BINOCULAR INTERACTION IN STRIATE CORTEX OF KITTENS REARED WITH ARTIFICIAL SQUINT

DAVID H. HUBEL AND TORSTEN N. WIESEL

Neurophysiology Laboratory, Department of Pharmacology, Harvard Medical School, Boston, Massachusetts

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INTRODUCTION

Before a kitten opens its eyes, and long before the eyes are used in visual exploration, single cells of the primary visual cortex respond to natural stimulation with the same specificity as is found in the adult (5). This suggests that the anatomical connections between retina and striate cortex are for the most part innate. During the first 3 months of life the connections are highly susceptible to the effects of visual deprivation, to the extent that exclusion of all form and some light from one eye leads to a severe decline in the ability of that eye to influence cortical cells. Anatomical and physiological evidence suggests that the defect is chiefly, though not entirely, a cortical one (7–9).

The object of the present study was to influence cortical connections by some means less drastic than covering one or both eyes. We wished if possible to alter the input in such a way that there would be no question of effects on the visual pathway below the level of the striate cortex. A method was suggested by the well-known clinical observation that a child with a squint (strabismus or nonparallel visual axes) may suffer a deterioration of vision in one eye (amblyopia ex anopsia). Since the visual pathways from the two eyes are for practical purposes separate up to the level of the striate cortex, it is unlikely that in these children the defect is in the retina or geniculate. An artificial squint therefore seemed to provide a possible means of obtaining a cortical defect while sparing the retina and lateral geniculate body. Accordingly, we produced a divergent strabismus by cutting one of the extraocular muscles in each of four newborn kittens, with the plan of testing vision and recording from single cortical cells after several months to a year.

When at length each eye was tested in these kittens by observing the animal’s behavior with the other eye covered the results were disappointing: there was not the slightest suggestion of any defect in vision in either eye. This was not entirely unexpected, since with both eyes uncovered the animals had appeared to fix at times with one eye and at times with the other. At this stage there seemed to be little point in proceeding further, for there

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was no reason to doubt that the cortical recordings would be entirely normal—especially since we had not yet studied the kittens with binocular lid closure, and had no idea of the extent of interdependence of the two eyes in sustaining normal function (9). Nevertheless, we decided to record from the kittens before abandoning the project. The results from all four animals were to our surprise quite abnormal; they are presented in the first part of this paper, together with a related set of experiments on alternating monocular occlusion. An incidental finding in the course of these experiments prompted us to re-examine the problem of distribution of cortical cells by ocular dominance in the normal animal.

**METHODS**

Four kittens were operated upon at 8–10 days after birth, just at the time the eyes were beginning to open. Periorbital tissues on the right were infiltrated with a local anesthetic (Xylocaine, 2%), which was sufficient to produce general anesthesia for 10–15 min. (7). The right eyeball was retracted laterally and the conjunctiva cut at its medial scleral attachment. The medial rectus muscle was caught with a blunt hook and cut. An asymmetry in the position of the two eyeballs was usually immediately obvious, and after recovery from anesthesia all four kittens had a marked divergent squint, with the sclera visible medial to the limbus. Except for some limitation in turning the eye inward there was no obvious reduction in movements of the right eye, which were as free and active as those of the normal one.

We recorded from three of the animals, no. 1, no. 3, and no. 4, at 3 months of age, and no. 2 was studied at 1 year. Two kittens (no. 5 and no. 6) were brought up from the time of normal eye opening with an opaque contact occluder covering one eye one day and the other eye the next. These animals were studied at 10 weeks. Two normal adult cats were recorded from as controls for the studies of distribution of cells by ocular dominance. Methods of stimulating and recording are described in other papers (1–4).

**RESULTS**

**Strabismus**

*Cortical penetrations*. Seven penetrations were made in four kittens. The first kitten (no. 1) was studied at 3 months of age. When it was anesthetized and paralyzed in the usual way the eyes diverged by 21°, as measured by the projected area centralis, instead of the normal 2–3°. We concluded that with the animal awake the eyes must have diverged by about 18°. In the remaining three animals the squints estimated in this way amounted to 29°, 12°, and 23°.

