from the radial direction above the active center and produce, in such directions, a "cone-of-avoidance" in the corpuscular streams with a width of about 4 to 5 days of solar rotation, or about 60°.

The purpose of this communication is to point out that, if this interpretation is correct, the process of using a running mean of perhaps 4 to 5 days in geomagnetic disturbance ratings will not adversely affect the detection of the M-region response in geomagnetic data, but it will decrease the sharp, irregular, and possibly accidental or local fluctuations in geomagnetic disturbance ratings that tend to mask the important features of the active region influence. As Fig. 1 shows, the use of a 5-day running mean simplifies the identification of the individual peaks of the trend curve that reveals the 27-day recurrence. For the period shown, practically every significant peak can be fitted into a recurrent series, members of the same series being represented by similar symbols. Moreover, the whole curve can be fairly well synthesized by adding curves for individual series, and these individual curves show reasonably smooth systematic trends in amplitude corresponding to the lifetimes of the recurrent series. Recurrent periods of other than about 27 days cannot similarly be fitted to the data.

Our work suggests that the use of 3- to 5-day running means as an index of the intensity of the hypothetical corpuscular beams has merit, at least for years of low solar activity, and may assist in other geophysical studies.

JEAN-CLAUDE PECKER
WALTER ORR ROBERTS
High Altitude Observatory, Boulder, Colorado

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Visual Receptor Lamellation and Active Rhodopsin

The paper by Wald (*1*) reconciling large changes in visual sensitivity with minute changes in concentration of photopigments apparently marks the end of an epoch. For more than 30 years the effort was made to explain various functions of vision by the reaction-kinetics of visual purple. This interpretation has appeared incompatible with the data of many workers, summarized in Wald's paper. The chemistry of mass-action can hardly operate as a measure of sensitivity, in terms of thresholds or of quantum demand, in the microlamellar environment of Wald's hypothesis. It will be interesting to see whether the formal deductions of the visual-kinetics school can find any *mechanism* in this new molecular-compartment theory.

Whether visual thresholds can be shown to depend solely on the properties of single receptors is also an open question. The photochemical approach frankly invokes the logical principle of parsimony in the attempt and tentatively denies other retinal mechanisms a role in this regard. Thus it excludes from the visual threshold mechanism considerations like receptors-per-fiber, receptive fields changing with adaption, and bipolar thresholds. With our increasing knowledge of retinal physiology, one wonders whether this logical principle is not a misleading guide.

Wald has now incorporated some of the data on receptor structure into visual theory, by relating the number of rhodopsin molecules to the number of Sjöstrand's submicrolamellae, and thence to the shifting quantum demand. His hypothesis, however, does not incorporate the striking major lamellation clearly demonstrated by M. Schultze in 1867, analyzed optically by W. J. Schmidt (*1*), but destroyed by the reagents used in Sjöstrand's technique. This coarser structure (*2*) has been shown alternately aqueous and lipid by chemical methods (*3*) and roughly periodic at 400 to 700 mμ (*2*). It interrupts the continuous stack of 1400 to 2800 microlamellae 4 to 8 mμ thick (as postulated by Sjöstrand) each contributing equally to vision in Wald's proposal. The coarser organization would give those protein microlamellae that contact the thicker lipid layers a special local sensitivity occurring at 400 to 700 mμ as proposed 10 years ago by Wald (*1*). Roughly each hundredth layer of rhodopsin protein would thus become the site not only of quantum absorption but of electric excitation—a hypothesis (*2*) based on several optical and electrochemical aspects of this interface. The thickness of the coarser lipid layers, yet unknown but lost in Sjöstrand's method, becomes a critical datum in the theory of excitation.

It would be interesting to have Wald's hypothesis related to several important details:

1) What is the electric trigger? That is, how can rhodopsin, after absorbing a quantum, create a local electric impulse by a known neuro-excitatory mechanism? The electric asymmetry required for depolarization of the stack (*2*) is not accounted for by "membrane breakdown" alone; nor is the considerable voltage and energy generated in the whole outer segment.

2) How do the protein microlamellae of Sjöstrand relate functionally to the coarser *lipid* layer and *its* microstructure shown by Schmidt?

