

## Cortical activity blockade prevents ocular dominance plasticity in the kitten visual cortex\*

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**Summary.** Recordings from single units in kitten primary visual cortex show that a reversible blockade of the discharge activities of cortical neurons and geniculocortical afferent terminals by intracortical infusion of the sodium channel blocker tetrodotoxin (TTX) completely prevented the ocular dominance shift that would normally be seen after monocular deprivation. The blockade of cortical plasticity, like the blockade of discharge activity, was reversible, and plasticity was restored following recovery from the effects of TTX. These results extend previous work suggesting involvement of electrical activity at the level of the cortex in the phenomenon of cortical plasticity by demonstrating an absolute requirement for discharge activities in the primary visual cortex.

**Key words:** Visual cortex – Plasticity – TTX – Area 17 – Ocular dominance

### Introduction

Monocular eye occlusion during a critical period in early life leads to a change in the responses of cortical neurons, shifting the predominantly binocular responses toward monocular responses to the eye that had remained open (Wiesel and Hubel 1963). Evidence from a number of sources suggests that the discharge activities of geniculate afferents and cells within the visual cortex are involved in these dramatic changes. Recent findings that support this notion include observations from three different laboratories (Cynader and Mitchell 1977; Rauschecker and Singer 1981; Carlson et al. 1986) of orientation-

dependent shifts in the ocular dominance of neurons following monocular exposure to a restricted range of orientations. These studies show that ocular dominance shifts are restricted to those populations of cortical neurons that were stimulated with the orientations appropriate to drive the cell. Another study supporting the notion that activity at the level of the visual cortex is responsible for plasticity is that by Shaw and Cynader (1984), in which intracortical glutamate infusions raised the spontaneous discharge rates of cortical neurons and drastically attenuated the effects of monocular deprivation.

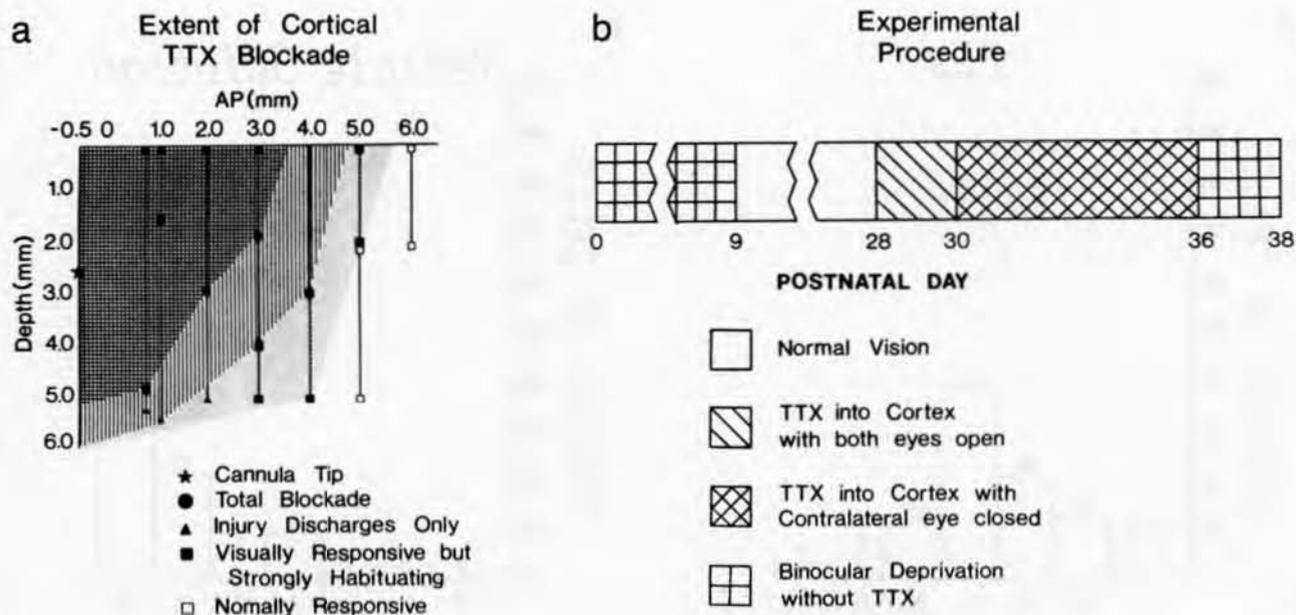
While these findings show that the cortex is involved, they do not answer the question as to whether cortical activity is essential for ocular dominance plasticity. We have addressed this question by blocking all discharge activity in a region of visual cortex during a period of monocular deprivation. If the discharge activity of cortical cells or geniculocortical afferent terminals is required for plasticity, the area of cortex subjected to activity blockade should show a normal ocular dominance distribution. A reversible activity blockade was produced by intracortical infusion of the sodium channel blocker tetrodotoxin (TTX) during a period of monocular deprivation. We report here that cortical activity blockade completely prevented the ocular dominance shift that would normally be seen after such a period of monocular deprivation.

