percent of the total incubation period. By contrast, the earliest measured retinally mediated response (the pupillary reflex) does not occur until day 15 (*4*).

A potential postnatal function of such sensitivity, if it is in fact maintained into adulthood, could be involved in the homing ability of pigeons. We are presently engaged in determining how widespread this form of photic sensitivity is in birds, but have thus far clearly demonstrated it only in the pigeon (*11*). Perhaps such a nonvisually mediated photic sensitivity aids in the orientation to the home loft. Experiments in which adult pigeons still return to the loft area when their visual input has been interfered with might be partially accounted for by this mechanism (*12*).

MARSHALL STEPHEN HARTH*
MARIETA BARROW HEATON
Neuroembryology Laboratory, Research Division, North Carolina Department of Mental Health, Raleigh 27611

References and Notes

1. M. Menaker, *Biol. Reprod.* **4**, 295 (1971).
2. A. V. Bursian, *Bull. Exp. Biol. Med. (Transl.)* **58**, 7 (1964).
3. G. Gottlieb, *Quart. Rev. Biol.* **43**, 148 (1968); M. B. Heaton, *Develop. Psychobiol.* **4**, 313 (1971).
4. M. B. Heaton and M. S. Harth, *J. Comp. Physiol. Psychol.*, in press.
5. W. J. Miller and L. S. Miller, *Anim. Behav.* **6**, 3 (1958); M. S. Harth, unpublished observations.
6. M. S. Harth, in preparation.
7. A second group of hatchlings was used to control for the possibility that the response was actually due to the slight temperature change induced by the stimulus light. An infrared heat lamp, creating a temperature change of 0.56°C in 5 seconds (greater than the change during light stimulation), was employed for this purpose. This heat lamp was positioned in the same relation to the hatchlings as the stimulus light had been, and trials were conducted in the same manner. No significant changes in response latency were found under these test conditions. The wavelengths of the heat and stimulus lamps were different, and whether wavelength plays any role in the pigeon's sensitivity to light requires further investigation.
8. N. Millott, *Symp. Zool. Soc. London No. 23* (1968), p. 1; A. Roberts, *Nature* **222**, 1265 (1969).
9. H. E. Becker and R. A. Cone, *Science* **154**, 1051 (1966).
10. For example, hatching occurs earlier in embryos that are artificially exposed to light stimulation prior to the onset of visual function [J. V. Shutze *et al.*, *Nature* **196**, 594 (1962); J. K. Lauber and J. V. Shutze, *Growth* **28**, 179 (1964); P. B. Siegel *et al.*, *Comp. Biochem. Physiol.* **28**, 753 (1969)].
11. M. B. Heaton and M. S. Harth, in preparation.
12. W. T. Keeton, paper presented at XIIth International Ethological Congress, Edinburgh (1971).
13. Supported by postdoctoral fellowships HD25527 and HD46944 from the National Institute of Child Health and Human Development. We thank Dr. R. W. Oppenheim for critical comments and helpful suggestions. Technical assistance from Dr. R. Foelix, L. Harth, and V. Riley is gratefully acknowledged.

* Present address: Ramapo College of New Jersey, School of Theoretical and Applied Science, P.O. Box 542, Mahwah 07430.

6 November 1972

Prism Adaptation: Control of Intermanual Transfer by Distribution of Practice

Abstract. *Correction of errors in localizing movements produced by laterally displacing vision by means of wedge prisms has been termed "prism adaptation." Intermanual transfer of prism adaptation from an exposed to an unexposed hand, with subject's head immobilized, has been reported not to occur. However, it was found that a standard learning variable, distribution of practice, controls the occurrence or nonoccurrence of transfer. When practice is massed, there is no transfer of adaptation; when it is spaced, the transfer is extensive. Spacing of practice also influences the amount of aftereffect displayed by the exposed hand.*

When wedge prisms are placed before the eyes, vision is displaced in the direction of the apex of the prisms. As a result, visually guided movements deviate at first to that same side. Under suitable conditions, the initial mislocalization can be corrected; this is termed "prism adaptation." When the prisms are then removed, this correction in orientation (now inappropriate) persists for a time, leading to errors in the opposite direction. The latter has been termed "prism aftereffect."

