abnormal responses from the eye with reduced acuity. The four patients with optic atrophy showed normal ERGs (amplitudes 0.18 to 0.34μV; implicit times 36 to 37 msec).

Discussion

Foveal cone ERGs recorded from the amblyopic eyes of fifteen patients with strabismic amblyopia were normal in amplitude and b-wave implicit time. Moreover, the differences in ERGs between the amblyopic and the normal fellow eyes were no greater than those observed between the left and right eyes of fifteen normal subjects of comparable age. The typical decrease in amplitude of the foveal cone ERG that occurs with eccentricity from the fovea in normal eyes, consistent with the known decrease in cone density in the parafovea compared with the fovea (Osterberg, 1935), was also present in amblyopic eyes.

Previous investigations that have reported abnormalities in the ERG of patients with amblyopia must be interpreted with caution. In his review of ERG studies in amblyopia, Burian (1967) disputed most of the reported abnormalities on technical or theoretical grounds. Even in recent ERG studies using patterned stimuli (Tuttle, 1973; Sokol and Nadler, 1979), it has not been certain that the stimulus was in focus and maintained on the amblyopic fovea throughout testing. With the stimulator-ophthalmoscope, these problems have been reduced to a minimum. The foveal nature of our stimulus was substantiated by the fact that patients with macular scars of one disc diameter gave undetectable responses. The technique proved sensitive for detection of foveal pre-ganglion cell retinal abnormalities in patients with juvenile hereditary macular degeneration and visual acuity of 6/15 or below (Sandberg and others, 1979). The cone specificity of the stimulus was confirmed when undetectable responses were obtained in recordings from a rod monochromat (Sandberg and others, 1979) and in the observation that decreasing ERG amplitude occurs with increasing eccentricity from the fovea.

The possibility exists that a test of even greater sensitivity than that used in the present investigation, such as a foveal cone ERG elicited with a stimulus smaller than 4°, could reveal a functional abnormality in the retina. The size of our stimulus was dictated by the fact that stimuli of smaller diameter yielded less reproducible responses with the current procedure. The adequacy of a 4° stimulus, however, is supported by previous psychophysical estimates of the diameter of the amblyopic scotoma; *e.g.* 'diameter of the scotoma varies between 2 and 15° (mean 4.7°)' (Duke-Elder and Wybar, 1973). In addition, a recent uniocular static perimetry study of patients with strabismic amblyopia (many from the present study), using the stimulator-ophthalmoscope and stimuli of 6′ to 1° in diameter (Jacobson and Sandberg, in preparation), showed elevated cone increment thresholds not only in the amblyopic fovea but also at perifoveal retinal loci (*i.e.* 10° nasal or temporal to the fovea).

The findings in the present study are consistent with the idea that pre-ganglion cell foveal cone function is normal in human strabismic amblyopia. Whether dysfunction exists at the retinal ganglion cell level in human strabismic amblyopia as suggested by the work of Ikeda and Tremain (1978) on experimental strabismic amblyopia in cats cannot be decided from the present investigation since the human ERG is known not to be a measure of retinal ganglion cell function (Berson, 1975). The normal foveal cone ERGs in our patients with optic atrophy serve to re-emphasize this point. In conclusion, the site of the lesion in human amblyopia remains unknown, but the results of the present study weigh against the possibility of a readily detectable physiological abnormality in the central retina distal to the ganglion cell layer.

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Functional development of geniculocortical pathways in normal and amblyopic vision

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Summary

The geniculocortical pathways of many mammals, including primates, are largely comprised of two neuron types. These are known as X and Y cells. Their functional significance is unclear, but the following highly speculative hypothesis is suggested. Y cells, because of their broad sensitivity to the crucial lower spatial frequencies as well as to the higher ones, are involved in a basic analysis of form vision. X cells, because of their fairly selective sensitivity to higher spatial frequencies, add to this certain details (*i.e.* enhanced acuity, etc.). If an animal develops with ametropia, Y cells should be adequately stimulated and as the lower spatial frequencies remain undistorted many Y cells would develop and reasonable form vision with mild amblyopia would result. Only X cells would be greatly affected by such an environment. However, if the animal develops with cataracts or is reared in an environment which abolishes all spatial frequencies, then neither X nor Y cells will develop. This would result in poor form vision and a deep amblyopia. Many of these phenomena have been observed in experimental studies of cats. The present relevance for clinical problems of this suggested functional dichotomy for X and Y cells is at best tenuous, and it is offered merely as a working hypothesis for future studies.

