SINGLE-CELL RESPONSES IN STRIATE CORTEX OF KITTENS DEPRIVED OF VISION IN ONE EYE[1]

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INTRODUCTION

IN THE FIRST PAPER OF THIS SERIES (11) we showed that in a kitten 2–3 months of monocular light and form deprivation can produce a marked atrophy of cells in the lateral geniculate body. The changes were confined to layers receiving projections from the deprived eye. Despite the atrophy, most of the cells recorded had normal receptive fields. The present paper extends this physiological study of monocularly deprived kittens to the next level in the visual pathway, the striate cortex. We wished to learn whether one could influence cortical cells from the deprived eye, and whether the receptive fields were normal.

At the cortical level any long-term effects of tampering with one eye might be expected to show up as a change in normal patterns of binocular interaction. It may therefore be useful to begin by summarizing some previous findings on binocular interaction in the normal cat. Approximately four-fifths of cells in the cat striate cortex are binocularly influenced. For any given cell the receptive fields mapped in the two eyes are similar in arrangement and occupy corresponding retinal positions. Although stimuli to corresponding retinal points thus produce qualitatively similar responses, the strengths of the responses from the two eyes are not necessarily equal: some cells respond best to the contralateral eye; others prefer the ipsilateral. Figure 1, reproduced from a previous study (7), shows the distribution of 223 cells according to eye-dominance. It will be seen from this histogram that on the whole the contralateral eye is decidedly the more influential. In the second paper of this series (9) we showed that very young kittens resembled adults in all of these respects. This was true even of animals with no previous patterned visual experience.

We have been particularly interested in learning whether the distribution of cells among the different ocular-dominance categories would be appreciably altered in recordings from monocularly deprived kittens. The histogram of Fig. 1 represents the lumped results of 45 penetrations, and cannot necessarily be used to predict the distribution of cells in a single penetration. To
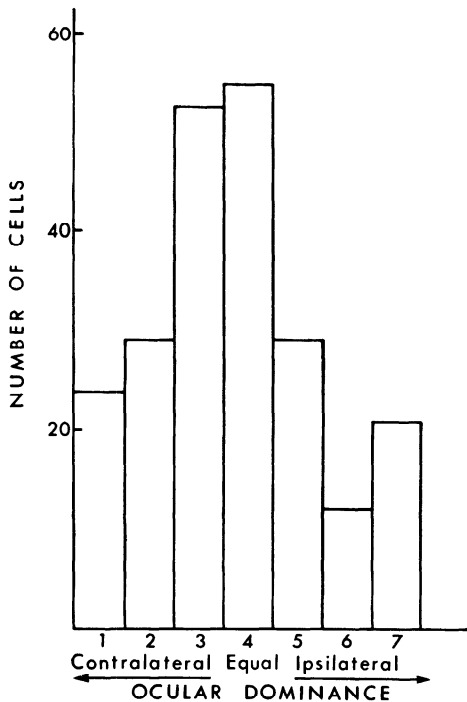
FIG. 1. Ocular-dominance distribution of 223 cells recorded from striate cortex of adult cats in a series of 45 penetrations (7). Cells of group 1 were driven only by the contralateral eye; for cells of group 2 there was marked dominance of the contralateral eye, for group 3, slight dominance. For cells in group 4 there was no obvious difference between the two eyes. In group 5 the ipsilateral eye dominated slightly, in group 6, markedly; and in group 7 the cells were driven only by the ipsilateral eye.

interpret the results of the present paper we need to know whether this distribution varies markedly from one penetration to the next. It might be much narrower and less constant from penetration to penetration if, for example, there were a strong tendency for cells with the same eye-preference to be grouped together in the cortex. To have a better idea of the variation in distribution from penetration to penetration we have therefore taken 12 separate consecutive penetrations from the series used to construct Fig. 1, and plotted each of their ocular-dominance distributions separately (Fig. 2). It appears from these histograms that there is no very marked tendency for cells to be anatomically grouped by eye-preference: penetrations *2* and *4* represent the extremes in the two directions, of dominance by the ipsilateral eye (*2*) or the contralateral eye (*4*), and even in these two penetrations both eyes make a substantial contribution. In judging an individual penetration it is therefore probably reasonably safe to take the distribution of Fig. 1 as the normal, regarding as probably significant only departures much greater than those of penetrations *2* and *4*.

METHODS

Seven kittens and one adult cat were used. The animals were monocularly deprived either by lid closure or by a translucent eye cover (11). Closure of lids prevented form vision and reduced the level of diffuse retinal illumination by about 4–5 log units. The translucent occluders also prevented pattern stimulation, but reduced the diffuse illumination by only about 1–2 log units. Four of the kittens were deprived from the time of normal eye-opening; the others had some prior visual experience. Deprivation was for periods of 1–4 months. Before some experiments the lids of the closed eye were separated or the translucent eye cover was removed. The normal eye was then covered with an opaque contact occluder and the animal's visual behavior was observed.