It has been reported that prism adaptation does not transfer from an exposed arm to an unexposed arm if the head is immobilized during the prism-exposure period (*1–5*). In contrast, several other studies have indicated that intermanual transfer can take place in the absence of head movement (*6–10*). An analysis of adaptation to sensory rearrangement in terms of a learning approach (*11*) allows us to account for this discrepancy.

In all experiments in which intermanual transfer has unambiguously occurred, a prism-exposure condition involving "terminal display" was employed; that is, the subject first pointed at a visual target with an unseen arm and was then allowed to view the terminal position of his responding finger in relation to the target. On the other hand, in the experiments of Hamilton (*2*) and of Mikaelian (*4, 5*), in which intermanual transfer with head immobilized was not observed, the prism-exposure period involved "continuous display," in which (i) the subject was provided with continuous view of self-produced hand movements and (ii) there were no targets to be pointed at; that is, the subject had no tasks other than to view his hand moving back and forth within a structureless visual field.

A number of explanations have been advanced for the disparity in results between the two sets of conditions, but none have been able to account adequately for all of the data (*12*). However, analysis of the two experimental designs within a learning context reveals that continuous display could be considered a type of massed-practice procedure. That is, the subject continually views his moving hand and is therefore being continuously trained. In contrast, the terminal-display procedure is, by definition, a spaced-practice procedure; that is, the hand is not always in view and, thus, training is not continuous.

If this difference in distribution of practice were responsible for the two conflicting sets of data, it follows that it should be possible to obtain intermanual transfer with a continuous-display procedure by introducing rest or blackout periods between periods of training, thereby spacing the practice. We report here confirmatory evidence.

The procedure involved measurement of the accuracy of pointing at visual-target positions with unaided vision before and after viewing self-produced, continuous movements of a hand through a 20-diopter prism which displaced vision 11.3° laterally. The hand could be seen only during the prism-exposure period and not when accuracy of pointing was tested. Half of the subjects (the nontransfer groups) were tested with the same hand that had been viewed through the prism, while the remaining subjects (the transfer groups) were tested with the unexposed hand. All testing and viewing were with the right eye (left eye occluded) and with the head stabilized by a biteboard. The deviation from baseline pointing values after prism removal provided a measure of the prism aftereffect.

Subjects (96 female undergraduates) were given one of three treatments during the exposure period. The first treatment consisted of spaced practice in contrast to the massed practice employed in previous continuous-display

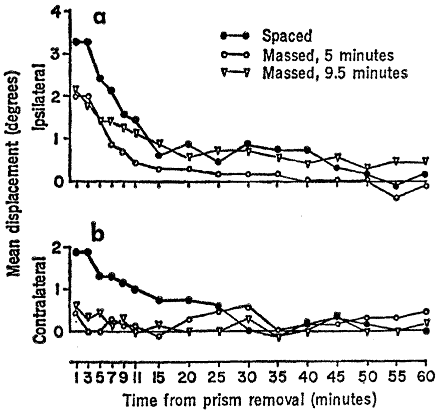

Fig. 1. Decay of prism aftereffect for ipsilateral and contralateral (transfer) testing following spaced and massed practice during prism exposure. Testing was always with the right arm. During prism exposure either the right arm (ipsilateral group) or left arm (contralateral group) was viewed. Each data point is the mean for ten trials each for 16 subjects. Magnitude of aftereffect was measured 1, 3, 5, 7, 9, 11, and 15 minutes after prism exposure, and every fifth minute thereafter through minute 60.