The current interest in the central visual pathways of animals raised with visual deprivation can be traced back to the already classic work of Wiesel and Hubel which began over 15 years ago with studies of kittens raised with one eye sutured shut (Wiesel and Hubel, 1963). This interest is directed not only at basic problems of neural development and plasticity but also at gaining insights into clinical problems, such as amblyopia, by studying appropriate animal models. The significance of these studies for clinical problems should certainly be questioned, particularly since cats are not humans and otherwise normal children rarely have one eye occluded (or any of the variety of artificial rearing conditions to which we subject animals). Nevertheless, such animal studies can provide insights into the types of developmental processes which are susceptible to environmental manipulation.

This thesis could be supported by consideration of any of dozens of research efforts in this area, but to achieve focus and brevity, the remainder of the paper will be limited to studies of geniculocortical cells in cats reared from birth with monocular suture. It is no coincidence that this happens to be the subject upon which our laboratory has focused. No attempt will be made to cover the vast body of research involving deprivation regimens other than lid suture, neural areas other than the geniculocortical pathways, or research animals other than cats. The reader is referred instead to recent reviews (Blakemore, 1978; Hirsch and Leventhal, 1978).

The geniculocortical pathways of the cat

One reason to study the cat's geniculocortical system is its basic similarity to that of man. Fig. 1 illustrates the wiring diagram for this system, and it can effectively be divided into X and Y pathways.[1] Retinal X and Y cells are functionally quite like their geniculate counterparts, but X and Y cells differ from each other in a number of important physiological and anatomical dimensions (see below and Stone and Dreher, 1973; Rowe and Stone, 1976; LeVay and Ferster, 1977; Rodieck, 1979; Friedlander, Lin, and Sherman, 1979). The functional significance of this division of the system into X and Y components remains unclear, and consequently we must substitute speculation for understanding. Since lid suture

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[1] A third system, involving W cells, also exists and is relayed *via* the most ventral C complex of laminae (see Fig. 1 and Wilson, Rowe, and Stone, 1976; see also Rowe and Stone, 1976; Rodieck, 1979). This appears to represent a relatively minor contribution to cortical innervation, but in fact little is known about this pathway in normal cats and nothing is known about the effects of visual deprivation upon W cell development.

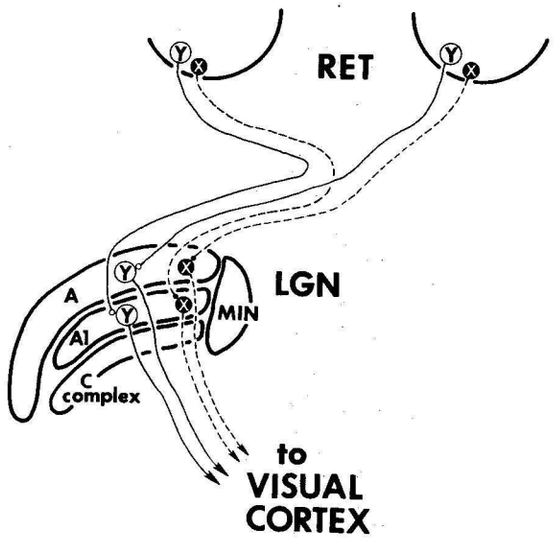

FIG. 1 *Pathways from retina (RET) to visual cortex in the cat. The lateral geniculate nucleus (LGN) is drawn in a coronal plane, and only pathways through the A laminae are shown in detail. Lamina A receives afferents from the contralateral retina; lamina A1, from the ipsilateral retina. These are the most thoroughly studied laminae and contain only X and Y geniculocortical cells. The medial interlaminar nucleus (MIN) and C complex of laminae also contain geniculocortical neurons which receive retinal afferents*

affects the X and Y pathways in cats quite differently, and because these effects probably underly the amblyopia suffered by these cats, it is worth speculating about the role normally played by X and Y cells.

The most common suggestion stems from the observations that, compared to Y cells, X cells tend to have smaller receptive fields, display more linear spatial summation, respond better to stationary targets, and are more concentrated in the central representation of the visual field. From these differences, some authors (*e.g.* Ikeda and Wright, 1972, 1975) have suggested that X cells analyse spatial patterns leaving an analysis of temporal features and/or movements to Y cells.

