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.

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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.

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