipsilateral fibre degeneration was dense around the lesion and could be seen radiating medially through the grey matter and cutting directly through the white matter to reach in slight amounts as far as the suprasplenial sulcus. This pattern of degeneration was seen well in cat W8. Lateral to the lesion preterminal degeneration was slight, increasing in amount down the medial wall of the lateral sulcus. There was a moderate amount of degeneration in the lower part of the lateral wall of the middle suprasylvian gyrus.

Contralateral degeneration was moderate in amount across the medial half of the lateral gyrus.

Cat W8 had a similar lesion (indicated by an arrow on Fig. 4) and pattern of degeneration to the previous cat with the exception that there was dense degeneration across the whole of the middle suprasylvian gyrus, which may be attributed to a plug of bone wax which indented this gyrus. A few fibres were found on the medial edge of the middle ectosylvian gyrus. Cat W9, with a less anterior lesion, showed similar degeneration to cat W7, but in addition a scant amount of degeneration was found on the lateral half of the middle suprasylvian gyrus contralateral to the lesion.

These last four cats (W5–9) show an ipsilateral projection to the lateral sulcus and to the lateral wall of the middle suprasylvian gyrus.

Cat W10 had a lesion on the posterior end of the intralateral accessory gyrus, not quite reaching the white matter, probably within visual II.

Ipsilateral degeneration: most of the degenerated fibres from the lesion entered the white matter, but a few extended laterally through the grey matter. A slight amount of fibre degeneration was seen on the medial wall and more anteriorly on the lateral wall of the middle suprasylvian gyrus.

Contralateral degeneration: occasional degenerated fibres were found on the medial extremity of the lateral gyrus, the intralateral accessory gyrus and the middle suprasylvian gyrus.

The next two lesions were more laterally placed along the lateral margin of the lateral gyrus and were therefore close to the horizontal meridian of the visual field

which separates visual II from visual III (Bilge *et al.*, 1967). A projection back into visual I was observed.

*Cat W*11. The lesion in this cat was about 12 mm anterior to the occipital pole. It reached, but did not damage, the white matter.

Ipsilateral degeneration: a considerable number of degenerating fibres ran into the white matter from the lesion but there was little in the adjacent cortex. A few degenerated fibres were seen on the crown of the splenial gyrus. Fibre degeneration was dense on the lateral wall of the middle suprasylvian gyrus.

Contralateral degeneration was scant at the corresponding point of the opposite hemisphere and on the lateral half of the middle suprasylvian gyrus.

*Cat W*12 had a lesion 6 mm more anterior than the last cat. This corresponds to a point near the horizontal meridian (visual II or III boundary) and even more peripheral along this meridian than the last cat.

Ipsilateral degeneration: a slight amount of fibre degeneration was seen in the depth and upper border of the splenial sulcus. Nauta degeneration was present around the lesion and a few fibres ran through the grey matter as far as the bottom of the lateral sulcus. Degeneration was dense in the lateral wall of the middle suprasylvian gyrus.

Contralateral degeneration amounted to only an occasional degenerated fibre opposite the lesion and a scant amount in the middle suprasylvian gyrus.

Layers of termination. Because of the smallness of the lesions and the sparseness of some of the projections the study was not well suited to reveal information on this point. Furthermore, it is difficult to set a deep level to the degenerated preteriminals because this is obscured by fibres of passage. Nevertheless, a few conclusions can be drawn. Irrespective of the site or hemisphere, degenerated fibres were found throughout the whole of lamina III and sometimes well into lamina II. The two bands of degeneration running in the inner and outer borders of lamina IV have already been described. Degeneration was also present in lamina V and VI. Thus degeneration extended throughout a greater depth of cortex than had been found in the previous study of the LGN projection to visual cortex (Wilson & Cragg, 1967).