Experimental procedures for preparing the animals and for stimulating and recording are given in previous papers (5, 6, 7, 11). Extracellular recordings were made with tungsten
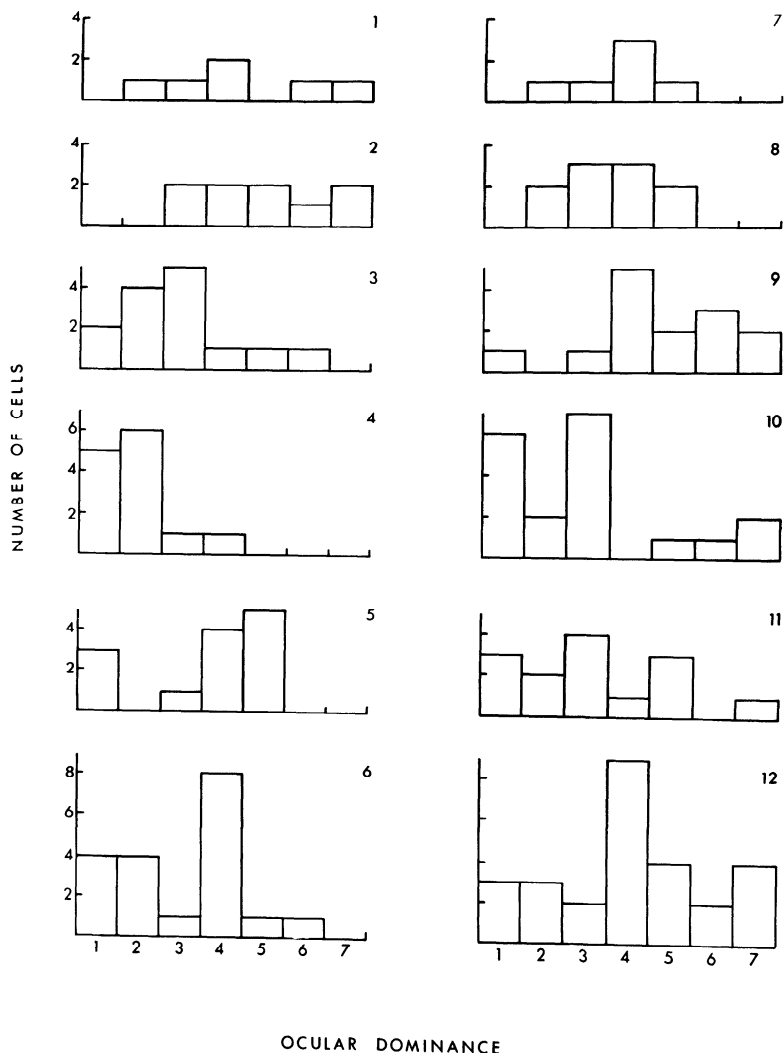
OCULAR DOMINANCE

FIG. 2. Ocular-dominance distribution of 12 separate consecutive penetrations from the same series as Fig. 1 (7).

microelectrodes in the part of the lateral gyrus receiving projections from the retinal area centralis. In one animal electroretinograms and evoked cortical potentials were recorded, the electroretinograms with chlorided silver electrodes placed on the limbus of the upper outer quadrant of the cornea, the evoked potentials with similar electrodes placed on the dura over homologous points on the two lateral gyri (Horsley-Clarke frontal plane zero, 1 mm. from the midline); the indifferent electrode was the frame of the Horsley-Clarke head holder. Responses were evoked with a Grass photostimulator (model PS 2) set at maximum intensity and held 6 in. from the animal's eyes. The eyes were stimulated separately by covering each in turn with a patch of thick black rubber.

All brains were subsequently examined histologically in order to reconstruct electrode tracks marked by electrolytic lesions.

RESULTS

Behavioral effects of monocular deprivation

Prior to some experiments in animals deprived from birth, the obstruction was removed from the right eye (by separating the lids or removing the translucent contact occluder), and an opaque occluder was placed over the normal left eye. Pupillary light reflexes were normal, and there was no nystagmus. No visual placing reactions could be obtained, though tactile placing was normal.

As an animal walked about investigating its surroundings the gait was broad-based and hesitant, and the head moved up and down in a peculiar nodding manner. The kittens bumped into large obstacles such as table legs, and even collided with walls, which they tended to follow using their whiskers as a guide. When put onto a table the animals walked off into the air, several times falling awkwardly onto the floor. When an object was moved before the eye there was no hint that it was perceived, and no attempt was made to follow it. As soon as the cover was taken off of the left eye the kitten would behave normally, jump gracefully from the table, skillfully avoiding objects in its way. We concluded that there was a profound, perhaps complete, impairment of vision in the deprived eye of these animals.

Physiological findings in kittens deprived of vision in one eye from birth

Of the 84 cortical cells recorded in kittens deprived from birth, 83 were completely uninfluenced by the deprived eye. The dominance of the normal eye in these cells was all the more striking since all but one of the five penetrations were made in the hemisphere contralateral to, and hence normally strongly favoring, the deprived eye (see Fig. 1).

