cits were reduced 33 percent by 1 month of postweaning enrichment of the standard type used by Rosenzweig *et al.* (*4*). In the other study, maze learning in severely deficient (propylthiouracil-treated) rats was significantly facilitated by superenrichment treatment on days 71 to 103.

Under the conditions of our studies, there was no significant facilitation of performance by enriched or superenriched experience in normal rats (*12*). This makes unlikely the possibility that our hypothyroid rats' performance was facilitated by transfer of specific associations from the enrichment settings to the criterion tasks. It also argues against the possibility that the facilitations were mediated by differences in exploratory tendencies, gross activity levels, emotionality, or other motivational functions that might have been induced by the two housing treatments. Some forms of transfer—either specific associations from the enriched environment or a generalized resistance to new learning stemming from the impoverished experience—could nonetheless have accounted for part of the differences between the performance of enriched and impoverished hypothyroid rats in maze acquisition. Our maze retention and bar-pressing extinction tasks would seem less subject to such influences, however. In both these cases, transfer mechanisms would tend to be overshadowed, in terms of their effects on long-term memory or resistance to extinction, by the training experience in the acquisition phases of these tasks and the fact that the various groups were trained to approximately equal levels of mastery (all groups were trained to the common criterion of learning in each maze problem and to virtually identical asymptotic levels in bar-pressing acquisition).

Our preferred interpretation of these results, therefore, is that postweaning enrichment resulted in enhancement of learning and memory capacities in the hypothyroid rats that was relatively generalized and enduring. In general, our data suggest that the reduced synaptogenesis and associated hypoplasia of the cortical neuropil which result from perinatal thyroid deficiency (*2*) may be in part reversed directly by growth-promoting effects of environmental stimulation in the central nervous system. In particular, the neurohistological changes which have been demonstrated in studies of enriched experience (*5*), including increased lengthening and branching of cortical dendrites, increased density of dendritic spines, and increased size of synaptic junctions, seem likely to be involved in such reversal. Our data are also consistent with results (*13*) showing persistence of brain changes for many weeks after rats are shifted from enriched to impoverished conditions (as in our studies) and demonstrations of larger neuroanatomical changes from superenriched than from standard enriched conditions (*8, 13*). It remains to be demonstrated that the effects of environmental experience and early thyroid deficiency interact at the neurohistological level suggested here.

Elsewhere (*6*) we have compared our findings with analogous results showing reductions of behavioral abnormalities by postweaning enrichment in malnourished rats (*14*) and rats with brain lesions (*15*). The significant remediation shown in all of these studies offers encouragement to those attempting to devise effective environmental therapies for certain human brain dysfunctions (*16*).

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10. In analyses of variance, drug-by-environment interaction effects were significant for maze acquisition errors ($F = 12.61$; d.f. = 1, 37; $P < .005$) and bar-pressing extinction responses ($F = 4.62$; d.f. = 1, 37; $P < .05$), but not for maze retention errors ($F = 3.34$; d.f. = 1, 37; $P < .10$); for the latter task the main effect of enriched versus impoverished environment was significant ($F = 8.01$; d.f. = 1, 37; $P < .01$). The effects of enriched versus impoverished rearing in the thiouracil-treated groups reported in the text were evaluated by conservative *t*-tests with the normal groups' scores excluded from the error variance because of differences in variability of normal and thiouracil-treated groups. No significant differences or interactions involving the sex variable occurred in the maze or extinction tasks.
11. The passive avoidance deficit in the hypothyroid rats probably does not represent a learning or memory deficit but rather a persistent form of hyperactivity which rats treated perinatally with thiouracil display in adulthood [J. W. Davenport (*6*); _____ and R. S. Hennies, *Dev. Psychobiol.*, in press].
12. See J. W. Davenport (*6*) for a discussion of the discrepancies between the results in our normal group and the many studies showing enrichment-facilitated learning in normal rats. Some of the latter suggest that the absence of environment effects in our normal rats may be attributed to our use of moderately severe hunger conditions and extensive adaptation of the animals.
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Binocular Interaction in Strabismic Kittens Deprived of Vision

Abstract. *Artificial strabismus in kittens decreases the proportion of binocularly driven units in area 17 of the cortex. This change in the binocular interaction of cortical cells also takes place if the kittens are deprived of vision from the day in which the strabismus is surgically produced to the day of the electrophysiological recording. Thus, altered motility of the eyes per se is sufficient to affect binocular interaction in the neurons of area 17 of the cortex.*

Hubel and Wiesel (*1*) have shown that artificial strabismus in kittens decreases the number of binocularly driven neurons in area 17 of the cortex. This is usually ascribed to asymmetry in the two visual inputs to the binocular neurons of the cortex. In that case, binocular interaction should not be decreased if strabismic kittens have no visual experience. The obvious alteration which remains in strabismic kittens reared in darkness is asymmetry of eye movements.

We tested the hypothesis that the altered motility of the eyes per se is sufficient to decrease the proportion of binocularly driven cells in the striate cortex. We induced surgical strabismus in kittens during their critical period (*2*) and simultaneously prevented them from seeing until the day of the electrophysiological recording (third month of life or later). The results supported the hypothesis.