### Methods

In 12 27–32-day-old kittens placed under anesthesia (1.5–2% halothane in 70 : 30 N<sub>2</sub>O-O<sub>2</sub>) in aseptic conditions, we implanted a sterile 33 ga stainless steel cannula into the right visual cortex (at Horsley-Clarke coordinates posterior 1, lateral 2, depth 2.5 mm from the dural surface). The cannulae were connected through silicon tubing to osmotic minipumps (Alza model 2002) that delivered 0.5 µl/h of a sterile solution of either 10 µM TTX

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**Fig. 1.** **a** Graphical illustration of the extent of cortex blocked by intracortical infusion of 10  $\mu$ M TTX in a citrate buffer solution. The star marks the tip of the cannula. The cross-hatched area denotes cortex devoid of any activity at all; while injury discharges, upon advancing the electrode, were seen in the immediately surrounding striped area. The dotted section refers to cortex containing visually responsive neurons that habituated strongly (responding to visual stimulation with no more than 1 spike in 10 s); in this region, which we interpret to be the region in which cortical cells were affected by marginal levels of TTX, there was no spontaneous activity. This figure refers to the activity of cortical neurons only; it disregards the activity of geniculate afferent fibers and terminals, which, though more resistant to the TTX effects, were also blocked in the immediate vicinity of the cannula. **b** Graphical illustration of the experimental procedure. The animals were allowed about three weeks of normal vision after the time of eye opening (here P9). At P28, a cannula was implanted stereotaxically into the primary visual cortex. Two days were allowed for cortical blockade to reach steady state before subjecting the contralateral eye to monocular deprivation for six days (P30–P36). On P36 the TTX delivery was terminated by disconnecting the minipump and simultaneously subjecting the animal to binocular lid suture. After a two-day “wear-off” period, single units were recorded in the previously blocked area of cortex.

(Calbiochem 584411) plus (in most cases) 1  $\mu$ Ci/ml  $^3$ H-proline (Amersham TRK.439) in 0.0035 M pH 4.8 citrate buffer or the citrate buffer solution alone (plus, in some cases, 1  $\mu$ Ci/ml  $^{14}$ C-proline). Following a period of 6–8 days of infusion and monocular visual deprivation, or infusion and recovery followed by visual deprivation, we prepared the kittens for acute single-unit recording from the previously infused visual cortex.

Kittens were initially anesthetized with halothane and a 70 : 30 mixture of  $N_2O$  :  $O_2$  until a venous cannula was inserted into the femoral vein. Thereafter, anesthesia was maintained with a combination of barbiturate infusion (10 mg/kg pentobarbital for surgery, 2–4 mg/kg maintenance) and ventilation with the nitrous-oxide/oxygen mixture. A tracheal cannula was inserted and a small skull opening (3  $\times$  7 mm) made anterior to the location of the cannula tip. The dura was then retracted.

After all surgical preparations, the animals were placed under neuromuscular blockade (0.1 mg/kg-h pancuronium bromide) and artificially ventilated at a rate and volume that maintained end-tidal  $CO_2$  at  $4.0 \pm 0.2\%$ . Temperature was maintained at  $38^\circ$  with a feedback-controlled heating pad and heart rate and e.g. were monitored.

