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VISUAL SENSITIVITY

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Selig Hecht is reported to have said: "I write not in order to add to the literature on vision, but to subtract from it." To do this, he tried to explain as many visual phenomena as possible in terms of photochemical processes in the receptors. Current practice in part perpetuates Hecht's attitude and in part has abandoned it. On the one hand, the attempt to discern links between visual experience and physiological processes dominates vision research now more than ever. In the effort to understand vision, the traditional search for simple descriptive principles and functional relations (e.g. Weber's Law, Ricco's Law, the Power Law) is taking second place behind the formulation of mechanistic, more or less explicitly physiological models. On the other hand, Hecht's ambition to subtract from the literature is not shared by many contemporary theorists (let alone experimentalists!). Few current advances in understanding vision take the form of theoretical integrations that encompass many phenomena within a simple conceptual framework. Instead, theories of visual phenomena are continually becoming more complex and more diverse. This is mainly because of a corresponding progressive increase in knowledge of the complex physiological substrate upon which such theories are built. Technical advances have made recent progress rapid in electrophysiology; there are notable examples of this in the areas of receptor behavior and of retinal circuitry. Sometimes these electrophysiological discoveries have provided answers to old questions, but more often they have raised new questions about the origin of the observed physiological events and about their functional role within the visual system as a whole. At the same time, psychophysical experiments are being designed to address increasingly specific physiological questions. And inevitably, as the jungle of visual fact thickens, theoretical statements are tending to become more restricted in range.

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Those who find a piecemeal accretion of knowledge disappointing may be reassured that the potential explanatory scope of some of the recent physiological discoveries is quite broad. For instance, the implications of the partitioning of the geniculo-striate pathway into parallel "sustained" and "transient" systems are the subject of much current speculation, and some would argue that this distinction may turn out to be as visually significant as the partitioning of retinal receptors into rods and cones (or perhaps much more significant). This and other physiological discoveries about central visual processes have encouraged visual psychophysicists to put central mechanisms into focus. Their efforts have revealed (or seemed to reveal) a splendid variety of specialized detectors, with different stimulus requirements, for each part of the visual field. The important advances in relating visual sensitivity and spatial vision to cortical organization have not been reviewed here. This topic deserves reviews of its own and is getting them (28, 96, 193).

In such a vigorously developing field, it is a little surprising to find that many old questions are not yet conclusively settled. Among these is the problem of the nature and varieties of visual adaptation. There is some evidence that even the simplest light adaptation phenomena involve multiple sites of action in the visual pathway. By ingenious methods, sensitivity modifying processes have been disclosed at various stages of the visual system without contamination by earlier stages; yet on the whole, the ways in which the successive stages of the visual pathway contribute to the variations of visual sensitivity are far from clear.

The theme in this review is the attempt to explain visual sensitivity in terms of physiological events, and especially the increasing recognition of the functional diversity of single cells at any particular level of the visual pathway and the significance of this diversity for vision. The years 1973 to 1976 are emphasized, and the review is designed to supplement previous reviews in this series (34, 116, 193). Invertebrate vision and animal psychophysics are not dealt with. Material that could be reviewed under Spatial Vison or under Color Vision has been given short shrift (or no shrift at all).

Useful Sources

Two new journals carry pertinent material: *Perception,* edited by R. L. Gregory (Pion Press), and *Sensory Processes,* edited by L. E. Marks (Academic Press). One established journal, *Investigative Ophthalmology,* is enlarging its scope to include more "basic vision" and has accordingly been rechristened *Investigative Ophthalmology and Visual Science.* The proceedings of the meetings of the Society for Neuroscience are now published under the title *Neuroscience Abstracts,* and each year's issue includes a substantial section on vision.

The two excellent textbooks by Kaufman and by Uttal (122, 212) should be useful for both graduate and undergraduate students. A recent introduction to general neurophysiology by Kuffler & Nicholls (133) puts the visual system at the center of the stage. More advanced general works include the second edition of Davson's multivolume treatise (51, 52). Volume 6 has a chapter by Werblin on the organization of the vertebrate retina and one by Dubin on retinal anatomy. Volume 2A includes a survey of visual psychophysics by Ripps & Weale that concentrates on

the more recently reported phenomena, an admirable account of retinal physiology by Arden, and an outline of central visual processing by Holden. Rodieck's book, *The Vertebrate Retina* (183), is an extraordinarily wide-ranging, thoughtful, and scholarly (but readable) survey, which anyone interested in vision could read with profit. Gazzaniga & Blakemore's *Handbook of Psychobiology* (88) includes interesting chapters by Blakemore and by Anstis. Carterette & Friedman's *Handbook of Perception* [especially Volume 5: Seeing (41)], and the Springer-Verlag *Handbook of Sensory Physiology* (the last few volumes of which should appear shortly) are valuable and comprehensive reference works.

Ditchburn (58) has assembled our knowledge about *Eye Movements and Visual Perception* in a useful compendium; much of it deals with the effective elimination of eye movements and the consequent elimination of visual perception. Relevant sections from *The Neurosciences: Third Study Program,* dealing with *Feature Extraction* (223) and with *Central Processing* (173) are available in paperback. Physiological development, not discussed here, is well covered by reviews in Gottlieb's book [(93); see also Barlow's paper (9)], and perceptual development by Cohen & Salapatek (46). But both these fields are moving so fast that rapid obsolescence of reviews seems guaranteed.