3) How incorporate the unique geometric (*2*) and chemical relationship of rhodopsin molecules to the thick lipid segments that separate the aqueous segments composed of approximately 100 protein microlamellae? That is, Wald's key proposal of 1944 deserves further exploration. If this is true, the relationship of total rhodopsin to molecules used in stimulation would be much more than 6 to 1—the main point at issue.

Now that Wald's unflagging and admirable series of investigations has begun to take quantitative ac-

count of the detailed structural organization already described, we may hope that the insight, which has so beautifully ordered the multiple reactions of the visual opsins, will eventually weave in with function the amazing complexity of structure in which these remarkable molecules are constrained to work.

S. A. TALBOT

Department of Medicine,
Johns Hopkins Medical School, Baltimore

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Of the interesting points raised by Talbot, I should like at present to comment upon only one. This is the relationship between the microscopic and the submicroscopic layering of the outer segments of the rods and cones, and the location of their proteins and lipids.

The microscopic layers, which exhibit periods of 300 to 700 mμ, are not visible in fresh intact preparations. They appear only after outer segments have lain for a time in the suspension medium or have swelled in acid or alkali (*1, 2*). It has been assumed that they indicate a preexisting submicroscopic layering of unknown dimensions.

The evidence that the outer segments are composed of alternate layers of protein and lipid rests upon their behavior in plane-polarized light. They exhibit lamellar birefringence, attributable to a regular alternation of layers of material, one component of which is extractable with fat solvents (*1*). The conditions for this type of birefringence, formulated by Wiener (*3*), are that the layers differ in refractive index and be thin relative to the wavelength of light. The microscopic layers of the rods and cones are too coarse to satisfy the latter condition. The presence of lamellar birefringence itself indicates a much finer submicroscopic layering.

Sjöstrand has now revealed this in the electron microscope (*4*). The outer segment of a rod or cone presents a pile of regularly spaced, parallel membranes, varying in thickness from about 40 to 160 A and separated by spaces about 65 to 130 A wide (guinea pig rods; perch rods and cones). There is no evidence of a coarser structure. In a personal communication, Sjöstrand expresses much the same opinion as Schmidt, that the microscopic cross-striation, when visible, probably represents some effect of swelling of the normal structures.

We have recently observed in the electron microscope that the coarse cross-striation exhibited by occasional outer segments arises by the deterioration of their fine structure. In such rods the submicroscopic layers have lost their integrity and regular spacing and have coalesced to form an intricate meshwork. This presents at times the appearance of surprisingly regular cross-striation (*5*).

I think it is clear that the microscopic layering under all conditions represents an artifact and that only the submicroscopic layers need to be considered. The method of preparing the ultrathin sections of retina so far examined removes lipids; the lamellar structure that remains appears to be composed primarily of protein. The visual pigments are probably located in these layers, in the thicker layers perhaps at their interfaces. The location of the lipids is still uncertain. Some lipids may be bound to protein and located either in the protein or lipid layers. The outer segments, however, exhibit a strong positive intrinsic birefringence which appears to be associated with highly oriented layers—probably double layers—of lipid, the carbon chains of which lie parallel with the long axes of the rods and cones (*1*). Such material presumably occupies, at least in part, the interstices between the protein membranes. It is significant that the narrowest interstices yet observed—about 65 A in guinea pig rods—could just accommodate a double layer of mixed myelin sheath lipids—63.7 A in the dry state (*6*).

GEORGE WALD

Biological Laboratories, Harvard University,
Cambridge, Massachusetts

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Water-Soluble Chelates in Histochemical Staining

Histochemical stains for metals have depended on the precipitation of an insoluble compound that was colored or could otherwise be made visible. In the case of the metal stains, such as the calcium stains, this has required removing the metal from its naturally bound state and reprecipitating it (or the anion associated with it) as the desired compound.

The use of water-soluble chelates provides another approach to the microscopic visualization of metals. Chelating compounds bind metals in a ringlike configuration through the formation of at least one coordinate bond and frequently one or more ionic bonds. For several years these agents have been used in analytic determinations of various metals (*1*). Weak chelating compounds that change color in the presence of the metal being analyzed have often been employed as titrimetric indicators.

This communication reports preliminary studies (*2*) on water-soluble, colored chelating agents as used in two types of histochemical stains. In type 1, the colored compound coordinates with the metal and is selectively bound only where the metal is present. In type 2, the agent impregnates the tissue as a whole but