DISCUSSION

It may be argued that part of what has been seen is retrograde degeneration stained by the Nauta method. So far this interpretation has only been put forward in a few sites (Guillery, 1959; Cragg, 1962; Powell & Cowan, 1964). The appearance they describe is quite different from the degeneration seen in the present study. The above workers used considerably longer survival times than the 7–8 d used here in all but two cats. Even in cats W4 and W8, which had 14 and 22 d survival periods respectively, only the typical anterograde characteristics of Nauta degeneration were seen.

Ipsilateral degeneration

The finding of three patches of degeneration ipsilateral to an area 17 lesion has confirmed the work of Hubel & Wiesel (1965). In cat W1 the first patch of degeneration was found on the middle third of the medio-lateral extent of the lateral gyrus and, although a little more medial than one might have expected, it is suggested that

it corresponds with visual II. A similar patch of degeneration was also present in this region in cats W4–6. In these three cats, because the lesion was near to the lateral edge of visual I, there was no degeneration free zone intervening between visual I and II. Furthermore, a lesion in this situation is likely to interfere with fibres running through the grey matter from visual I in the upper medial wall of the hemisphere to visual II more laterally situated on the hemisphere, producing a greater extent of degeneration in visual II than expected.

The second patch of degeneration was more laterally placed and it is suggested that it lies within a region which corresponds to the representation of visual III. Cat W1 showed degeneration on the medial half of the intralateral accessory gyrus and as the lesion is near the horizontal meridian in visual I, the degeneration probably lies close to the medial edge of visual III. The lateral extent of visual III depends on its position along the length of the brain. In the most anterior lesion, near the vertical meridian (cat W7), the degeneration is in the bottom of the lateral sulcus. The more posterior lesions in cats W5 and 6 show a projection to the lateral wall of the postero-lateral sulcus. As the visual I/II boundary is placed obliquely across the lateral gyrus it seems reasonable to find that the lateral boundary of visual III is also obliquely placed, riding up on to the lateral wall of the postero-lateral sulcus. This is in agreement with Hubel & Wiesel (1965).

The third patch of degeneration was found on the lateral half of the middle suprasylvian gyrus. This degeneration was found for 2–3 mm anterior or posterior to the coronal plane of the lesion; localization was not apparent within the depth of the wall of the sulcus. In the cats with the more posterior lesions (cats W5, W6) the suprasylvian projection was to an area 3–4 mm rostral to the lesion. This confirms the electrical observations by Clare & Bishop (1954).

The degeneration found in the medial wall of the hemisphere (visual I) of cats W11 and 12 following lesions in the lateral gyrus is consistent with the mapping of the visual fields by Bilge *et al.* (1967). The lesion in cat W12 was placed close to the horizontal meridian (visual II/III) towards the periphery of the field and the degeneration was found in the bottom of the splenial sulcus which represents the corresponding point of the visual field in visual I. The lesion in cat W11 was less peripherally placed near the horizontal meridian than in cat W12 and the degeneration found in visual I (crown of the splenial gyrus) was correspondingly less peripherally placed. Thus, a projection into area 17, from areas 18/19 has been demonstrated.

It can be concluded that support is given to the idea that cortical areas dealing with the same point in the visual field are interconnected within the same hemisphere.

The projections in either hemisphere are not strictly point-to-point but extend for one or two millimetres, gradually becoming less dense, on either side of the centre of the patch of degeneration. The degeneration found in visual III and the middle suprasylvian gyrus is more widespread than in visual II. Hubel & Wiesel (1965) suggest that each complex cell in visual II receives its input from the simple cells in visual I. As complex cells have larger receptive fields than simple cells, one might speculate that the simple cells in visual I supply not just one, but many, complex cells of visual II covering an area corresponding to at least a few degrees of visual field. Similarly, as hypercomplex units have larger receptive fields than complex units (Hubel & Wiesel, 1965), one would expect this dispersion to be even more

marked for the hypercomplex cells of visual III. Also, since the degeneration is poorly localized in the lateral edge of the middle suprasylvian gyrus one would expect the cells there to have large receptive fields, although electrophysiologically their centres may be well localized and topographically arranged.