To begin with, the cortical activity seemed perfectly normal. The penetrations were unusually rich, spikes from a new unit growing up to replace those of a declining one each time the electrode was advanced. The unitary discharges were seen against a background of almost continuous unresolved activity. Each cell was briskly responsive to one or the other eye and had the normal preference for a slit, edge, or dark bar in a particular orientation, which varied from one column to the next.

As more and more cells were studied it became obvious that the amount of binocular interaction was far less than normal. Most cells were driven by one eye only, some by the ipsilateral, others by the contralateral. Even more startling was the finding that there were regions of complete contralateral or
ipsilateral dominance in which one eye drove cell after cell as well as the unresolved background activity, with no trace of a response to stimulation of the other eye. As the electrode was advanced a region dominated by one eye would give way to a region dominated by the other. Mixed regions were also seen, containing cells driven from one eye and cells driven from the other, interspersed with an occasional cell driven from both eyes.

One of the two penetrations made in cat 2 is reconstructed in Fig. 1. To the left of the figure the long vertical lines separate the 7 ocular-dominance groups. The 61 cells are represented as short horizontal lines placed in the appropriate spaces at points corresponding to the electrode depths as interpolated from the two lesions, L' and L". In this penetration, except for cells 7–9 and cell 13, all of the first 33 cells were completely dominated by the contralateral eye. The electrode then entered a region of strong, almost exclusive ipsilateral dominance which extended to cell 46. After a brief transitional phase (cells 47–50), the electrode entered a third region, from 51 to the end, which showed complete contralateral domination.

Similar results were seen in the other six penetrations. In these, however, the regions of mixed dominance were more prominent. Figure 2 shows reconstructions of two penetrations made in cat 3, at an age of 3 months. In the penetrations made in the left hemisphere (Fig. 2A) an area of mixed dominance occurred between cells 22 and 28, and another brief episode between cells 40 and 42. The penetration in the left hemisphere (Fig. 2B) showed even more extensive mixed zones. After an initial region of ipsilateral domination (cells 50–59) the first mixed region extended from 60 to about 91. There was then another ipsilaterally dominant area from about 91 to 106, followed by a second mixed area. In the first mixed zone there were several examples of dual-unit recordings (indicated by dots) in which one cell was group 1 and the other group 7. Cell 64 in group 1 was recorded with 65 and 66, both in group 7. In the single penetration made in cat 4 at 3 months (Fig. 3) the middle groups (no. 3–5) were especially poorly represented. Here the mixed areas consisted almost entirely of cells of group 1 intermixed with cells of group 7 (cells 49–67; cells 90–95). It thus appears that in these kittens the cortex is subdivided into regions of three types, one containing contralaterally dominated cells, the second containing ipsilaterally dominated cells, and the third containing cells of both types as well as a few binocularly driven cells. As shown in a later section of this paper, the three types of region represent an exaggeration of regional variations in ocular dominance that occur in normal animals.

Ocular-dominance distribution. The ocular-dominance distributions of cortical cells recorded in four of the penetrations in animals with strabismus are given in Figs. 1–3 below each track reconstruction. In all these penetrations the distributions were abnormal, with the extreme groups (no. 1 and 7) well represented and the inner ones (no. 3–5) poorly represented. To be sure of this result we recorded from many cells in each animal, 106 in one penetration in cat 4 (Fig. 3) and 116 in two penetrations in cat 3 (Fig. 2). In all
seven penetrations deviations from normal were large, but there was some variation; the two least abnormal penetrations were seen in cat 1, summarized in Fig. 4. Even in this experiment 50 of 81 cells, or roughly 60%, were driven from one eye only, compared with about 20% in the normal cat.

The distribution of the 384 cells from all four kittens is given in Fig. 5B. This histogram is to be compared with the distribution of 223 cells previously obtained in 17 penetrations in the normal adult, shown in Fig. 5A. Of the 384 cells recorded from animals with squint, 302, or 79%, were monocularly driven, as opposed to 20% in the normal.

That the difference represented by Fig. 5, A and B, has nothing to do with age differences is clear from a previous study in which normal kittens were found to have an ocular-dominance distribution similar to that of adult cats (ref. 5, Fig. 2). Finally, another measure of ocular-dominance distribution of an entirely different sort is described below and shown in Fig. 9B.

