and Holton, however, considered a multichromatic wave source so that it was unnecessary for them to invoke eddy viscosity to smear out the critical level singularity which occurs for a monochromatic wave. The depth scale for the critical layer in (*3*) was determined by the spectral distribution of the IGW's rather than by viscosity as in (*1*). Since atmospheric IGW's are generally multichromatic, Lindzen and Holton's model is likely the more realistic. Moreover, the assertion by Bekofske and Liu that a decrease of the Richardson number at the critical level allows a larger portion of the wave momentum to pass through without being absorbed is incorrect. It is shown in (*4*) that if the upward flux of momentum due to the IGW is a constant A far below the critical level, then an amount of momentum given by

$$A\{1 + \exp[-2\pi(\mathrm{Ri} - 0.25)^{1/2}]\}$$

is absorbed at the critical level. Here Ri is the Richardson number. Thus, the momentum absorption actually increases as the Richardson number decreases. The reason for this behavior is that the momentum flux changes sign at the critical level so that even though a larger fraction of wave energy passes through the critical level as the Richardson number decreases, the momentum flux convergence nevertheless must increase. Bekofske and Liu's contrary conclusion apparently arises from their failure to properly normalize the IGW momentum flux to remain constant (independent of Ri) far below the critical level. It should be added that at small values of Ri all analyses are somewhat questionable, but the assertion of Bekofske and Liu about momentum passage is impossible.

We do agree that a quasi-steady balance should eventually occur in which the mean wind shear is limited to the value for which $\mathrm{Ri} \simeq 0.25$ over the depth of the critical layer. However, this balance should occur not from decreased absorption of the IGW momentum flux but rather from increased momentum diffusion due to the turbulence produced by breaking Kelvin-Helmholtz waves.

J. R. HOLTON
Department of Atmospheric Sciences, University of Washington, Seattle 98195

R. S. LINDZEN
Center for Earth and Planetary Physics, Harvard University, Cambridge, Massachusetts 02138

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22 January 1973; revised 9 March 1973

Dioptrics of the Periphery of the Eye

Leibowitz *et al.* (*1*) described an improvement in peripheral motion detection with correction of refractive error of the peripheral dioptrics of the eye. Retinoscopy was used on three subjects to determine the refractive error to eccentricities of 80°. When these errors were corrected they found a decrease in the motion threshold and in individual differences. They interpreted these data as implying that motion perception in the periphery was dioptrically rather than retinally limited, and they stated that the relative degradation of off-axis viewing is less for motion than for resolution and that as dioptric variables had been eliminated, the behavioral data and the relevant neurological substrate could be compared.

Their findings are at variance with those expected theoretically for light entering the optical system of the eye at an oblique angle (*2*, *3*). Taking a typical example, LeGrand (*2*) calculated that at 50° in the temporal field 7.52, diopters of astigmatism would be induced. At that same eccentricity Ferree *et al.* (*4*), using a refractometer, recorded an average astigmatism of 4.82 diopters for their type A (12 of 18 subjects) and 1.90 diopters for their type B. Leibowitz *et al.* (*1*) found a 0.50-diopter change in astigmatism at 50°. The theoretical data and both types described by Ferree *et al.* have progressively increasing amounts of astigmatism. Ferree *et al.* also recorded a change in axis of 90° in 16 of their 18 subjects. Thus, although Leibowitz *et al.* agreed with Ferree *et al.* in there being large refractive errors and wide individual differences, they did not demonstrate the increase in the amount of astigmatism or change in axis expected.

We repeated measurements of the refractive error by using retinoscopy, refractometry, and a subjective method on three subjects. We were unable to record repeatable values beyond 50° with retinoscopy and 60° with the refractometer and subjective methods. With the refractometer the mean refractive error was similar to that of type A in Ferree *et al.* There was a shift toward hyperopia with retinoscopy, as expected with the eye at a known and constant focus, and due to the error inherent in retinoscopy (*5*). The mean results for the three subjects were (*6*) $+2.58/-3.08 \times 90$, $+1.50/-2.92 \times 90$, and $+2.08/-3.50 \times 90$ for retinoscopy, refractometry, and the subjective method, respectively, at 50° eccentricity and when adjusted to give zero refractive error along the visual axis.

In the report by Leibowitz *et al.*, it would seem that either their subjects did not conform to the norm of theory and other experimenters, or insufficient attention was paid to the determination of the expected large astigmatism by the retinoscopist. Perhaps the marked improvement in the motion threshold obtained after correction might be attributed to training if testing without correction were always done before testing with correction. It is somewhat difficult to conceive that for subject TI of their study, at 50°, a refractive change of $0.13/-0.50 \times 15$ would affect the results to the extent shown. And these results are of the same order of magnitude as those obtained with larger changes, for example $+4.50/-0.50 \times 30$ for subject KS at 50°.

Thus, it is difficult to draw a conclusion linking behavioral data and the relevant neurological substrate on the assumption that optics have been eliminated.

