averaged for 24 time periods of 1 hour each (three samples per subject per period); sleep onset was zero time. These hourly means were expressed as a percentage of that subject's mean prolactin concentration, and the percentages were averaged for all six subjects (Fig. 2). This calculation demonstrated for the group as a whole the prominent diurnal variation in relation to nocturnal sleep which was seen in each subject individually. A similar hour-by-hour analysis of human growth hormone concentrations in the same samples contrasted with that of prolactin (Fig. 2). Each subject had the expected nocturnal rise in growth hormone 1 to 2 hours after sleep onset; elevated concentrations lasted 2 to 3 hours, with an ultimate return to undetectable concentrations (Fig. 1). With the exception of this rise in growth hormone concentration during early sleep and the initial rise in prolactin concentration after sleep onset, no consistent relation between episodes of prolactin and growth hormone release could be identified throughout the 24-hour period. The initial rise in prolactin concentration after sleep onset usually coincided with the first elevation in growth hormone concentration, but the prolactin peak occurred an average of 40 minutes later than the first growth hormone peak.

This study of the 24-hour pattern of release of prolactin in humans provides evidence that prolactin secretion, like that of growth hormone, ACTH, cortisol, and gonadotrophins, is episodic and not constant over the sleep-wake period. The pattern of prolactin release, however, could not be directly correlated in temporal sequence with those of the other hormonal systems.

A number of stimuli have been demonstrated to result in prolactin release, including stress of various kinds, hypoglycemia, strenuous exercise, suckling in postpartum women, and administration of psychotropic drugs (9). None of these appeared to be operative in producing the patterns seen in our subjects. Rather, the overall 24-hour changes in prolactin in this study appear related to factors underlying the sleep-wake or light-dark cycle. It is not possible to determine from these data whether the initiation of the nocturnal rise in plasma prolactin is entirely dependent on sleep or whether it has free-running circadian properties to some degree independent of physiological state or environmental events. Shifts of the sleep-wake cycle and light-dark patterns are necessary to clarify this issue.

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References and Notes

1. Y. Takahashi, D. M. Kipnis, W. H. Daugha-day, J. Clin. Invest. 47, 2079 (1968); Y. Honda, K. Takahashi, S. Takahashi, K. Azumi, M. Irie, M. Sakuma, T. Tsushima, K. Shiaume, J. Clin. Endocrinol. Metab. 29, 20 (1969); J. F. Sassin, D. C. Parker, J. W. Mace, R. W. Gotlin, L. C. Johnson, L. G. Rossman, Science 165, 513 (1969).

- E. Weitzman, H. Schaumberg, W. Fishbein, J. Clin. Endocrinol. Metab. 26, 121 (1966);
 L. Hellman, F. Nakada, J. Curti, E. Weitzman, J. Kream, H. Roffwarg, S. Ellman, D. Fukushima, T. Gallagher, *ibid.* 30, 411 (1970);
 E. Weitzman, D. Fukushima, C. Nogeire, H. Befürger, T. Gallagher, *ibid.* 32, 410 (1970); Roffwarg, T. Gallagher, L. Hellman, *ibid.* 33, 14 (1971).
- S. Kapen, R. Boyar, L. Hellman, E. Weitzman, *Psychophysiology* 7, 337 (1970); R. Rubin,
 A. Kales, A. Adler, T. Fagan, W. Odell, *Science* 175, 196 (1972).

- Science 175, 196 (1972).
 A. G. Frantz and D. L. Kleinberg, Science 170, 745 (1970).
 P. Hwang, H. Guyda, H. Friesen, Proc. Nat. Acad. Sci. U.S.A. 68, 1902 (1971).
 K. Van Kirk and J. Sassin, Amer. J. Electroencephalogr. Tech. 9, 143 (1969).
 A. Rechtschaften and A. Kales, Eds., A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects (Government Printing Office, Washington, D.C., 1968).
 P. Hwang, H. Guyda, H. Friesen, J. Biol. Chem. 247, 1955 (1972).
 A. G. Frantz, D. L. Kleinberg, G. L. Noel,
- A. G. Frantz, D. L. Kleinberg, G. L. Noel, Recent Progr. Horm. Res. 28, 527 (1972).
 K. Laue, C. Gottlieb, V. Herbert, Proc. Soc. Exp. Biol. Med. 123, 126 (1966).
 L. Sumorted by Difference AM, 11204 AM.
- EXP. BIO. Mea. 123, 120 (1900).
 11. Supported by PHS grants AM 11294, AM 5397, CA 11704, AM 14282, HD 06209, and OH 00331 and NASA contract 9-BB32-79-2-1461. Sleep studies were done in the Montefiore Hospital and Medical Center Clinical Rech Center (PHS grant RR 53). We thank H. Friesen for supplying the human prolactin reparation and antibody used in these studies. R. Sundeen, J. Finney, B. Ward, L. Liotta, K. Tucker, and P. McGregor provided tech-nical assistance.
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Peripheral Motion Detection and Refractive Error