studies. View of the moving limb was restricted to ten 30-second periods during which the subject moved her arm in time with a metronome (90 beats per minute). These practice periods were alternated with 30-second intervals of rest, during which the subject sat in complete darkness without moving her arm. Thus, the entire exposure period lasted for 9.5 minutes, of which 5 minutes (ten 30-second periods) were used for practice. Exposure for subjects in the second treatment was similar to that in other experiments using continuous display (*2, 4, 5, 13*). The arm was viewed constantly as it was moved from side to side in time with the metronome for 5 minutes. These subjects received the same amount of training as did the groups with spaced practice; however, since there were no rest intervals, the exposure period lasted only 5 minutes. In a third treatment, exposure was also massed, but in this case it was for 9.5 minutes, the total length of the practice session for the first treatment. This group was included to control for the possibility that differences between the first two treatments might be due to the total amount of time in the exposure period. Thus, there were one spaced treatment and two massed treatments. For each treatment, there was one transfer group and one nontransfer group. The prism was oriented with the base to the right for half of each group and to the left for the remaining subjects (*14*). Significant adaptation took place for all three nontransfer (ipsilateral) groups (Table 1). However, transfer of adaptation was significant only for the spaced-practice condition, in which it was 59 percent of the ipsilateral aftereffect. For the two massed treatments, transfer was not significant ($P > .05$). Another finding of interest was that for ipsilateral testing, the initial aftereffect for the group with spaced practice was significantly greater than it was for either of the groups with massed practice ($P < .05$ in both cases) (*15*).

The decay functions for aftereffects during 60 minutes after prism removal are presented in Fig. 1. For transfer testing (Fig. 1b), the decay function for the first 20 minutes for the spaced-practice group was different from those of the two massed-practice groups, whose functions did not differ from baseline. The effects of distribution of practice on the ipsilateral aftereffect are also clear (Fig. 1a); the aftereffect for the spaced-practice group was greater for at least the first 7 minutes after prism removal.

Thus, we have replicated here the previously observed lack of intermanual transfer with continuous display (*1–5*), but have shown that it is a special case which obtains when practice is massed. When continuous display periods were spaced by interpolating intervals of rest in darkness, there was significant intermanual transfer. Intermanual transfer has now been shown to occur in all of the types of exposure conditions tested (*16*).

Cohen (*17*) reported that viewing only the terminal position of a pointing finger resulted in intermanual transfer, but continuous view of the limb in essentially the same situation did not result in transfer. This difference was attributed to a disparity in the type of visual feedback available in the exposure session, the implicated factor being whether the hand moves in and out of the field of view (resulting in transfer) or is continually visible (producing no transfer). In contrast, Walsh and Freedman (*7*) demonstrated that by reducing the spacing of trials during terminal display (the situation in which Cohen obtained transfer), it is possible to eliminate the intermanual transfer that is normally found. In terms of the present experiment, Cohen's results could be explained on the basis of the massed nature of his continuous-display procedure (in which intermanual transfer was not obtained), as compared with the more spaced nature of his terminal-display condition (in which intermanual transfer was obtained).

Table 1. Magnitude of aftereffect during the first minute after prism removal for trained and untrained hands following spaced and massed practice. Data are means for the 16 subjects in each group; I, ipsilateral; C, contralateral.

Practice	I (deg)	C (deg)
Spaced	3.2*	1.9*
Massed, 5 minutes	1.9*	0.4
Massed, 9.5 minutes	2.1†	0.5

* $P < .001$, *t*-test. † $P < .01$, *t*-test.

The apparent lack of intermanual transfer of adaptation with the head held rigid has been used by Harris (*3*) to support, in part, the theory that adaptation is the result of a change in the "felt location" of adapted body parts. While the present results do not contradict Harris' theory, they do negate this aspect of his argument. In addition, since it is claimed that conditioned responses and learned skills do transfer easily between limbs, it has been argued that adaptation to visual rearrangement does not involve a learning process (*4*). The present results void this argument.

This experiment also provides positive evidence for a learning interpretation of prism adaptation in two ways: (i) Spaced practice was found to be more effective than massed practice in producing adaptation of the ipsilateral (exposed) hand, a relation found to apply in most learning situations. (ii) Spaced practice was also more effective in producing intermanual transfer of adaptation. Spatz (*18*) showed that after training on the pursuit-rotor task, interlimb transfer occurred when there was a delay between ipsilateral training and contralateral testing, but not when testing followed training immediately. Thus, massing of practice would seem to affect intermanual transfer similarly in prism adaptation as it does in the pursuit-rotor task, a classic motor-learning situation.

