Stereoblind Monkeys Have Few Binocular Neurons

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Infant monkeys wore prisms before their eyes, lost binocular neurons from their visual cortex, and were stereoblind to dynamic random dot stereograms. Three years later, the authors recorded from 880 neurons of the V1 and V2 visual cortex and found only 22% binocular neurons as compared with 81% for normal monkeys. These results (1) demonstrate, for the first time in the same subjects, the strong association between cortical binocular neurons and primate stereopsis; (2) show that congenital binocular neurons, once lost, do not recover even with extensive binocular visual experience; and (3) stress the vulnerability of the primate binocular system to abnormal early visual experiences.


Binocular neurons of the visual cortex occupy an unquestioned central position in current concepts of global stereopsis and depth perception.1–7 Binocular neurons of striate as well as prestriate cortex have been accepted widely as the neuronal entity mediating stereopsis; but evidence has been lacking that binocular neurons are necessary for this purported function. We have shown recently that the population of cortical binocular neurons can be depleted from the brains of infant monkeys8 and that such monkeys show no binocular summation and are unable to detect depth in dynamic random-dot stereograms,9 the sine qua non test for global stereopsis.6,10 Until now, the appropriate behavioral and electrophysiologic experiments have not been conducted on the same animals. Here we show that these same stereoblind monkeys have few binocular neurons in either striate (V1) or prestriate (V2) cortex, thereby providing the experimental link between a brain mechanism and the behavioral function.

Materials and Methods

Eight hundred and eighty neurons were recorded from the visual cortices of seven rhesus (Macaca mulatta) monkeys, three of which had had optically induced strabismus early in life produced by viewing the world continuously through prisms (27ΔBI) for 30 days between 30 and 60 days of age. There followed a 3-year period of normal binocular visual experience (no strabismus) and then extensive behavioral testing. The three experimental animals had no strabismus and gave every behavioral evidence of being stereoblind to random-dot stereograms prior to the electrophysiologic experiments.9 All procedures were consonant with guidelines for care and use of laboratory animals as set forth in National Institutes of Health (NIH) publication #80-23. The monkeys (three experimental and four normal controls) were prepared in the usual way for single-unit recordings from visual cortex.11 Paralyzed and under Pentothal anesthesia, both eyes were focused by contact lens upon a rear projection screen at 1 m distance. Oblique and normal penetrations into the foveal striate cortex, extending through the fiber layer and into the underlying prestriate (V2) cortex were made by a tungsten microelectrode.5,12 Single neurons throughout were resolved and tested for orientation selectivity, grating sensitivity, and eye dominance.
Results

Samples of neurons recorded from V1 and V2 cortex in the experimental monkeys were normal in all regards except for the degree of binocularity. Control monkeys had large numbers of neurons (81%) with input from both eyes; in the stereoblind monkeys, only 22% of the cells were binocular neurons. These differences are seen readily in the eye-dominance histograms of Figure 1, and the differences are contrasted in the lower right histogram, which indicates the missing binocular neurons by the darkened bars.

Discussion

These results are consistent with the purported role of the binocular neurons in global stereopsis. Monkeys (and presumably all primates) are born with binocular neurons, and the proportion of such cells approaches adult levels by 30 days of age. With normal visual experience, binocular neurons serve stereopsis and depth discrimination throughout life. However, if the early visual environment prevents simultaneous stimulation of the two eyes by compatible images, cortical binocular neurons lose functional connections with one eye, becoming exclusively monocular in service to the other eye. In addition, if an eye is further disadvantaged by poor optical quality or restriction in motility, an amblyopia may result. Here it is shown that binocular neurons can be removed experimentally from the visual cortices and that the loss of these binocular neurons results in stereoblindness.

These results, therefore, have assumed that the binocular neurons of the striate cortex, in turn, project to higher order binocular neurons of prestriate cortical areas. If so, loss of binocular neurons from striate cortex would evoke a similar deficit in prestriate cortical areas. This does not have to be the case; prestriate areas could, in theory, form binocular neurons from monocular striate inputs. The results presented here suggest that (1) binocular neurons of the striate cortex (V1) project to binocular neurons of V2, or (2) binocular neurons of striate cortex and prestriate cortex in common are both severely depleted by our experimental procedures. Either way, it is clear that under these conditions, there are few binocular neurons remaining in V1 or V2 visual cortex.

These results (1) demonstrate, for the first time in the same subjects, the strong association between cortical binocular neurons and primate stereopsis; (2) show that congenital binocular neurons, once lost, do not recover even with extensive binocular visual experience; and (3) stress the vulnerability of the primate binocular system to abnormal early visual experience.

Key words: stereopsis, binocular neurons, monkey cortex

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References

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