The one third of recordable cells in three-month-old binocularly sutured animals which you describe as "normal" could only be so called if one used the the lowest common denominator of selectivity from visually experienced adult cortex.

JULESZ: Bower claims that about 25 per cent of newborn human babies can detect depth in random dot stereograms, suggesting that disparity detectors are built in genetically. My second comment concerns experiments I've done on disparities one never uses in normal vision. If you increase disparities in a random dot stereogram and present them tachistoscopically to obviate convergence, then after many, many trials some people begin to see depth in them. This implies that units which were never used can be made functional later in life. My last comment bears on Blakemore's claim that his work on deprivation in cats indicates that there is cortical reorganization rather than the dying out of certain cells because he did not find silent areas in the cortex. Do you agree with this?

PETTIGREW: The last time I asked a newborn baby whether he could see depth in a stereogram I received an unintelligible reply. If stereopsis is present so early, it must be gross, since binocular coordination of eye movements takes some months to develop. I suppose it is conceivable that a kitten uses the binocular neurons I have described for a very crude form of depth discrimination on first opening its eyes.

The psychophysical evidence you cite for plasticity in the human disparity-detecting apparatus is interesting, but has various interpretations.

Yes, I agree that there may be cortical reorganization. Since I maintain that adult-type organization is not present in a young kitten, it follows that there must be some degree of reorganization even in the normal animal. With selective visual experience, whole populations of neurons appear to change rather than fall out, as was first pointed out by Hubel and Wiesel in their squint and alternating occlusion experiments and later by Blakemore and Cooper (1970) and Hirsch and Spinelli (1970).

Visual development in cats

*S. Murray Sherman**

Much interest has recently been focused on animals reared with visual deprivation. One could attack two problems by comparing the visual systems of such animals with those of both newborn and normally reared animals. First, there is the basic aim of this work of gaining an understanding of how the environment affects the developing visual system. Second, there is the prospect of a better understanding of the normal visual system from correlations among the abnormal physiology, anatomy, and behavior of visually deprived cats. Examples of an approach to both these problems will be shown in the following discussion, which is limited to studies of monocularly and binocularly deprived cats (MD and BD cats, respectively) and, for comparison, newborn and normally reared cats. The absence of crucial data still hinders any attempt to either correlate physiologic, anatomic, and behavioral effects of visual deprivation or formulate models which explain the action of the environment on the immature visual system. Because of this, the correlations and model outlined in this paper are tentative and incomplete.

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Abnormalities in MD and BD cats

Before discussing these experiments, it is useful to consider a division of the visual system into its *binocular* and *monocular* segments, because this has lately become important in studies of visually deprived

cats.[1-3] The cat's dorsal lateral geniculate nucleus (LGNd) can be divided into a larger, laminated portion medially, which is the binocular segment of the nucleus, and a smaller, unlaminated portion laterally, which is its monocular segment.[1] The former includes the full mediolateral extent of lamina A_1 and the adjacent parts of laminae A and B. Lateral to lamina A_1, laminae A and B merge to form the monocular segment. The binocular and monocular segments of the visual field are defined as those portions which are mapped, respectively, onto the binocular and monocular segments of the LGNd.[4] Likewise, these binocular and monocular segments of the visual field are mapped onto the binocular and monocular segments of the retina, striate cortex, and superior colliculus.

Neurophysiology.

Retina. Relatively little interest has been directed toward retinal physiology in visually deprived cats, presumably because LGNd neurons in such cats seemed to possess essentially normal properties.[5, 6] However, after a recent study which pointed to a cell type missing from the LGNd in MD and BD cats[2] (see also below), several properties of retinal physiology were studied in visually deprived cats. In the normal cat, the retina and optic tract have units with concentric ON or OFF center receptive fields, and among these units can be found slow-conducting X-cells[4, 7] (sustained cells[8]) with linear receptive-field characteristics and Y-cells[4, 7] (transient cells[8]) with nonlinear receptive-field characteristics. Also, the normal retina has a small proportion of retinal ganglion cells with nonconcentric receptive fields and conduction velocities slower than those of X-cells.*[9] All of the above features were found to be normal in the retina and optic tract of MD and BD cats.† At least for the present, therefore, the major defects due to visual deprivation in the feline visual system are assumed to be central to the optic tract.