Single-unit recordings were made using lacquer-coated tungsten microelectrodes (Hubel 1957) driven along vertical penetrations into the visual cortex using a stepping-motor microdrive. Receptive fields, all within 15 degrees of the area centralis, were

plotted with the help of a hand-held lamp. For each cell we determined the optimal orientation and directional preference, and we then assigned it to an ocular dominance group on the basis of Hubel and Wiesel's seven-point scale (1962). About thirty single units were recorded from each kitten, and the data used to construct ocular dominance histograms.

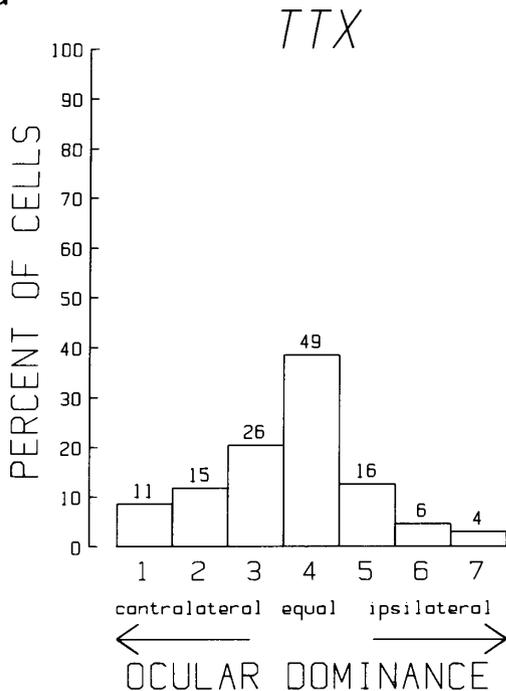
A scalar index was devised to describe the bias toward one eye or the other in each animal's ocular dominance distribution. This contralateral bias index (CBI) was calculated according to the following formula:

$$CBI = 100 \times [(1-7) + ((2/3) \times (2-6)) + ((1/3) \times (3-5)) + n] / (2 \times n)$$

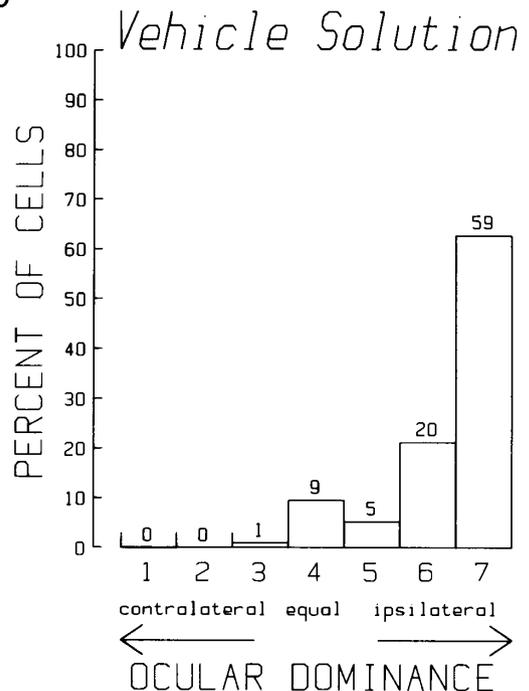
where italicized numbers (1...7) equal the number of units in each ocular group and n equals the total number of visually responsive units.

This index takes a value of 100 if all cells are driven exclusively by the contralateral eye, a value of 0 if all cells are driven exclusively by the ipsilateral eye, and intermediate values for conditions in between these extremes. The virtues of this index are (1) that its values correspond to the “weight” of the ocular dominance distribution toward one eye or the other, and (2) that it is affected to an equal extent by errors in the assignment of ocular dominance between any two adjacent categories. The values of this index have been calculated from published ocular dominance histograms (see Table 1) as well as from our own data.

a



b



**Fig. 2. a** Histogram of ocular dominance distribution of 127 units recorded in four kittens after TTX treatment. Percent of units (ordinate) classified in each ocular dominance group (abscissa). Number of units in each category is written above appropriate bar. Cortical activity blockade completely prevented the ocular dominance shift that would normally have been seen after 1 week of monocular deprivation. As in normal kittens of this age there is a slight bias in favor of the contralateral eye, which is the deprived eye in the TTX treated kittens. **b** Histogram of ocular dominance distribution of 94 units recorded in three control kittens with vehicle solution infusions only. Most units were dominated by the open, ipsilateral eye, an effect which clearly differs from that seen with TTX infusions