Anatomical findings. Histological sections of the lateral geniculate and the striate cortex showed no sign of any abnormality. In one animal 50 cells from the dorsal layer of the geniculate (layer A) were measured on each side, and no significant difference was found between the two sides.

Ocular-dominance distribution in kittens raised with alternating monocular occlusion

In the experiments just described, the strabismus kept the two eyes from working together without cutting down the input to either eye. It seemed worthwhile to ask whether a similar cortical defect would result if one were to stimulate the two eyes alternately, blocking light from entering one whenever the other was in use, and thus keeping the eyes from working together without introducing the possibility of antagonistic interaction between them. We therefore placed an opaque contact occluder over one eye one day, and the other eye the next, alternating eyes each day from the time of normal eye opening up to an age of 10 weeks. At that point the animals seemed to see perfectly well with either eye, and both eyes when uncovered moved together without obvious strabismus.
FIG. 2A. Reconstruction of penetration through right hemisphere of cat 3, a 3-month-old animal with divergent strabismus from birth. To right of figure is a photomicrograph of a Nissl-stained coronal section through the postlateral gyrus.
Fig. 2B. Reconstruction of penetration through left hemisphere of cat 3. Conventions as in Fig. 1.
Fig. 3. Reconstruction of penetration in cat 4, through right hemisphere. Kitten, age 3 months, strabismus from 3 days. Conventions as in Fig. 1.
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Fig. 4. Ocular-dominance distribution of 81 cells recorded in two penetrations, one in each hemisphere. Kitten 1, age 3 months, right medial rectus cut at 8 days. (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.)

Fig. 5. A: ocular dominance of 223 cells recorded from a series of normal adult cats (3). B: ocular dominance of 384 cells recorded from all four strabismus experiments.
Three penetrations were made in these animals and are reconstructed in Fig. 6. The ocular-dominance histograms, shown below in the figure, are even more abnormal than those of the squint animals, with 176 of 194 cells (91%) driven by one eye only. The track reconstructions again show strong evidence for spatial aggregation of cells according to ocular dominance. We conclude that the results of the strabismus experiments do not depend on the fact that the two eyes were both open at the same time.

Spatial distribution of cells according to ocular dominance in normal cats

We were naturally interested in whether the division of striate cortex into areas according to ocular dominance was an abnormal state produced by squint, or alternating occlusion, or whether it also existed in the normal animal and was merely made more obvious by the exaggerated ocular dominance of the cells. Our previous studies of normal striate cortex had suggested that there was some tendency to aggregation of cells according to ocular dominance (ref. 3, p. 140 and text-Fig. 13). To help settle this problem we made four penetrations in two normal adult cats and reconstructed the results in Figs. 7 and 8. Here there indeed seemed to be a subdivision of cortex according to eye dominance. In penetration 1 of Fig. 7, the first 13 cells favored the ipsilateral eye; this was followed by a small area of mixed dominance (cells 14-17), and then the contralateral eye prevailed to the end (cell 25). In penetration 2, the first few cells favored the ipsilateral eye and from then on there was a mixture. In Fig. 8, the penetration in the right hemisphere was first predominantly ipsilateral in emphasis, and at the end contralateral (cells 24-26), cells 3 and 4 giving the only hint of intermixing in the early part. Penetration 2, made in the left hemisphere, was largely mixed.

This tendency for cortical cells in the normal cat to be segregated according to ocular dominance complicates the assessment of cortex as normal or abnormal in animals with strabismus. But while the ocular-dominance distributions for the individual penetrations in the squint and alternating monocular occlusions vary to some extent (Figs. 1-4; Fig. 6), the marked preponderance of cells in the two end groups (no. 1 and 7) is common to all of them. For the normal cat, on the other hand, an idea of the variation from one penetration to the next can be obtained from the histograms of 12 individual penetrations, shown in a previous paper (ref. 8, Fig. 2) or from the histograms of the 4 normal penetrations in Figs. 7 and 8. None of these normal penetrations, and no others we have made, have shown ocular-dominance distributions with anything like the shape of those from kittens with strabismus or alternating monocular occlusion.