Published symposia include one devoted to the exciting collaboration between theoretical physicists and retinologists (and an occasional psychophysicist) in the study of photoreceptor optics (201), a topic too technical for this review. Enoch's review contributions to this and two other recent symposia (67, 68) are necessary (but enjoyable) reading for anyone interested in optical aspects of retinal function. Receptor electrophysiology and quantum efficiency are the prominent topics in a recent symposium on *Photoreception* (11). The symposium edited by Langer (136) deals with receptor structure and function as well as with visual pigments. The ARVO symposium on retinal circuitry (62, 132, 162, 165, 177) gives a current picture of an active field, with physiology as well as anatomy. Zettler & Weiler's symposium (230) deals with peripheral neural mechanisms in vertebrates and invertebrates. Different approaches to a model vertebrate system have been brought together by Fite (84). Ditchburn has assembled an issue of *Optica Acta* devoted to visual detection and discrimination [see, for instance, Kelly (126)]. A recent discussion of *The Visual Field* (172) includes attempts, mainly by physiologists, to relate single unit activity at all levels of the visual pathway to visual function. A published symposium on eye movements (159) has a section on eye movements and vision. The Leningrad symposium on information processing in vision (90) has some interesting contributions but may be hard to find. The issue of the collected papers of H. K. Hartline and his colleagues (176) is noteworthy, not least for Ratliff's topical introductions in which he integrates vertebrate physiology and psychophysics with the invertebrate work. Psychophysicists will be pleased that the papers of W. S. Stiles are being brought together from the various dark corners where they have stood over the years (203).

"Physiology" in this review is nearly synonymous with "single unit recording in the retina or geniculo-striate pathway." Readers devoted to the electroretinogram might turn to Armington's book (5) or the annual ISCERG symposia published as

supplements to *Documenta Ophthalmologica.* The state of our knowledge about cortical evoked potentials is captured by Regan (178, 179) and by Desmedt's volume (56); a second edition of Regan's book is on the way. Some readers will also be interested in recent discussions of the role of midbrain centers (91, 114) or in the retinohypothalamic system, implicated in the generation and control of circadian rhythms (160).

RECEPTOR BEHAVIOR

Electrophysiological recording from receptors has brought some big surprises over the past few years. The polarity of the response to light and its dependence on intensity and on stimulus geometry all exhibit features that are more or less unexpected.

In the dark, a continuous current of sodium ions flows radially in the retina in the space outside each receptor, moving from the inner segment of the receptor to its outer segment where the sodium ions enter the receptor. Light absorbed in the outer segment blocks this inward sodium current, and as a result the interior of the receptor becomes more strongly negative relative to the exterior [(170); or see Arden's review in (51)]. This hyperpolarization of rods and cones in response to light came as a surprise because it is depolarization that is required for generating action potentials, and because invertebrate receptors were known to be typically depolarized by light. The paradoxical polarity of rod and cone response is also expressed in the chemical signals they release; though the transmitters involved have yet to be identified (23), it has recently been established (181) that the receptors release transmitter rapidly in the dark, more slowly in the light. Perhaps one polarity of signal is as good as another? But Hodgkin (108) suggests that a dark object against a lighter background may be a more interesting stimulus than vice versa; an object like this would stimulate transmitter release.

A second surprise is that studies of the vertebrate retina have not upheld the view (based mainly on early invertebrate studies) that the signal is logarithmically related to light intensity. Rather, the shift in membrane potential in response to a brief flash is roughly proportional to flash intensity, provided there are less than about 40 photons absorbed per rod, that is up to many thousand times psychophysical threshold (49, 79, 170). For the less sensitive cones, the proportionality extends to correspondingly higher flash intensities (21, 79). If these levels are exceeded, the response of the rod or cone is driven close to the maximum achievable hyperpolarization and even the brightest flashes fail to produce responses greater than this saturating value. According to Penn & Hagins (170), the saturation limit corresponds to the closure of all the sodium-permeable channels in the receptor. The psychophysical phenomenon of rod saturation, a failure of discrimination at high light levels in rod vision, is probably due to saturation of the receptor signal, and is the object of renewed interest (103, 188) now that its physiological substrate has been identified. Similar effects have been observed in cone vision (e.g. 197), but the correlation with cone electrophysiology is less well documented. The blue-sensitive cones are particularly intriguing since, like the rods but unlike other cones, these cones and/or their

associated pathways remain saturated under prolonged exposure to a constant illumination (157).

The approximate linearity of the receptor response to transient stimuli of less than saturating intensity helps to explain a number of otherwise puzzling visual phenomena: linear temporal integration for reaction time (147, 187), additivity of heterochromatic luminosity, and the Talbot-Plateau Law. With long test flashes, compressive nonlinearity may appear at levels below saturation (21, 27, 168); this effect, a manifestation of time-dependent adaptation in receptors, helps to account for the reduction in brightness exponent with increased duration (146).

Receptors of the same type appear similar in sensitivity (20, 49). The greatest sensitivity reported for rods (for diffuse brief flashes, in turtles) is 700 microvolts per photon absorbed; for cones, 25 microvolts per photon (49). By this criterion, rods are thus considerably more sensitive than cones, and the longer duration of their response may give them a further advantage. The difference, together with optical factors, might account for most of the superiority of human rod vision over cone vision in detecting large test flashes [documented by Ronchi (184)]. Differences in spatial summation between rod and cone pathways may therefore not be as critical as previously thought, and recent experiments on ganglion cells (72) support this conclusion. But as Hood & Hock (110) point out, many uncertainties make it difficult to compare receptor sensitivities with human visual sensitivity.

The large responses of receptors to small numbers of photons imply enormous amplification by the receptor. In part, this amplification is at the outer membrane of the receptor, where the effect of light is to modulate a spontaneous process, the inflow of sodium. But nearly all photons are absorbed in the interior of the rod, away from the cell membrane, so there must be an agent released at the site of absorption which can migrate to the membrane to block the sodium channels. It has been argued (170) that this transmitter must be released in large numbers by a single photon, thereby providing an additional stage of amplification within the cell. Various lines of evidence suggest that the transmitter is calcium; for instance, calcium introduced inside the cell roughly mimics the effect of light (33).

These minutiae of receptor function may not be without significance for vision. In cones, the random opening and closing of sodium-permeable channels in the dark seems to be a more important source of variability in the cone signal, and hence more important in limiting reliable detection, than the spontaneous isomerization of pigment molecules traditionally postulated to account for "dark light" (11, 135). The afterimage seen after exposure to intense light has a physiological counterpart in a persisting hyperpolarization of the receptor in the dark (20, 131, 170), and Sakitt (188) plausibly suggests that both are due to the continued presence of internal transmitter to close the sodium channels.

