from treated animals. No parasites or parasite debris were seen 24 hours after treatment.

Mice that were infected with *T. b. rhodesiense* and that showed both the LS forms and SS forms of the parasite in the blood were also injected with SHAM and glycerol. One hour after treatment only a monomorphic SS form was present in the blood, and after 24 hours no parasites were observed.

Six days after the treatment of rats and mice infected with *T. b. brucei*, the parasites reappeared in the blood and the animals died within a few days. Without treatment, the animals would have died within a few hours. Mice infected with *T. b. rhodesiense* also showed a recurrence of parasitemia after treatment. We have found no regimen of treatment that prevents the recurrence of parasitemia, although the response of the parasite population to successive treatments remains the same and the parasites do not appear to become resistant to the treatment. Two possible explanations for the recurrence are (i) the entire population of parasites contains a few resistant cells with a partial TCA cycle, and (ii) the effective trypanocidal levels of SHAM and glycerol are not reached in some tissues and parasites in those tissues survive. The second hypothesis seems more probable, because temporal separation of SHAM and glycerol administration by 5 minutes abolishes their therapeutic value, suggesting that they are cleared very rapidly from the blood. By finding a substitute for glycerol or a different method of administration, it might be possible to maintain therapeutic levels of the drugs long enough for all infected tissues to be reached. Similarly, other hydroxamic acids or iron chelators might be more effective in blocking the activity of glycerophosphate oxidase.

The results presented here promise a rational approach to trypanosome chemotherapy based on knowledge of the peculiar carbohydrate catabolic pathways of these parasites.

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13. We thank A. Balber and C. Patton for sharing their observation that in the presence of SHAM *T. b. brucei* glucose transport is inhibited by glycerol causing loss of motility and cell death in vitro. We thank M. R. Rifkin for her cloned isolate of *T. b. brucei* EATRO strain 110 from which we derived the monomorphic strain, and W. Trager, D. M. Dwyer, and P. A. D'Alesandro for their encouragement and criticism. This work was supported by training grant AI-00192 from the U.S. Public Health Service.

21 May 1976; revised 6 July 1976

Depletion of Brain Catecholamines: Failure of Ocular Dominance Shift After Monocular Occlusion in Kittens

Abstract. *Monocularly deprived kittens were compared with littermates that had had their eyelids sutured for the same time but that had, in addition, been treated with 6-hydroxydopamine to deplete their forebrains of catecholamines. The visual cortices of all the catecholamine-depleted kittens showed high proportions of binocular neurons, in contrast to the control group, most of whose visual cortical neurons were driven exclusively by the nondeprived eye. Catecholamines may play an important role in the maintenance of cortical plasticity during the critical period.*

A valuable paradigm for the effects of experience on brain function is the change in binocularity of kitten visual cortical neurons which occurs after eye occlusion. Monocular visual experience results in increased numbers of neurons which respond exclusively to the previously open eye, with a loss of the normal, binocularly activated neurons. These changes in ocular dominance are virtually permanent (*1*), occur rapidly (*2*), and are confined to a well-defined postnatal period of susceptibility or "critical period" (*3*). A key question concerns the factors which determine the beginning and end of this period of neural plasticity.

In this report we present a preliminary test of the hypothesis that the catecholamine neurohormones are required for the maintenance of visual cortical plasticity during the critical period. Our research was stimulated both by the hypotheses of Kety (*4*) and Crow and coworkers (*5*) that the brainstem monoamine pathways are involved in the forebrain's plasticity, and by recent work linking the monoamines with brainstem pathways which have powerful effects on the visual pathway (*6*), and which appear to mature during the critical period (*7*). To examine the role of catecholamines we used 6-hydroxydopamine (6-OHDA), a specific neurotoxin that is taken up from the cerebrospinal fluid by axon terminals which contain norepinephrine or dopamine and results in their destruction (*8*). Electrophysiological recording supports the hypothesis to the extent that the usual changes in binocularity of cortical neurons do not follow monocular occlusion in kittens whose cortices have been depleted of catecholamines by 6-OHDA.