Abstract. Motion thresholds were determined for the fovea and peripheral retina with and without correction for peripheral refractive error. With correction, motion thresholds decreased and individual differences disappeared. These results imply that under normal observation conditions, peripheral sensitivity is limited mainly by dioptric rather than retinal variables.

The potential contribution of the periphery to the functional performance of the eye can be appreciated when one considers that the fovea occupies only a small fraction of the entire visual field. Although the resolution of the peripheral regions is much less than that of the fovea, it is generally assumed that off-axis stimulation provides an important cue in directing voluntary eye movements. In spite of the functional importance of peripheral stimulation. little attention has been given to the possible influence of the refractive characteristics of the peripheral visual fields. Since both the resolution of the retina and the quality of the retinal image are degraded with increasing eccentricity, it is not possible to state which factor is limiting peripheral vision. Studies by Ferree, Rand, and Hardy (1) have demonstrated that very large refractive errors are present in the peripheral visual fields and furthermore that the direction of these errors-that is, toward hyperopia or myopia-varies considerably among individuals. The purpose of the study reported here was to determine the effect of the correction of peripheral refractive errors on the threshold for motion perception, a function which plays an important role in peripheral vision.

Three male graduate students, experienced in visual psychophysics, served as observers. As an aid to maintaining fixation, a stimulus was imaged in the blind spot so that involuntary movement of the eye would be immediately apparent. While a subject maintained monocular fixation with his dominant (right) eye, thresholds for motion perception were determined for the temporal visual field for a 1.0-second exposure at eccentric angles ranging from 0° to 80° in 10° steps. The stimulus was a white (reflectance 80 percent) square, 1.3 cm on a side, with luminance 4.3 mlam, viewed against a black (reflectance 0.8 percent) background at a distance of 78.7 cm. The subjects reported whether the

stimuli had moved to the right or left, or were stationary during the exposure periods. The staircase method was used, and the thresholds were calculated on the basis of a 50 percent correct criterion.

Since subjects typically show improvement with practice in this task, it was necessary to stabilize each subject's performance by repeated testing on four occasions. When the motion thresholds had been thus stabilized, the spherical and astigmatic refractive errors for each subject at each eccentricity were determined by dynamic retinoscopy. While the subjects maintained fixation in the experimental apparatus, the examiner positioned the retinoscope at the same location at which the motion stimulus was presented during the experiment. The results of the peripheral refraction are listed in Table 1. It will be noted, in confirmation of the results of Ferree et al. (1), that the peripheral refractive error can be extremely large and that wide individual differences are observed.

In the second phase of the experiment, motion thresholds were again obtained, with correction for the previously measured refractive errors. This was accomplished by placing ophthalmic trial lenses to correct for both spherical and astigmatic errors in front of the eye, normal to the axis of stimulation. Figure 1 presents the mean motion threshold as a function of eccentricity, both with and without correction for refractive error. In the data obtained without correction, there is a progressive increase in the motion threshold with increasing eccentricity. A typical value of 1 minute of arc per second was found with foveal fixation (2), with the thresholds increasing by approximately a factor of 10 at 80° of eccentricity. A comparison of the refractive data with the data for the uncorrected condition reveals a correlation between refractive error and the thresholds for peripheral motion perception (3).

With the introduction of the peripheral correction, the sensitivity of all subjects is improved. The threshold velocity required for the far periphery is only six times that required at the fovea. The increase in threshold with eccentricity is described by a slight negatively accelerated function. Furthermore, the data for all the subjects are very similar-the individual differences observed without correction have disappeared.

arc/second) a c c = × KS 4 TI ч 7 6 (minutes threshold Without correctio With correction 50 -1-20 30 40 Stimulus eccentricity (degrees of arc)

Fig. 1. Motion thresholds as a function of stimulus eccentricity with and without correction for peripheral refractive errors. CJ, KS, and TI refer to subjects.