## Results

### *Preliminary experiments*

Preliminary experiments in the initial 3 animals revealed the cortical area in which action potentials were blocked by the TTX infusion. In these animals, studied 1.5, 6 and 7 days after beginning the infusion, the cortex 10 mm anterior and 6 mm lateral to the cannula was exposed and mapped while the infusion continued. The results are summarized in Fig. 1a. The area of cortex blocked by the infusion extended 4–5 mm anterior and at least 2 mm lateral to the cannula, to a depth of more than 5 mm, and was of similar size in the 3 animals tested. The success of the infusion was verified autoradiographically in 3 cases by demonstrating the cortical distribution of the isotopically-labelled proline contained in the infusion solution (not shown).

After the TTX infusion was stopped, spike activity returned to the region of cortex that had been blocked over the next 1–2 days. In 2 animals recorded 2 days after the end of the infusion, the blockade appeared to have reversed completely.

### *Monocular deprivation during intracortical infusion*

Having determined the region affected by the TTX infusion and the time course of these effects, we began the experiment to see whether such a cortical activity blockade would prevent the ocular dominance shift that would ordinarily be produced by monocular deprivation. The procedure for this experiment is illustrated in Fig. 1b. In 4 kittens, we sutured the lid of the left (contralateral) eye two days after beginning the TTX infusion. After an additional 5–6 days, we terminated the TTX infusion and sutured the right (ipsilateral) eyelid as well, producing a period of binocular deprivation while the activity blockade was allowed to wear off. After a final 1–2 additional days, we recorded from about 30 cortical neurons in the previously blocked area in a series of vertical penetrations spaced 400  $\mu\text{m}$  apart between 1 and 3 mm anterior to the cannula. Recordings concentrated on determining the eye preference of single units spaced at intervals of at least 100  $\mu\text{m}$ , expressing this preference on the 7-point ocular dominance scale of Hubel and Wiesel (1962).



Fig. 3. Nissl-stained coronal section of TTX-treated visual cortex showing microelectrode track. Arrow indicates lesion at the end of the track. Scale bar = 250  $\mu$ m

Three control kittens were fitted with a cannula that delivered vehicle solution only and were then subjected to a procedure otherwise identical to that used for the TTX kittens. These control animals were studied using a "blind" procedure in which we did not decode which animals had received vehicle solution infusions until after the physiological experiments were completed.

Visual cortical responses in the TTX-treated cortex had returned to near-normal vigor by the time of recording. Figure 2a shows the ocular dominance distribution of 127 units recorded in four kittens in which cortical activity was blocked with TTX during the period of monocular deprivation. This ocular dominance distribution is indistinguishable from that reported for normal kittens of about the same age (Stryker and Harris 1986). As in normal kittens, there is a slight bias in favor of the contralateral eye, which in the experimental kittens was the deprived eye. Thus, activity blockade completely prevented

the ocular dominance shift expected after 1 week of monocular deprivation during the height of the sensitive period (Hubel and Wiesel 1970; Olson and Freeman 1975; Movshon and Duersteler 1977).

The results from the control kittens that received citrate buffer infusions are shown in Fig. 2b. These animals showed an ocular dominance shift similar to that reported for untreated kittens of this age that were subjected to about one week of monocular deprivation. Most units in these control animals were dominated by the open (ipsilateral) eye, an effect very different from that seen in the TTX-treated animals.