More recent studies in several laboratories, including our own, have suggested a different and more complicated interpretation. Our visual world is a rich one composed of a variety of spatial and temporal patterns. The range of these features is typically large. That is, spatial patterns range from very coarse, large objects to fine details; similarly, temporal changes in our world vary from slow movements or changes in brightness to very fast ones. A more precise way to describe these patterns is in terms of spatial and temporal frequencies. Low spatial frequencies are coarse patterns, while high frequencies are fine patterns; similarly, low temporal frequencies represent slow changes in the visual scene, while high frequencies represent fast changes. Recently, psychophysicists have begun to analyse vision by measuring sensitivity to stimuli comprised of single spatial and temporal frequencies. This is accomplished by using sine wave gratings, which are similar to the common stripe-like square wave gratings except that the luminance profile is a sine wave instead of a square wave. Such a sine wave grating has a single spatial frequency which is the number of complete sine wave cycles per degree of visual angle (cpd). These gratings are usually temporally modulated by gradually interchanging the light and dark areas, again in a sinusoidal fashion (see Fig. 2 of Sekuler, Pantle, and Levinson, 1978), so that the temporal frequency can also be expressed in cycles per second. Throughout this process, the mean luminance is constant and is half the sum of the maximum and minimum luminance values along the sine wave profile [1/2 (Lmax+Lmin)]. The contrast is (Lmax−Lmin) divided by (Lmax+Lmin).

In a typical experiment, a single spatial and temporal frequency is employed and contrast is adjusted until the threshold value for detecting the grating is obtained. By plotting these threshold contrast values as a function of spatial or temporal frequency, contrast sensitivity functions (CSFs) are constructed. These functions are analogous to the modulation transfer functions often used to describe physical systems. Just as Fourier analysis and synthesis permits complex waveforms to be analysed and synthesized from their sine wave components, complex visual scenes can be analysed and synthesized from their component sine wave gratings. Consequently, one can infer many aspects of the responses of a single cell or entire visual system to complex visual scenes from these CSFs. For a more complete discussion of the methods and rationale of this approach, see Braddick, Campbell, and Atkinson (1978) and Sekuler and others (1978).

Once psychophysicists demonstrated the value of this approach, neurophysiologists followed and began to apply the same stimulus/response measures to single visual cells. When these data from cat geniculate X and Y cells (*cf.* Lehmkuhle, Kratz, Mangel, and Sherman, 1980a; and numerous others) are compared to psychophysical data from cats and humans, some interesting correlates emerge. As expected, X cells tend to respond to higher spatial frequencies than do Y cells, but the mean difference is small and there is considerable overlap since some Y cells are more sensitive to higher frequencies than are some X cells. In the same way Y cells tend to respond to higher temporal frequencies than do X cells; but again, the mean difference is small and overlap exists. Much sharper differences would be seen if these cells were functionally specialized for spatial (X cells) or temporal (Y cells) processing.

A much more dramatic difference between X and Y cells is seen in their responses to lower spatial frequencies (*i.e.* coarser targets). Y cells are sensitive to lower spatial frequencies and exhibit a monotonic sensitivity decrease as spatial frequency increases. X cells, on the other hand, respond best to a middle range of spatial frequencies (roughly 1–2 cpd) and display marked attenuation in sensitivity for frequencies higher *and* lower than this. As mentioned above, X cells are slightly more sensitive than are Y cells to higher spatial frequencies, but at lower frequencies Y cells are responsive whereas X cells are relatively unresponsive. This difference at lower spatial frequencies represents one of the major differences between X and Y cell responses to spatiotemporal stimulation (Lehmkuhle and others, 1980a).