LGNd. Three physiologic features established for the LGNd of normal cats have been studied in detail in MD and BD cats. First, normal LGNd neurons have concentric ON or OFF center excitatory receptive fields associated with the dominant eye[10] (i.e., the eye from which these neurons receive direct retinal afferents), and this feature remains essentially normal in MD and BD cats.[2, 5, 6] Second, most neurons in normal cats have nonspecific inhibitory receptive fields associated with the nondominant eye[11]; whereas the LGNd in MD cats appears normal in this respect,[6] fewer LGNd neurons in BD cats have this inhibitory, binocular interaction.[6] Third, as in the retina (see above) most LGNd neurons can be classified as X- or Y-cells by their receptive-field characteristics[4, 8]; the LGNd of visually deprived cats suffers a loss of Y-cells.[2] This loss is most severe in MD cats in "deprived" laminae (i.e., those receiving direct retinal afferents from the deprived eye) of the binocular segment of the nucleus, intermediate in severity *throughout* the LGNd of BD cats and not apparent in MD cats either throughout nondeprived portions or in the deprived, monocular segment.[2]

Striate cortex. Studies of the striate cortex have been largely limited to its binocular segment. In normal cats, the great majority of these cortical neurons are binocularly driven and have receptive fields with stimulus specificity for orientation and, often, direction.[12] In MD cats, however, these cells are driven with normal properties almost exclusively by the nondeprived eye.[13-15] Cortical deficits in BD cats are less severe than those related to the deprived eye in MD cats and include large numbers of both unresponsive neurons and neurons with nonspecific, although binocular, receptive fields. Only about half of the cortical neurons in BD cats appear to retain all of their normal properties.[15, 16]

It seems likely that these cortical deficits in MD and BD cats are related to the loss of Y-cells from the LGNd, both because the LGNd is the principal source of visual

*Stone, J., and Hoffmann, K.-P.: Unpublished observations.

†Sherman, S. M., and Stone, J.: Unpublished observations.

afferents in the cortex and also because the severity of the loss of Y-cells seems to correlate with the severity of the cortical deficit. This would imply both that in MD cats the deprived eye drives cortical cells normally in the appropriate monocular segment and that in BD cats the deficits would occur throughout the cortex. These predictions await future work for confirmation or rejection.

Superior colliculus. Most units of the superior colliculus are normally binocularly driven, with an ocular dominance pattern much like cortical units, and most have receptive fields which are directionally selective for stimulus movement.[17] In normal cats, these properties seem to be largely provided by input from the striate cortex. After long-term cortical removal, most superior collicular neurons are driven only by the contralateral eye without direction selectivity.[18, 19] In MD cats, units in both colliculi are driven normally and almost exclusively by the nondeprived eye,[20] as are striate cortical neurons.[13-15] After decortication, however, these units in MD cats are driven mostly by the contralateral eye, whether deprived or not, without direction selectivity.[20] Conversely, in BD cats the receptive field properties of the superior colliculus do not reflect the properties of cortical receptive fields. Rather the superior collicular properties resemble those found in the superior colliculus of a normally reared cat following decortication: The cells are driven mostly by the contralateral eye without direction selectivity.[21] However, *preliminary* data indicate that electrical stimulation of the visual cortex in BD cats evokes in the superior colliculus field potentials and single unit responses which appear essentially normal.* It is possible that in the BD cat the corticotectal pathway can drive superior collicular cells when the cortex is electrically stimulated, but not when visual stimuli are used. For instance, the cortical cells which give rise to this pathway in the BD cat may be among those which are abnormally unresponsive to visual stimulation.[15, 16] Of course, alternatives exist, and more data are needed to resolve this point.

As in the cortex, the monocular segment of the superior colliculus in visually deprived cats remains to be studied. Yet insofar as the MD superior colliculus reflects the cortical physiology in that cat, one might expect its monocular segment to be normal (see also above), and insofar as the BD superior colliculus appears "decorticate" with respect to visual stimulation, one might expect its monocular segment to be much like its binocular segment.

Anatomy. Wiesel and Hubel[5, 16] first noted that the average neuronal size in the LGNd of MD and BD cats is smaller than normal. This "shrinkage" involves the largest neurons and perhaps smaller neurons as well. Recent work[1] has shown both that this loss in MD cats is limited to the deprived binocular segment of the nucleus and that the loss in BD cats is relatively small but occurs throughout the nucleus.*[15] Since this loss of large neurons correlates with the loss of Y-cells in MD and BD cats, it has been suggested that the Y-cells are the large neurons.[2]

Studies of cortical anatomy in visually deprived cats are few. There have been comments that the Nissl-stained cortex in MD[5] and BD[16] cats seem qualitatively normal and a report[22] that BD cats suffer a loss from the striate cortex of mainly third order dendrites of stellate cells, whereas the pyramidal cells there seem normal. Since it has been shown that the stellate cell of the cat's striate cortex normally receives thalamic input,[23] this abnormality in BD cats might be a consequence of fewer LGNd synapses due, for instance, to the loss of Y-cells.