The ocular-dominance histogram of a kitten whose right eye was sutured at 8 days for a $2\frac{1}{2}$-month period is shown in Fig. 3. Of the 25 cells examined in a single penetration of the left striate cortex, 20 were driven exclusively by the normal (ipsilateral) eye, and none could be influenced from the deprived (contralateral) eye. The 5 remaining cells could not be driven from either eye, and would have gone unnoticed had it not been for their spontaneous activity. The presence in most of these penetrations of a small number of unresponsive cells is worth stressing, since in normal adult cats it has been possible to drive all cells with appropriate visual stimuli (7). The electrode track of this penetration, reconstructed from the histological slides, is shown in Fig. 4. The 20 cells whose receptive fields were mapped in the normal eye all responded to line stimuli, and each strongly favored one orientation and failed to respond to a slit, edge, or dark bar placed at right angles to the optimum. The receptive fields were arranged in the usual "simple" or "complex" manner (in the sense that we have previously used these terms (7)) and varied in their orientation in a way consistent with a columnar arrangement. Unresolved background activity was present throughout the

penetration, and, like the isolated units, it was strongly influenced by the normal eye but not at all by the deprived one.

In another 3-month-old kitten whose right eye had been closed from birth, penetrations were made in homologous regions in both hemispheres. The ocular-dominance histograms of these penetrations are shown in Fig. 5, and again illustrate a failure to drive any cell from the abnormal eye. Again there were several cells that could not be driven from either eye.

The one cell in the series that could be driven from the deprived eye was recorded from a third kitten, lid-sutured from birth to an age of $2\frac{1}{2}$

FIG. 3. Ocular-dominance distribution of 25 cells recorded in the visual cortex of a $2\frac{1}{2}$-month-old kitten. Experimental procedures are indicated beneath; during the first week the eyes were not yet open; on the eighth day the lids of the right eye were sutured, and they remained closed until the time of the experiment (cross-hatched region). The left eye opened normally on the ninth day. Recordings were made from the left visual cortex, contralateral to the eye that had been closed. Five of the cells, represented by the interrupted column on the right, could not be driven from either eye. The remaining 20 were driven only from the normally exposed (left, or ipsilateral) eye, and were therefore classed as group 7.
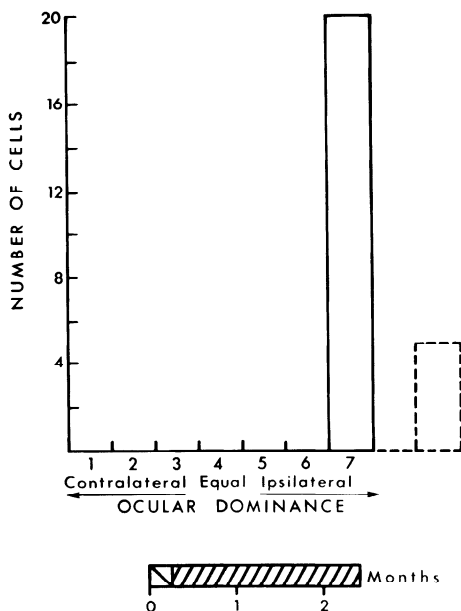
months. This cell was unusual in having abnormal fields in both eyes. Unlike other cells in the same penetration, which were normal except for their unresponsiveness to stimulation of the deprived eye, this cell had no particular orientation-preference, and the responses were more sluggish than those of the other cells. The receptive fields were in roughly corresponding parts of the two retinas.

To try to evaluate the relative importance of form deprivation as against light deprivation we raised one kitten from birth to an age of 2 months with a translucent contact occluder over the right eye. This prevented patterned retinal stimulation and reduced the general retinal illumination by about 2 log units, as opposed to 4–5 for the sutured lids. The ocular-dominance distribution of 26 cells recorded from the left hemisphere (contralateral to the occluded eye) is shown in Fig. 6. Just as in the lid-suture experiments, all cells were driven exclusively from the normal (left) eye, except for three which could not be driven at all. The cells that could be driven had normal

receptive fields. From the electrode track reconstruction shown in Fig. 7, the electrode is seen to have traversed three columns, both shifts in orientation being small, discrete, and anticlockwise. Such orderly sequences have been seen in penetrations in adult cats (8), and support our impression that in parts of the striate cortex the columns are arranged in an orderly manner. As in the first two kittens there was continuous unresolved activity throughout the penetration, briskly responsive to stimuli of the left eye, but with no hint of a response from the right.
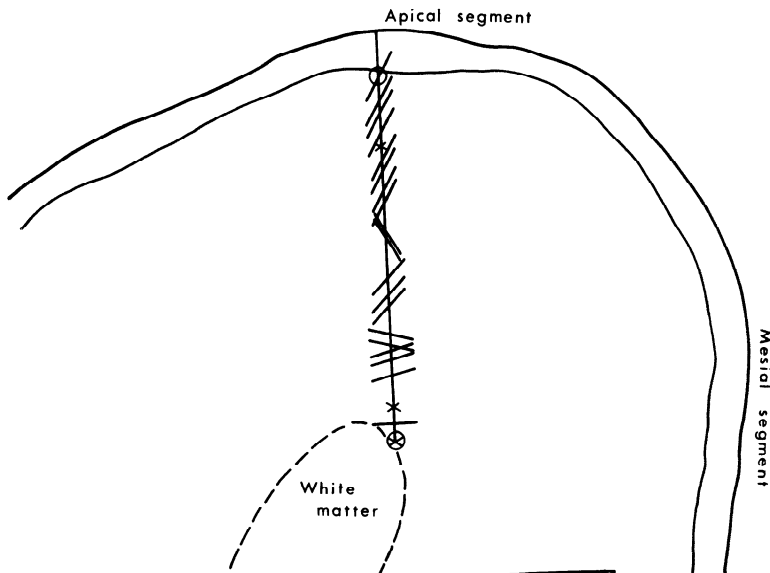