Four pairs and one trio of littermates were obtained from our quarantined cat colony, a partially inbred line of tabby cats. Each pair included a control and an experimental kitten, and the trio (JT4, JT2, and TJ12; see Table 1) included two different kinds of controls as well as an experimental animal. Each of the 11 kittens had a fine stainless steel cannula with trocar implanted in the right lateral ventricle under ketamine anesthesia (30 to 40 mg/kg) during the fourth to seventh week after birth. The right eyelid of all kittens was sutured under Fluothane anesthesia (for rapid recovery) during the same period, some time after ventricular cannulation (Table 1). Each procedure was timed to coincide as closely as possible for both members of an experimental-control pair (Table 1).

Using the permanently implanted cannula as a guide, we injected into the ventricle a dose of 6-OHDA plus vehicle (16 μg of 6-OHDA per microliter of 0.05 to 0.1 percent ascorbic acid in Ringer solution), or the vehicle solution alone (*9*). Our choice of dose was guided by previous studies on neonatal rats and adult cats (*10*) and by close observation of the behavioral effects following injection (*11*). Because we do not have reliable data on the lower limits of catecholamine levels achieved by our treatment (*12*), and because some evidence indicates that the nerve terminals recover rapidly after cessation of treatment (*13*), we gave repeated large injections for more than 1 week. For example, 200 μg of 6-OHDA

was injected on day 1 and the dose was doubled each day thereafter until the daily dose reached 1.6 mg on day 4. The daily dose was then kept at or below this level until a total dose of about 10 mg had been administered (Table 1 and Fig. 1G). With these doses there were clear behavioral effects in the experimental group, including a rage reaction to slight provocation, pupillary constriction, compulsive turning to the left (*11*), and, on rare occasions, gross seizures (*14, 15*). The experimental group was sometimes handfed with milk to maintain a normal curve for the increase in body weight. In one of the control animals (JT2 in Table 1) we injected 5,7-dihydroxytryptamine (5,7-DHT) which has a small effect on catecholamine-containing terminals but destroys those containing the indoleamine transmitter, serotonin (*16*).

One to two weeks after eye occlusion, the kittens were prepared for single unit recording with standard techniques (*17*). Preparation was routine except that when the anesthetic was first administered we noticed that the experimental group was more sensitive to anesthesia and could be adequately maintained on 0.5 percent Fluothane or less (as opposed to 1 to 1.5 percent Fluothane required for the controls). Single neurons were recorded extracellularly with Levick's tungsten-in-glass microelectrodes (*18*) which were driven down the medial bank of the left postlateral gyrus. Recording sessions lasted about 20 hours. Each neuron was assigned to an ocular dominance group from one to seven based on the criteria of Hubel and Wiesel (*19*) (see Fig. 1, E and F). Electrolytic lesions were made for later reconstruction of the electrode tracks from frozen lesions stained with cresyl violet (Fig. 1, C and D).

The principal finding was that closure of the right eye failed to modify the binocularity of cortical neurons in each experimental kitten. All of the 6-OHDA–treated animals had significant numbers of binocularly activated neurons and none showed the strong bias toward neurons driven exclusively by the left eye which was apparent for each member of the control group. This is shown in Table 1, where experimental details and ocular dominance data are given for all 11 kittens, and Fig. 1, where we have picked out a particular experimental (TJ9)-control (TJ11) pair to show the result in detail. Some variation in the degree of preservation of binocularity can be traced to differences in the dose of 6-OHDA and the time of the first injection with respect to that of eye occlusion. For example, the neurons of TJ3 show a slight bias toward the left eye which may reflect the fact that eye occlusion was started with the first injection and the kitten may therefore have had 1 or 2 days of monocular experience before the 6-OHDA was exerting its full effect. Similarly, both kittens (TJ3 and TJ12) receiving lower doses of 6-OHDA show a slight left eye bias. In addition to the marked differences in ocular dominance distributions shown in Fig 1, E and F, we also observed differences (not shown) between the two preparations in the selectivity of neurons for the orientation of the stimulus. Kitten TJ9 tended to have larger numbers of the nonselective neurons characteristic of young or visually inexperienced kittens (*20*). We believe that the nonselective and nonresponsive cells may in fact be further evidence that catecholamine depletion prevents visual experience from exerting its usual effect on cortical development because the numbers of such neurons normally decrease as a result of visual experience (*20*). Moreover, treatment of a normal adult with 6-OHDA did not result in the appearance of nonresponsive or nonselective neurons (*21*).