No detailed studies have to date been reported concerning the anatomy of the superior colliculus in visually deprived cats,

*Hoffmann, K.-P., and Stone, S. M.: Unpublished observations.

*Guillery, R. W.: Personal communication.

although Wiesel and Hubel[5, 16] noted that this structure in MD and BD cats appears qualitatively normal in Nissl-stained material.

Interocular alignment. Visually deprived cats tend to develop strabismus, but an MD cat's strabismus can be of any type, whereas a BD cat usually develops a divergent strabismus.[24] It is interesting to note further that indirect evidence (based on interocular alignments after anesthetizing and paralyzing these cats) suggests that *both* eyes of MD cats as well as BD cats are symmetrically misdirected.[24]

Behavior. It has been realized for some time that both MD and BD cats are deficient in visual behavior while using their deprived eyes.[13, 15, 16, 25, 26] This of course generally correlates with the physiologic deficits outlined above.

A recent study[3] with the use of a visual field perimetry test[27-29] on normal, MD, and BD cats has strengthened these correlations. In the normally reared cat, the visual cortex and superior colliculus interact to subserve the visually guided behavior used for this perimetry testing, and if one of these structures is ablated the other can continue to maintain this behavior.[27-29] The binocular visual field of the normal cat extends to about 100 degrees on either side of the midline, and each monocular visual field extends from about ipsilateral 100 degrees to contralateral 45 degrees.[3, 27-29] From this, the binocular segment of visual field extends bilaterally from the midline to about 45 degrees, and the monocular segment on each side extends from about 45 to 100 degrees. For the MD cat, both the binocular visual field and the nondeprived eye's monocular visual field are fairly normal, but the deprived eye's monocular visual field is limited to the monocular segment.[3] Likewise, the binocular visual field of a BD cat is fairly normal, but each monocular visual field is limited to the ipsilateral hemifield.[3]

The visual field perimetry of MD cats correlates well with the physiology outlined above, although it is stressed that the monocular segments of striate cortex and superior colliculus, which are predicted to be normal, have yet to be studied. In BD cats the correlation between visual field perimetry and physiology is less clear, because most cortical units in these cats are binocularly activated.[15, 16] The perimetry results are, however, consistent with the idea[3] (see above) that in BD cats the visual cortex is nonfunctional for visually guided behavior, although it might function for other visual behavior such as discrimination learning.[30] The responses to perimetry testing would then be determined by the retinotectal pathway, which is mostly crossed.

Critical period. Hubel and Wiesel have looked for a decrease in many of the above deficits (i.e., in LGNd anatomy and cortical physiology) in cats which were reared under conditions of visual deprivation but which were allowed extended periods of a normal visual environment for their previously deprived eyes. They[31] found the deficits to be permanent. Likewise, Wickelgren and Sterling[21] report that the superior collicular deficits in MD cats were permanent. Conversely, cats deprived of normal vision for extended periods after being normally reared for the first three months were without functional deficits.[32] From this study, Hubel and Wiesel[32] have defined a *critical period* consisting of the second and third postnatal months and during which visual deprivation of only a few days results in permanent deficits. In support of this, deficits both in visual behavior[3, 25, 26, 31] and interocular alignment[24] remain permanent in animals reared as MD and BD cats but subsequently given extended periods of a normal visual environment for the previously deprived eyes. Conversely, a recent study[15] indicates that the decrease of LGNd neuronal size in MD and BD cats is reversible. Many of the deficits described above, including the physiologic deficits both for the LGNd in MD and BD cats and for the superior

colliculus in BD cats have not yet been checked for permanence.

Newborn kittens

The newborn kitten is very different from normal and MD cats, yet in many ways is remarkably similar to the BD cat. These similarities are elaborated below.