For many years the importance to vision of low spatial frequencies, which would activate Y cells but not X cells, has been overlooked. Perhaps this is because the most common clinical assessment of visual capacity is an acuity test which measures sensitivity only to the highest spatial frequencies seen. Recent psychophysical work (Kabrisky, Tallman, Day, and Radoy, 1970; Hess and Woo, 1978) has emphasized the importance of the lower spatial frequencies. These studies, in fact, suggest that basic form vision travels in the lower spatial frequencies, and that the higher ones are used to analyse fine detail. Most of us have had experience which supports this conclusion. Blurring of an image (by a poorly focused camera, missing spectacles, etc.) fairly selectively blocks transmission or reduces contrast of higher spatial frequencies with little effect upon lower ones. We can easily recognize basic shapes (houses, landscapes, faces, etc.) in a moderately defocused image. Diffusion of an image (by dirty spectacles, heavy fog, viewing through waxed paper, etc.) depresses transmission or contrast for all spatial frequencies roughly equally. If such diffusion reduces transmission of low spatial frequencies sufficiently, spatial vision is severely hampered. In fact, if the same visual scene is distorted by blurring or diffusion so that higher spatial frequencies are more affected by the former than by the latter, that scene typically is much more readily recognized if blurred than if diffused (Hess and Woo, 1978). Again, this emphasizes the importance of low spatial frequencies to form vision.

It is now possible to offer an hypothesis regarding the functional role of X and Y pathways, an hypothesis which differs from the common one based on a spatial and temporal distinction. It seems clear that low spatial frequencies are important to, and at least sufficient for, good form vision. Generally, Y cells seem responsive to these frequencies while X cells are not. Perhaps Y cells are involved in a basic analysis of spatial patterns, and X cells are involved in adding important details to this, such as higher acuity, stereopsis, colour vision, etc. (for a detailed discussion of this see Lehmkuhle and others, 1980a).

Support for this view comes from the study of lesions of the striate cortex (area 17). In the cat, geniculate X cells project exclusively to striate cortex, but the Y cells project extensively to striate plus numerous extrastriate cortical areas (Stone and Dreher, 1973; Rodieck, 1979). (This represents one important difference between carnivores and primates; in monkeys, it has been shown that all geniculate fibres exclusively terminate in striate cortex and do not reach extrastriate areas directly: Tigges, Tigges, and Perachio, 1977.) Berkley and Sprague (1979) have shown that lesions carefully removing all striate cortex (thus destroying all geniculate X cell connections but sparing many or most of the Y pathways) produce cats with excellent form vision and only a slight acuity loss. Just as low spatial frequencies are sufficient for good form vision, so apparently are the surviving Y pathways.

It must be vigorously emphasized that this speculation is based on a woefully incomplete understanding of the visual process. Nonetheless, it serves as a useful framework within which to consider the consequences of early monocular lid suture upon form vision and X and Y cell development.

Development of X and Y cells in lid-sutured cats

Anyone who has worked with monocularly deprived cats will attest to their deep amblyopia when forced to use the deprived eye, and as expected, the nondeprived eye seems capable of providing normal vision. If the above hypothesis were true, one would predict that pathways related to the deprived eye must have serious abnormalities in Y cells; since destriate cats without X pathways have excellent form vision, the poorer vision than this seen after rearing with lid suture implies Y cell deficiencies. As indicated below, evidence for this exists. Two preliminary points will be made before considering this:

(1) The neural defects seem to occur central to the optic tract in lid-sutured cats, since retinal ganglion cells in these animals seem entirely normal (Sherman and Stone, 1973; Kratz, Mangel, Lehmkuhle, and Sherman, 1979).[2]

(2) The defects described below develop only if lid suture is present during the first few months of life. That is, later lid suture has little or no effect on normal X and Y cells; and likewise, later normal vision cannot correct defects established by early lid suture (J. R. Wilson and S. M. Sherman, in preparation). These phenomena closely resemble the 'critical period' defined by Hubel and Wiesel (1970) for striate cortex.

Geniculate cells do not develop normally in deprived laminae (*i.e.* laminae innervated by the sutured eye). Compared to neurons in the nondeprived laminae, in deprived laminae very few Y cells can be recorded electrophysiologically while fairly normal X cells are recorded in normal numbers (Sherman, Hoffmann, and Stone, 1972). This severe deprivation effect upon Y cell development also has anatomical correlates (LeVay and Ferster, 1977; Garey and Blakemore, 1977; Lin and

[2] However, this conclusion must be qualified by the claim of Ikeda and Tremain (1979) that cats reared with esotropia develop abnormalities in retinal X cells located in the area centralis.

Sherman, 1978), although the detailed relationship between the physiology and anatomy of this defect is as yet unclear. X cells in deprived laminae are not completely normal, but have a subtle defect (Lehmkuhle, Kratz, Mangel, and Sherman, 1980b). Although responsive to middle spatial frequencies with fairly normal receptive field organization, they are relatively insensitive to higher spatial frequencies. That is, they suffer an acuity loss although they retain normal responsiveness to middle spatial frequencies.

