INTRODUCTION

The importance of normal sensory stimulation in the development and maintenance of the nervous system is now generally recognized. In the visual system this problem has usually been approached by examining the effects of sensory deprivation on structure and behavior (see reviews by Hebb (12) and Riesen (28)). An obvious way of extending this work would be to examine electrophysiologically the functional effects of visual deprivation, but such experiments require some knowledge of normal function. During the last 10 years single-cell responses have been examined and receptive-field arrangements compared at several levels in the cat's visual pathway: in the retina (21), the lateral geniculate body (18), and the visual cortex (17, 19). This information provides the necessary background for a study of the immature and the stimulus-deprived visual system.

The results of a physiological and anatomical study of the visual pathways in normal and visually deprived kittens will be presented in a series of three papers. In the present paper we describe single-unit recordings in the optic tract and lateral geniculate body of kittens in which one eye had been deprived of vision, and an anatomical examination of the visual pathways in these animals. The second paper (20) will describe single-unit recordings in the striate cortex of newborn kittens. The final paper (32) will deal with responses of cells in the visual cortex of visually deprived animals.

METHODS

Nine kittens and one adult cat were used in studying the morphological effects of 1 4 months of monocular visual deprivation. In two of the kittens recordings were made from the lateral geniculate body and the optic tract. In addition, three kittens were used in a study of the development of lateral geniculate cells. Table 1 summarizes the procedures carried out in the experimental animals.

Deprivation procedures. The most common method of deprivation was to suture together the lids of the right eye. Under local or general anesthesia the lid margins were trimmed and then sutured end to end. The reduction in intensity of a light beam passing through the eyelid, measured with a photoelectric cell, was found to vary between 4 and 5

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log units, depending on fur color. The lid-sutured eyes were thus completely deprived of form stimulation, and also, to a large extent, of stimulation by diffuse light.

In two kittens a translucent plastic contact lens was placed over the right eye. This type of cover allowed more light to enter the eye but still excluded pattern stimulation. In a third kitten, to overcome this difficulty, the nictitating membrane was brought across the cornea of the right eye, and the edge of the membrane was trimmed and sutured to the abraded conjunctiva of the upper lid. These two types of covering were found by direct measurement to reduce retinal illumination by only about 1-2 log units.

Physiological methods. Single-unit recordings were made from the optic tract and lateral geniculate bodies in two kittens. Both animals were 3 months old and had had the right eyelid sutured before the time of normal eye-opening. The recordings were done under general anesthesia. Procedures for preparing the animals and details of methods for stimulating and recording have been described elsewhere (15, 18, 19). In both kittens, after separating the previously closed lids, we observed that the cornea was moist and clear; the fundus, including the optic disc, appeared normal, and the direct and consensual light reflexes were normally active.

Single units were recorded in the lateral geniculate body with tungsten microelectrodes (14), and electrode positions were marked by electrolytic lesions (15). Receptive fields were mapped by projecting small spots of white light on a wide screen which the animal faced from a distance of 1.5 m. The screen was lit diffusely by a moderately bright background light ($-1$ to $+1$ log cd/m$^2$). Intensities of stimulus and background lights were adjusted so that the difference between the two did not exceed 2 log units.

Anatomical methods. After the acute experiments all animals were perfused with normal saline followed by 10% formalin. The eyes were also perfused separately with formalin according to a method described by Polyak (25). Brains and eyes were embedded in celloidin; the brains were serially sectioned at 26 $\mu$ and the sections stained with cresyl violet; the retina was sectioned at 10 $\mu$ and the sections stained alternately with hematoxylin and eosin and with cresyl violet.

A quantitative method for measuring areas of the entire cell, the nucleus, and the nucleolus was adapted from the method of Matthews, Cowan, and Powell (23). Cell outlines were traced on 1-mm. graph paper after a linear magnification of 1,000. The number of square millimeters within a particular outline gave a direct measure of the projected area in square microns. Only cells showing a distinct nucleus and nucleolus were traced. Measurements in the two dorsal layers (A and A$'$) did not give rise to any difficulties since these layers are clearly defined, relatively thick, and rich in cells. On the other hand, the ventral layer (B) was comparatively thin in coronal section. Here special care was taken to avoid the large intralaminar cells since they are considered by some to have binocular input (10). In the two dorsal layers cell density was estimated by counting the number of cells within a given area, using a Whipple net micrometer disc.