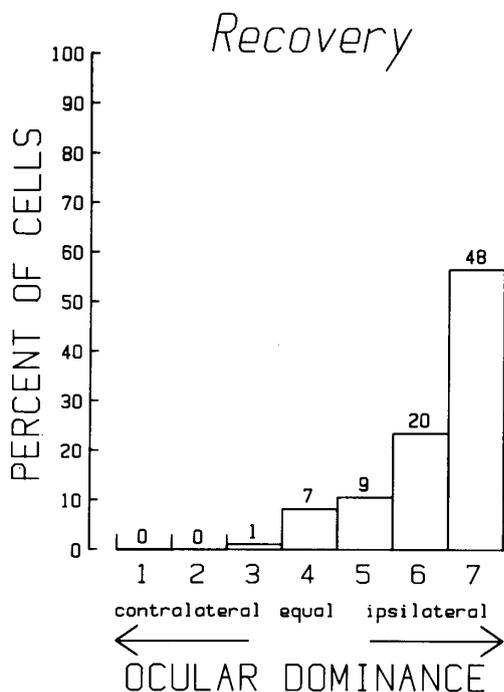
Histological sections of the region of the cortex affected by the TTX infusions did not disclose anatomical abnormalities. Although there was damage to the cortical laminar structure in the immediate area of the cannula tip, Nissl-stained sections like those shown in Fig. 3 from the area of our electrode tracks (1–3 mm anterior to the cannula) were not distinguishable from those from the untreated side.

#### *Monocular deprivation following recovery from intracortical TTX infusions*

One interpretation of the results of the previous section is that the TTX infusion may have produced its effect in blocking cortical plasticity by some unspecified mechanism other than its effect on the discharge activities of cortical neurons and their afferent inputs. Under this hypothesis, the TTX infusion abolished plasticity in ways that were not evident from our single-unit recordings or Nissl histology. As a control for the interpretation of these results as such a non-specific artifact, we performed the following experiment to determine whether, upon termination of the TTX infusion, plasticity was restored to the cortex along with the discharge activity of its neurons.

Three kittens were fitted with cannulae as described above, but were subjected to monocular deprivation only *after* (rather than *during*) the one-week infusion period. At the end of the infusion period, the minipump was removed (while the disconnected cannula remained in place) and the kitten was then subjected to 5–9 days of monocular deprivation. Following this time of monocular deprivation, the kittens were prepared for microelectrode recording as described above.

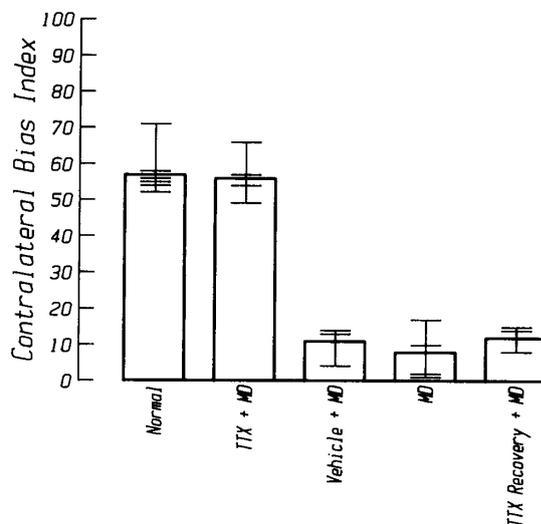
Figure 4 shows the results of 85 units recorded in 3 kittens. The ocular dominance shifts were as severe as those reported elsewhere for intact kittens subjected to a one-week deprivation period. They are similar as well to those following monocular depriva-



**Fig. 4.** Histogram of ocular dominance distribution for 85 units recorded from 3 kittens subjected to a week of intracortical TTX infusion followed by a week during which the animal was subjected to monocular deprivation. Abscissa and ordinate are the same as in Figs. 2a, b. The shift in ocular dominance toward the open (ipsilateral) eye is similar to that shown in Fig. 2b for monocular deprivation during vehicle solution infusion and to that shown in the literature for short-term monocular deprivation only (Hubel and Wiesel 1970; Olson and Freeman 1975; Movshon and Duersteler 1977)

tion during the period of infusion of the vehicle solution as described above. Therefore, the plasticity of cortical neurons (as measured by their ability to change their responsiveness in favor of the non-deprived eye after a period of monocular deprivation during the critical period) is not affected adversely by the TTX infusion beyond the duration of its inhibitory action in blocking discharge activities in the cortex.