FIG. 4. Reconstruction of a microelectrode penetration through the postlateral gyrus of the left hemisphere. This 2½-month-old kitten had its right eye covered from birth by lid suture. Lines intersecting the electrode track represent cortical cells; directions of these lines indicate the receptive-field orientations. Crosses indicate cortical cells uninfluenced by light stimulation. Simultaneous recordings from two units, which occurred three times in this penetration, are each indicated by only one line or cross. A lesion was made while recording from the first unit, and another at the end of the penetration: these are marked by small circles. The ocular-dominance distribution of units recorded in this penetration is shown in Fig. 3. All fields positioned 5–6° to the left of the area centralis, slightly below the horizontal meridian. Scale, 0.5 mm.

In order to have a gross over-all impression of the retinal and cortical activity in this kitten, bilateral electroretinograms and evoked potential recordings were made (Fig. 8). The corneal electroretinograms evoked from either eye by a brief flash showed the normal a- and b-waves (Fig. 8, A and B). Successive responses were almost identical, and with stimulation rates of 1/sec. there was no sign of fatigue. (The amplitude was, if anything, greater from the deprived eye, but this could easily be due to minor differences in stimulating conditions.) This is consistent with the findings of Zetterström (12), that the electroretinogram develops normally except for a

somewhat delayed time course in kittens raised in darkness. On the other hand, cortical potentials evoked from the two eyes were far from equal. This was true for either hemisphere (Fig. 8, *C–F*). Responses from the previously occluded eye showed an initial positive component, but the later negative wave, so prominent in responses from the normal eye, was almost completely lacking. Moreover, the latency to stimulation of the deprived eye was 35–40 msec., as opposed to 25–30 msec. from the normal eye. That any cortical wave was evoked from the deprived eye indicates that some impulses originating from the eye must be relayed to the cortex, a finding that is not surprising since normal geniculate receptive fields were found in these
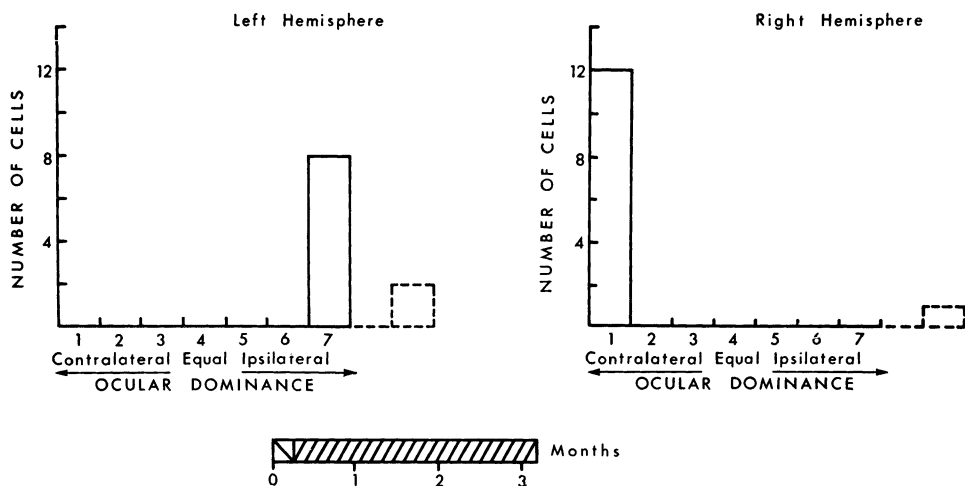

FIG. 5. Histograms showing ocular-dominance distribution of cells recorded in two penetrations, one in the left visual cortex and one in the right. A 3-month-old kitten with right eye closed by lid suture at 8 days (i.e., prior to normal eye-opening). Of a total of 23 cells, 3 were not influenced from either eye (interrupted lines). The remaining 20 could be driven only from the left (normally exposed) eye: 8 were recorded in the left hemisphere, and are therefore classed as group 7; 12 were recorded in the right hemisphere, and are classed as group 1.

animals. The striking differences in the negative phase support the single-unit findings in indicating that form deprivation and perhaps also moderate light deprivation during the first 3 months after birth can cause marked changes in the normal physiology of the striate cortex.