### Table 1. Experimental animals

<table>
<thead>
<tr>
<th>Procedure, Right Eye</th>
<th>No. of Animals</th>
<th>Age at Onset of Deprivation, weeks</th>
<th>Duration of Deprivation, weeks</th>
<th>Age at Time of Experiment, weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lid closure</td>
<td>4</td>
<td>Just before normal eye opening</td>
<td>9-13</td>
<td>10-14</td>
</tr>
<tr>
<td>Lid closure</td>
<td>1</td>
<td>9</td>
<td>16</td>
<td>25</td>
</tr>
<tr>
<td>Lid closure</td>
<td>1</td>
<td>9</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>Plastic translucent occluder</td>
<td>2</td>
<td>Adult</td>
<td>12</td>
<td>Adult</td>
</tr>
<tr>
<td>Occlusion with nictitating membrane</td>
<td>1</td>
<td>Just before normal eye-opening</td>
<td>8-10</td>
<td>9-11</td>
</tr>
</tbody>
</table>
RESULTS

Single-unit recordings

Single units were studied in the lateral geniculate bodies of two kittens monocularly deprived of light and form by lid suture. The kittens were 3 months old and had had the right eyelids sutured together just before the time of normal eye-opening. Records were made from the left lateral geniculate body, and detailed receptive fields were mapped for 34 geniculate cells. Twenty of the cells were recorded in layers receiving input from the deprived eye, 19 in the dorsal layer (A) and one in the ventral layer (B); the remaining 14 were recorded in the middle layer (A1), whose input was from the normal eye.

All cells, whether they received their input from the normal or from the deprived eye, had the usual concentric receptive-field arrangement consisting of an excitatory or inhibitory center and a peripheral region of the opposite type (18). With a few exceptions, which will be described below, the cells had field centers of normal size. They responded well to stimuli restricted to the field centers, and if the antagonistic periphery was included in the area stimulated by using large spots or diffuse light, there was a marked decrease in the response. From this it is clear that cells can have normal receptive-field arrangements in immature (3-month-old) kittens, and that patterned-light stimulation is not required for the development of the necessary connections.

Records were also made from seven optic-tract fibers, which were recognized by their firing pattern, response characteristics, and spike shape (16). Two of the fibers were activated from the normal eye and five from the stimulus-deprived eye. All fibers had normal concentric receptive-field arrangements, with "on" centers and "off" peripheries, or the reverse (21). The field centers were well defined and of normal size, and the units responded strongly when the centers alone were stimulated. The normality of the receptive fields of at least some retinal ganglion cells in these kittens is hardly surprising since, as already described, lateral geniculate cells with normal fields were found in the same animals.

In previous studies (18) we showed that geniculate cells were very similar to retinal ganglion cells in their receptive-field arrangements, the main difference being an increase in the peripheral suppression at the geniculate level. This difference was demonstrated directly in simultaneous recordings from a geniculate cell and its main excitatory afferent: the probability that the excitatory afferent would trigger the geniculate cell varied in such a way as to render diffuse light less effective at the geniculate level. In the present study simultaneous recordings were made of a geniculate cell and its main excitatory afferent on three occasions; in two of these the driving was from the normal eye and in the third it was from the deprived eye. In all three cases there was a clear increase in the peripheral suppression for the geniculate cells, indicating that the lateral geniculate body in a 3-month-old kitten, like that of the adult cat, is not merely a relay station, but modifies the input in
an important way. Moreover, geniculate cells possess this power of modifica-
tion even in animals that have not previously been exposed to patterned
light.

Although the majority of the geniculate cells receiving input from the
previously closed eye appeared normal, there were a few exceptions. Four
cells, all in the dorsal (A) layer, were strikingly sluggish in their responses to
light stimulation. These had field centers four to six times the diameter of
neighboring cells—indeed, the centers were larger than any we have seen in
the lateral geniculate body of the normal adult cat—and they showed less
than normal peripheral suppression. Furthermore, in the two kittens in
which recordings were made from the geniculate, the over-all activity
seemed to be less in the visually deprived layers than in the normal ones;
fewer units could be isolated, and the unresolved background activity was
unusually sparse. Despite these signs of abnormal function, it was our general
impression from these recordings that the geniculate physiology was rela-
tively normal, a somewhat surprising finding in view of the striking anatomi-
cal changes to be described below.

