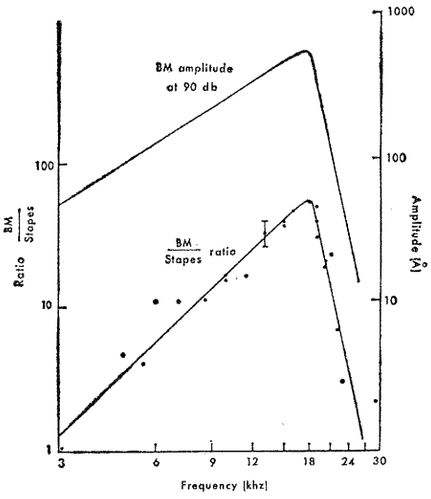


Fig. 1. Tuning curve of the basilar membrane at 1.4 mm from the stapes. The lower curve is referred to a constant stapes amplitude. The upper curve is for a constant sound-pressure level of 90 db SPL; the amplitudes are peak ($\sqrt{2}$ times the root mean square) values.

Tuning curves were obtained for the membranes of ten animals, and all had maximums between 18 and 21 khz. Figure 1 shows the result for one guinea pig. Other animals gave very similar curves, but the results were not so complete: in one case, measurements were continued down to 350 hz; in another case, more accurate data were recorded at 0.5-khz intervals in the region of the peak. The correction of each basilar measurement to constant stapes amplitude completely eliminated the large fluctuations of sound pressure introduced by standing wave patterns in the vicinity of the ear; this was particularly important at the shorter wavelengths in the region of the peak.

The amplitude of the stapes motion for constant sound pressure was observed to fall at 4 db per octave with increasing frequency. With this result it was possible to calculate the absolute amplitude of the basilar membrane movement; the upper curve in Fig. 1 shows the result for a constant sound-pressure level of 90 db SPL. The uncertainty of this level would not exceed 5 db.

The tuning curves, illustrated by the lower curve of Fig. 1, resemble those obtained by Bekesy but are more sharply peaked, with a Q (resonance frequency divided by the half-power band width) of 2.5 (most of Bekesy's curves have a Q around 1.6 or less). This value of Q is still smaller than that for a typical first-order neural tuning curve. The slope on the low-frequency side is 13 ± 1 db per octave, and this is maintained down to about 3 khz; from here down to 350 hz the slope is slightly reduced to around 10 db per octave. The high-frequency slope is 70 ± 10 db per octave. The maximum ratio of basilar membrane amplitude to stapes amplitude is about 50 to 1, and the maximum basilar membrane amplitude at 90 db is 600 Å at 18 khz. The main systematic error in the amplitude ratio will arise from the uncertainty in the angle between the direction of motion of the source on the stapes and the direction of the line from the source to the detector. It is unlikely that this angle exceeded 60° to 70°, and, since the detector subtended an angular range of about 45°, the maximum correction to the ratio would be by about one-half. The shape of the tuning curve and the absolute amplitude curve are not affected.

Bekesy (*3*) gives a value for the maximum amplitude of motion of the basilar membrane measured at high sound-pressure levels in the human ear; in the range of 2 to 3 khz his results would suggest a value of 400 Å at 90 db SPL. Bekesy (*4*) also gives some values for the ratio of basilar membrane to stapes amplitude derived from his work (*1*); at 3 khz the ratio is about 10, and Bekesy remarks that it should increase with increasing frequency as the surface area of the membrane that is set in motion becomes smaller. Both these results are quite consistent with our work.

Changes in the source mass by a factor of 2 did not produce any significant change in our results. Furthermore, it did not seem to matter whether the first turn of the scala tympani was dried or full of perilymph, a result in accord with the model experiments of Bekesy (*1*).

B. M. Johnstone
A. J. F. Boyle
*Departments of Physiology and Physics, University of Western Australia, Nedlands*

### References and Notes

1. G. von Bekesy, *J. Acoust. Soc. Amer.* **21**, 233 (1949).
2. P. Hillman, H. Schechter, M. Rubinstein, S. Shtrikman, *Rev. Mod. Phys.* **36**, 360 (1964).
3. G. von Bekesy, *Acta Oto-Laryngol.* **42**, 197 (1952).
4. G. von Bekesy, *Experiments in Hearing* (McGraw-Hill, New York, 1960), pp. 173–174.
5. We thank Wendy Gardner, T. K. McNab, and T. J. Edwards for technical assistance.