Developmental mechanisms of X and Y cells

Another difference between deprivation induced defects in X and Y cells provides important clues regarding mechanisms which control their development. This difference lies in the pattern of the above-mentioned abnormalities (see Fig. 2). The loss of recordable geniculate Y cells is limited to the deprived binocular

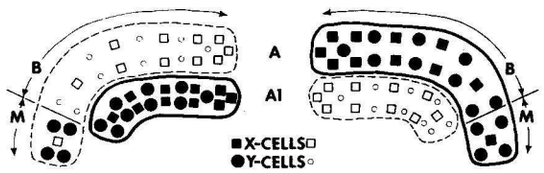

FIG. 2 *Summary diagram of laminae A and A1 from the lateral geniculate nucleus of a cat reared with uniocular suture. The dashed outlines indicate the deprived laminae, and the binocular and monocular segments are shown. ■ and ● represent normal X and Y cells, and □ and ○ deprived cells which fail to develop normally. Nondeprived laminae contain apparently normal cells throughout. X cells throughout deprived laminae exhibit a subtle abnormality: they are somewhat insensitive to the higher spatial frequencies, but seem otherwise normal. Few normally responsive Y cells can be located anywhere in the deprived binocular segment, but the deprived uniocular segment contains these cells in normal numbers*

segment[3] of the nucleus, whereas the deprived monocular segment[3] has Y cells normal in response properties and numbers (Sherman and others, 1972; Lehmkuhle and others, 1980b). The acuity loss for geniculate X cells, on the other hand, is seen throughout the nucleus, and is roughly equal in magnitude in the deprived binocular

[3] The binocular segment of the central visual pathways, including the lateral geniculate nucleus, is that portion in which the neurons have receptive fields within the binocularly viewed portion of the visual field. The monocular segments contain neurons of which the receptive fields are in the peripheral, monocularly viewed crescents of the visual field. The cat's visual field horizontally subtends roughly 180°. The central 90° is the binocular segment and the peripheral 45° on each side form the monocular segments (see Sherman, Hoffmann, and Stone, 1972; Sherman, Guillery, Kaas, and Sanderson, 1974).

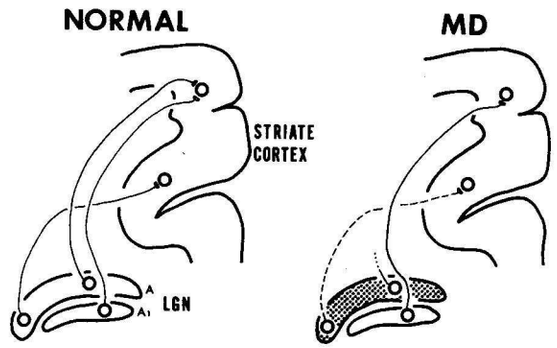

FIG. 3 *Diagram to illustrate binocularly competitive and noncompetitive mechanisms of development. The monocularly deprived (MD) example is drawn as if the right eye (and thus left lamina A) were deprived. The competitive interactions are thought to occur among geniculocortical terminals, although the site of these interactions has not been experimentally verified, and other locations are possible. It is suggested that terminals from geniculocortical cells can compete with one another during postnatal development. If no cell groups are given an advantage, as during normal binocular rearing conditions (left), a balance is struck and a normal, binocular visual system emerges. If geniculocortical cells are somehow placed at a disadvantage by lid suture (i.e. cells in lamina A), they fail to compete successfully, do not establish or maintain many cortical synapses, and do not develop normal responsiveness to visual stimuli. However, these cells are placed at a disadvantage only in the binocular segment; in the monocular segment, no lamina A1 cells are present to develop at the expense of the deprived lamina A cells. Thus these deprived neurons can develop normally here. To the extent that, in deprived laminae, neurons in the monocular segment develop completely normally while those in the binocular segment do not, a mechanism of binocular competition can completely account for the results. Such is the case for Y cell development. On the other hand, if deprivation induced abnormalities are seen equally in binocular and monocular segments, as is the case for X cell development, some form of binocularly noncompetitive mechanism is implicated. That is, the lid suture itself affects development of deprived cells without regard to the development of their nondeprived counterparts. Note that with data only from the binocular segment, it would be extremely difficult to distinguish between binocularly competitive and noncompetitive mechanisms. Finally, it should be noted that a combination of these processes is possible. This combination would be manifested by abnormalities in both binocular and monocular segments of deprived laminae, but the abnormalities would be less pronounced in the monocular segment.*
MD = monocularly deprived

and monocular segments (Lehmkuhle and others, 1980b).