Morphological changes in the lateral geniculate body
induced by light and form deprivation

*Kittens monocularly deprived from birth by lid closure.* In the two kittens
described above and in two others also deprived for about 3 months the
lateral geniculate bodies showed striking histological changes. In all four
animals there was a profound atrophy in the geniculate layers receiving
input from the covered eye. This is illustrated for one kitten in Fig. 1:
coronal sections of the left lateral geniculate body, contralateral to the
closed eye, show atrophy in the dorsal (A) and ventral (B) layers (Fig. 1A);
in the right lateral geniculate body there is atrophy in the middle (A1)
layer (Fig. 1B). These changes were observed throughout the entire extent
of the lateral geniculate bodies.

The abnormal layers stood out by virtue of several morphological
changes (Figs. 1–3). Throughout their entire extent these layers were thinner
than normal and appeared somewhat collapsed. The collapse was in part
produced by a general reduction in cell size; a lack of Nissl substance gave
most cells a pale, often ghost-like appearance, though a few normally stained,
healthy-looking cells were interspersed among the mass of atrophic cells.
Also contributing to the thinness of these layers was a reduction in the vol-
ume of the apparently homogeneous space between cell bodies, as a result of
which the cells seemed to be more thickly packed. There was no obvious
glial infiltration.

Cross-sectional areas of lateral-geniculate cell bodies, nuclei, and nucleoli
were estimated for 50 cells in each layer. As shown in Table 2, cell bodies and
nuclei were much smaller for layers receiving their input from the closed eye.
Comparison of corresponding layers on the two sides showed a reduction of
about 40% in mean cell area for the left dorsal (A) layer, and a similar reduction for the right middle (A₁) layer. The reduction in the left ventral (B) layer was about 25%. These differences are highly significant \((P < 0.001)\). Comparable shrinkages were found for the nuclei and nucleoli.

The distribution of cell areas in the different layers is shown in the histograms of Fig. 4. In the normal layers the variation in cell size was relatively large, ranging from about 180 to 600 \(\mu\). In contrast, in the layers receiving input from the light-deprived eye, cell areas ranged from 150 to 300 \(\mu\), reflecting a marked reduction in the number of large cells. In all three abnormal layers the proportion of cells with small areas was greatly in-

<table>
<thead>
<tr>
<th>Layer</th>
<th>Area of Cells</th>
<th></th>
<th>Area of Nuclei</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Left, (\mu^2)</td>
<td>Right, (\mu^2)</td>
<td>Shrinkage, (%)</td>
<td>Left, (\mu^2)</td>
</tr>
<tr>
<td>A</td>
<td>198 ± 7</td>
<td>365 ± 20</td>
<td>46*</td>
<td>88 ± 4</td>
</tr>
<tr>
<td>A₁</td>
<td>340 ± 15</td>
<td>201 ± 6</td>
<td>41</td>
<td>136 ± 6</td>
</tr>
<tr>
<td>B</td>
<td>236 ± 7</td>
<td>303 ± 14</td>
<td>22</td>
<td>106 ± 3</td>
</tr>
</tbody>
</table>

All \(t\) values exceed 4.0 \((P < 0.001)\).

* Sample calculation: \[
\frac{\text{difference of means}}{\text{mean of normal}} \times 100 = \frac{365 - 198}{365} \times 100 = 46\%.
\]

increased. Values given here for upper limit and average cell areas in normal layers varied to some extent from one cat to the next, probably because of differences in details of fixation and histological processing. These variations emphasize the advantage of using monocular deprivation, since here the normal layers furnish the control in each animal. That cells in the “normal” layers were not hypertrophied was checked by measuring cell areas in a 3-month-old kitten raised with both eyes open.

The determination of cell areas, while useful in comparing the degree of atrophy produced by different schedules or types of deprivation (see below), did not seem to us as sensitive an index of abnormality as ordinary microscopic examination of the Nissl-stained sections. Histological changes in layers whose mean area was decreased by only 10% were obvious at a glance. This was probably because the measurements were based on only one criterion, the area, whereas in examining the slides one can to some extent unconsciously compare many features such as cell area, staining properties,
Fig. 2. High-power photographs of dorsal and middle layers. Same sections as in Fig. 1.
Fig. 3. Same sections as in Figs. 1 and 2, but still higher magnification.
Fig. 4. Distribution by layers of cell areas in the two lateral geniculate bodies; kitten deprived by right eye-closure from the time of normal eye-opening to an age of 3½ months. Same kitten as in Figs. 1–3. Continuous lines, left lateral geniculate body; interrupted lines, right lateral geniculate body.
cell density, and layer thickness, both in adjacent layers and in corresponding layers on the two sides.