EDWARD TAUB
ISRAEL A. GOLDBERG
Institute for Behavioral Research,
Silver Spring, Maryland 20910

References and Notes

1. H. B. Cohen, *Amer. Psychol.* **18**, 411 (1963).
2. C. R. Hamilton, *Amer. J. Psychol.* **77**, 457 (1964).
3. C. S. Harris, *Science* **140**, 812 (1963); *Psychol. Rev.* **72**, 419 (1965).
4. H. H. Mikaelian, paper read at the meeting of the Eastern Psychological Association, New York, April 1963.

5. ———, paper read at the meeting of the Eastern Psychological Association, New York, April 1966.
6. I. A. Goldberg, E. Taub, A. J. Berman, paper read at the meeting of the Eastern Psychological Association, Boston, April 1967; R. E. Kalil and S. J. Freedman, *Percept. Mot. Skills* **22**, 123 (1966).
7. T. M. Walsh and S. J. Freedman, paper read at the meeting of the Eastern Psychological Association, New York, April 1966.
8. S. J. Freedman, in *The Neuropsychology of Spatially Oriented Behavior*, S. J. Freedman, Ed. (Dorsey, Homewood, Ill., 1968), pp. 63–76.
9. I. P. Howard, in *ibid.* pp. 19–36.
10. T. M. Walsh and S. J. Freedman, paper read at the meeting of the Eastern Psychological Association, Boston, April 1967.
11. E. Taub, paper read at the Conference on Adaptation to Rearrangement, Massachusetts Institute of Technology, Cambridge, June 1965; in *The Neuropsychology of Spatially Oriented Behavior*, S. J. Freedman, Ed. (Dorsey, Homewood, Ill., 1968), pp. 77–106.
12. For example, since in some earlier experiments (*6, 7*) error-corrective feedback was available to the subject, it was suggested that this "cognitive" factor was responsible for the intermanual transfer. However, in other studies (*9, 10*) employing what Howard (*9*) termed a "prism-shaping procedure," this argument was disproved by the demonstration that intermanual transfer can occur with terminal display even though the subject is not aware that he has made any errors.
13. R. Held and A. V. Hein, *Percept. Mot. Skills* **8**, 87 (1958).
14. Further details of the procedure and apparatus were reported by I. A. Goldberg and E. Taub (paper read at the meeting of the Eastern Psychological Association, Washington, D.C., April 1968).
15. Duncan's new multiple-range test. All statistical analyses are for the data from the first minute after prism removal only. Analysis of variance indicated that the effect of prism base was not significant ($P > .05$). The data are, therefore, combined for presentation here.
16. The present results should not be interpreted as indicating that intermanual transfer could never be demonstrated to occur with massed practice in a continuous-display situation, nor is this suggested by the position that prism adaptation is a learning phenomenon. It seems quite possible that some change in training or stimulus conditions, perhaps one which served to increase the massed-ipsilateral effect, might result in some transfer.
17. M. M. Cohen, *Percept. Mot. Skills* **24**, 299 (1967).
18. K. C. Spatz, thesis, Tulane University (1966); *Percept. Mot. Skills* **26**, 95 (1968).
19. Supported by grant AF-1042 from the Air Force Office of Scientific Research and by NIH grant MH18821. We thank E. E. Coons for his advice and aid, and P. B. Taub for her technical assistance. Based on a paper read at the Eastern Psychological Association meeting, Washington, D.C., April 1968.

