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INTEROCULAR INTERACTIONS IN ESOTROPIA

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INTRODUCTION

The visual system of adult mammals is characterized by an highly topographic ordering of projections (for a review see Ref. 37). Since the pioneering studies of Hubel and Wiesel (24) experimental work, particularly on the cat, has provided a large body of evidence indicating that the highly ordered set of visual connections is in large part genetically programmed yet its ultimate arrangement is shaped by visual experience (45) . At birth, in fact, the visual system is still immature and most susceptible to changes in its morpho-functional organization. Indeed, the early postnatal life represents a 'critical period' for the normal development of connections between the visual afferents and their cortical and subcortical targets. If during the critical period the individual's eyes are submitted to unequal stimulation as a result of experimental manipulations or ocular pathologies, binocular convergence of the two eyes' inputs is lost or severely impaired and binocular functions are damaged as well (for a review see Ref. 5).

Clinical evidence indicates that congenital unilateral strabismus is one of the conditions that leads in humans to permanent binocular sensory anomalies and to monocular spatial vision deficits. Our understanding of the neural alterations responsible for the functional vision defects associated with ocular misalignment is based primarily on the findings from investigations on the animal models of strabismus. Naturally occurring strabismus is rare in animals but surgical procedure have typically been employed to create an ocular misalignment in young animals.

I. *Different susceptibility to strabismus of the binocular interactions in striate and extrastriate cortex.*

Hubel and Wiesel (25) were the first to demonstrate that strabismus induced during the critical period of plasticity modifies the functional architecture of the primary visual cortex. In normal animals, owing to the convergence of retinothalamic inputs from the two eyes onto single neurons of area 17, the majority of striate cells is binocularly activated. Cortical binocularity, that is already present at 3 weeks of age, is however invariably lost when unilateral ocular deviation is induced in the early postnatal life (25, 43, 29). The absence of binocular interactions in the primary visual cortex is likely to be responsible for many of the binocular vision deficits found in strabismic subjects (e.g. reductions in stereopsis, binocular summation and interocular transfer of masking and adaptation phenomenon; Ref. 1, 22, 33).

It is generally agreed that the deleterious effect of strabismus -either convergent