In normal and abnormal animals the cortical subdivisions defined by ocular dominance seem to be quite independent of the columns defined by receptive-field orientation. Within an orientation column there may be more than one region defined by ocular dominance (Fig. 1, cells 33-40 and cells 50-58; Fig. 2A, cells 21-28; Fig. 2B, cells 77-99) and, conversely, an ocular-
FIG. 6. Schematic reconstruction of three penetrations in the striate cortex of two 10-week-old kittens (no. 5 and no. 6) raised from the time of normal eye opening with an opaque contact occluder covering one eye one day, and the other eye the next. Each penetration extended into cortical gray matter for about 1.5 mm. Conventions as in Fig. 1.
Fig. 7. Two penetrations in the right striate cortex (postlateral gyrus) of a normal adult cat. Conventions as in Fig. 1.
Fig. 8. One penetration in each hemisphere through striate cortex (post-lateral gyrus) of a normal adult cat. Conventions as in Fig. 1.
dominance region may contain a large number of receptive-field orientation columns (see especially Figs. 1 and 3).

To study the interrelationship of the two kinds of aggregations we re-examined a series of experiments made originally for the purpose of mapping receptive-field orientation columns (4). Each experiment consisted of a number of superficial cortical penetrations. A typical map is redrawn in Fig. 9A

![Diagram](image)

**Fig. 9. A:** normal adult cat. Map showing receptive-field orientations and ocular dominance of first cells encountered near the surface, in 31 penetrations. The entire map covers a region of the right striate cortex measuring about 1 x 3 mm. Interrupted lines separate regions of relatively constant receptive-field orientation, partly outlining 3 columns. The numbers refer to ocular-dominance groups. Continuous lines separate areas of strong ipsilateral dominance from areas of mixed or contralateral dominance (redrawn from ref. 4, text-Fig. 3 and Plate 1). **B:** ocular-dominance distribution of the first units recorded in 167 superficial penetrations made in five normal adult cats.

(Ref. 4, text-Fig. 3). The columnar regions of constant receptive-field orientation are roughly outlined as before by interrupted lines. Superimposed upon and cutting across these lines, the continuously drawn contours outline areas of marked ipsilateral eye dominance. Presumably, in an extensive enough mapping one might also outline regions of contralateral and mixed dominance, but in this experiment there were too few points and they were too far apart for comfort. The map does, however, reinforce the impression that the system of parcellation by ocular dominance is independent of, and cuts across, that of the orientation columns so that the same surface of cortex is simultaneously subdivided in two different ways.
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The ocular-dominance distribution of cells in normal cat cortex has been estimated from a relatively small number of deep penetrations (3, 5, and 8). To obtain an entirely different measure of this distribution we tabulated the ocular dominance of the first units recorded, in all 167 superficial penetrations made in the mapping experiments (4). The resulting histogram, given in Fig. 9B, agrees reasonably well with those obtained from deep cortical penetrations (Fig. 5A; and ref. 5, Fig. 2).

We are still not absolutely clear about the shape and size of the regions of one-eye or mixed eye dominance. Penetrations like that of Fig. 1, together with the surface-mapping experiments described above, suggest that the regions may be large, extending along the cortex for some millimeters. Deep microelectrode penetrations (ref. 3, text-Fig. 13) indicate that they can, at least sometimes, extend from surface to white matter. Thus it seems likely that the regions are columnar, though evidence that the walls separating them are perpendicular to the cortical layers is still lacking.

The spatial segregation of cells by ocular dominance may have a relatively simple anatomical explanation. Geniculate axons as they enter the cortex may be grouped into small bundles, all of the fibers in a given bundle coming from the same geniculate layer and consequently all connected with the same eye. These fiber groups, entering the cortex and fanning out, could establish certain regions in which one or the other eye strongly dominated, and other regions of mixed dominance in which groups of the two types intermingled. The ocular dominance of a given cell in the cortex would then be determined by the relative regional concentration of the two types of axon.

DISCUSSION

The results presented here show that it is possible to produce abnormalities in neural functioning by alterations in sensory input that are relatively subtle, compared with light or form deprivation or cutting an afferent nerve. The neural abnormality was a severe decline in the number of cells that could be driven by both eyes in area 17 of the cortex. In the case of strabismus the sensory impairment was simply a misalignment of the two eyes; on the average, both retinas must have received the same sensory input. Thus it seems fairly certain that the squint produced no impairment of traffic along the two paths emanating from the separate eyes, up to the point of their convergence in the cortex. The changes in cortical function must therefore have been produced by the abnormal relationship between signals in the two paths. The nature of this faulty relationship can best be seen by considering what is known about binocular convergence at the cortical cell.