Several other parts of the visual system were examined for anatomical changes. The normal and light-deprived retinas showed no gross morphological differences: there were no obvious differences in the thickness of the entire retinas or of the various layers, and the size and staining properties of retinal ganglion cells appeared normal. The two optic nerves, stained with osmium, appeared identical, as did the two superior colliculi and the striate cortex on the two sides. While more specialized histological methods might have shown abnormalities at various levels in the visual system, the most obvious changes undoubtedly occurred at the level of the lateral geniculate body.

Monocular light deprivation in animals with previous visual experience. Monocular lid closure was performed in two kittens aged 2 months. In one of the kittens, deprived for 4 months, the lateral geniculate layers receiving input from the closed eye showed strong atrophic changes which were, however, less severe than those found in kittens deprived from birth. The other kitten had the right eye closed for only 1 month. In this animal the appropriate layers showed even less atrophy, although the changes were still very clear both on microscopic inspection and on measuring cell areas. Thus deprivation by lid closure caused atrophic geniculate changes in spite of the previous visual experience; furthermore, the changes could be observed after only 1 month of lid closure.

Finally, it seemed important to ask whether similar changes would occur in mature cats after similar periods of monocular light and form deprivation. Accordingly, the right eye was closed for 3 months in an adult cat. In this animal there was no difference between corresponding geniculate layers on the two sides: layers that received input from the closed eye were of normal thickness and contained cells normal in size and staining properties. Apparently there is an important difference between a growing kitten and an adult cat in susceptibility to these atrophic changes.

Visual deprivation with translucent covers. In the experiments described so far the kittens were visually deprived of form and also, to a large extent, of light. To assess the relative importance of form and light deprivation some experiments were done using translucent eye covers. In two kittens the cornea of the right eye was covered with a contact occluder made of translucent plastic. This was kept in place from the time of normal eye opening to an age of 2 months in one kitten, and 2 1/2 months in the other. The occluder reduced retinal illumination by about 2 log units (compared to 4-5 log units in kittens with lid suture) and excluded all form vision. In both kittens the geniculate layers receiving input from the covered eye showed histologically obvious cell atrophy, with a reduction in cell area of 10-15% for the appropriate dorsal and middle layers. This may be compared with the reduction, following lid closure for a similar period, of 40%. The histological changes in the lateral geniculate body thus seemed to vary with
the degree of diffuse light deprivation, strong light deprivation giving severe atrophy, moderate deprivation giving moderate atrophy.

In one of the experimental kittens morphological changes were completely absent. This kitten was raised in a normal environment for the first 5 weeks after birth and was then deprived for 3 months by suturing the nictitating membrane across the cornea. This procedure reduced retinal illumination by about 1 log unit and excluded form vision, but unlike translucent plastic occluders, it caused no infection or irritation of the conjunctiva. The lateral geniculate bodies of this kitten appeared quite normal: layers with input from the covered eye had the same thickness and the same mean cell areas as the corresponding layers with normal input. In the case of deprivation by lid suture, as described in a previous section, clear atrophy was found in kittens normally raised for 2 months and then deprived for as little as 1 month. This again supports the conclusions of the last paragraph, that for the production of geniculate atrophy the amount of light deprivation is an important variable.

Growth of geniculate cells during the first weeks after birth. A few experiments were done with the object of learning whether the geniculate cells of light-deprived kittens were small because of failure to grow at the normal rate or because of a decrease in size following previous growth. We therefore examined brains to determine geniculate cell size in visually naïve kittens at 1 day of age and at 8 days (i.e., before the time of normal eye-opening), and at 16 days in a kitten with plastic translucent contact occluders kept on both eyes from the eighth to the sixteenth day.

In the 1-day-old kitten the layering of the lateral geniculate body was poorly developed. The cells were densely packed and small, with a mean cell area for the dorsal layers of about $100 \mu^2$, as opposed to about $300 \mu^2$ in a normal 3-month-old kitten. In the 8-day-old kitten the layers were better differentiated: the cells were not so densely packed, and they had increased in size so that those of layers $A$ and $A_1$ had reached a mean area of $170 \sim 180 \mu^2$, and those of layer $B$ an area of $130 \mu^2$. At 16 days the mean cell areas for layers $A$ and $A_1$ had further increased to about $200 \mu^2$, and for layer $B$ to about $170 \mu^2$.