7 August 1967

## Dissociation of the Visual Placing Response into Elicited and Guided Components

Abstract. *Kittens reared without sight of their limbs extended their forelimbs when carried down toward the edge of a horizontal surface. However, unlike normally reared kittens, they were not capable of guiding their paws accurately to the solid parts of an interrupted surface. This fractionation of the visually controlled placing response reveals that the guided reach requires an integration of sensorimotor systems not necessary for development of the elicited extension response.*

As a cat is carried down toward a visible surface, its forelimbs extend automatically as if to prevent collision. This response, called visual placing, is common to many mammalian species. It has been used as an indicator of integrity of the neural mechanisms underlying visuomotor behavior since Rademaker's report in 1931 (*1*). More recently, it has served to measure deficits resulting from special conditions of rearing, removal of brain tissue, and combinations of the two (*2*). By the use of a special rearing procedure and a refined testing technique, we have been able to show that, even though a cat will extend its forelimbs on approach to a surface, it may be quite unable to guide these limbs toward particular objects. This hitherto undescribed dissociation of two aspects of the placing response can serve to distinguish gross from subtle deficits in the visual control of response. As a consequence, new distinctions may be made among underlying neural mechanisms.

Visually controlled placing is conveniently tested without special apparatus or training of the animal. Typically, the hindlimbs and torso of an animal are supported in the experimenter's hands while the forelimbs and head are free. The animal is carried down toward the edge of a horizontal surface. A normal animal extends its forelimbs when close enough to reach the edge, but prior to contact of the head or paws with the surface. To eliminate tactual cues from the whiskers, they are cut prior to testing. Several alternative experimental procedures may be used: (i) The animal may be moved either forward or sideways toward an edge; (ii) the surface, rather than the animal, may be moved to

eliminate nonvisual cues produced by movement of the animal; or (iii) the animal may be held near the surface and allowed a predetermined time period within which to place. If an animal fails to show visually elicited placing, its ability to perform tactually elicited placing may be tested by contacting the dorsal surface of the paw with an edge. This procedure can determine the availability of the placing response as such when visual stimuli do not elicit it.

We first began to suspect that the traditional procedure for testing visual placing did not assess visual guidance of the forelimb when we found that kittens reared without sight of their forelimbs showed visual placing (*3*). The results of research going on in our laboratory had led us to believe that the accurate visual guidance of reaching requires a certain amount of prior experience in viewing the actively moving limbs (*4*). Consequently, if a kitten had been reared without sight of limbs, it should not show accurate reaching, even though reared with no other restriction. Thus, we were left with the task of testing these animals for the hypothesized deficit in the visually guided response. Development of our procedure and associated apparatus was guided by the following logic. Although the edge of a surface is effective in eliciting extension of the paw, the edge need not be continuous. If it is suitably interrupted by cutouts, in the absence of guidance by the eye, the paw is as likely to drop into the cutout as it is to strike the surface. On the other hand, if visually guided, the paw should almost always strike the surface. This notion was embodied in the following apparatus (Fig. 1). A discontinuous edge was made from a board 1.0 cm thick, with prongs 2.5 cm wide, 20.0 cm long, and 7.5 cm apart. On the average, a prong should be contacted during half the number of unguided reaches if the paw is 2.5 cm wide and if reaching movements are both perpendicular to the pronged surface and equally distributed along its length. Both conditions were approximated by our 4-week-old kittens during tests of paw-placing.

During testing trials, the torso, hindlimbs, and one forelimb were supported (as shown in Fig. 1) while the animal was carried downward toward the interrupted edge. The experimenter directed the kitten's head alternately either toward one of the prongs or

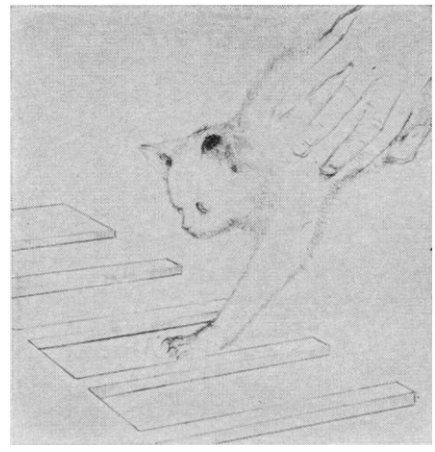

Fig. 1. Apparatus for testing visually guided reach.

toward the middle of a space between the prongs. In the test procedure we gave 12 trials, six for each forelimb. A trial was repeated if no visually elicited extension was observed. We recorded hits and misses only when both the experimenter, who carried the animal, and an observer agreed that: (i) the animal's head was directed toward the pronged surface, (ii) a visual extension response was performed, and (iii) the paw either hit or missed a prong. We scored an extension as a hit if one or more claws contacted the upper surface of a prong. When the head of a normally reared kitten was directed toward a prong, almost all its placing responses were directed to that prong. When the head was moved toward the intervening space, most reaches were to the prong nearest the extending limb. Occasionally, a limb crossed the midsaggital plane of the body to contact a prong. Normally reared kittens hit a prong on 95 percent of the trials.