In the normal cat, the two receptive fields of a single cortical cell are similar in arrangement and occupy corresponding retinal positions in the two eyes. If the eyes fix normally on a flat object, the two retinal images, falling on corresponding parts of the retinas, will affect the cell in the same qualitative way by either eye, exciting it through both or inhibiting it through both. The amount of influence, excitatory or inhibitory, may differ
for the two eyes, and when it does the direction and degree of the difference decides which ocular-dominance group the cell occupies in our rough and arbitrary scheme of classification. There is indirect evidence (3; and ref. 9, DISCUSSION) to suggest that impulses originating from the two eyes converge mainly upon simple cells in the cortex. If that is so, any changes in ocular dominance in a complex cell would merely reflect, in a passive way, interaction effects at the simple cell.

Now suppose that the retinas are exposed to an ordinary, real-life visual stimulus, and consider the response of a cell in group 2. In the normal animal the images fall on corresponding parts of the retinas (neglecting parallax) and the response will be determined mainly by impulses coming in from the dominant (here, the contralateral) eye, though there will be some help from the nondominant one. In the animal with strabismus this relationship is completely changed: the cell will tend to follow the commands of the dominant eye, and whether the other eye helps, hinders, or has no effect at all will be more or less a matter of chance, depending on the make-up of the stimulus and the amount of squint. It must be this lack of synergy between the two afferent paths that somehow, over a period of time, gives rise to the changes in over-all ocular-dominance distribution.

The new ocular-dominance distribution could result from a simple dropping out of binocularly driven cells. This seems unlikely experimentally, because of the wealth of responsive cells and absence of unresponsive cells, and because a dropping out of binocularly driven cells would leave short alternating sequences of group 1 and group 7 cells, rather than the very long sequences actually observed. A far more likely possibility is that the lack of synergy in the two paths causes the ocular dominance of cells to change, with an over-all increase in the number of group 1 and group 7 cells at the expense of the others. This would happen if there were a decrease in the effectiveness of the nondominant eye. There might also be an absolute increase in the effectiveness of the dominant eye, but that would be difficult to detect because of differences in the responsiveness from one cell to the next. In any case, given the initial normal tendency for grouping of cells by ocular dominance, a shift in ocular dominance, cells of groups 2 and 3 becoming group 1 and 5 and 6 becoming group 7, would explain very well the long sequences of cells of groups 1 or 7.

One may ask whether in these experiments it is the mere absence of synchronous visual input that produces the result, or whether it is the presence of asynchronous inputs. It seems reasonably clear that absence of synchrony by itself is not enough, since binocular occlusion in the early months of life did not give the marked loss of binocular driving found in strabismus. On the other hand, the alternating occlusion experiments gave substantially the same result as the squint experiments, showing that the result does not depend on simultaneous nonsynchronous activation of the two eyes. What does seem necessary to produce the result is absence of
synchrony, and activation of at least one of the two afferent pathways at any one time.

Regardless of detailed mechanisms, the results of this paper are interpreted as suggesting that, in some systems at least, the maintenance of a synapse depends not only on the amount of incoming impulse activity but also on a normal interrelationship between activity in the different afferents. That two sets of synapses on the same cell can be interdependent is also suggested by a previous study comparing the effects of monocular and binocular eye closure (8, 9). In attempting to imagine how an organism can be influenced by experience—to account in synaptic terms for learning, imprinting, and other phenomena that demand neural plasticity—the possibilities would seem to be greatly increased by adding, to the ordinary use-disuse concept, that of the interdependence of different synapses on a single cell.