The receptor response to a brief flash shows a comparatively slow buildup and decay, and although formal models have been developed to describe the time course of the response (22, 170), there is as yet no definite physical or chemical answer to the question: why is visual transduction so slow? Whatever the reason, the slowness of rods in particular is remarkable. Turtle rods take at least 600 msec to reach their peak response after a brief flash and often much longer (49), and primate rods are

only a little less sluggish than those of the turtle (224), suggesting that the rod response may be even slower to develop than the visual sensation. This apparent paradox is resolved by the observation (195) that subsequent neurons may approximately differentiate the receptor response over time. The neural pathways from the receptors also introduce significant temporal integration. Interestingly enough, these pathways are matched to the receptors that feed them, the rod pathways integrating for longer than the cone pathways to the same ganglion cells (18). Yet under some conditions psychophysical measures of temporal resolution may agree well with physiological measurements on primate receptors (26). Both rod and cone responses are accelerated by increasing the intensity or area of a flashed stimulus, and for large bright stimuli, the responses exhibit a pronounced transient at onset along with a sustained response (19, 21, 49). The transient and sustained components of the rod response could perhaps be the basis of the recently discovered duplex behavior of flicker resolution in rod vision, observed physiologically in the skate (99) and psychophysically in man (47).

Crosstalk Between Neighboring Receptors

The assumption that individual receptors generate their response independently and that spatial integration is the job of the higher order neurons with which they communicate was once accepted without question. It now turns out to be badly wrong. Baylor, Fuortes & O'Bryan (19) recorded from single cones during stimulation with tiny spots and discs of light, and were able to show that the consequences of stimulating one cone or group of cones may be recorded in its neighbors, which hyperpolarize in the dark much as if they had been stimulated themselves. The signal elicited by a focal stimulus spreads from one receptor to another across retinal distances which may be quite large: in the turtle, 120 microns for cones, 300 microns for rods (49, 191). Thus, receptors themselves have receptive fields wide enough to significantly affect the receptive field dimensions of the later cells through which the signal is relayed.

Anatomically, networks of contacts have been seen to link the synaptic endings and the inner segments of different receptors; in many cases, freeze-fracturing has identified the connections as "gap junctions" that allow ions to pass from one receptor to the other (177). Most physiological investigations (49, 80) suggest that the connections are primarily between receptors of the same type. Two reports, however, suggest strong rod-cone interactions at the receptor level: Schwartz (192) finds that cones excite rods in the turtle, and Nelson (165) reports successfully penetrating a cat cone and finding a conspicuous rod input. The primate retina does exhibit gap junctions between cones and rods (177), but they are much smaller than the cone-cone junctions. It has been suggested (80), on the basis of experiments on the toad, that the rod-cone contacts would provide, at best, a weak coupling, unlikely to be visually important. Though psychophysical evidence for a convergence of signals from rods and cones continues to accumulate (85, 115, 138, 228), it is not clear at what point in the visual pathway these convergences occur.

The probable greater spreading of rod signals must be kept in mind when comparing the sensitivities of rods and cones. In psychophysical measurements, rod sensitivity decreases to approach cone sensitivity as the size of the test flash is reduced to approach a point. This has been taken to mean that rods are not inherently more sensitive than cones, but owe their greater sensitivity to summation in postreceptoral pathways. However, the use of a punctate test flash might now be considered to place the stimulated rods at a disadvantage by dispersing their signal among hundreds of neighboring unstimulated rods so that no individual rod can deliver its full signal. It is the diffuse stimulus that, by equally stimulating all receptors within a large area, obviates the need for current flow between one receptor and its neighbors and allows each rod or cone to generate the same response that it would if functionally isolated from its neighbors (80). And to a diffuse stimulus, individual rods are about 30 times as sensitive as cones, as was noted above (49).

But in applying such arguments to human vision, it is important to be wary of species differences. There seems to be no clear anatomical evidence for rod-rod contacts in mammals, though cone-cone and cone-rod contacts are conspicuous (177). However, rod-rod contacts in the cat have been suggested on the basis of physiological evidence (139).

The existence of receptor coupling in any animal poses an intriguing teleological puzzle. If the coupling does not improve sensitivity, what does it do? No really convincing answers are forthcoming (80, 135). Perhaps the retina is more subtle than the brains of its investigators.

NEURAL CIRCUITRY

The complexities of spatial organization at the receptor level are not limited to the crosstalk described above. The hyperpolarization in response to light is passed between neighboring receptors by sign-conserving lateral connections, but in cones there is also an opposite, depolarizing effect of more remote surround illumination, the result of inhibition by horizontal cells (19). Individual cones therefore exhibit center-surround organization, making them crude contour or nonuniformity detectors of the sort envisaged by Mach; crude because the antagonism from the surround only fractionally reduces the net hyperpolarization when a diffuse stimulus is presented. The horizontal cell antagonism may be entirely absent in rods (49, 192). Surround antagonism is much more prominent in bipolar cells than in receptors, and this has been taken to mean that horizontal cell antagonism is mainly applied by feedforward onto the bipolars, rather than by feedback onto receptors (180, 221). In support of this interpretation, Werblin (221) points out that the delayed antagonism from the horizontal cell makes the bipolar response somewhat transient, yet the horizontal cell's own response is sustained. If the horizontal cell were acting by feedback upon its own input (the receptor signal), it would itself exhibit the same transient response as the bipolar cell. A feedforward role is also indicated by the fact that the center-surround antagonism of bipolar cells is not found in horizontal cells [though a nonadditive, facilitatory surround is reported in lower vertebrates (137)]. The horizontal cells apparently oppose the receptor signal not by attenuating it, but by subtracting from it linearly (195, 221). To the extent that this is true, these cells cannot importantly contribute to the changes of sensitivity associated with light and

dark adaptation. Their function might be to contribute the "zero adjustment" found in ganglion cells (12, 73, 74, 156) by canceling the effects of uniform steady light. Under some conditions, their canceling effect can prevent overloading of the bipolar cell and so produce an improvement in sensitivity (221).

Primate horizontal cells, like one type in the cat, link rods to cones via a long thin axon (25); but apparently there is no significant direct communication between the compact cone end and the distant rod end of such cells (165, 166). Rod/cone segregation is thus preserved at this stage, but it may be violated at the receptor level (165, 192).