ANNE LAMONT
MICHEL MILLODOT
School of Optometry, University of Montreal, Montreal 101, Canada

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6. The notation $+2.58/-3.08 \times 90$ is an optical prescription which means $+2.58$ diopters spherical power and -3.08 diopters cylindrical power (astigmatism) at axis 90°.

3 May 1973; revised 27 June 1973

The choice of standard clinical static retinoscopy in our study was made on the assumption that a relevant and accurate assessment of refractive error must be made empirically under the same conditions as were present during the motion experiment. There are very few data on peripheral refractive error, and those which exist demonstrate large individual differences. Thus, the use of this well-known standard technique by an experienced refractionist was the method selected. Since the nature of stimuli in the peripheral visual field has been shown to influence accommodation (*1*), testing was carried out in the motion apparatus under the same conditions of peripheral stimulation, illuminance, head position, and so forth which obtained in the motion experiments. These determinations by an experienced clinical refractionist were difficult and tedious, requiring about 1 hour per subject, but were repeatable. Whether other refractive methods under different stimulus conditions would produce similar results would be of interest, but is beyond the scope of the present study. Although we did not encounter patterns of refractive errors characteristic of the type A reported by Ferree *et al.* (*2*), increasing divergence in orthogonal meridians, the data from our subjects are highly similiar to the pattern which they refer to as type B, that is, increasing spherical error in the periphery with a small increase in divergence between the orthogonal meridians. In view of the paucity of investigations concerned with peripheral dioptrics, and the small subject samples used by Lamont and Millodot and by us, it does not seem fruitful at this point to speak of normal or average magnitudes or patterns of peripheral refractive errors. Further investigations in this area are necessary.

We are reasonably certain that the observed improvement in motion thresholds is independent of learning. The data reported in the original paper were obtained only after practice produced no further change in motion detection. In a more extensive study, we have systematically compared improvement in motion detection resulting from practice and feedback both with and without correction as opposed to changes induced only by correction of refractive errors (*3*). Under all conditions, correction of refractive error produces the greatest and most consistent improvement.

We had also noted the relatively large improvement in the ability to detect motion with correction for the subject TI, for whom relatively small peripheral refractive errors were recorded. In addition, we noted that the larger the refractive error, the greater the improvement when correction was introduced. The data would be consistent with the hypothesis that perhaps a small refractive error exerts a proportionately greater deleterious effect than larger errors. Substantiation of this hypothesis would be of considerable theoretical as well as practical importance. In the case discussed here, the significant fact is that introduction of correction for every subject at all eccentricities lowered motion thresholds. Whether or not we have entirely eliminated dioptric variables, our data obtained with correction reflect the contribution of the neurological substrate in the periphery more accurately than any data hitherto available.

HERSCHEL W. LEIBOWITZ
CHRIS A. JOHNSON
Department of Psychology, Pennsylvania State University, University Park 16802

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8 August 1973

Hemostasis and Blood Coagulation

Ratnoff and Bennett in their article on the genetics of hereditary disorders of blood coagulation (*1*) significantly omitted the term hemostasis. Biggs (*2*) has stated: "If blood clotting is essential to haemostasis, then the sequential hypothesis does not fit well with clinical observation. The facts suggest that if blood fails to start clotting or fails to finish the process off, the defect is less notable than if the chain is interrupted in the middle." Macfarlane (*3*) makes the comment: "It has already been pointed out that the clotting of blood is of little importance in the control of bleeding from small wounds, and if the platelets are also excluded, what factor remains that is capable of producing haemostasis? Only the action of the vessels themselves, a factor that has been almost completely ignored from the point of view of a possible haemostatic function."

The waterfall or cascade description of blood coagulation is still a theory, as was Howell's concept 40 years ago. It should therefore be considered and compared with other explanations such as that of Seegers (*4*), who postulates a multifaceted prothrombin complex and states "prothrombin itself contains all that is required to have thrombin" and that "it activates itself whenever it is in a suitable environment," or mine (*5*), which I have described as the "expanded Morawitz theory." In this scheme coagulation begins with the liberation of the platelet clotting factors which require thrombin as the catalyst. When native platelet-rich plasma carefully kept from contact with glass is clotted in a silicone-coated container, clotting occurs but no detectable amount of prothrombin is consumed; but if a minute amount of thrombin is added, normal consumption occurs (*6*).

The answer to Ratnoff and Bennett's inquiry: "An unsolved genetic puzzle is why these four proteins [factors II, VII, IX, and X], all involved in blood coagulation, are the only proteins known whose synthesis requires vitamin K" is that these are the only proteins for which definitive tests are available. Thus, factor VII can only be identified by the one-stage prothrombin time with the use of a purified tissue thromboplastin free of factors VII and X.

ARMAND J. QUICK
Hemostasis Research Laboratory, Medical College of Wisconsin, Milwaukee 53233

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