We think it most likely that the striking effect we have observed is a product of catecholamine depletion rather than some nonspecific effect of 6-OHDA. The occurrence of seizures in some animals could be of concern in this regard, but a role for this side effect appears to be ruled out by two observations. First, all of the experimental animals, except for TJ9, failed to show seizures and yet remained binocular after eye occlusion. Second, the indoleamine-depleting agent, 5,7-DHT, can produce seizures, yet was associated with perhaps the greatest shift in ocular dominance after eye occlusion (JT2; Table 1). This differential effect of 6-OHDA versus 5,7-DHT further supports the thesis that the failure of cortical modification is attributable specifically to catecholamine depletion, because the two neurotoxins appear to have similar modes of action and

Table 1. Summary of data for the 11 kittens grouped as four pairs and one trio. Numbers under the entry of ocular dominance refer to cells in one of three categories, that is, binocular, groups 2 to 6 of Hubel and Wiesel's classification (*19*); left eye, group 7; and right eye, group 1. The total number of cells recorded was 156 in the controls and 175 in the experimental kittens. The columns for day of injection indicate the first day and the last day after birth on which injections were given; the column for days of occlusion indicate the days after birth during which the right eye was occluded.

Cat, sex	Body weight (g)		6-OHDA			Days of occlusion	Ocular dominance		
	At first injection	At recording	Total dose (mg)	Days of injection			Binocular	Left eye	Right eye
				First	Last				
TJ1* F	800	945				47 to 61	8	27	6
TJ2 M	730	550	8.7	47	55	47 to 58	38	8	2
TJ4 M	240	260				32 to 40	4	15	1
TJ3 M	300	290	5.0	32	38	32 to 39	16	16	5
TJ5† M	365	250	2.2	35	38	39 to 48	1	5	0
TJ6 F	325	295	13.4	35	47	39 to 47	16	3	6
TJ11* M	425	570				39 to 47	7	23	0
TJ9 M	490	515	12.4	33	44	39 to 46	23	3	3
JT4* F	380	660				35 to 44	13	14	0
JT2‡ M	405	580	(3.5)	(28)	(35)	35 to 43	2	30	0
TJ12 M	415	580	7.0	28	35	35 to 42	24	11	1
Total control							35	114	7
Total experimental							117	41	17

*TJ1, TJ11, and JT4 were recovered and reverse-sutured (*15*). †The small sample size in TJ5 was due to the accidental loss of this kitten during the recording session. ‡JT2 was treated with 5,7-DHT in place of 6-OHDA. In this kitten, the behavioral changes were distinct from those characteristic of 6-OHDA–treated kittens except for a trend toward seizures (of the grand mal type) at high doses of 5,7-DHT. For example, kittens treated with 5,7-DHT had dilated pupils (as opposed to constriction of the pupils in the 6-OHDA–treated group) and tended to seek a hiding place after injection in contrast to the compulsive turning shown by the 6-OHDA–treated group.

differ principally in the transmitter system each affects (*8*, *16*). We have additional controls that appear to rule out the role of other side effects of 6-OHDA in maintaining binocularity (*15*). Particularly convincing is the observation on TJ12 who shows significant binocularity (67 percent) after a period of eye occlusion which began after the injections of 6-OHDA had ceased and therefore after the behavioral side effects had disappeared.

In conclusion, we think that the catecholamine neurohormones may play a major role in the maintenance of cortical plasticity. We do not know which of the two catecholamines, dopamine or norepinephrine, is more important for this effect, nor whether it is possible to enhance plasticity outside of the "critical period" by appropriate treatment.