Deprivation of kittens with previous visual experience

In the first paper of this series (11) we showed that the geniculate atrophy resulting from 2–3 months of visual deprivation is much less if the animal has had 1 or 2 months of normal visual exposure, and that in the adult cat no detectable atrophy results from 3 months of monocular lid closure. The effects of delayed deprivation on the responses of cortical cells closely paralleled these anatomical findings. Figure 9 shows the ocular-dominance histo-
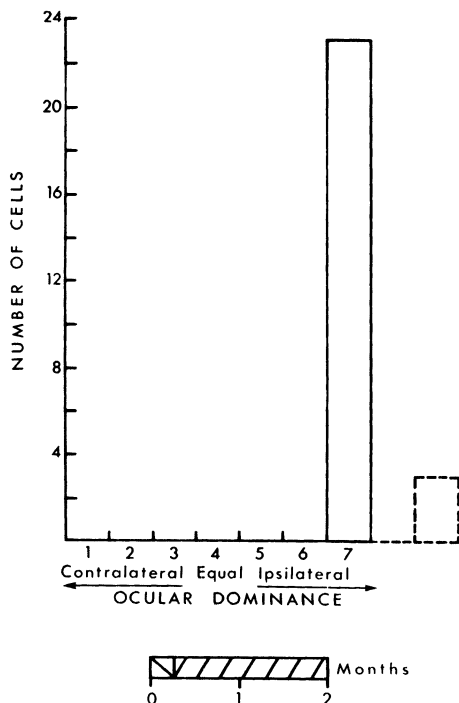
FIG. 6. Ocular-dominance distribution of 26 cells recorded in the visual cortex contralateral to the deprived eye. This 2-month-old kitten had had its right eye covered from the time of normal eye-opening by a translucent contact lens; the left eye was normally exposed. Twenty-three cells were driven by the normally exposed (left, or ipsilateral) eye, and were therefore assigned to ocular-dominance group 7. Three cells could not be activated by either eye (interrupted lines).

grams from a kitten deprived by lid closure at the age of 9 weeks, for a period of 4 months. For both hemispheres the eye that had not been occluded was again abnormally dominant. Particularly abnormal was the large number of cells driven exclusively by the normal eye. Now, however, some cells (11 of the 34) could be driven also by the deprived eye, and while the deprived eye was dominant in only 3 of these, it clearly exerted far more influence than the deprived eyes of kittens operated on at birth. The responses of all cells seemed normal, and, in contrast to experiments done in kittens deprived from birth, there were no cells that could not be driven.

A second kitten was light-deprived by lid closure at the age of 2 months for a period of only 1 month. The ocular-dominance distribution of a penetration contralateral to the deprived eye was clearly abnormal (Fig. 10), though it was less so than that of the previous kitten. Again, all cells were responsive to patterned-light stimulation and had normal receptive fields.

In one kitten the nictitating membrane was sewn across the right eye at 5 weeks, for a 3-month period. It will be recalled that this animal showed no geniculate atrophy (11). Nevertheless the ocular-dominance distribution was clearly abnormal (Fig. 11), suggesting that a decrease in effectiveness of the deprived eye in driving cortical cells is not necessarily of geniculate origin. Once again, the ocular-dominance distribution of cortical cells was less distorted than that of a kitten deprived by a translucent occluder from birth, for an even shorter time (Fig. 6).

Finally, a single penetration made in the left hemisphere of an adult cat whose right eyelids had been sewn for 3 months was completely normal. Here again it will be recalled that the lateral geniculate bodies were histologically normal (11). The ocular-dominance distribution of 26 cells (Fig.

12) shows the usual over-all dominance of the contralateral eye—in this cat the eye that had been deprived. A few months of monocular lid closure was thus not enough to cause any obvious change in cortical function; whether a longer period would have is not known.

DISCUSSION

The results of the present paper show that in kittens monocular deprivation of light and form from birth can produce both behavioral blindness
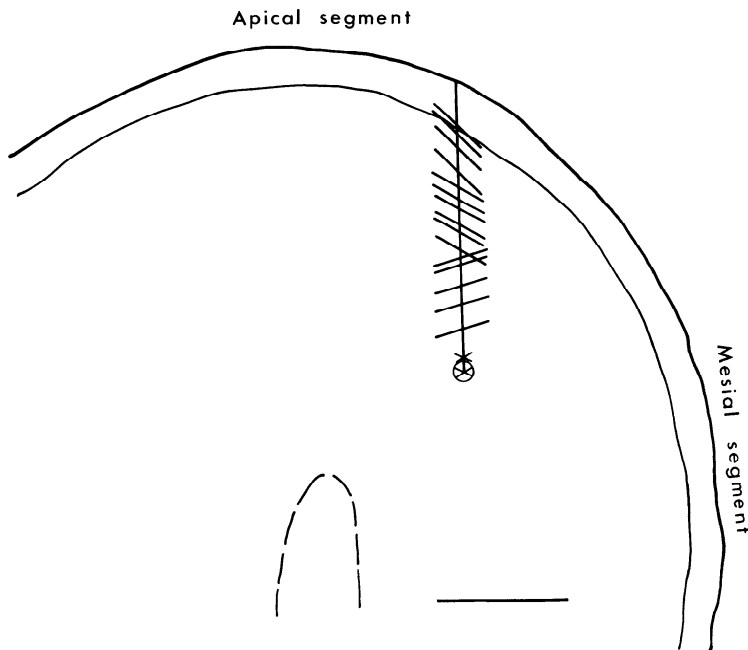
Apical segment

FIG. 7. Reconstruction of a microelectrode penetration through the left postlateral gyrus; ocular-dominance distribution of this penetration is given in Fig. 6. Conventions as in Fig. 4. Of the 17 recordings indicated, 8 were single-unit and 9 were 2-unit. Scale, 0.5 mm.

in the deprived eye and a failure of cortical cells to respond to that eye. These findings must be viewed in the light of those reported in the preceding paper (9), that the specific responses seen in cortical cells of normal adult cats are present also in newborn and very young visually inexperienced kittens. We conclude that monocular deprivation produces physiological defects in a system that was once capable of functioning.