Figure 5 compares the findings in the individual animals of this series to those in normal (Stryker and Harris 1986) and monocularly deprived (see above) animals from the literature. The results are described in the form of an index of the bias in the ocular dominance distribution toward one eye or the other (see Methods). Note that the average values and ranges of this index are very similar for normal and TTX-plus-monocular-deprivation animals. An equal similarity holds among all 3 groups of animals in which the cortex was active during monocular deprivation: vehicle-solution-plus-monocular-deprivation;



**Fig. 5.** Averages and ranges of the contralateral bias indices (see Methods) calculated from ocular dominance distributions for the individual animals that make up Figs. 2a, b, 3, and for animals from the literature. The five groups illustrated are normal kittens, kittens with TTX infusion plus monocular deprivation, kittens with vehicle-solution infusion plus monocular deprivation, kittens with short-term monocular deprivation only (Hubel and Wiesel 1970; Olson and Freeman 1975; Movshon and Duersteler 1977), and kittens with short-term monocular deprivation following recovery from TTX infusion

monocular-deprivation-only; and mono-cular-deprivation-following-recovery-from-TTX. The results from the first two groups, however, are quite different from those from the latter three groups.

## Discussion

These results demonstrate that cortical TTX infusions can prevent the shift in ocular dominance typically caused by monocular deprivation. While these results do not yet tell us which elements in the cortex must be active for plasticity to take place, they do imply (1) that the primary visual cortex is the *locus* where competition between the two eyes leads to physiological changes in cortical responsiveness as a result of monocular deprivation, and (2) that *activity* at the level of the cortex is an absolute requirement for ocular dominance plasticity.

We may compare these results to those from other studies in which substances have been infused into the cortex in order to affect cortical plasticity in response to monocular deprivation. Many substances have been reported to attenuate cortical plasticity, but unlike activity blockade with TTX, these treatments do not completely abolish plasticity. Table 1 presents an analysis of results from the literature

**Table 1.** Comparison of contralateral bias indices calculated from published ocular dominance histograms

Authors	Procedure	Number of Units	CBI
Stryker and Harris (1986)	normal kittens (36–51 d. of age)	372	57
Kasamatsu et al. (1979)	intracortical 6-OHDA infusion	62	46
Daw et al. (1983)	intracortical 6-OHDA infusion	93	48
Paradiso et al. (1983)	intracortical 6-OHDA infusion	153	44
Shaw and Cynader (1984)	intracortical glutamate infusion	173	66
Bear and Singer (1986)	cortical depletion of NE and ACh by:		
	a) 6-OHDA and NMA lesions and	a) 99	39
Reiter et al. (1986)	b) cingulate lesion	b) 88	39
	intracortical TTX infusion	127	56

This table compares results from normal kittens (top line) with those from monocularly deprived kittens treated by the various procedures noted in column 2. Column 3 shows the number of visually responsive single units. Column 4 shows the contralateral bias index (see Methods) calculated for each study. Note that the deprived eye is contralateral to the experimental hemisphere, causing a shift toward lower than normal values, in all cases except for the 173 units in Shaw and Cynader (1984), for which the ipsilateral eye was sutured, causing a shift toward higher than normal values. Note also the absence of a shift only in the present study (bottom line). To analyze the 5-point ocular dominance histograms in Bear and Singer (1986) a formula modified as follows was used:  $CBI = 100 \times [(1-5) + (1/2 \times (2-4)) + n] / (2 \times n)$

using the contralateral bias index that we have devised to describe the present findings (see Methods). It is evident from this analysis that monocular deprivation produced a shift of 9 points or more from the value of this index in normal kittens (57) under all experimental treatments except for activity blockade using TTX.

Shaw and Cynader, for example, reduced, but did not eliminate plasticity with intracortical glutamate infusions. In contrast, the contralateral bias index obtained in our TTX-treated kittens is equal to the value obtained in normal kittens, showing the naturally occurring bias in favor of the contralateral eye. The contralateral bias index of various monocularly deprived control animals (for example, Shaw and Cynader: 12), however, is almost identical to that in our own: (11).