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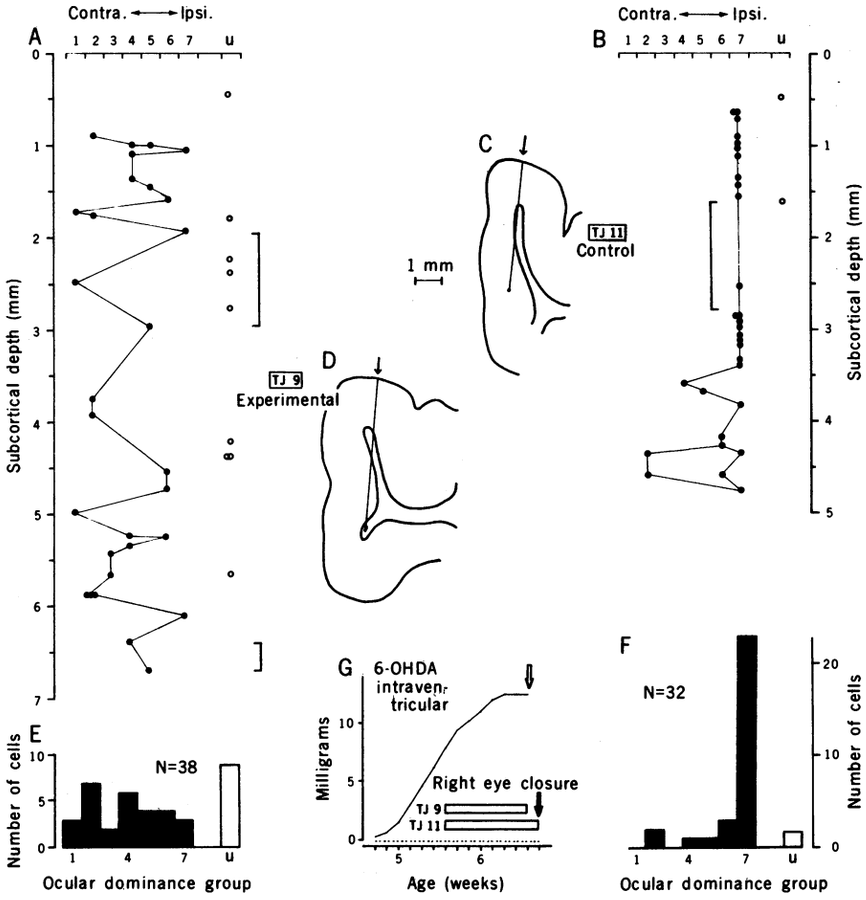

Fig. 1. Diagrams of binocularity of visual cortical cells in a pair of kittens (TJ9 that was treated with 6-OHDA and TJ11 that was a control) showing the failure of ocular dominance shift after eye occlusion in the kitten treated with 6-OHDA. (A and B) The location of individual cells from the cortical surface was plotted successively on the ordinate (in millimeters). The binocularity of each cell was scored semiquantitatively as one of seven groups according to Hubel and Wiesel (*19*) (abscissas). Briefly, the group 1 cell can be excited only through the contralateral eye. The group 7 cell can be excited only through the ipsilateral eye. The group 4 cell is influenced equally by both eyes. The group U (unresponsive) to the right of group 7 designates visually unresponsive cells encountered in the same electrode track. After monocular occlusion the majority of cells in TJ11 responded, as expected, exclusively to ipsilateral eye stimulation (group 7), whereas only 3 of 29 visually responsive cells (closed circles) in TJ9 were found in group 7. Open circles are for visually unresponsive cells. The incidence of binocular cells is also higher in TJ9 than in TJ11. These differences are not attributable to sampling bias, as shown by the electrode tracks (see D and C, respectively). Although there was a certain difference in the sampling efficiency between TJ9 (180 μm per cell, electrode track 7 mm in length) and TJ11 (118 μm per cell, electrode track 5 mm in length) both electrode tracks were in the projection area of the central gaze and (see diagrams in C and D) both tracks had chances to cross ocular dominance columns several times except for the white matter (indicated by brackets in A and B). The high proportion of visually unresponsive cells and lower sampling efficiency in TJ9 as opposed to TJ11 was not observed in other experimental kittens. Recording sites with at least one neuron (and sometimes two or three) were separated by a mean of 114 μm for four control kittens. The numbers of unresponsive neurons for the other experimental kittens TJ2, TJ3, TJ6, and TJ12 were, respectively, 3, 1, 2, and 2. Lines indicate recording sequence. (C and D) Drawings of two frontal sections containing the lesions at the end of track. The orientation of penetration was angled 5° medially and anteriorly from the vertical. (E and F) Ocular dominance histograms for TJ9 and TJ11, respectively. The difference in experimental and control animals is apparent. (G) The curve of the cumulative dose (solid line) of 6-OHDA administered intraventricularly into TJ9 plotted against the age of the animal. The daily dose of 6-OHDA for TJ9 started with 200 μg, and was doubled every day until it reached some upper limit determined empirically (1.6 mg) on day 4 after the start of drug injection. The total dose for 2 weeks was 12.4 mg. Kitten TJ11 (control) received the same volume of the vehicle only (dashed line). The timing and duration of monocular eye closure (right) was indicated by open squares at the bottom. The cortical recording was made on postnatal day 46 for TJ9 (open arrow) and day 47 for TJ11 (solid arrow).