Experiments were performed on eight kittens. In six of them, under halothane

anesthesia, we severed the right rectus medialis muscle (*N*, 5) or the left rectus medialis muscle (*N*, 1) (Table 1). At the same time we sutured the eyelids following the technique of Wiesel and Hubel (*3*); in addition, we drew the nictitating membrane across the cornea, and sutured it to the conjunctiva along the upper lid. Three of these animals were reared in complete darkness. In two kittens we simply sutured the eyelids during the third week of life. The sutures were inspected daily in order that any possible small openings in the eyelids could be promptly repaired.

The recording experiments were performed when the kittens were at least 3 months old. A plastic chamber was positioned around two small openings of the skull overlying area 17 of the cortex and fixed firmly to the skull with dental cement. On the day of the experiment the cat was anesthetized with halothane and endotracheally intubated; a venous cannula was inserted. Areas of incision were infiltrated with a long-lasting local anesthetic. Anesthesia was terminated after the dura was removed, and the cat was immobilized with curare and given artificial respiration with room air.

In order to fix the animal to the recording table, we bolted the rim of the plastic chamber to a suitable metal mounting. After the animals' pupils were dilated with atropine, contact lenses with artificial pupils 4 mm in diameter were applied to both eyes, and refraction was corrected with additional lenses.

The action potentials of neurons in area 17 were recorded extracellularly with tungsten microelectrodes, and receptive fields were mapped by projecting the images of bright slits or edges onto a tangent screen placed 57 cm from the cat's eyes. Receptive fields were plotted separately for the two eyes, and the capacity of the cell to be excited by both eyes was ascertained.

We recorded from 228 cells, the receptive fields of which were located within 15° of the area centralis. The position of the area centralis on the tangent screen was derived from the position of the optic disks, which were projected ophthalmoscopically (*4*).

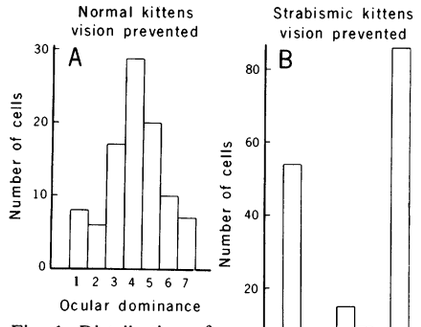

Fig. 1. Distribution of neurons of the striate cortex according to ocular dominance. (A) Distribution of 96 cortical neurons recorded from two kittens in the third month of life. The vision of these two kittens was prevented from the third week of life to the day of the electrophysiological recordings, during the 14th week of life. (B) Distribution of 182 cortical neurons recorded from six animals. In these animals the eye muscle, rectus medialis, was sectioned in the third week of life (Table 1) and both eyelids were sutured closed until the day of the experiment (third month or later). Three animals were also kept in complete darkness. The figures on the abscissa indicate the ocular dominance group according to the classification of Hubel and Wiesel: cells activated only by the contralateral eye (1) or by the ipsilateral eye (7), binocular cells having a strongly dominant input from the contralateral eye (2) or from the ipsilateral eye (6), binocular cells having a slightly dominant input from the contralateral eye (3) or from the ipsilateral eye (5), and cells having balanced inputs from the two eyes (4).

We noted that the divergence of the visual axis of the strabismic animals exceeded by several degrees the values reported for normal animals. This observation is in keeping with the fact that in these cats, divergent strabismus was produced by severing one of the medial recti.

The location of the electrodes in area 17 was confirmed by histological examination following electrolytic lesions.

Cortical cells were distributed in seven groups of ocular dominance according to the classification of Hubel and Wiesel (*5*) (Fig. 1). In agreement with previous results (*6*, *7*) the ocular dominance distribution of kittens deprived of vision but not made surgically strabismic is very close to normal (Fig. 1A). The loss of binocularly driven neurons in the strabismic kittens prevented from seeing (Fig. 1B) is comparable to that occurring in strabismic kittens living in a normally lighted environment. In each of the strabismic animals there was a slight bias in favor of the monocular neurons driven by the normal eye. In two of the cats this bias was clear. In cat 1, out of 26 monocular units 19 were driven by the normal eye, and in cat 4, out of 38 monocular neurons 26 were driven by the normal eye.

We conclude that asymmetrical eye movement in kittens resulting from artificial strabismus, even in the absence of vision, is sufficient to affect cortical binocular interaction. This suggests that symmetry in the flow of information from the proprioceptive receptors of oculomotor muscles to visual centers is important for maintaining the binocularity of cortical neurons.

This conclusion is complemented by recent findings of Maffei and Fiorentini (*8*), who found that in adult cats the diminution of binocular interaction in the neurons of area 17 produced by surgical immobilization of one eye (*9*) also occurs when the animals are prevented from seeing. Furthermore, they observed (*8*) that if both eyes are immobilized, which reestablishes artificial symmetry in the motor mechanisms of the two eyes, there is little or no loss of binocular interaction in the neurons of area 17.

Table 1. Ocular dominance distribution of cortical neurons of six cats in which the right (R) or left (L) oculomotor muscle (rectus medialis) had been sectioned and the eyelids sutured. Some of the cats were reared in complete darkness (D).

Animal	Treatment	Age (days)	Cells (No.)	Number of cells in each ocular dominance category						
				1	2	3	4	5	6	7
1	R	21	30	7	2	1	1	0	0	19
2	R	20	30	11	0	1	1	1	0	16
3	R	28	38	13	2	2	5	2	0	14
4	R D	25	57	12	2	2	5	5	4	26
5	R D	27	16	5	0	1	3	1	0	6
6	L D	20	11	6	0	0	0	0	0	5

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