The kitten's geniculostriate system has many of its functional connections by the eighth postnatal day.[33, 34] However, compared to the normal adult striate cortex there is a general lack of responsiveness, binocular interaction, and stimulus specificity in neurons of the neonate striate cortex.[33, 34] These are markedly similar to the deficits already noted for BD cats.[15, 16]

There is considerable cell growth postnatally in the LGNd,[35] and the LGNd in BD cats has cells smaller than normal.*[15, 16] Postnatal changes in feline neocortical histology have been carefully documented by Noback and Purpura,[36] and, although their methods do not permit a direct comparison with the analogous study in BD cats,[22] it is interesting that they speculate that stellate cells are the last to mature. The only reported anatomic abnormality for the BD cortex is in the dendritic branching of stellate cells.[22]

Kittens are born with a large divergent strabismus which they normally correct during the second postnatal month,[24] that is, during their "critical period" of development.[32] BD cats also have a divergent strabismus, and it has been suggested that the only postnatal changes in interocular alignment for the BD cat are due to normal growth and maturation of orbital tissues.[24]

Differences between MD and BD cats

Wiesel and Hubel[16] first noted the paradoxical differences in cortical physiology between MD and BD cats, namely that, despite the fact that the BD cat underwent more severe deprivation (i.e., twice as much), its cortex in many ways seemed less abnormal than that of the MD cat. They sought to explain this on the basis of a single mechanism—"binocular competition" among geniculostriate synapses for control of cortical neurons during development.[16] In the normal cat, according to this model, LGNd neurons driven by each eye compete for synaptic sites on cortical neurons, and in the course of normal development a balance is struck between these synapses resulting in the normal pattern of ocular dominance of cortical neurons.[12] In the MD cat, due to the unbalanced environment, synapses from the nondeprived eye become dominant. They subsequently seize total control of cortical neurons, thus causing the deprived synapses and their cell bodies to cease growth and/or atrophy.[1, 5] In the BD cat, the balanced though impoverished environment results in a relatively balanced and normal geniculostriate synaptic arrangement.[16]

However, recent findings seem to suggest different mechanisms for visual development in MD and BD cats. First, binocular competition among geniculostriate synapses of Y-cell axons, with resultant functional and anatomic atrophy of the deprived Y-cells, could account for the LGNd anatomy[1] (if Y-cells are normally larger than X-cells) and physiology[2] in an MD cat. The same mechanism applied in a BD cat would produce a normal monocular segment in the LGNd as in an MD cat. Yet in the monocular segment of the LGNd in a BD cat, the cells are smaller and include fewer Y-cells than in the corresponding part of the MD nucleus. Although Wiesel and Hubel made no attempt to extend their concept of binocular competition, it can be noted that such a generalization of the concept does not explain the finding[6] that the LGNd in BD cats has fewer binocular neurons than that in MD cats; and this becomes another difference which must be accounted for in any model of visual development. Second, the superior colliculus also appears fundamentally different between MD and BD cats in that properties in the MD cat appear to be

*Guillery, R. W.: Personal communication.

dominated by a functioning corticotectal pathway,[20] while properties in the BD cat appear to be dominated by a normal retinotectal pathway.[21] Third, there is a qualitative difference between MD and BD cats in visually guided behavior,[3] as outlined above. Finally, BD cats consistently present a divergent strabismus, while MD cats can have any of a wide range of interocular misalignments.[24]

A tentative model for visual development

Different mechanisms have been proposed to explain the difference between MD and BD cats in the development of interocular alignment,[24] and an extension of this concept serves as a tentative model for feline visual development. This model proposes that a *patterned-light* visual environment, either monocular or binocular, is necessary to initiate the mechanisms which normally guide development in the feline visual system. The emphasis is placed on patterned light because Wiesel and Hubel[13] have pointed out that cortical abnormalities of cats reared with translucent occlusion of one eye (i.e., a *diffuse-light* visual environment for one eye) appear essentially identical to those of a cat reared with monocular eyelid closure (i.e., a relatively dark environment for one eye). Yet they point out that the LGNd cell shrinkage is less, though still considerable, in cats with translucent occlusion than it is in cats with eyelid closure,[5] and this raises the possibility that some LGNd development can take place with diffuse light. With this qualification in mind, patterned light can be considered as essential for normal development of the visual system. This concept of the developing feline visual system must remain very general, because little can be said concerning the above-mentioned, presumed mechanisms. However, to clarify the hypothesis, a tentative scheme for geniculostriate development is presented below.

Geniculostriate development. In the geniculostriate system, a patterned-light environment could initiate some form of binocular competition[16] among Y-cells and possibly also X-cells, but to a considerably lesser extent. With the normal *binocular* patterned input during development, the typical feline geniculostriate system emerges having a binocularly balanced drive of cortical neurons[12] and the normal proportion of X- and Y-cells in the LGNd.[2] With *no* patterned input, as in BD cats, this binocular competition never takes place and relatively little postnatal development occurs in the geniculostriate system. Y-cells thus fail to develop properly throughout the LGNd,[2] causing the cell sizes there to remain somewhat less than normal*[15, 16] and cortical neurons retain many neonate features.[15, 16, 33, 34] With a *monocular* patterned input, as in MD cats, binocular competition is brought to bear on development. In the binocular segment the competitive advantage belongs to the nondeprived eye, but in the appropriate monocular segment the deprived eye develops a normal drive of neurons because it has no opposition from the nondeprived eye. Thus the asymmetric environment in an MD cat results in a deprived eye which on the one hand drives few LGNd Y-cells[2] and cortical cells[13-15] in the binocular segment but on the other hand drives the normal complement of Y-cells[2] and, it is predicted, cortical cells in the monocular segment. In those parts of the LGNd where Y-cells fail to properly develop, the cell sizes remain smaller than normal.[1, 5]