Six experimental kittens were reared in the dark until they were 4 weeks old. Subsequently they were permitted to move freely for 6 hours daily in an illuminated and patterned environment. During this time they wore lightweight opaque collars which prevented sight of limbs and torso but had little effect on locomotion (Fig. 2). For the remainder of the day they were kept without collars in a lightless room. When these animals were tested after 12 days of this treatment, all of them extended their paws to a continuous surface. At this time, they received 12 trials on the interrupted surface. The extension responses of the experimental animals tended to be individually stereotyped. Some animals repeatedly positioned the paw directly in front of the nose; others, in front of the shoulder joint. Thirty-six of the 72 extensions performed by the six kittens hit the prongs, with a range of five to seven hits for each animal. In another test of eye-paw coordination, we observed reactions of the experimental animals to a ball dangling at the end of a string. Orientation of the head and eyes to the ball appeared normal, but striking responses directed at the ball were remarkably inaccurate when compared with those by the normally reared kittens. After removal of the collar, the animals required an average of 18 hours of freedom in a normally illuminated laboratory room before they showed guided reaching on the test apparatus.

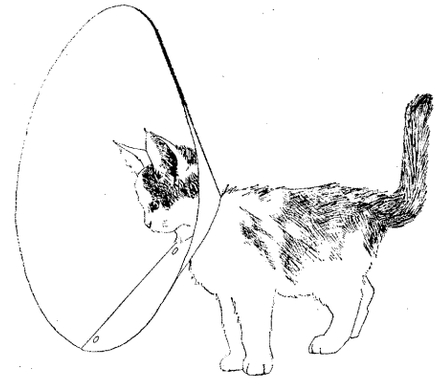

Fig. 2. Kitten wearing a collar that prevents sight of limbs and torso.

These results confirmed our prediction and demonstrated the dissociability of the two kinds of visually controlled placing in kittens. Elicited extension develops without sight of the forelimbs, but guided placing seems to require prolonged viewing of these limbs. A related study with infant monkeys revealed a similar dependence upon sight of the hands for the development of visually guided reaching (*5*). Visual placing tested by the traditional method may not be taken as adequate evidence for the integrity of visually guided behavior. The ability to match the position of the paw to the position of a visible target requires an integration of sensorimotor systems not necessary for the elicited extension response. Our new test allows a more precise assessment of visuomotor capability.

A. HEIN
R. HELD

*Department of Psychology, Massachusetts Institute of Technology, Cambridge 02139*

**References and Notes**

1. G. G. J. Rademaker, *Das Stehen* (Springer, Berlin, 1931).
2. D. Denny-Brown and R. A. Chambers, *Res. Publ. Ass. Nerv. Ment. Dis.* **36**, 35 (1958); J. A. V. Bates and C. Ettlinger, *Arch. Neurol.* **3**, 177 (1960); R. W. Doty, in *The Visual System: Neurophysiology and Psychology*, R. Jung and H. Kornhuber, Eds. (Springer-Verlag, Berlin, 1961), pp. 228–247; A. Reisen, *J. Nerv. Ment. Dis.* **132**, 21 (1961); A. Hein and R. Held, in *Biological Prototypes and Synthetic Systems* (Plenum Press, New York, 1962), pp. 71–74; R. Held and A. Hein, *J. Comp. Physiol. Psychol.* **56**, 872 (1963); P. M. Meyer, J. A. Horel, D. R. Meyer, *ibid.*, p. 402; M. W. Fischman and T. H. Meikle, Jr., *ibid.* **59**, 193 (1965); A. B. Wetzel, V. E. Thompson, A. Janes, P. M. Meyer, *Psychon. Sci.* **3**, 381 (1965); J. J. Braun, *Brain Res.* **1**, 381 (1966); E. C. Gower, paper presented at Eastern Psychological Association, New York, 1966; A. Hein, in *Symposium on Perception and Action* (Proc. 18th International Congress of Psychology, Moscow, 1966), A. V. Zaporozhets, Ed. (Moscow Univ. Press, 1966), pp. 197–202; J. M. Sprague, in *The Thalamus*, D. P. Purpura and M. D. Yahr, Eds. (Columbia Univ. Press, New York, 1966), pp. 391–417; J. M. Sprague, *Science* **153**, 1544 (1966).
3. R. Held and A. Hein, *J. Comp. Physiol. Psychol.* **56**, 872 (1963).
4. Background to this work is discussed by A. Hein and R. Held, in *Biological Prototypes and Synthetic Systems* (Plenum Press, New York, 1962), pp. 71–74; R. Held and J. Bossom, *J. Comp. Physiol. Psychol.* **54**, 33 (1961); R. Held and A. Hein, *ibid.* **56**, 872 (1963); R. Held, *Sci Amer.* **213**, 84 (Nov. 1965).
5. R. Held and J. A. Bauer, Jr., *Science* **155**, 718 (1967).