In view of the anatomical findings described in the first paper of this series (11), the results of the present study may at first seem paradoxical: that deprivation by lid closure from birth should produce in the geniculate marked atrophy of cells with only mild physiological effects, and in the cortex just the reverse, no obvious anatomical changes but profound phys-

iological deficits. The reasons for the differences in morphological effects in the two structures are easy to imagine. Each geniculate cell receives visual input almost exclusively from one eye, and the cells receiving afferents from a given eye are aggregated in separate layers, so that any anatomical change is easy to see, the more so since it tends to contrast with the normality of
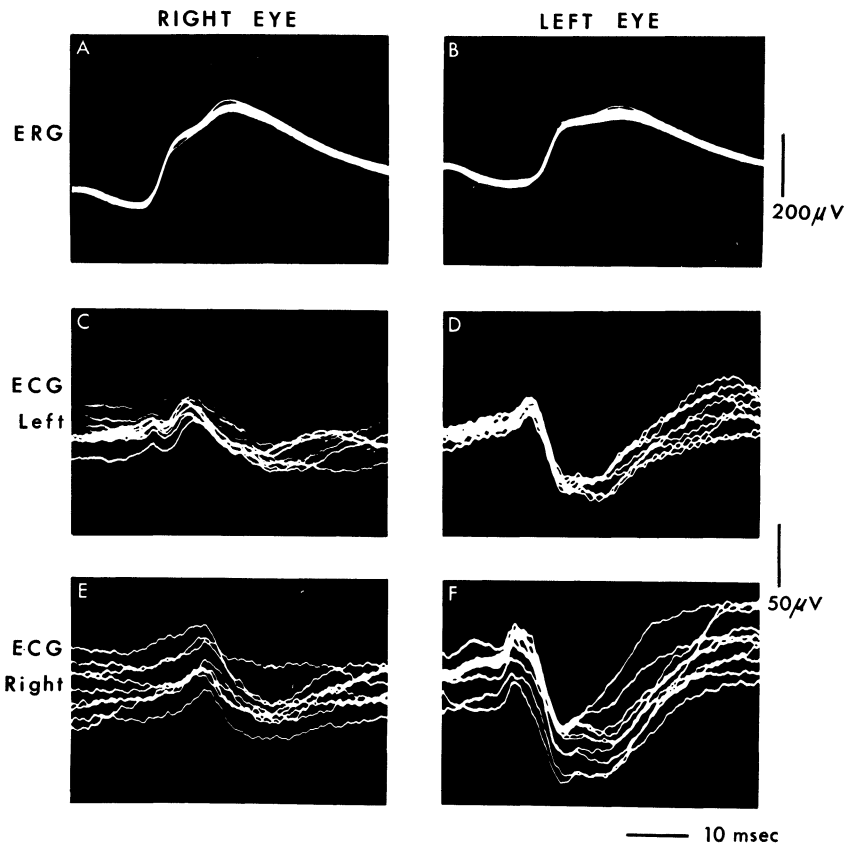

FIG. 8. Corneal electroretinograms and cortical evoked potentials (ECGs) in a 2-month-old kitten whose right eye had been covered by a translucent contact lens from the time of normal eye-opening (same kitten as in Figs. 6 and 7). A, C, E: stimulation of right (previously occluded) eye. B, D, F: stimulation of left (normal) eye. A, B: electroretinograms. C, D: cortical evoked potentials, left hemisphere. E, F: cortical evoked potentials, right hemisphere. Positive deflections upward.

the adjacent layers. The majority of cortical cells, on the other hand, have a binocular input (7), and the relatively few cells that are fed exclusively from one eye are intermixed with the others. One would therefore not expect a selective atrophy of the monocularly driven cells to stand out histologically.