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6. The link between catecholamines and specific brainstem pathways to the visual system is supported by the following pieces of evidence: (i) Single neurons in the visual pathway show changes in excitability at the time of PGO (ponto-geniculo-occipital) waves, which originate in the pons synchronously with saccadic eye movements [E. V. Evarts, *J. Neurophysiol.* **25**, 812 (1962); H. Sakakura and K. Iwama, *Proc. Jpn. Acad.* **42**, 418 (1966); E. Bizzi, *J. Neurophysiol.* **29**, 1087 (1966); H. Sakakura, *Jpn. J. Physiol.* **18**, 23 (1968); R. W. McCarley and J. A. Hobson, *Science* **167**, 901 (1970); T. Kasamatsu and W. R. Adey, *Brain Res.* **55**, 323 (1973); T. Kasamatsu, *ibid.*, in press]. (ii) The anatomical structures responsible for PGO waves, though not yet well defined, largely overlap with the ascending catecholaminergic projections from the locus coeruleus and the nucleus subcoeruleus of the pons [M. Jouvet and F. Michel, *C. R. Soc. Biol.* **153**, 422 (19→ T. Maeda, C. Pin, D. Salvert, M. Ligier, M. Jouvet, *Brain Res.* **57**, 119 (1973); J.-P. Laurent, R. Cespuglio, M. Jouvet, *ibid.* **65**, 29 (1974)]. (iii) Catecholaminergic neurons in the locus coeruleus show firing correlated with PGO waves [N.-S. Chu and F. E. Bloom, *Science* **179**, 908 (1972)]. Moreover, conditioning stimulation of the locus coeruleus enhances the responsiveness of single geniculate neurons to the optic tract stimulation. This facilitation is not observed after catecholamine depletion with reserpine and is reduced in cats treated with dopamine-β-hydroxylase inhibitor to reduce norepinephrine. Both of these effects are reversed by intraventricular injection of norepinephrine [Y. Nakai and S. Takaori, *Brain Res.* **71**, 47 (1974)]. (iv) High levels of catecholamine, especially norepinephrine, are correlated with the occurrence of PGO waves in the visual system [A. Buguet, F. Petitjean, M. Jouvet, *C. R. Soc. Biol.* **164**, 2293 (1970); R. Laguzzi, F. Petitjean, J. F. Pujol, M. Jouvet, *Brain Res.* **48**, 295 (1972)]. There is also recent evidence for innervation of the cerebral cortex by dopamine, another important catecholamine in the brain [A. M. Thierry, G. Blanc, A. Sobel, L. Stinus, J. Glowinski, *Science* **182**, 499 (19→ A. M. Thierry, L. Stinus, G. Blanc, J. Glowinski, *Brain Res.* **50**, 230 (1973); T. A. Reader, J. De Champlain, H. Jasper, *ibid.*, in press; M. Fossel, M. Ptito, M. C. Lassonde, K. H. Pribram, *Neurosci. Abstr.* **1**, 191 (1975)].
7. The gradual increase in the concentration of brain monoamines such as norepinephrine and

serotonin reaches a peak around 4 weeks postnatally in cats [W. A. Himwich, in *Sleep and the Maturing Nervous System*, C. D. Clemente, D. P. Purpura, F. E. Mayer, Eds. (Academic Press, New York, 1972), p. 125]. This approximately coincides with the start of the critical period for consolidation of binocularity in kitten visual cortex [see (*3*)]. Furthermore, the mean frequency of PGO waves in kitten geniculate nucleus (in REM sleep) increases sharply during the third and fourth weeks. No PGO waves are observed before postnatal day 15 [C. Bowe-Anders, J. Adrien, H. P. Roffwarg, *Exp. Neurol.* **43**, 242 (1974)].