The pattern of deficits seen for Y cells can most readily be explained by a developmental mechanism of binocular competition (*cf.* Fig. 3). That is, during development, Y cells from the different laminae somehow compete with one another, perhaps for synaptic sites onto cortical neurons. If no population of Y cells has a competitive advantage, as in normal binocular

rearing conditions, a balance is struck, and Y cells throughout can develop normally. Monocular suture can upset this balance and somehow confer a competitive advantage upon Y cells in nondeprived laminae; perhaps this advantage is obtained through higher peak firing rates. Because of the competitive disadvantage, Y cells in deprived laminae cannot develop or maintain functional integrity and become abnormal. Since such competition can occur only in the binocular segment, the loss of recordable Y cells is seen only there. Since deprived Y cells in the monocular segment are spared the deleterious consequences of being placed at a competitive disadvantage, they develop normally. Furthermore, their normal development in the deprived monocular segment indicates that deprivation alone, without binocular competition, does not retard Y cell development. See Sherman, Guillery, Kaas, and Sanderson (1974) for a more detailed discussion of binocular competition.

By this same reasoning, one can forcefully argue that X cells do not develop by way of binocular competition. The observation that deprived X cells are equally abnormal in both monocular and binocular segments (Fig. 2) tends to rule out this mechanism. Some other binocularly noncompetitive mechanism must determine X cell development. That is, it may be that some result of the deprivation, such as reduced peak firing rates, directly prevents X cells from developing or maintaining sensitivity to high spatial frequencies, and the relative development of nondeprived X cells is irrelevant.

Therefore, X and Y cell development seems to differ in two important ways:

(1) The qualitative defects seem quite different between the massive loss of recordable Y cells and the subtle loss of acuity in otherwise normal X cells.

(2) The developmental mechanisms seem different; binocular competition dominates Y cell development while a binocularly noncompetitive mechanism determines X cell development. These two differences may be related. The more serious consequences of lid suture upon Y cell development could relate to the more serious consequences of being on the losing side in a competitive struggle. Furthermore, geniculate Y cells seem to take longer than do X cells to develop under normal conditions (Daniels, Pettigrew, and Norman, 1978), and this, too, could make them more susceptible to environmental manipulation.

Application to clinical amblyopia

The above account has been limited to cats reared with lid suture. The pronounced amblyopia seen in lid-sutured cats has the predicted correlate in a severe defect in Y cell development. The lids effectively diffuse light so that practically no spatial patterns survive the passage to stimulate the retina. Thus, neither X nor Y cells would be effectively stimulated during development with lid suture. Defocusing, however, is another matter. Remember that moderate blur will attenuate only the higher spatial frequencies. The blurred image, with low but not high spatial frequencies, would be an excellent stimulus for Y cells but not for X cells. One might then predict that rearing conditions which create moderate blur, such as limited ametropia or certain forms of strabismus, would adequately stimulate Y cells but not X cells. The Y pathways would develop fairly normally, ensuring reasonable form vision, while the X pathways would develop with reduced responsiveness to higher spatial frequencies. One would predict form vision in such a cat to be reduced in acuity but otherwise adequate. Such results have, in fact, been obtained in cats reared with strabismus or ametropia (Ikeda and Tremain, 1978).

Such an hypothesis can also account for some of the variability observed in clinical studies of amblyopia. Some amblyopes have reduced acuity but otherwise good form vision, and they are presumably sensitive to lower spatial frequencies. Other amblyopes have much worse vision, presumably because of poor sensitivity to most or all spatial frequencies. Perhaps an early visual environment which adequately stimulates Y cells (*i.e.* moderate defocus, some types of strabismus, etc.) produces the less severe amblyopia. An early environment which does not adequately stimulate Y cells (*i.e.* diffusion from cataracts, ptosis, corneal oedema, etc.) produces the more severe amblyopia.