Conclusions. With no patterned light (i.e., the BD cat), parts of the visual system may continue some development while others may undergo some degeneration, but these would represent events which take place without specific influence from the visual environment. In general, the visual system in such a BD cat would continue to resemble that of the immature kitten. With patterned light, certain mechanisms would be triggered in some as yet unspecified way and result in a strong influence by the visual environment on the developing visual

*Guillery, R. W.: Personal communication.

system. Thus in the normal cat the typical binocularly balanced visual system emerges and in the MD cat a highly asymmetric visual system develops. In this sense, the BD cat develops without specific visually influenced mechanisms, whereas the MD and the normal cat develop by way of the *same* visually influenced mechanisms. The difference between the MD and normal cat would thus result from different visual environments (i.e., monocular versus binocular) operating on the *same* mechanisms.

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Discussion

DAW: In your movie the cat was always making a choice between 2 stimuli—essentially one straight ahead and one to the side. Is that correct? I find it rather puzzling that given two stimuli he should go off to the side.

SHERMAN: (The movie referred to was a short film clip demonstrating visual perimetry testing in MD and BD cats.) That is correct. This seems to be a feature of the cat's visual behavior, whether the cat is normal or visually deprived. The straight-ahead stimulus was the "fixation object" to which the cat had been visually attending for several seconds, and the stimulus to the side was the "novel stimulus" which was introduced only about one second before the cat was allowed to make its choice. Cats seem sufficiently curious to prefer always to investigate a new visual object instead of a familiar one.

LUND: Have you tried your technique to test cortical function in deprived animals?

SHERMAN: No, and this is a very interesting problem to which I hope to address myself shortly. Presumably, this perimetry technique tests the combined functional capabilities of the cat's visual cortex and superior colliculus. One way to separate out cortical function in visually deprived cats would be to test them both before and after having made cortical or collicular lesions. If my guesses are correct, for instance, I would predict that cortical lesions would have little effect on the BD cat's visual perimetry, but such a lesion (possibly combined with a transection of the commissure of the superior colliculus[29]) could return behavioral function for the binocular segment of the MD cat's deprived eye.

SPRAGUE: Could I just ask for what the physiologic interpretation is here? The anatomy and the single unit work reveal a decrease in the number of Y units at the LGN level. The visual field responses in the monocularly deprived animals showed a clear dropout in the binocular portion of the field. Is the conclusion that this is the type of response mediated by the Y units?

SHERMAN: I think it's much too early to be very precise about the physiologic bases of this behavior. I would expect that the loss of Y cells from the LGNd leads directly to severe abnormalities in the visual cortex and indirectly to superior collicular abnormalities via the corticotectal pathway. Therefore, I believe that the behavioral results are closely linked to the loss of Y-cells, although I wouldn't care to be any more specific than I already have been.

BURKE: Shouldn't there be some changes in the pattern of discharges of retinal ganglion cells also to explain the geniculate abnormalities? Do you have any information about this?

SHERMAN: There need be no differences in the discharge patterns of retinal ganglion cells to explain the loss of Y-cells from the LGNd, because it's possible that all of these abnormalities occur central to the optic tract. Also, as I've already stated, the retina and optic tract in visually deprived cats seem to have the normal complement of X- and Y-cells. To answer your specific question, however, we have gathered no data concerning possible abnormal discharge patterns in the optic tract of visually deprived cats.

MACKAY: Were you able to observe any deficits in eye movements in these animals?

SHERMAN: I didn't notice deficits in eye movements in the visually deprived cats, but I wasn't really looking very hard for them. However, these cats generally have a mild strabismus.[3, 24] I have estimated the strabismus in each of the visually deprived cats that I tested for visual perimetry to be less than 15 degrees. If I could make one further point: The loss of central visual field for MD and BD cats was not associated with any increase in the lateral boundaries of the visual field. This, together with the relatively small strabismus, makes it highly unlikely that the results could be an artifact due to eccentric fixation.