Kasamatsu, Pettigrew and co-workers attempted to affect the noradrenaline (NA) system with intraventricular and intracortical infusions of 6-hydroxydopamine (6-OHDA) (Kasamatsu and Pettigrew 1976, 1979; Kasamatsu et al. 1979). They

reported that both methods prevented an ocular dominance shift in the visual cortex of kittens so treated. Other workers (Daw et al. 1983a; Paradiso et al. 1983) have confirmed the 6-OHDA finding with respect to intracortical infusions, but not when cortical NA stores were depleted by intraventricular injections (Adrien et al. 1982; Daw et al. 1985). Several other methods, all of which deplete cortical NA to levels comparable to those achieved by intracortical 6-OHDA infusions, fail as well in preventing ocular dominance shifts (Bear and Daniels 1983; Daw et al 1984; Daw et al. 1985; Daw review 1985).

Kasamatsu and Shirokawa (1985) have attempted to offer an explanation for this apparent paradox, by focusing their attention not on the absolute level of cortical NA content, but rather on the state of activation of  $\beta$ -adrenergic receptors. Their attempt to influence ocular dominance plasticity with  $\beta$ -adrenergic receptor blockers, however, again only attenuated the degree of ocular dominance shift one would normally see.

A possible explanation for the conflicting data may come from a recent study by Bear and Singer (1986) suggesting an important role not only for the noradrenergic but for the cholinergic system as well. Bear and Singer found that interfering with either system by itself was not sufficient to attenuate the expression of cortical plasticity significantly, while interfering with both systems simultaneously greatly attenuated the ocular dominance shift in response to monocular deprivation. In addition, Bear and Singer's study offered an explanation for the effectiveness of intracortical infusion of 6-OHDA by showing that iontophoretic application of 6-OHDA affects adrenergic as well as cholinergic transmission in the kitten visual cortex. Nevertheless, this study showed that ocular dominance plasticity was not *completely* prevented by interference with both the cholinergic and adrenergic systems (see Table 1).

In view of the controversial and inconsistent results obtained with NA depletion in visual cortex in connection with monocular deprivation, Daw has suggested that one must use care in interpreting the effects of intracortical infusion experiments, because of the possibility of non-specific damage to the region surrounding the injection site caused by the infused substance.

In our TTX experiments, we have controlled for this in the following ways: (1) the visual responses in the formerly blocked area were studied with care and found to be nearly normal in vigor and quality, (2) histology of the region containing electrode penetrations appeared normal and (3) plasticity was restored to the cortex when the discharge blockade was lifted.

Therefore, we believe than non-specific damage is unlikely to account for the results of this study.

Experiments that reveal orientation-dependent ocular dominance shifts (Cynader and Mitchell 1977; Rauschecker and Singer 1981; Carlson et al. 1986) or reversal of orientation-dependent ocular dominance shifts (Rauschecker and Singer 1979) also provide strong evidence that the activity of cortical cells contributes to ocular dominance plasticity, for only cortical cells (and not LGN units) are markedly orientation-selective (except at high spatial frequency limits of LGN neurons, Vidyasagar and Heide 1984). These experiments too, however, do not show that cortical activity is an absolute requirement for plasticity, because slight ocular dominance shifts were still observed in those cells which one supposes not to have been stimulated by appropriate orientations. In other words, while exposure to restricted orientations overwhelmingly favored an ocular dominance shift in cells whose preferred orientation corresponded to the orientation which was viewed, cells which preferred the orthogonal orientation also displayed a slight shift.

The present study thus extends previous findings by showing that complete blockade of activity in the visual cortex also leads to a complete blockade of any expression of ocular dominance plasticity in response to monocular deprivation.

## References

Adrien J, Buisseret P, Fregnac Y, Gary-Lobo E, Imbert M, Tassin J-P, Trotter Y (1982) Noradrenaline et plasticité du cortex visuel du chaton: un réexamen. *CR Acad Sci Paris III* 295: 745-750

Bear MF, Daniels JD (1983) The plastic response to monocular deprivation persists in kitten visual cortex after chronic depletion of norepinephrine. *J Neurosci* 3: 407-416

Bear MF, Singer W (1986) Modulation of visual cortical plasticity by acetylcholine and noradrenaline. *Nature* 320: 172-176

Carlson M, Hubel DH, Wiesel TN (1986) Effects of monocular exposure to oriented lines in monkey striate cortex. *Dev Brain Res* (in press)

