Binocular Neurons and Binocular Function
in Monkeys and Children

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Electrophysiologic studies have shown that binocular neurons of the striate cortex of monkeys are irreversibly lost when the visual axes of the eyes are optically dissociated during the first weeks of life. The present study was a behavioral investigation of monkeys reared with optical dissociation. After brief periods of experimental strabismus, the monkeys showed poor binocular summation performance and were unable to see random dot stereograms even after two years of post-treatment normal binocular vision. These monkeys’ performance on the stereopsis task was virtually identical to the stereoperformance of clinically diagnosed stereoblind children. Invest Ophthalmol Vis Sci 24:491–495, 1983

It has been proposed that binocular neurons of the primary visual cortex are involved in functions of binocular vision and stereopsis.1,2 Although this hypothesis has become commonly accepted, no direct relationship between the existence and function of binocular cortical neurons, on the one hand, and stereopsis, on the other hand, has been demonstrated in primates. For obvious reasons, such a relationship cannot be tested in human patients who are stereoblind from various forms of visual deprivation (eg, strabismus, ocular occlusion, anisometropia, etc) that occurred early in life. However, since the rhesus monkey (Macaca mulatta) has a visual system comparable, functionally and structurally, to that of humans, has stereopsis,3,4 and an abundance of binocular cortical neurons,5,6 investigation of the relationship between binocular cortical neurons and behaviorally tested stereopsis seemed in order.

We report in this study data from contrast sensitivity, binocular summation and stereopsis testing in monkeys reared with prism-induced strabismus and from normally raised controls. The results are compared with those of children with normal and abnormal binocular vision tested on the same apparatus.

Materials and Methods

Five monkeys (Macaca mulatta) were used in these experiments. Three monkeys (L 46, L 51, and L 58) viewed the laboratory environment continuously through a pair of dissociating optical prisms from 30 to 60 days of age. Two prisms (17 and 10 diopters) were held base-in before the two eyes, with the base of one prism rotated downward by about 30° to prevent fusion in any position of gaze. The optical dissociation method to disrupt binocular vision has been previously shown to decrease drastically the number of binocular cortical neurons in monkeys.8,9 The prism goggles were then removed and the monkeys exposed to a normal visual environment until they reached the age of 1 year, when testing was begun. There was no suggestion of eye misalignment throughout the period of recovery as measured by video tape recordings of the corneal reflexes as the monkeys fixated targets at different viewing distances. Two control monkeys (L 38 and 418 T) were reared without visual restrictions and underwent the same testing procedure.

Three human observers were tested for stereopsis on the same apparatus. ME, a 15-year-old girl, noted in infancy to have intermittent esotropia. During childhood, the esotropia became manifest and surgery on the extraocular muscles was performed when she was 12 years old. At the time of testing the patient was orthophoric on the cover test, and her uncorrected visual acuity was 20/20 in each eye. JM had an esotropia noted shortly after birth. Treatment con-
sisted of glasses, and surgery was performed on the extraocular muscles at the ages of 10 and 13 years. At the time of testing, she had 20/20 visual acuity in each eye and was orthophoric on the cover test. Both patients had periods of alternate patching in childhood to prevent amblyopia. A third observer was an 8-year-old girl with normal eye findings and a negative history of past eye diseases, including strabismus.

Contrast sensitivity data were obtained for each eye on one normal and three experimental monkeys using the criterion reaction time paradigm described previously. The procedure was then repeated under conditions of binocular viewing. The degree of binocular summation was determined as the logarithm ratio of the binocular to monocular contrast sensitivity at each spatial frequency tested.

The monkeys were then trained on a disparity grating detection task using dynamic random dot stereograms with or without an associated luminance cue. The dynamic random dot stereogram was produced on a color television tube that had been modified to gain individual control over the color guns. Laboratory-designed TTL logic signal generators permitted a controlled horizontal spatial disparity between the red and green signals. When viewed at 1 M, through Wratten filters #29 and #58, the disparity-controlled section of the screen appeared as a square-wave grating standing in front of (or behind) a field of randomly moving noise. The square color video display subtended 20° visual angle, and the grating patterns could be adjusted over a wide range of spatial frequencies. The grating was made to disappear by desynchronizing the red and green signals for 100 ms. Normal monkeys have been shown to be able to detect images embedded in static random dot stereograms, and the well-documented use of the technique of random dot stereograms for human observers has presumed the presence of binocular neurons as a prerequisite to detecting the monocularly invisible test target.

All five animals were trained to depress and hold down a lever while viewing the color video display through the red and green filters. The cue to release the lever was a 100-ms disappearance of the grating pattern. If the monkey released the lever within 500 ms, the correct response was recorded, a tone sounded and an occasional squirt of orange juice was given. To help obtain stimulus control of the monkey's behavior, the monkeys were trained to a 90% performance criterion using a small red or green light emitting diode (LED) centered on the face of the video display. The LEDs were matched to the two Wratten viewing filters (#58 and #29) so that only one eye could see the appropriately colored LED. The size of the small LED approximated that of the individual dot elements that made up the stereogram, subtending about 2 min of arc. When given the associated luminance cue of the grating-paired LED, all five monkeys easily exceeded the 90% detection criterion. Equally high detection rates were obtained when the monkeys were permitted to observe the display without the anaglyphic filters in place. Such a procedure demonstrates that the monkeys could see the color and luminance-dependent gratings and could respond appropriately.