22 May 1972; revised 8 January 1973 ■

Histamine: Effect on Self-Stimulation

Abstract. *When injected discretely into the lateral hypothalamus of rats, histamine inhibited electrical self-stimulation at the injection site without affecting self-stimulation in the contralateral lateral hypothalamus. This effect was blocked by prior treatment with antihistaminics. Histidine, the amino acid precursor of histamine, produced a similar effect after a delay of 6 to 10 minutes.*

In the course of screening substances whose intracranial administration might affect electrical self-stimulation or stimulus-bound eating, we have found histamine to be capable of influencing both behavioral modalities. The presence of histamine in the mammalian central nervous system has been known for some time (*1*), although its role in behavioral homeostasis is currently obscure. The regional distribution of histamine in the brain is virtually identical to that of noradrenalin and serotonin (*2*), and its subcellular localization is predominantly synaptosomal (*3*). The brain also contains abundant quantities of the enzymes that govern histamine metabolism: histidine decarboxylase, histimine *N*-methyltransferase, diamine oxidase, and monoamine oxidase (*4*). At least one of these enzymes, the *N*-methyltransferase, is predominantly synaptosomal (*3*).

We examined the effects of small intracranial doses of histamine on self-stimulation in rats and found it to be a potent inhibitor of this phenomenon. Intracranial cannula electrodes were implanted in 20 250-g male Sprague-Dawley rats with the use of a Kopf stereotaxic apparatus. Nembutal was the anesthetic. Cannula electrodes, made from No. 23 hypodermic electrodes insulated except for the tip of the cannula insert, were placed in the left perifornical region of the lateral hypothalamus. With the tooth bar set 3.1 mm above the ear bar, the electrodes were implanted 0.0 mm posterior to bregma, 1.3 mm lateral to the midline, and 8.5 mm below the skull. A bipolar electrode of comparable diameter was placed in the contralateral perifornical area. After the experiment half of the animals were perfused with formalin and prepared for histological examination. Sections 50 μm thick were cut and stained with cresyl violet. Placements were verified to be in the perifornical area.

The cannula electrode permitted injection of compounds into the site of electrical self-stimulation. The contralateral electrode was used as a control. Cannula electrodes in the septum were 3.0 mm anterior to bregma, 0.0 mm lateral to the midline, and 6.0 mm perpendicular to the skull. Electrodes in the lateral ventricle were 1.0 mm posterior to bregma, 2.0 mm lateral to the midline, and 5.1 mm perpendicular to the skull.

Thresholds for self-stimulation were obtained by determining that amount of current which would maintain lever-pressing for 30 seconds. Whenever an animal failed to reach this criterion the current was raised 10 percent. When the animal met the criterion the current was lowered 10 percent.

Chemicals were obtained from commercial sources and were of the highest quality available. All substances were dissolved in physiologic buffer and injected with Hamilton syringes in volumes no greater than 1 μl. Repeat injections were made only after thresholds had returned to the level before treatment. Control injections were appropriate volumes of physiologic buffer alone, with osmolarity appropriately adjusted for drug concentration with NaCl.

Over the range of 1 to 8 nmole, histamine injected into the perifornical area caused an immediate dose-related elevation of self-stimulation thresholds, which returned over the next 20 minutes to within 10 percent of the level before treatment (Fig. 1). No change in threshold was obtained with buffer alone. All changes were independent of the order of administered dosages. Thresholds to self-stimulation on the contralateral perifornical electrode were unchanged by the ipsilateral injection.

Prior treatment with cannula injections of 1 μl of an antihistaminic (either diphenhydramine, 40 nmole; or chlorpheniramine, 36.5 nmole) completely prevented the effect of 8 nmole of histamine injected 5 minutes later. At these dosages the antihistaminics alone did not alter thresholds. At higher dosages, chlorpheniramine (60 to 200 nmole) consistently lowered thresholds whereas diphenhydramine (45 to 180 nmole) consistently elevated thresholds, both in a dose-related fashion.

Injection of 100 nmole of histidine, the amino acid precursor of histamine, elevated the threshold to self-stimulation but only after an initial delay of 6 to 10 minutes (Fig. 2). Prior treatment with either antihistaminic prevented this effect. The histamine metabolite imidazoleacetic acid (30 nmole) failed to alter self-stimulation thresholds, whereas the metabolites methylimidazoleacetic acid (30 nmole) and