The relations between bipolars, amacrines, and ganglion cells present an intricate and rapidly changing picture. In two current models, developed from experiments on mudpuppy (156) and on catfish (162), the connections of bipolar cells to ganglion cells are always sign-conserving (so that impuslses are generated when the bipolar is depolarized); on-center cells are driven by depolarizing bipolars, off-center cells by hyperpolarizing bipolars, and on-off cells by both these and by amacrines (156). The division into on and off systems as early as at the bipolar level is given visual justification by Marr (149). This retinal organization is consistent with the reported segregation of on- and off-center ganglion cells in the cat into different retinal strata where they connect with invaginating (presumed depolarizing) and flat (presumed hyperpolarizing) bipolars respectively (81, 132), but it is not clear how the proposed correlations between form and function could be applied to the different morphology of primates.

Amacrine and ganglion cells come in different shapes and sizes, and the physiological properties of the different types are now becoming clear (44, 142). "Alpha" ganglion cells with large cell bodies and large dendritic fields are the "brisk transient" or Y type; "beta" cells with smaller cell bodies and dendritic fields are the "brisk sustained" or X type (for more on X and Y, see below). The cells with smallest bodies form the heterogeneous physiological class known as "sluggish" or W cells.

Although amacrine cells were at first thought to depolarize transiently at both onset and offset, sustained "on" and "off" types have also been found (38, 42, 222). Chan & Naka (42) believe that only the sustained types may be true amacrine cells that form a lateral transmission line within the retina, but others prefer to associate each amacrine type with the corresponding type of ganglion cell (156). In any event, Dubin's account (52) of anatomical differences between species at the inner plexiform layer suggests the primate retina could be simpler than those investigated physiologically.

Where investigated, the polarity of the amacrine influence has always been sign-inverting (156, 222). Delayed inhibition from amacrines nicely accounts for transient ganglion cell responses (156, 222), but the sustained ganglion cells are thought to derive their inhibitory surrounds from horizontal cells (through bipolars) rather than from amacrines (222). Since X cells in mammals are not only sustained but also linear, and Y cells are nonlinear, the highly nonlinear characteristics of transient amacrine cells (42) suggest they influence mainly Y cells (107); pharmacological evidence supports this (130).

A new type of cell, the interplexiform cell, that provides feedback from the amacrines to the outer plexiform layer has attracted attention in a number of laboratories but has not yet been studied electrophysiologically (62, 132). Dowling, Ehinger & Hedden (62) show pharmacologically that depolarization of these cells in goldfish opposes the effect of illumination on horizontal cells. To embroider upon their suggestion that "interplexiform cells in goldfish may regulate center-surround antagonism," it is worth noting that a feedback loop of this sort could be valuable in ensuring that the influence of horizontal cells on bipolars is kept nearly equal to the opposing influence of receptors (when averaged over the receptive field of the interplexiform cell), thus ensuring an even balance of center-surround antagonism. The effect of light in increasing center-surround antagonism (12, 73) could be explained if light tends to hyperpolarize the interplexiform cell, which would then increase the antagonistic input from horizontal cells so as to redress the balance and keep activity in the inner plexiform layer relatively constant. A system like this might exhibit a transient excess of surround antagonism when presented with a flashed background, and some psychophysical studies of spatial integration (101, Fig.7) suggest that this does occur. The oscillatory response of ganglion cells to intense large flashes and the associated afterimages (40) could have the same origin.

In comparing psychophysics with ganglion cell responses it may be important to know how many ganglion cells are actually stimulated by a single punctate stimulus. Fischer's estimate that about 35 centers are stimulated in the cat [at any point in the visual field (82, 155)] makes it surprising that a single cell can be as sensitive as the whole animal (8); but when the diversity of ganglion cells is taken into account, only a few centers of any cell type overlap at any point (142, 182). Analogy with the trichromatic theory of color discrimination shows that three overlapping receptive fields in any small neighborhood would suffice for spatial discrimination in two dimensions (besides intensity) far finer than receptive field dimensions might at first suggest.

Though lateral inhibition is traditionally associated with the retina, a notable recent development is the increasing documentation of inhibitory interactions in the brain. Relay cells in the lateral geniculate nucleus (LGN) exhibit a spatially opponent receptive field organization of their own. Current models (45, 65, 169, 202) explain this by invoking excitation of inhibitory interneurons (as well as the relay cells) in the LGN by an all-excitatory input from ganglion cells. Directly antagonistic influences of neighboring ganglion cells, a central postulate of previous models, are no longer invoked. Receptive fields of LGN cells are thus shaped (to the extent that they differ from those of ganglion cells) by inhibitory influences originating within the LGN, and control of this inhibition from other brain sites may cause changes of sensitivity correlated with alertness (45, 59).

The principle that all afferent synapses are excitatory appears to hold in cortex (209) as well as in the LGN (169), and there are indications that all interneurons in the cortex, as in the LGN, are inhibitory (50, 104, 189). The importance of intracortical inhibition is all the greater because according to some recent views (50, 189), each cortical cell collects excitation from at most a tiny group of LGN cells, making the geometry of the afferent input only a weak constraint on the form of

cortical receptive fields, the variety of which is ascribed to differences in intracortical connections. In support of the critical role of intracortical inhibition in forming the responses of cortical cells, it has been shown that their receptive fields may be considerably modified by blocking inhibition with bicuculline (198), and even abnormalities due to deprivation during development can be reversed in this way (66). Intracortical inhibition may eventually inspire as many psychophysical experiments as lateral interaction in the retina (e.g. 53, 143, 164, 217).

In both retina and brain, then, the preferred organization is one of sharply localized excitatory projections, with inhibitory interneurons running laterally at each level. The question remains: why is this a good arrangement?