It remains to account for the striking unresponsiveness of cortical cells to stimulation of the deprived eye, compared with the relative normality of geniculate responses and receptive fields. The cortical impairment was just
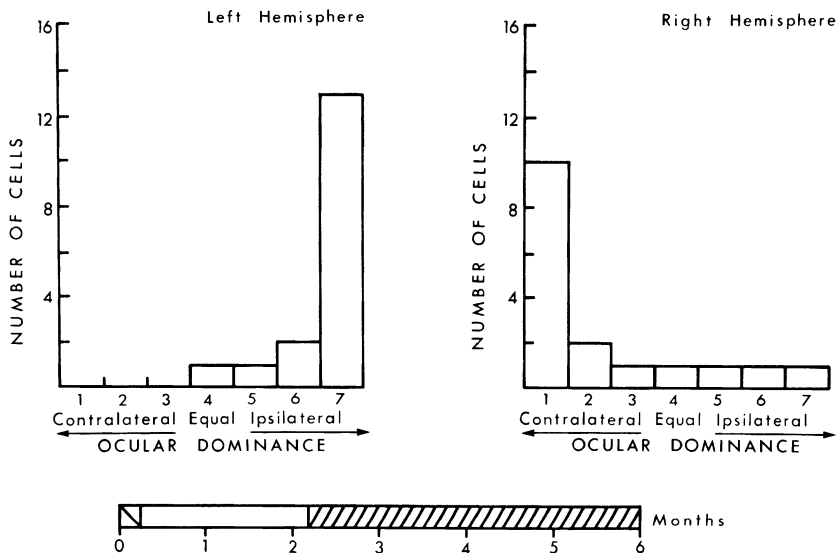
FIG. 9. Histograms of ocular-dominance distribution of 34 cells recorded in two penetrations, one in the left visual cortex and one in the right. Kitten whose right eye was closed by lid suture at 9 weeks, for a period of 4 months. Seventeen cells recorded from each hemisphere. All cells were influenced by patterned-light stimulation.

as marked after deprivation with a translucent occluder as with lid closure an especially surprising result since in the lateral geniculate the amount of light deprivation seemed to be important in determining the degree of anatomical change (11). These differences between geniculate and cortical cells can perhaps be best understood if we recall that in the normal cat cortical cells are much less responsive than geniculate cells to diffuse light (5, 6). Any light reaching the retina would thus help to keep geniculate cells active. On the other hand, most cortical cells would be practically uninfluenced regardless of the type of occlusion, and over a long period they might become unable to respond even to patterned stimulation of the deprived eye.

In discussing morphological changes in the lateral geniculate following visual deprivation (11), we pointed out that the maintained activity persisting in the retina under these conditions of lid suture or translucent occlusion is insufficient to prevent the atrophy. The same obviously applies to the present cortical recordings: the functional pathway up to the cortex was evidently not maintained by whatever activity persisted in the lateral geniculate body.

Though we have no direct way of knowing the exact site of the abnormality responsible for cortical unresponsiveness, the main defect must be central to the lateral geniculate body, since geniculate cells respond well to stimulation of the deprived eye. Nevertheless these geniculate impulses have no apparent effect on cortical cells, even though the cortical cells fire per-
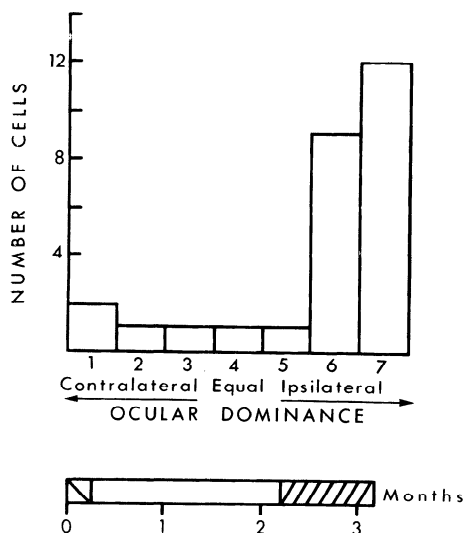
fectly well to stimulation of the normal eye. This suggests that the abnormality is in the region of the synapse between the axon terminals of geniculate cells (those receiving input from the deprived eye) and the cortical cells on which these terminals end.

Though the most abnormal feature in the physiological studies was the ocular-dominance distribution, there was another consistent difference from normal penetrations: in every experiment in kittens deprived from birth there were a few cells that could not be driven from either eye. This probably cannot be explained by the immaturity of the kittens, since in a previous study of even younger animals all of the cells could be driven by appropriate stimuli (9). A more likely explanation is that the unresponsive cells were connected exclusively with the covered eye—at least the proportion of nondriven cells was about what one would expect on that assumption. Had it not been for their maintained activity these cells would probably have gone undetected, and one wonders if the maintained firing does not reflect some other, nonvisual input.

While the site of the physiological defect may be within the cortex, as suggested above, it would probably be wrong to assume that abnormalities at the geniculate level did not also contribute to the unresponsiveness of the cortical cells. Many of the geniculate cells were atrophic; in recordings from the geniculate there was an over-all decrease in activity in the layers connected with the deprived eye, and a few fields seemed abnormal (11). The cortical evoked responses to a flash made it clear that impulses in at least some geniculate fibers associated with the deprived eye reached the cortex of both hemispheres, but the marked increase in latency suggests that this input may have been abnormal. Thus it is probable that defects at several levels, from retina to primary visual cortex, contributed to the observed physiological abnormalities. At present we have no direct way of assessing their relative importance.