The plasticity demonstrated in the occlusion and strabismus experiments has two obvious limitations. First, it is probably confined to the early months of life. This was clearly shown for the occlusion experiments, inasmuch as three months of deprivation produced no changes in an adult cat (7, 8) and even several months, deprivation starting at 2–3 months was less severe in its effects than deprivation for a similar period from birth. Similarly, the failure to obtain full recovery on opening the eyes after 3 months' deprivation from birth may be a matter of age rather than of irreversibility as such (10). The effects of cutting an eye muscle in older cats have not been studied, but one would probably find a similar age dependence, in view of the common clinical experience that strabismus acquired in the adult produces effects on mechanisms for fusion of images or for stereoscopic depth perception. A second limitation concerns the pathological nature of the changes. In all of the experiments of this series, both deprivation and strabismus, normal, fully formed connections were rendered abnormal by distorting the sensory input. The next step would be to look at more central parts of the visual path for changes in connections resulting from normal experience. The changes could involve the development of entirely new connections, or simply a modification—a relative strengthening or weakening—of innately determined ones, as in the present experiments. Here also the influence of age on plasticity would obviously be of interest.

In all of the experiments of this series there has been a certain correspondence between the sensory deprivation employed and the nature of the defect produced. Monocular form deprivation with only minor light deprivation led to an unresponsiveness of cortical cells to stimulation of the deprived eye, with very minor anatomical changes in the lateral geniculate body; whereas monocular deprivation of both form and light gave similar cortical unresponsiveness plus marked morphological changes in the lateral geniculate. This fits very well with the reactions of cells at the two levels to diffuse light—the virtual unresponsiveness of cortical cells and the brisk responses of most geniculate cells. With strabismus the result was similar:
here the defect was precisely in the area of binocular interaction, with other
cortical functions apparently intact. All of this makes one wonder whether
more subtle types of deprivation—an animal brought up in isolation or a
bird kept from hearing the call of another bird of the same species—may not
likewise exert their ill effects through the deterioration of complex central
pathways that either were not used or else were used inappropriately.

Finally, the results of these studies may have some bearing on the
problem of strabismus in man. It is recognized that a squint in a child must
be corrected in the first few years of life if capability of using both eyes in
binocular vision is to be retained. This correlates well with our finding that
in cats a mechanical misalignment of the two eyes early in life produces a
deterioration in cortical connections. We have made no attempt at testing
the reversibility of the damage by straightening the eyes surgically, but our
failure to produce any significant recovery in the occlusion experiments
(10) would make us pessimistic. Furthermore, given even a normal mecha-
nical apparatus for aligning the eyes, perfect binocular fixation presumably
depends also upon a normal set of neural connections in the visual pathway,
possibly the very connections concerned with binocular interaction that are
lost in these experiments. In that case even a perfect mechanical repair
would not guarantee the realignment necessary to promote recovery of
binocular vision.

**Summary**

In four kittens the right medial rectus was severed at about the time of
normal eye opening, producing an obvious divergent squint. The animals
were raised under normal conditions for periods of 3 months to 1 year. When
the two eyes were then tested separately no behavioral visual defects were
seen. Recordings from the striate cortex were normal, except for a marked
decrease in the proportion of binocularly driven cells: instead of about 80%,
only 20% could be influenced from the two eyes. The cortex appeared nor-
mal microscopically. In a given penetration there was a marked tendency for
cells driven from a particular eye to occur in long uninterrupted sequences.
These results suggest that the strabismus caused cells to shift in their ocular
dominance, a given cell coming to favor more and more the eye that domi-
nated it at birth, ultimately losing all connections with the nondominant
eye. We conclude that a lack of synergy in the input from the two eyes is
sufficient to cause a profound disruption in the connections that subserve
binocular interaction.

In two kittens an opaque contact occluder was placed over one eye one
day and the other eye the next, alternating eyes each day from shortly after
birth to an age of 10 weeks. This kept the eyes from working together with-
out introducing the possibility of antagonistic interaction between them.
Vision in either eye seemed normal. Penetrations in the striate cortex gave
results similar to those obtained in squint animals; if anything, the shift in
ocellar dominance was more extreme, 91% of cells being driven by only one eye. Again cells were spatially aggregated according to ocular dominance.

Recordings from normal adult cats indicate that besides being grouped according to receptive-field orientation, cells in the striate cortex are grouped by ocular dominance into regions of ipsilateral, contralateral, and mixed dominance. The exaggeration of eye dominance of individual cells, in animals raised with squint or alternating monocular occlusion, produces an accentuation of these cortical subdivisions.

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REFERENCES