LIGHT ADAPTATION

Adaptation in Receptors

When the first recordings were made from vertebrate receptors, it was uncertain whether or not they would be capable of light adaptation. It has since become clear that most receptors, if not all, contain efficient sensitivity-regulating mechanisms. The initial evidence (27, 170) suggested a fixed nonlinear relation between the prevailing stimulus intensity and the response generated by the receptor. The nonlinear response-intensity relation was a compressive or saturating one, and this made it possible to explain reduced sensitivity in the presence of background illumination: the additional light required to shift the receptor potential by a certain number of millivolts would be greater in the presence of a background that had driven the receptor signal to a point where the slope of the intensity-response function was reduced. This simple Fechnerian conception of adaptation at the level of the receptors has had to be abandoned [for most receptors recently studied; likely exceptions are mammalian rods (97, 170) and possibly mudpuppy rods (168)] in favor of something more complicated and more interesting. For turtle and mudpuppy cones (20, 168) and for turtle, gecko, and toad rods (20, 78, 131), it has been shown that the signal generated depends not only on the prevailing light intensity but also on the recent history of stimulation. Any change of light intensity is quickly registered, but during the next minute or less the membrane potential drifts back toward the dark level. During exposure to a steady light, the response settles down to a level well below the maximum available, and so, according to the response compression model, the receptor should be excitable; in fact, however, the light-adapted receptor is much less sensitive to changes of intensity than the response compression model would suggest (78, 131, 168). This reduced sensitivity in the light-adapted state means that the dynamic range of the receptor has been changed in accordance with the prevailing conditions of illumination.

The simplest alternative model compatible with these observations is a sensitivity-scaling model, in which light adaptation reduces the effectiveness of all stimuli, background and test alike, by the same factor (as if the stimuli were being delivered through the photochromic dark glasses now available, which increase their opacity in response to light). This is the model favored by Normann & Werblin (168) on the basis of their results with mudpuppy cones. It is also roughly (though not exactly) consistent with the relation between background response and test sensitivity in

more recent reports (78, 131). In particular, there is a range of intensities where Weber's Law is approximately satisfied at the receptor level; that is, the response in millivolts is proportional to the ratio of the test light to the background intensity. Within this range the steady membrane potential increases only slowly with increasing background intensity; this supports the "dark glasses" model, according to which the steady potential should be independent of the background, if Weber's Law holds.

CHANGE IN INTEGRATION TIME However, the "dark glasses" model too is a gross oversimplification, as has been shown in a recent monumental analysis of turtle receptors (20, 22). Backgrounds do not merely decrease the amplitude of the response of flashes; they also profoundly affect the time course of the response. When the response to a flash is displayed as a function of time, the peak responses satisfy Weber's Law, but the simplicity of Weber's Law is misleading because the peak occurs earlier in time the brighter the background. The early part of the flash response is less affected by light adaptation than Weber's Law implies. Baylor, Hodgkin & Lamb [(21, 22); for related models, see (86, 229)] explain this complicated behavior by means of a beautifully simple and concrete hypothesis, namely that the transmitter (probably calcium) which is released by an absorbed photon and which closes the sodium channels in the receptor membrane, is quickly inactivated by an autocatalytic reaction. The lifetime of the transmitter molecules is inversely proportional to the concentration of the catalyst, which in turn is linear with light intensity, since it is absorbed light that produces the catalyst. Because the amount of transmitter initially released by a test flash is unaffected by adaptation, and is proportional to flash intensity, the earliest part of the response is proportional in rate of rise to flash intensity and is essentially unaffected by background; but during exposure to a background, or during the response to a flash, a quantity of catalyst will be generated which will reduce the later part of the signal by shortening the lifetime of the transmitter.

The predicted linearity of the early phase of the receptor response, and its resistance to adaptation, are supported by tests using flickering stimuli applied to frog rods (210) and primate cones (14). In these studies, steady backgrounds decrease sensitivity to flicker by a factor that decreases with increasing flicker frequency: slow fluctuations are attenuated roughly in accordance with Weber's Law, but the most rapid flicker signals are not attenuated at all by the addition of a steady background. In the autocatalytic model, this immunity of rapid flicker to adaption is understandable because detection of the rapid flicker depends on the most rapidly changing phase of the flash response, which is the early, linear part, whereas detection of slow fluctuations requires a relatively sustained signal, the amplitude of which is reduced by any adaptive reduction in transmitter lifetime. The immunity of high-frequency flicker rules out a simple response compression model for adaptation, even for primate cones, for which the response compression mechanism was first proposed (27).

High-frequency linearity is also observed psychophysically (124), making it an important principle linking psychophysics with electrophysiology. Its empirical basis has been questioned (122), perhaps because it is so counterintuitive, but the

results of Roufs (186) should dispel any doubts. A model proposed by Kelly (124) succeeds in explaining high-frequency linearity but requires some assumptions that are perhaps not very plausible. In Kelly's model, the response to the flickering test stimulus pulls itself down by its own bootstraps: an inhibitory feedback loop integrates the response over a short time and subtracts the result from the input to the inhibitory stage. The effect of a background is to increase (by an unspecified mechanism) the inhibitory feedback elicited by the flicker. Perhaps an autocatalytic scheme might provide an alternative model in which the role of the background has an intuitively plausible mechanistic basis. In any case, future models of adaptation will probably tend to abandon the mathematically tractable but physiologically unrealistic static nonlinearities of Fechner's Law and Stevens' Law (148) in favor of time-dependent formulations.

Light adapting by changing integration time has at least two advantages: first, the receptor can preserve a large fraction of its operating range for registering increments above the adapting level, something not generally possible in a simple response compression mechanism; and second, instead of simply wasting light as in the "dark glasses" type of mechanism, the light adapted receptor is able to gain in speed of reaction while sacrificing sensitivity that it does not need.

Postreceptoral Processes: Interaction Between Nearby Receptors

Recognition of the adaptive capabilities of receptors has been accompanied by accumulating evidence that postreceptoral processes are also important—in some cases perhaps all-important. The familiar observation that light adaptation does not proceed independently at nearby points in the visual field has been confirmed in the cat (77) and extended to cone vision in that animal (72). Enroth-Cugell & Shapley (77) have shown that in cat ganglion cells, light adapting influences are collected over an area roughly coextensive with the receptive field center, even though this area varies from one ganglion cell to another. These observations show that light adaptation is not achieved by individual receptors working independently. Nor can receptor crosstalk create an "adaptation pool" at the receptor level, for adjacent receptors do not much modify each others' sensitivity [(19, 191); but see (20, p. 741)]. The pooling of adaptive influences is unlikely to be the work of the ganglion cell itself since local adaptation has been demonstrated within the receptive field centers of ganglion cells in frog (39) and rat (100) and within the receptive field surrounds in cat (43), but it does imply a postreceptoral process. The implication of a postreceptor site for adaptation is supported by the adaptive changes in spatial integration familiar to psychophysicists, which are absent at the receptor level (80, 135).