In summary, in newborn kittens geniculate cells are smaller than in the adult, and even smaller than cells in the atrophic layers of 3-month-old kittens deprived from birth. Moreover, even in the absence of patterned-light stimulation growth occurs, so that at 16 days the cells reached about the size of those in kittens deprived for 3 months. It would thus seem that under these conditions of deprivation cells continue to grow up to some time between the eighth and sixteenth days, and then remain stationary. It is conceivable that growth continues beyond this time, and that atrophy occurs subsequently; to rule this out would require more studies in animals deprived for intermediate periods. That visual deprivation can cause atrophy in the strict sense of a diminution in cell size was, of course, demonstrated in the
experiments already described, in which light deprivation of 2-month old kittens was found to cause a clear decrease in cell area.

**DISCUSSION**

Our recordings from visually deprived kittens show that geniculate cells and optic-tract fibers can have normal receptive-field arrangements, indicating highly complex and functionally normal connections, even though the associated retinas have never been exposed to patterned-light stimulation. That this is also true for cells in the primary visual cortex will be shown in a subsequent paper (20). Consistent with these results are behavioral experiments in the rat by Lashley and Russel (22), Hebb (11), and Walk and Gibson (30), which show that at least in some mammals certain simple visual discriminations are not necessarily learned.

Such abnormalities as were found in lateral-geniculate receptive fields were minor but nonetheless interesting. The large center size seen in a few of the fields may be the result of some derangement in the lateral geniculate body, but it may also reflect a similar abnormality in the fields of incoming optic-nerve fibers. While no such abnormal optic-nerve fields were seen, the sampling was far too meager to rule out their existence. The apparent enlargement of some geniculate-field centers might seem to suggest some kind of proliferation of connections. It might also be accounted for by a diminution of input opposing the center response, from the transitional zone between field center and surround. Whatever the explanation, the observation suggests that sensory deprivation can lead to a distortion of function.

That there were at least some physiological abnormalities is not surprising in view of the marked atrophic changes observed in the lateral geniculate body of visually deprived kittens. The atrophy, present in all layers receiving input from the closed eye, was most pronounced in kittens deprived from birth, less marked in kittens deprived at later ages, and absent in the deprived adult cat. The exact age beyond which cat geniculate cells are no longer sensitive to light and form deprivation of several months duration was not determined. This age may represent the end of a critical period in the development of the nervous system, a period during which an animal is not only more sensitive to sensory deprivation, but also can more easily make new nervous connections, adapting itself to variations in sensory stimulation.

The marked anatomical changes in the lateral geniculate body were especially surprising since similar findings have not previously been reported. In 1889 von Gudden (9) introduced the method of depriving animals of visual stimulation by suturing the lids. Later Berger (1), using this technique in newborn kittens, could find no anatomical changes in the lateral geniculate body after \(2\frac{1}{2}\) months of binocular deprivation. Goodman (6) raised rabbits in darkness from birth to an age of 6 months, and Chow (3) kept immature monkeys (age not specified) in darkness for 8 months; no
geniculate changes were found in either of these two studies. All of this work, then, has been unanimous in finding no geniculate abnormalities by conventional histological techniques. There may be several reasons for this discrepancy, besides the obvious one of species difference. In our study the deprivation was monocular, whereas all of those referred to above were binocular. The monocular technique tends to be more sensitive, since in each experiment the structures to be compared, deprived and nondeprived, appear in the same animal and on the same slide. But beyond this there is the possibility that in some way monocular deprivation may be very different from binocular: we might have found no atrophy had we closed over both eyes. Such a possibility is of course easy to test experimentally, and is now under study.

Apparently in kittens it is especially the lateral geniculate body that is sensitive to light deprivation, since similar atrophic changes could not be found in other parts of the visual system. In the retina, however, certain studies have demonstrated clear structural changes following light deprivation. An X-ray micrographic study of single retinal ganglion cells by Brattgård (2) showed marked reduction of cellular proteins in rabbits kept in darkness from birth to an age of 10 weeks; the same ganglion cells appeared normal when examined with ordinary histological techniques. Marked abnormalities, even complete disappearance of retinal ganglion cells, were first shown by Chow, Riesen, and Newell (4) in 2- to 3-year-old chimpanzees kept in darkness from birth. Less pronounced retinal changes have been reported in the cat (31, 27, 26).