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9. Eight milligrams of 6-OHDA · HBr (Sigma) was dissolved in 0.5 ml of Ringer solution containing 250 to 500 μg of ascorbic acid to give a final concentration of 16 μg/μl of 6-OHDA. The *p*H was about 6.0. The low *p*H and presence of ascorbic acid prevent the auto-oxidation of 6-OHDA which turns the color of the solution reddish-brown on contact with air. The solution was kept frozen at −20°C.
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11. The first sign of drug effects began 2 days after the start of 6-OHDA injection for TJ9. This was a sham rage reaction to the sudden presentation of any sort of sensory stimulus [K. Nakamura and H. Thoenen, *Psychopharmacologia* **24**, 359 (1972); L. J. Poirier, P. Langelier, A. Roberge, R. Boucher, A. Kitsikis, *J. Neurol. Sci.* **16**, 401 (1972)]. This reaction may be due to the acute release of catecholamines from degenerating catecholamine terminals since it is prevented by prior treatment with α-methyl-*p*-tyrosine, a specific inhibitor of the rate-limiting enzyme in catecholamine synthesis, tyrosine hydroxylase [K. Nakamura and H. Thoenen, *Psychopharmacologia* **24**, 359 (1972)]. On day 4 after injecting 6-OHDA, we noted pupillary constriction of both eyes and a poor light reflex. On the next day, there was a compulsive rotation of the body toward the left (the side opposite injection), after the injection of 6-OHDA. It was very fast (40 rotations per minute, if the rotation took place on the same spot) and lasted for 30 minutes or so with a regular break every 0.5 to 1 minute. This rotation may be explained as an effect of 6-OHDA on the dopamine receptors in the caudate nucleus at the floor of the lateral ventricle near our injection site [U. Ungerstedt, in *The Neurosciences, Third Study Program*, F. O. Schmitt and F. G. Worden, Eds. (MIT Press, Cambridge, Mass., 1974), p. 695]. When the animal was held during rotation he showed large amplitude nystagmus whose quick phase was in the same direction as body rotation. This rotation response to injection of 6-OHDA started as early as the third day in some kittens. Gross behavioral manifestations such as compulsive rotation and the prodromata of seizures (twitching of ear tips, whiskers, and eyelids) were confined to the time of injection. In between injections, the 6-OHDA–treated kittens showed normal visual placing and following reactions, and both the experimental and control kittens spent comparable periods of time alert, awake, and interacting with the environment. While it was sometimes possible for one of us to distinguish the experimental from the control animal between injections, on the basis of his more sluggish pupillary response or a tendency to be "jumpy," there were no gross differences in behavior which might alter the amount of visual experience obtained by the experimental kittens. In other words there were no indications that 6-OHDA–treated kittens suffered from a decreased visual input.
12. Nissl-stained slides did not show any gross changes in the visual cortex of 6-OHDA–treated kittens.
13. One indirect measure of the activity of the catecholamine pathway may be the frequency of PGO waves [see (*6*)]. When 6-OHDA is administered, we have found that the frequency of PGO waves in rapid-eye-movement sleep drops sharply. However, high doses are required to maintain the suppression and PGO wave frequency returns to control levels 1 week after stopping the 6-OHDA injections. Further evidence for rapid recovery of catecholamine terminals may be found in a recent review [R. Y. Moore, A. Björklund, U. Stenevi, in *The Neurosciences, Third Study Program*, F. O. Schmitt and F. G. Worden, Eds. (MIT Press, Cambridge, Mass., 1974), p. 961].
14. The total amount of 6-OHDA given to TJ9 was 12.4 mg. After the accumulated dose attained 11.0 mg on the tenth day, the kitten started to show some signs of seizure such as hypersalivation, widely dilated pupils, twitches of whiskers, and blinks and jaw movements which were followed finally by mewing. Another dose of 1.0 mg on the next day made the situation worse resulting in a fit of the grand mal type involving the whole body [G. Chen, C. R. Ensor, B. Bohner, *Proc. Soc. Exp. Biol. Med.* **86**, 507 (1954); A. Lehmann, *Life Sci.* **6**, 1423 (1967); K. Schlesinger, W. Boggan, D. X. Freedman, *ibid.* 7, 437 (1968)]. As shown by a sudden drop of body weight, the general condition of TJ9 deteriorated, but he recovered on cessation of drug treatment. This seizure occurred in several episodes after the injection. Seizures were not observed in any of the other 6-OHDA–treated kittens in this report, although we have since observed them after high doses of 6-OHDA in another study (*15*).
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17. Briefly, the kitten was anesthetized (N_2O, O_2, CO_2; 75 : 27.5 : 2.5 by volume, respectively) and immobilized (Flaxedil, 5 mg/hour). A small amount of dexamethasone (0.5 mg/hour) was added to the infusion solution. Body temperature and the rate of heartbeats were monitored continuously. The cornea was covered by contact lenses of zero power. The background illumination was kept at the photopic level and the brightness of visual stimuli was at about 1 to 2 log units above it. The pupil was dilated by topical application of Cyclogyl (1 percent). Visual stimuli were presented on a tangent screen at 57 cm from the animal's eye by a specially designed rear projection system. A joystick controlled the movement of images in the *X* and *Y* axes as well as their rotation.
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20. J. D. Pettigrew, *ibid.* **237**, 49 (1974); C. Blakemore and R. C. Van Sluyters, *ibid.* **248**, 663 (1975).
21. We have also tested the effects of short- and long-term administration of 6-OHDA on the visual response properties of neurons in normal kittens and adults (*15*). 6-OHDA had little effect on the few neurons we studied both before and after injection. Long-term administration in normal visually experienced animals (in contrast to very young kittens) has little effect on binocularity and appears to have subtle effects which include a general increase in the sharpness of orientation tuning. This latter effect is difficult to understand but it at least supports our interpretation that the direct effects of 6-OHDA on neuron response properties play a minor role in comparison to its effects in reducing the sensitivity of the cortex to monocular deprivation.
22. Supported by NIMH grant MH25852 and by the Spencer Foundation. We thank Lederle Laboratories for providing us with Flaxedil.