Psychophysically, the reduced spatial integration of the light adapted eye expresses itself in the validity of the DeVries-Rose square root law, rather than Weber's Law, for light adaptation when the task is detection of a small test stimulus or resolution of fine detail (e.g. 126). The square root law now presents a theoretical problem in view of reports that receptors more closely satisfy Weber's Law (20, 27, 78, 131). The same problem appears in comparing psychophysical observations with observations on mammalian ganglion cells. Individual mammalian ganglion cells, like receptors, show a smaller change in spatial integration than what is observed psychophysically, and even with small tests they are slightly (12) or appreciably (76) more susceptible to light adaptation than the square root law implies. Probably the preferential desensitization of large-field ganglion cells, as compared with small-field cells, by uniform backgrounds (77) is also an important factor contributing to the psychophysical change in spatial integration with light adaptation.

"Interaction" between receptors of different types (a vague concept, seldom precisely defined though frequently disputed) is relevant here. The critical issue where adaptation is concerned is: when only one class of receptors detects a test light, can visual sensitivity be affected by the action of an adapting light on the remaining classes of receptor? If it can, there must be an adaptive mechanism or other nonlinearity fed by signals from both receptor types; if not, the observed variations of sensitivity are due to processes at a stage where the signals are still segregated. Stiles' successful model (203) does not explicitly incorporate interactions in this sense, and has served as a point of departure in the current search for interactions between receptors having different spectral sensitivities. With spatially uniform, steadily exposed adapting fields, Pugh (175) finds interactions between different cone types suggestive of two successive stages of sensitivity regulation. Rod-cone interactions are also reported in this situation, but they are comparatively weak (e.g. 115), and cat ganglion cell recordings are consistent with rod-cone independence (72). A report of a strong influence of monkey cones on rod sensitivity (225) is weakened by the subsequent discovery (213) that halothane, the anaesthetic required to produce the effect, retards recovery from strong light exposures; the disappearance of the rod response attributed to cone influence (225) could reflect light adaptation of the rods themselves to the bright recycled test stimuli.

The intriguing phenomenon of transient tritanopia (insensitivity of "blue" cones during early dark adaptation) has been rescued from obscurity by Mollon & Polden (158) and by Augenstein & Pugh (6), who tentatively explain it by changes of sensitivity at the opponent process level due to signals from other types of cone. Again, no comparably strong rod-cone interactions are reported during dark-adaptation (103, 228), but the first few seconds of dark adaptation have still to be examined. The absence of strong rod-cone interactions seems consistent with the view that the postreceptoral processes that supplement receptor adaptation with spatially uniform adapting stimuli are not more central than the bipolars (103). But it is not yet clear whether this is consistent with King-Smith & Carden's evidence (128) that a white background selectively depresses the sensitivity of the nonopponent system relative to the opponent system.

Electrophysiological recording from different levels of the visual pathway has continued to yield evidence for a postreceptoral site of visual adaptation. Green et al (98) show that bipolars and ganglion cells in the skate can be enormously desensitized by conditions that leave receptor and horizontal cell sensitivity almost unaffected. This is presented as evidence against the proposal (76) that horizontal cells have a role in postreceptoral adaptation, and the case against the horizontal cell is strengthened by the apparent linearity of center-surround interactions likely to be mediated by horizontal cells (73, 74, 195). But of course there are no other cells that

could introduce a loss of sensitivity between the receptors and bipolars. A way out of this impasse is offered by Dowling & Ripps (63) with their observation that potassium can profoundly affect b-wave sensitivity without doing much to the receptors. Unfortunately, sensitivity control by freely diffusing potassium would not be compatible with the approximate independence of different receptor types (especially of rods and cones) in light adaptation, so perhaps the horizontal cell hypothesis for postreceptoral light adaptation should be retained. If the horizontal cells control bipolar sensitivity by feedforward, there is nothing perplexing in the fact that they evade the sensitivity losses that they impose on subsequent neurons.

Dark Adaptation

According to Pugh (174), a very brief flash of high intensity regenerates a substantial fraction of the initially bleached pigment, yet the persisting loss of sensitivity after such an exposure is the same as after a more prolonged exposure of equal energy that leaves far more pigment bleached. This result, designated Rushton's paradox, dashes hopes for a comprehensive correlation between the sensitivity loss and the concentration of photoproducts. At the same time, the correlation between sensitivity and the fraction of rhodopsin bleached continues to be supported under other sets of conditions (171), and we await a theory that can explain the validity of the correlation under some conditions and its failure under others.

A physiological basis for prolonged afterimages seen in total darkness (188) has been discovered in the persisting hyperpolarization of receptors after exposure to strong light (20, 131, 170). Even the latent period of the afterimage (167) has its correlate at the receptor level in a silent period of a few seconds immediately after the offset of the bleaching light, during which there is no hyperpolarization (20). The existence of a persisting hyperpolarization in bleached receptors qualitatively supports the idea that the effects of bleaching are physiologically akin to the effects of illumination, but quantitatively the loss of receptor sensitivity imposed by bleaching is often greater or longer lasting than the persisting hyperpolarization might suggest (20, 131, 170). Because of the persisting hyperpolarization, postreceptoral processes (as well as receptoral processes) may be implicated in dark adaptation as well as light adaptation, and indeed the importance of postreceptoral processes in dark adaptation has long been recognized. Recently, studies of intensity-time tradeoff have provided new evidence on this point. Green et al (98) report that dark adaptation of the skate ERG depends more on the duration of the adapting exposure than on its intensity, and Virsu & Laurinen (216), and Loomis (143a) report analogous observations using afterimages. Both the afterimage phenomena and the ERG results could be explained if the persisting cause of insensitivity during dark adaptation depends on a neural response related to intensity by a saturating nonlinearity.