Contralateral degeneration

The densest contralateral degeneration was found on the medial third of the lateral gyrus in the region corresponding to the visual I/II boundary (i.e. the vertical meridian). This was produced by lesions close to the visual I/II boundary of the other hemisphere (cats W4–9). This is in agreement with the cross sections published by Ebner & Myers (1965) and Hubel & Wiesel (1965). Contrary to Ebner & Myers (1965), a slight projection was found well down the medial wall of the hemisphere (cats W 1–3). This was only scanty but it was definite; in the presence of dense degeneration in the rest of the brain it could easily be missed. It confirms the finding of Polley & Dirkes (1963).

A contralateral projection was found to the lateral wall of the postero-lateral sulcus (i.e. the vertical meridian of visual III) in cats W5 and 6 which had lesions close to the representation of the area centralis of visual I/II.

In one cat Hubel & Wiesel (1965) describe degeneration on the lateral half of the middle suprasylvian gyrus contralateral to the lesion in visual I. In the present experiments a scant projection to this region was found in one cat from a visual I lesion.

Cats W11 and 12 with lesions laterally situated on the lateral gyrus (visual II/III boundary) showed a scant projection to the corresponding point, and to the lateral half of the middle suprasylvian gyrus of the opposite hemisphere. The contralateral projection was least for cat W 12, which had the lesion farthest from the area centralis.

This study complements the one of Ebner & Myers (1965), who described the total Nauta degeneration picture after either removing one hemisphere or cutting the corpus callosum. This gives no information about the source of the various projections. The present study gives this information, but because the degeneration is often slight from a small lesion and the staining and the size of the lesion is variable, it is less reliable for demonstrating the relative density of the termination of the projections.

Choudhury, Whitteridge & Wilson (1965) could only record photically evoked potentials mediated by the corpus callosum in the region of the visual I/II boundary, i.e. the vertical meridian and the representation of the area centralis (Vesbaesya, Whitteridge & Wilson, 1967). This corresponds with the region seen to receive the most dense callosal projections by the Nauta method. Although degeneration could be seen on either side of this boundary, the projection was presumably insufficient to dominate the behaviour of the cells, so that electrophysiologically they appeared unresponsive.

The following explanation is suggested for the presence of this contralateral degeneration distant from the vertical meridian. Each cortical unit receives its input from several LGN neurones. These LGN neurones might be in different halves of the brain if the receptive field of the cortical unit crosses the vertical meridian. Therefore to complete the input to a cortical unit one would expect the existence of transcallosal fibres, to carry over information about the other part of the field of the unit. This

would be likely to occur most frequently for units lying close to the vertical meridian and occasionally for the few units with large receptive fields which lie further away. Hubel & Wiesel (1965) have found a small overlap of receptive fields across the vertical meridian, although they were unable to determine whether this was real, or an artefact due to an error in lining up the eye. However, more distant regions may influence the cells although this effect may be too slight to be observed in their experimental situation. For example, Burns & Pritchard (1964) using a cerveau isolé preparation found cells with much larger receptive fields. For cat W1 this explanation implies a cell with a very large receptive field, perhaps 60° wide. However, an alternative explanation is that some of the callosal connexions are inhibitory and supply the information that a stimulus has not extended that far into the contralateral visual field.

SUMMARY

Small lesions have been made in the three cortical visual areas of the cat. The ensuing degeneration was stained with the Nauta method and related to the representation of the visual field on the cortex (Bilge *et al.*, 1967). The chief projections were:

1. Within the same cerebral hemisphere (*a*) from visual I to visual II, visual III and the lateral half of the middle suprasylvian gyrus; (*b*) from the visual II/III boundary into visual I.

2. To the opposite cerebral hemisphere: (*a*) a scant projection from visual I, particularly from the visual I/II boundary to the corresponding point; (*b*) from the region of the visual I/II boundary concerned with central vision to visual III; (*c*) a scant projection from the visual II/III boundary to the visual II/III boundary and the lateral half of the middle suprasylvian gyrus.

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