The morphology of the deprived geniculate layers is strikingly similar to the transneuronal changes that occur some time after sectioning the optic nerve (24, 5, 23). In both conditions the abnormal layers are thin and contain small cells which lack Nissl substance. Comparing our results with those of Cook, Walker, and Barr (5), it appears that for similar periods of deprivation and deafferentation the amounts of cell atrophy were of the same order. Such a result might at first glance seem surprising, since the geniculate cells of light-deprived kittens, in contrast to the deafferented cells, may receive input from spontaneously active retinal ganglion cells (7, 21, 8); this input should, if anything, tend to limit the atrophy. The difference in age between the two types of preparation must, however, be taken into consideration: in the adult cat, in which optic-nerve section produces marked geniculate changes, visual deprivation was without discernible effect. Furthermore, transneuronal atrophy is known to be more pronounced and to develop faster in immature animals (13, 29), so that in a newborn kitten optic-nerve section might well produce a geniculate atrophy more severe than that which follows form and light deprivation. Preliminary studies indeed suggest that this is so (unpublished). In any case it is clear that such activity as does remain in optic-nerve fibers after prolonged deprivation is insufficient to maintain the cells in their normal healthy state.
We speak of the changes in deprived kittens as an "atrophy" because of the close histological resemblance to transneuronal atrophy found in adult cats, but it is important to realize that for the kittens one may be dealing at least in part with failure to grow rather than an atrophy in the strict sense. Ideally one would wish to compare two growth curves, one for normal cells, the other for visually deprived ones. In the present work we have shown that geniculate cells of newborn kittens are small by adult standards, and that they do increase in size even without benefit of visual stimulation. What is still not known is whether cells in the deprived cat grow to anything like a normal adult size and then atrophy, or whether they simply grow at a slower rate. In any case, in the normal kitten of about 2 months the geniculate cells are of about adult size, so that the changes observed when these kittens are then visually deprived can be termed an atrophy without any reservation.

From these experiments the exclusion of light seems to be an important factor in the development of atrophy in lateral geniculate cells, since the amount of atrophy varied with the amount of light deprivation, and, furthermore, a visually experienced kitten when subsequently light deprived showed atrophy, whereas there was no atrophy in a comparable animal subjected to a similar period of form deprivation. These results are in line with the findings of Chow, Riesen, and Newell (4), that a few hours of diffuse light stimulation each day is enough to prevent degeneration of retinal ganglion cells in animals raised in darkness. It remains to be established whether geniculate atrophy can be caused by form deprivation alone, and also whether it can be caused by diffuse light deprivation without form deprivation.

**Summary**

1. Kittens were subjected to deprivation of form and light in one eye, at various ages and for various periods. Deprivation was accomplished either by suturing the lids together or by placing a translucent contact occluder over the cornea.

2. In kittens with the lids of one eye sutured from birth for 3 months, most geniculate cells with input from the deprived eye had normal receptive fields, with an on-center and an off-periphery, or the reverse. The normal process by which the peripheral suppression demonstrable in retinal ganglion cells is increased at the geniculate level was observed. The overall activity of cells in layers fed by the deprived eye was, however, diminished, and a few cells had sluggish responses and receptive fields with abnormally large centers.

3. Marked histological changes were present in layers fed by the deprived eye. Mean cell areas were decreased by about 40% for the dorsal and middle layers and 25% for the ventral layer, and nuclei and nucleoli were also shrunken. No obvious histological changes were found in the retinas, optic nerves, superior colliculi, or striate cortex.

4. Lid closure for comparable periods in 2-month-old, visually experi-
enced kittens produced similar but less severe histological changes in the lateral geniculate bodies. No changes were seen in an adult cat visually deprived by lid suture of one eye for 3 months.

5. A translucent contact occluder placed over one eye from birth for 2 to 2\(\frac{1}{2}\) months produced similar histological changes, but again these were less marked, with 10–15\% reduction in mean cell area, in the appropriate dorsal and middle layers. In one kitten a translucent occluder was placed over one eye at 5 weeks for a 3-month period; there was no atrophy of geniculate cells.

6. Geniculate cells measured in a newborn kitten are smaller than those in the adult, and are even smaller than cells in the atrophic layers of kittens deprived from birth by lid suture for 3 months, indicating that some growth of cells occurs subsequent to birth, in spite of visual deprivation.

Acknowledgment

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References

LATERAL GENICULATE: VISUAL DEPRIVATION