Cynader M, Mitchell DE (1977) Monocular astigmatism effects kitten visual cortex development. *Nature* 270: 177-178

Daw NW, Rader RK, Robertson TW, Ariel M (1983a) Effects of 6-hydroxydopamine on visual deprivation in the kitten striate cortex. *J Neurosci* 3: 907-914

Daw NW, Robertson TW, Rader RK, Videen TO, Cosica CJ (1984) Substantial reduction of cortical norepinephrine by lesion of adrenergic pathway does not prevent effects of monocular deprivation. *J Neurosci* 4: 1354-1360

Daw NW, Videen TO, Rader RK, Robertson TW, Cosica CJ (1985) Substantial reduction of noradrenaline in kitten visual

cortex by intraventricular injections of 6-hydroxydopamine does not always prevent ocular dominance shifts after monocular deprivation. *Exp Brain Res* 59: 30-35

Daw NW, Videen TO, Parkinson D, Rader RK (1985) DSP-4 (N-(2-chloroethyl)-N-ethyl-2-bromobenzylamine) depletes noradrenaline in kitten visual cortex without altering the effects of monocular deprivation. *J Neurosci* 5: 1925-1933

Daw NW, Videen TO, Robertson TW, Rader RK (1985) An evaluation of the hypothesis that noradrenaline affects plasticity in the developing visual cortex. In: *The visual system*. AR Liss, New York, pp 133-144

Hubel DH (1957) Tungsten microelectrode for recording from single units. *Science* 125: 549-550

Hubel DH, Wiesel TN (1962) Receptive fields, binocular interaction and functional interaction in the cat's visual cortex. *J Physiol (Lond)* 160: 106-154

Hubel DH, Wiesel TN (1970) The period of susceptibility to the physiological effects of unilateral eye closure in kittens. *J Physiol (Lond)* 206: 419-436

Kasamatsu T, Pettigrew JD (1976) Depletion of brain catecholamines: failure of ocular dominance shift after monocular occlusion in kittens. *Science* 194: 206-208

Kasamatsu T, Pettigrew JD (1979) Preservation of binocularity after monocular deprivation in the striate cortex of kittens treated with 6-OHDA. *J Comp Neurol* 185: 139-162

Kasamatsu T, Pettigrew JD, Ary M (1979) Restoration of visual cortical plasticity by local microperfusion of norepinephrine. *J Comp Neurol* 185: 163-182

Kasamatsu T, Shirokawa T (1985) Involvement of  $\beta$  adrenoreceptors in the shift of ocular dominance after monocular deprivation. *Exp Brain Res* 59: 507-514

Movshon JA, Duersteler MR (1977) Effects of brief periods of unilateral eye closure on the kitten visual system. *J Neurophysiol* 40: 1255-1265

Olson CR, Freeman RD (1975) Progressive changes in kitten striate cortex during monocular vision. *J Neurophysiol* 38: 26-32

Paradiso MA, Bear MF, Daniels JD (1983) Effects of intracortical infusion of 6-hydroxydopamine on the response of kitten visual cortex to monocular deprivation. *Exp Brain Res* 51: 413-422

Rauschecker JP, Singer W (1979) Changes in the circuitry of the kitten visual cortex are gated by postsynaptic activity. *Nature* 280: 58-60

Rauschecker JP, Singer W (1981) The effects of early visual experience on the cat's visual cortex and their possible explanation by Hebb synapses. *J Physiol (Lond)* 310: 215-239

Shaw C, Cynader M (1984) Disruption of cortical activity prevents ocular dominance changes in monocularly deprived kittens. *Nature* 308: 731-734

Stryker MP, Harris WA (1986) Binocular impulse blockade prevents the formation of ocular dominance columns in the cat visual cortex. *J Neurosci* 12: 2117-2133

Vidyasagar TR, Heide W (1984) Geniculate orientation bias seen with moving sine wave gratings: implications for a model of simple cell afferent connectivity. *Exp Brain Res* 57: 196-200

Wiesel TN, Hubel DH (1963) Effects of visual deprivation on morphology and physiology of cells in the cat's lateral geniculate body. *J Neurophysiol* 26: 978-993