In view of the recent work by Virsu & Laurinen (216), Sakitt (188), and others (e.g. 92, 103, 126, 143a), the obituary on afterimages published by Ripps & Weale (see 51) is clearly premature. Far from being dead, the study of afterimages has been rejuvenated by the modern approach of treating them as clues to the mechanisms of light and dark adaptation.

Sensitivity Against Flashed or Contoured Backgrounds

When adapting stimuli are spatially uniform and are not rapidly changing in intensity, the possible loci of sensitivity regulation are confined to the early stages of the visual system, because steady uniform backgrounds like this have little capacity to affect the maintained response of ganglion cells and more central neurons (71, 150). But when the adapting stimulus is flashed or is locally nonuniform near the test field, the spatial opponency and transience of response that characterize these cells would less effectively block signals from the adapting stimulus, so allowing more central factors to come into play. Current reports suggest that even at this primitive level of complexity, the physiological substrates of changes in visual sensitivity cannot be identified with confidence.

The transient exaggerated elevation of threshold at the onset and offset of a uniform background, long familiar psychophysically ("Crawford masking"), has recently attracted the attention of retinal physiologists. Enroth-Cugell & Shapley (76) did not find this effect in cat retinal ganglion cells, but their luminances were lower than those at which the psychophysical effect is prominent. Afanador & Adams (1) found a transient elevation in the goldfish retina, but the time constant was of the order of minutes rather than seconds as in psychophysics; in this study, however, the luminances were perhaps excessively high, enough to bleach considerable pigment. The mudpuppy has yielded a more promising physiological substrate for Crawford masking (48, 222); in this animal a transient insensitivity is found in ganglion cells but is absent in bipolars. Werblin & Copenhagen (48, 222) attribute the effect to inhibition by amacrine cells, on the grounds that an adapting field flashed at a position outside the receptive field can produce it without strongly exciting the ganglion cell itself.

When a tiny test flash is superimposed on a concentric steady background, the reduction of sensitivity caused by the background may be greater if the background is small than if it is large. This "sensitization," brought about by enlarging the background, will here be taken up from the point where Brown (34) left it. Like the decreased sensitivity close to the edge of a large background field (214), the increased threshold at the center of a small background is formally a spatial analog of the Crawford masking by temporal transients, but its physiological basis is at present not so clear. Sensitization therefore makes an interesting test case in the correlation of physiological and psychophysical observations and illustrates some of the problems that hinder that correlation.

A first issue is whether large and small backgrounds act upon the same sensitivity regulating mechanism with different effectiveness or whether small backgrounds introduce an additional loss of sensitivity by their action on some more central mechanism which large backgrounds stimulate only weakly. On either view, the center-surround organization of the receptive fields of single cells would be a prerequisite for sensitization, but on the first view, which has dominated thought about the problem until recently, sensitivity is determined simply by how much the background (whether large or small) excites some type of cell with center-surround

organization, such as the bipolar cell; the critical cell might be rendered most insensitive by its strong response to a "small" background big enough to fill the center without encroaching on the antagonistic surround. There is now physiological support for this simple scheme [(37, 48, 221), but see below]. But some recent psychophysical observations speak against it. The difference in sensitivity between small and large backgrounds is reported to disappear, or almost disappear, when the retinal image of the background is stabilized against eye motion (13, 211), even though stabilizing the boundary of a large background has little effect on threshold. This suggests that the small background, but not the large one, may elevate threshold by its effect on cells, for instance amacrine cells (211), that are much more responsive to moving contours than to stationary ones. Another indication that small and large backgrounds may act through different mechanisms is the observation that rod-cone interactions may be much more conspicuous with small backgrounds than with large ones (85, 138, 140). The finding of interaction with small backgrounds and the importance of stabilization could both be explained if sensitivity regulation occurs at different stages of the visual pathway; a stage without center-surround antagonism might be activated by small and large backgrounds alike, and a later stage exhibiting center-surround antagonism, perhaps the inner plexiform layer (211) or perhaps the visual cortex (140), would be activated more strongly by the small unstabilized background. Since there are many monocular cells in visual cortex, the hypothesis of a central or cortical process is not inconsistent with the reported absence of strong dichoptic sensitization effects (118, 205).

It might be expected that recording from the retina would show whether sensitization originates in the outer plexiform layer, in the amacrine cell layer, or more centrally. Unfortunately the picture is confused by probable species differences, possible differences between rods and cones, and differences between stabilized and unstabilized vision. In lower vertebrates at light levels high enough for cone vision, sensitization is conspicuous in retinal ganglion cells (37, 48) and in bipolar cells (37, 221); but at scotopic levels, less frequently examined, it has been reported only in off- and on-off center ganglion cells (120). In the mammalian eye on the other hand, most experiments have been done at low light levels, and here evidence continues to accumulate (12, 73) that under most conditions, cat retinal ganglion cells of whatever type show no sensitization. Enroth-Cugell, Hertz & Lennie (72) extend this result to photopic conditions in the cat. Only if the test flash is large enough or intense enough to itself elicit a significant antagonistic response from the surround of the receptive field can sensitivity be improved by enlarging a background (74), and this is unlikely to be the basis of the psychophysical effect, which works best with small test stimuli (2, 154). These negative results have led to the conclusion (12, 140) that sensitization does not originate in the eye but in the brain. But if indeed the psychophysically observed effect is abolished by stabilizing the retinal image, current physiological evidence does not, in fact, exclude a retinal basis for it; perhaps physiological experiments involving real or simulated eye movements would reveal sensitization in the retina.

Thus it remains unclear at what stage in the visual pathway small and large backgrounds exert their different effects on sensitivity. Any correspondence between

sensitization and receptive field properties is not a simple one; a ganglion cell may show conspicuous center-surround antagonism and yet show no sensitization under the same conditions (74). The apparent brightness of a background stimulus may begin to decrease with increasing size, presumably owing to center-surround antagonism, while sensitivity at its center continues to decrease (105). The fact that antagonism from the surround need not reduce the level of light adaptation suggests that the lateral inhibition involved does not precede the sensitivity regulating process in the mammalian retina, and so supports the conclusion that if horizontal cells regulate sensitivity, they do it by feedforward.