6. Supported by grants NSF GB-2728, NIMH M-7642, and NASA NSG-496.

10 August 1967


## Enhancement of Flicker by Lateral Inhibition


Abstract. *Sinusoidal modulation of illumination on the compound eye of the horseshoe crab,* Limulus, *produces a corresponding variation in the rate of discharge of optic nerve impulses. Increasing the area of illumination decreases the variation at low frequencies of modulation, but unexpectedly enhances—or "amplifies"—the variation at the intermediate frequencies to which the eye is most sensitive. Both effects must result from inhibition since it is the only significant lateral influence in this eye.*


The appearance of flicker in a light that varies periodically depends upon several variables (*1*). For example, whether a flicker will be observed in a light with sinusoidally varying luminance depends on both the amplitude and frequency of the variation. Results typical of those obtained by DeLange in 1957 from a human observer viewing a small sinusoidally modulated 2-deg field of white light are represented by the solid curve in Fig. 1A. Increase in frequency, up to about 10 cycle/sec, decreases the percentage of modulation about the mean luminance required for the observer to see flicker. At this point the modulation to reach threshold is minimal; that is, flicker sensitivity is maximal. With additional increases in frequency, the percentage of modulation required to reach threshold increases rapidly, and at some point between 50 to 75 cycle/sec even a light modulated 100 percent does not appear to flicker.

The size of the retinal area stimulated has a marked effect on the threshold of flicker. Measurements made by Kelly in 1960, using a large, 85-deg field, are represented by the dashed curve in Fig. 1A. The dash-dot curve, extending from 1 to 10 cycle/sec, represents some results obtained by Thomas and Kendall in 1962 in an experiment in which a sinusoidal modulation was applied to the illumination of the entire room in which the subject was seated.

The increase, at low frequencies, in the percentage of modulation required to reach threshold that results from an increase in the area of the stimulus has been attributed to the effects of lateral inhibition (*1*). In order to test this hypothesis by direct electrophysiological observations we have carried out some comparable experiments with sinusoidal stimuli (*2*) on the compound eye of the horseshoe crab, *Limulus*, in which lateral influences are predominantly inhibitory (*3*).

Modulation of the intensity of light shining on an ommatidium of the *Limulus* eye causes a modulation in the rate of discharge of impulses in the optic nerve fiber from that ommatidium. The amplitude of this modulation of the discharge may be taken as a measure of the "flicker response" of the receptor. Figure 1B shows that this response varies in a characteristic manner with frequency of modulation, and that it is affected by varying the area of the eye illuminated by the modulated light.

In order to obtain the results illustrated in Fig. 1B, a *Limulus* eye was excised and mounted in a moist chamber. A single active optic nerve fiber, arising from an ommatidium near the center of the eye, was dissected from the optic nerve and placed on wick electrodes in the input circuit of an amplifier. A spot of light was centered on the ommatidium from which the nerve fiber arose. The mean intensity of this spot was set by neutral density wedges inserted in the beam. The intensity was then varied sinusoidally, with a constant modulation about this mean, by a rotating polarizer and a fixed analyzer. (Other experiments showed that the polarization itself played no role in the effects reported here.)

The modulated light was turned on for 20 seconds every 3 minutes. The discharge of impluses by the single optic nerve fiber was recorded on line by a small digital computer (*4*). In order to avoid the transient response at the onset of the illumination, only the final 15 seconds of each record were processed. The sum of a constant plus a sine was fitted, by the method of least squares, to the reciprocals of the intervals between successive impulses. In this way average discharge rate (impulses per second) and amplitude and phase of the response modulation were determined (*5*).

The results for a small field (a spot of light about 0.25 mm in diameter which is slightly larger than the facet of one ommatidium) are represented by the solid curve in Fig. 1B. The results that were obtained with a large field (a spot about 1.5 mm in diameter that covered about 20 ommatidia) are represented by the dashed curve. In Fig. 1B *modulation* refers to the various peak-to-peak amplitudes of the neural flicker responses to stimuli of constant amplitude; in Fig. 1A *modulation* refers to the various amplitudes of the stimuli required to produce a constant response—that is, to reach the observer's threshold of flicker. Note that the scale of frequency in Fig. 1A is three times that in Fig. 1B.

Increasing the area of illumination produced two major effects. (i) There was the expected decrease in the amplitude of the flicker response to low-frequency modulation of the light. (ii) At its maximum, the dashed curve in Fig. 1B actually exceeds the maximum of the solid curve. This enhancement or "amplification" of the amplitude of the flicker response to intermediate modulation frequencies was unexpected. Both effects are the result of lateral inhibition.