The behavior of our monocularly deprived kittens, under conditions in

FIG. 11. Ocular-dominance histogram of 22 cells recorded from the left visual cortex; kitten with nictitating membrane sutured across the right eye at an age of 5 weeks, for 3 months. The left (ipsilateral, normal) eye dominated markedly, and all cells were influenced by patterned-light stimulation.
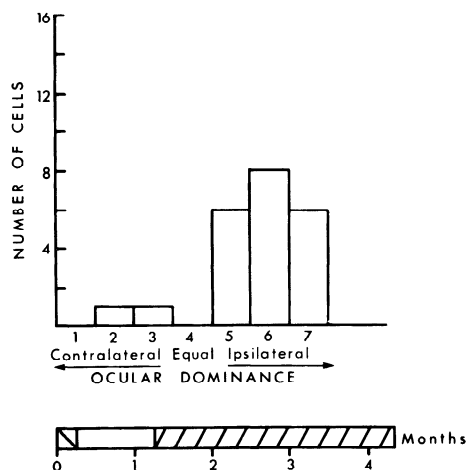
which they could use only the deprived eye, suggested the presence of gross visual deficits. This confirms the binocular-deprivation studies of other observers in several different mammalian species (2, 4, 10, 3). In monocularly deprived animals the visual pathway beyond the point of convergence of impulses from the two eyes was presumably intact, since cortical cells were actively driven from the normal eye, and since the animals were able to see with that eye. This suggests that also in bilaterally deprived animals the defect may not necessarily be at a far central level: e.g., the defect need not be in visually guided motor function, or the result of some emotional disturbance. There may indeed be such defects, but our results make it likely that abnormalities exist at a more peripheral level as well.

In interpreting the blindness resulting from raising animals in darkness it has generally been assumed that some of the neural connections necessary for vision are not present at birth, and that their development depends on visual experience early in life. Our results suggest the alternative possibility, that certain connections are intact at birth and become defective through disuse. One must, however, make one reservation: deprivation of one eye may be quite different in its morphological and physiological effects from binocular dark-raising. Conceivably if one eye is not stimulated, the fate of its projections in the central visual pathway may partly depend on whether or not the other eye is stimulated. This question of a possible competition between the eyes can only be settled experimentally, by studying animals that have been binocularly deprived of vision. For example, Baxter (1), comparing records from the visual cortex of normal and dark-raised kittens, could find no striking difference in the size or shape of the evoked potentials in the two groups. This finding contrasts sharply with the difference we saw in cortical potentials evoked from the normal, as opposed to the deprived eye (Fig. 8), and it remains to be learned whether or not the discrepancy reflects a difference in the two types of preparation.

The susceptibility of very young kittens to a few months of visual deprivation apparently does not extend to older animals, since there is a detectable lessening of effects when deprivation is begun at 2 months, and an absence of behavioral or physiological effects in adults. This is a clear demonstration of a pronounced difference between kittens and adults in susceptibility to deprivation, a difference one might have expected from the profound visual defects observed after removal of congenital cataracts in man, as opposed to the absence of blindness on removal of cataracts acquired later in life.
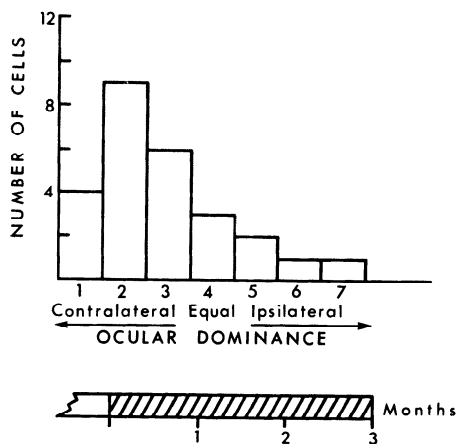

FIG. 12. Ocular-dominance distribution of 26 cells recorded in the left visual cortex of an adult cat whose right eye had been covered by lid suture for a period of 3 months. As in the normal cat, the contralateral eye—in this case the deprived eye—dominated.

SUMMARY

1. Single-unit recordings were made from striate cortex of kittens in which one eye had been deprived of vision either from birth or subsequently, and for various periods of time.

2. Kittens deprived from birth for 2–3 months showed profoundly defective vision in the deprived eye. Visual placing and following reactions were absent, and there was no hint of any ability to perceive form. Pupillary light reflexes were nevertheless normal.

3. In kittens deprived from birth, either by suturing the lids of one eye or by covering the cornea with a translucent contact occluder, the great majority of cortical cells were actively driven from the normal eye, with normal receptive fields. On the other hand, only 1 cell out of 84 was at all influenced by the deprived eye, and in that cell the receptive fields in the two eyes were abnormal. A few cells could not be driven from either eye.

4. In one 2-month-old kitten monocularly deprived with a translucent contact occluder, the corneal electroretinograms were normal in the two eyes. On flashing a light in the previously occluded eye the slow-wave potentials evoked in the visual cortex of the two hemispheres were highly abnormal, compared with responses from the normal eye.

5. One to two months of normal visual experience prior to monocular deprivation by lid suture or with a translucent occluder reduced the severity of the physiological defect, even though the ability of the deprived eye to influence cortical cells was still well below normal. On the other hand, 3 months of deprivation by lid closure in an adult cat produced no detectable physiological abnormality.

6. We conclude that monocular deprivation in kittens can lead to unresponsiveness of cortical cells to stimulation of the deprived eye, and that the defect is most severe in animals deprived from bith. The relative normality of responses in newborn kittens (9) suggests that the physiological defect in the deprived kittens represents a disruption of connections that were present at birth.

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