Results

The results of contrast sensitivity testing are shown in Figure 1. The contrast sensitivity functions were found to be essentially normal except two of the experimental animals (L 46 and L 58) showed a mild, unilateral amblyopia. For these monkeys, the high spatial frequency cut-off values were approximately 20 cycles per degree of visual angle for the nonamblyopic eyes and approximately 15 cycles per degree for the amblyopic eyes. These spatial frequency values correspond to Snellen acuity notations of 20/30 and 20/40, respectively. The third experimental monkey (L 51) showed normal acuity balance between the two eyes.

None of the optically dissociated monkeys showed clear evidence of binocular summation (Fig. 1). On the other hand, the normal monkey (L 38) had a consistently superior performance of approximately 40% (indicated by the dashed line) with binocular viewing as compared to monocular viewing. This is virtually the same as has been reported for human observers with normal binocular vision.

The ability of all subjects to detect form embedded in dynamic random dot stereograms is shown in Figure 2. The first histogram of each row shows that neither the normal monkeys (L 38, 418 T) nor the normal human observer (MM) experienced difficulties in performing the behavioral detection task when there was a paired luminance cue (the LED). Likewise, detection performance for these subjects remained comparably high (second histogram) when this luminance cue was removed and the stereomage alone provided the sole cue to release of the lever.

These results are in contrast with those obtained from the experimental monkeys (L 46, L 58, L 51) and human subjects (ME, JB) with a history of strabismus during infancy. Equally high detection performance was obtained with the luminance cue but performance plunged to near chance when this group was required to detect the change from retinal dis-
Fig. 1. Binocular (□) and monocular (●, ○) contrast sensitivity functions for four rhesus monkeys. L 38 was a normally reared monkey, while monkeys L 51, L 46, and L 58 were binocularly dissociated for 30 days early in life. Shown below each contrast sensitivity curve(s) is the binocular summation performance that is the logarithm of the ratio of binocular contrast sensitivity to the mean monocular sensitivity at each spatial frequency of the contrast function. The dashed line represents the 40% binocular summation predicted from data for normal human observers.
Fig. 2. The percentage of correct detection of dynamic random dot stereograms by monkey and human observers with and without an associated luminance cue. L 38, 4187, and MM had normal vision histories, while L 46, L 51, L 38, ME, and JB had histories of disruption of normal binocular function by strabismus early in life.

parity cues alone. The conclusion that this group, monkeys and human subjects alike, is stereoblind is compelling.

Discussion

In the newborn rhesus monkey (and presumably the human infant) a proportion of cortical neurons have innate connections from the two eyes. In the course of normal visual experience during the months following birth, the subpopulation of binocular neurons increases and many individual neurons become rather precisely tuned to small retinal disparities of the relative positions of visual objects seen from the perspective of the two eyes. Contrariwise, if their normal visual experience is marred by an eye misalignment, poor image quality in one eye, or by one eye being occluded, the animal’s binocular neurons rapidly disappear from the visual cortex and only monocular neurons serving only one of the two eyes can be identified. In past experiments we have described the course of these events produced by abnormal visual experience in monkeys and kittens by misaligning the visual axes optically to mimic a concomitant strabismus. These experiments have shown that as a result of this type of strabismus the binocular neurons disappear from the striate cortex as a monotonous function of the duration of prism wear. For instance, within 30 days of experimental strabismus, as used in this study, the percentage of binocular neurons drops from a normal 75–80% to 25–30% and to less than 10% within 60 days. Moreover, we have also shown that once lost, binocular neurons do not recover. For example, in eight monkeys having had optical dissociation from 30–60 days of age, no animal had more than 40% binocular cells (N = 8; X = 24%; SE = 5) following as much as 90 days of subsequent binocular viewing. We can assume, therefore, from these early experiments that the monkeys used in the present study had suffered a substantial loss of binocular cortical neurons through optical dissociation of their eyes in early infancy. It is reasonable to conclude from the data presented in this study that a normal population of cortical neurons is necessary for stereopsis and that there exists a direct relationship between the presence and function of binocular neurons and stereopsis. Moreover, this study shows that only a brief period of binocular dissociation in infant monkeys suffices to cause a permanent loss of stereopsis in spite of a subsequent prolonged period of normal binocular visual input. Further experiments are in progress to define the percentage of binocular striate neurons necessary for a criterion level of performance on stereopsis testing.

One may argue that stereoblindness in two of the monkeys (L 48 and L 78) may be a function of amblyopia caused by the artificially induced strabismus. However, the contrast sensitivity studies showed that amblyopia, if present at all, was of an extremely mild degree and thus not likely to interfere with stereopsis. Moreover, one of the monkeys (L 51) had no evidence of any amblyopia, yet was totally stereoblind.

The similarity of the results of monkeys and humans with a history of binocular dissociation early in life not only emphasizes that the monkey is a valid animal for the study of strabismus but also suggests that infantile strabismus in humans may cause a loss of binocular cortical neurons similar to that demonstrated electrophysiologically in monkeys with experimental strabismus.

Key words: binocular vision, stereopsis, dynamic random-dot stereograms, monkeys, optical dissociation, binocular summation
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References