Conclusions and qualifications

This should be recognized as a highly speculative and narrow account that may well be incorrect. This paper has covered X and Y cell development only in cats and it is not at all certain to what extent these results can be extrapolated to humans. The few relevant comparative studies do suggest that most of these phenomena can be extrapolated to many mammals, including primates (Sherman, Wilson, Kaas, and Webb, 1976; Dreher, Fukuda, and Rodieck, 1976; Norton, Casagrande, and Sherman, 1977; see also Rowe and Stone, 1976; Blakemore, 1978; Hirsch and Leventhal, 1978; Rodieck, 1979). Perhaps much of the normal and abnormal development of visual capacity may depend to a large extent on development of X and Y cell properties as outlined here. The main point, however, is the potential usefulness of animal models for the understanding of clinical conditions such as amblyopia. Eventually, we can expect a more detailed and confident explanation of the developmental processes which underlie amblyopia. Therefore, this paper should be treated more as a logical exercise than as a statement of accepted hypotheses.

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Development of the neural basis of visual acuity in monkeys

Speculation on the origin of deprivation amblyopia

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The effects on the developing visual cortex of even brief periods of uniocular deprivation in kittens and baby monkeys (Wiesel and Hubel, 1963; Hubel and Wiesel, 1970; Crawford, Blake, Cool, and von Noorden, 1975; Hubel, Wiesel, and Le Vay, 1977; Blakemore, Garey, and Vital-Durand, 1978) provide an appealing model for the profound amblyopia that follows early occlusion of one eye in human infants (von Noorden, 1973; Awaya, Miyake, Imaizumi, Shiose, Kanda, and Komuro, 1973). Cortical neurones, the majority of which are normally responsive to visual stimulation of either eye, rapidly lose their functional input from the deprived eye. This phenomenon has been interpreted in terms of some kind of competitive interaction between inputs from the two eyes at the level of the striate cortex, where the two pathways converge for the first time in the retino-geniculo-cortical system.

There are a number of delightfully simple analogies to be drawn between the changes in ocular dominance of cortical neurones and the changes of acuity in amblyopic eyes. For instance, in a kitten or baby monkey rendered amblyopic by uniocular deprivation, the ocular dominance of cortical cells can be shifted back in favour of the initially deprived eye by a subsequent period of reverse-suturing—opening the deprived eye and closing the other (Blakemore and Van Sluyters 1974; Blakemore and others, 1978), just as alternate closure of first one eye and then the other in babies, leaves amblyopic the eye that is closed second (Awaya and others, 1973).

But are things as simple as they seem? Blakemore and Eggers (1978) have pointed out that it is naive to argue from population shifts in the ocular dominance of neurones to relative acuity in the two eyes: as long as an animal has even a tiny fraction of all cortical cells still responsive through its deprived eye it *might* in principle have quite normal vision through that eye. It is surely the *performance* of visual neurones, not their mere number, that determines an animal's acuity.

More important, the suggestion has been made, at least for strabismic amblyopia, that subtle changes in neuronal properties *earlier* in the visual pathway than the cortex itself might explain the reduction in acuity that characterizes amblyopia (see Ikeda, 1979).

Ikeda and her colleagues (who first emphasized the importance of testing the spatial resolving power of neurones in any attempt to analyse the neural basis of amblyopia) have found that the surgical induction of paralytic esotropia in kittens retards the normal process by which retinal ganglion cells (Ikeda and Tremain, 1979) and neurones of the lateral geniculate nucleus (Ikeda and Wright, 1976) gradually improve their spatial resolving power during the first 2 or 3 months of life. Thus strabismic amblyopia may be essentially a peripheral defect. Could the same be true for occlusion amblyopia?

Maffei and Fiorentini (1976) and Lehmkuhle, Kratz, Mangel, and Sherman (1978) have indeed reported that uniocular deprivation in kittens leads to an inability of cells of the lateral geniculate nucleus (LGN) to respond to grating patterns of high spatial frequency presented to the deprived eye. Interestingly, however, the spatial resolving power of retinal ganglion cells in the deprived eye is not affected (Kratz, Mangel, Lehmkuhle, and Sherman, 1979).

We, in this study, have examined this question for the first time in monkeys—a species of much higher visual acuity than the cat, in which the effects of visual deprivation on the development of acuity might therefore be expected to be even more severe. We have recorded from cells in the LGN of monkeys, from the day of birth onwards, and have examined their ability to resolve fine spatial detail. The results show that the normal improvement in neuronal 'acuity' parallels the monkey's actual behavioural visual development, but that, surprisingly, uniocular deprivation has little or no effect on this process.

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