The data of the present study are of interest from several points of view.

1) They demonstrate that correction of peripheral refractive error does result in an improvement in performance. To our knowledge, in previous studies of peripheral vision the refractive error has not been corrected over a wide range of eccentricities. These data imply that for motion perception the limiting factor in peripheral perform-

Table 1. Refractive data for subjects KS, TI. and CJ under the conditions of the experiment.

Stimulus eccen-	Correction (diopters)		Cylin- der
tricity (deg)	Spher- ical	Cylin- drical	axis (deg)
Subject KS			
0	- 2.25	- 0.50	180
10	- 2.25	- 0.50	180
20	- 2.00	— 0 .50	180
30	- 1.50	- 0.50	180
40	- 0.50	- 0.50	170
50	+ 2.25	- 1.00	150
60	+ 3.50	- 1.00	150
70	+ 4.00	1.00	150
80	+ 5.50	- 1.00	145
Subject TI			
0	$+ 1.00^{\circ}$	- 1.25	90
10	+ 1.00	- 1.25	90
20	+ 1.00	1.50	85
30	+ 1.00	- 1.50	80
40	+ 1.00	- 1.75	80
50	+0.87	- 1.75	75
60	+ 0.87	- 2.00	70
70	+ 0.87	- 2.00	70
80	+0.87	-2.25	65
Subject CJ			
0	+0.87	- 0.25	90
10	+0.87	- 0.25	90
20	+0.87	- 0.50	85
30	+0.87	- 0.50	80
40	+0.87	— 0 .50	80
50	+ 3.25	- 0.75	75
60	+ 7.50	- 0.75	70
70	+ 7.50	- 0.75	70
80	+ 8.00	- 1.00	65

ance may be dioptric rather than retinal. Also, there is a marked reduction of intersubject variability resulting from the correction of peripheral refractive error.

2) These data also provide a measure of the functional relation between the motion threshold and off-axis viewing. Even without the correction for refractive error, motion sensitivity increased by only a factor of 10 over a range of 80° of arc. On the other hand, the most conservative measure of visual acuity degradation reveals that a change by a factor of 10 occurs at 30° of off-axis viewing (4). For eccentricities comparable to those used in the present study, the uncorrected acuity is reduced to 1 to 2 percent of its value at the fovea (5). This comparison clarifies the common observation that the peripheral regions are very sensitive to motion. From these data it is clear that the relative degradation with off-axis viewing is less for motion than for resolution. In the periphery, all visual functions are degraded, but motion suffers the least.

3) It is of theoretical interest to determine the relationship between the performance of the eye and the subserving neurological structures, for example, receptive field sizes or ganglion cell activity (6). Because the effect of dioptric variables has been eliminated, the present data permit a direct comparison between the behavioral data and the relevant neurological substrate.

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References and Notes

- 1. C. E. Ferree, G. Rand, C. Hardy, Arch. Ophthalmol. 5, 717 (1931).
- C. E. Ferree, G. Rand, C. Hardy, Arch. Ophthalmol. 5, 717 (1931).
 C. H. Graham, in Vision and Visual Percep-tion, C. H. Graham, N. R. Bartlett, J. L. Brown, Y. Hsia, C. G. Mueller, J. A. Riggs, Eds. (Wiley, New York, 1965), p. 575.
 No correction was made for the change in effective pupil size with oblique viewing since the luminance level employed was assumed to be on the flat portion of motion-luminance.
- be on the flat portion of motion-luminance function. This was confirmed by testing at both 4.3 and 43.0 mlam with and without correction 4.3 and 43.0 mlam with and without correction at 60°, 70°, and 80°. No differences were ob-served as a function of luminance.
 J. Kerr, Percept. Psychophys. 9, 375 (1971).
 Y. LeGrand, Form and Space Vision, M. Millodot and G. G. Heath, Transl. (Indiana Univ. Press, Bloomington, rev. ed., 1967), pp. 135-145
- Univ. Press, pp. 135-145.
- pp. 135-145.
 6. W. Richards, Brain Behav. Evol. 4, 162 (1971);
 H. Ikeda and R. M. Hill, Nature 299, 556 (1971).
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