1 March 1976; revised 7 June 1976

Responses of Infants to Visually Presented Objects

Abstract. *The reaching behavior of some 60 infants between 7 and 23 days of age was studied. Contrary to some other reports, the infants did not respond differently to a visually presented, graspable, solid object than to its two-dimensional representation.*

Recent advances in the study of perception of very young infants have revealed abilities undreamed of 10 years ago (*1*). It has been claimed, with some justification, that the more sophisticated the methods of investigation have become, the more the infant's perceptual capacities have apparently grown (*2*). The study of infant perception is not a new phenomenon, but earlier evidence on perceptual development (*3*) generally supported the view that the initial stages of perceptual activity are diffuse and poorly articulated, and that intersensory coordination, particularly between vision and touch, develops slowly over time.

Bower (*4*) has pointed out that demonstrating an infant's ability to make visual discriminations does not necessarily tell us anything about its apprehension of the nature of the distal (physical) stimulus, for example, whether it is a real object. He argues that investigating an ecologically valid response—one that has utility for the organism, such as grasping at small objects presented visually and not too far from the body—would give more information about the infant's actual perceptual world. He reported that infants less than 2 weeks old do differentiate with an appropriate gesture between graspable and nongraspable objects presented visually (*4*). This finding is so revolutionary and goes against such a well-established tradition in perceptual psychology that it should be validated. We therefore planned to replicate Bower's experiment and then to investigate the conditions under which visually guided reaching develops with respect to speed and precision.

Table 1. Comparison of Bower's results (*4*) with those of experiment 1. Infants 7 to 23 days old were exposed for 4 minutes each to either an object (O) or its two-dimensional representation (picture) (P). Entries are the mean number of responses per infant in each observation period for each target.

Experiment	Contacts		Reaches	
	O	P	O	P
Bower	12.0	0.0	53.0	0.5
This report	0.46	0.39	2.2	1.9