Seeking alternative to the retinal inhibition explanation, Lennie & MacLeod (140) pointed out that a known central process—the reduction of sensitivity in cortical cells by an adapting stimulus that strongly excites them—could account for sensitization. The size-selectivity of cortical neurons might be sufficient to prevent a large background from exciting the neurons required for detection of the tiny test spot. On this view, sensitization could be regarded as the removal of a desensitizing influence of contours near the test area, contours that make the small background an effective stimulus for central neurons. But the idea that contours determine sensitization is questioned by Enoch and Johnson (69), who show that with a windmill-shaped background, many narrow vanes in the sensitizing region are equivalent to a few broad ones.

An interpretation of sensitization in terms of size-selective channels (2, 140, 154) can easily explain the important observation (2, 154) that spatial integration of test flash energy occurs across larger areas with a small background than with a large one: with a small background reducing the sensitivity of the "smaller" channels, the test may have to be detected by "larger" channels with larger areas of spatial integration. To account for this observation on the alternative view that the test spot is always detected by the same cell, the receptive field center must be assumed to contract with increasing background size (154). This hypothetical contraction could also account for sensitization, if sensitivity modification were at a stage after the one at which the change in spatial organization occurs, for then the reduction in spatial integration would be equivalent to a reduction in adapting intensity (154). Such complexity has yet to be demonstrated in the retina. To complicate matters further, Vassilev (214) reports that the elevation of threshold near an edge is least evident with small test stimuli and thus involves an *increase* in spatial integration near the edge. Since this is the opposite of the effect found with concentric backgrounds, any common mechanism underlying both phenomena must be quite a complicated one.

In summary, recent studies of sensitization have only served to increase uncertainty about its origin, with a growing awareness of the possible importance of amacrine cells and of more central factors. The relationship of sensitization to uniform field adaptation on the one hand, and to masking on the other, represents an intriguing current problem.

A Psychophysics of the Inner Plexiform Layer?

Werblin & Copenhagen (222) describe an ingenious method of investigating lateral interactions in the inner plexiform layer while holding more peripheral processes

relatively constant. They placed a test spot at the center of a windmill pattern. As the windmill turns, there is a rotation of activity among the horizontal cells in the outer plexiform layer, but the total signal averaged over all cells might be expected to stay the same as the sustained response to a stationary windmill. In the inner plexiform layer, however, many amacrine cells respond only weakly to steady stimulation but are excited by changes from light to dark or from dark to light. These cells are much more strongly excited by a spinning windmill than by a static one. Werblin and Copenhagen found that "on" type ganglion cells did not differentiate between the static and the spinning windmill, but "on-off" type ganglion cells were more strongly inhibited by a spinning windmill than by a static one. The implication is that amacrine cells antagonize the response to change found in on-off cells, but have little effect on the response of sustained ganglion cells. Enoch, Lazarus & Johnson (70) have adopted a similar technique in psychophysical experiments. A rotating windmill always gave less sensitivity than a static windmill, presumably owing to inhibition from transient-type amacrine cells. Interestingly enough, this difference was found only when the test spot was periodically interrupted and the observer's task was to detect the interruptions. A sustained perception of the test spot could still be obtained with full sensitivity despite the rotation of the windmill, perhaps because the sustained-type ganglion cells are free from inhibition by amacrines, as the physiological observations of Werblin & Copenhagen (222) and others (107, 130) suggest. A windmill seen by cones can raise rod threshold (115) but interocular transfer is not conspicuous (117).

THE NEW VISUAL DUPLICITY

Physiological Identification of X and Y Systems

Anatomists have long been concerned with cataloging the diversity of cell types at any particular level of the visual system. The anatomical distinction between rod and cone receptors was given visual significance long ago, but the extent of the diversity among afferent neurons has only recently been recognized physiologically, and still more recently psychophysically. The switch to "multiple channel thinking" amounts to a revolution in our outlook, and has created an era of frenetic classification in which each class of cell at each level of the visual system is given its own physiological characterization and if possible its own role in vision.

Over the past few years, a distinction between two systems involving cells known cryptically as "X" and "Y" cells, and recognizable at retinal, thalamic, and perhaps cortical levels, has become increasingly prominent. The two types are easily distinguished on the basis of the linearity of their spatial summation, which was their original defining property (75). X cells are practically linear in spatial summation [though nonlinear in other respects (75)], so that when a grating stimulus is suitably positioned, an X cell will not respond if the light and dark bars are exchanged. For Y cells, no such null positions can be found: no change escapes their notice (75, 106). The two cell types can also be distinguished on the basis of their response latencies or conduction velocities (64, 106), though the concordance between a conduction velocity criterion and a linearity criterion may not be perfect (55). They also differ

in the time course of their responses to a stimulus switched on and left on (so that in popular parlance X cells are "sustained" and Y cells are "transient"), but light adaptation tends to jeopardize classification on this basis, for in the cat retina, Y cells become sustained at sufficiently low light levels, while at high light levels even X cells acquire a transient response to stimulus onset with only a slight sustained component (117).

Electrophysiological evidence in the cat has tended to suggest that X and Y cells are differently distributed over the retina, with Y cells rare in central vision but abundant in the periphery. Surveys by anatomical methods have cast doubt on this, suggesting that Y cells comprise an almost uniform 2–4% of cells at all retinal locations in the cat (142), but one recent study of the cat lateral geniculate (141) supports the differential distribution. In the monkey, the recordings of DeMonasterio & Gouras (54) show X cells predominating in the fovea and Y cells in the periphery, but Schiller & Malpeli (190), with a bigger sample, but from a more restricted range of eccentricities, report a uniform distribution. Cell bodies and dendritic fields of Y cells are larger than those of X cells at the same retinal eccentricity, and the receptive field centers of X cells are correspondingly smaller than those of Y cells in cat (44, 142) and monkey (54).

The X/Y distinction was discovered in the cat (75), and until recently, as Jacobs (116) points out, there has been no clear indication of its existence in primates. The ambiguities of categorization make it hard to be certain that classifications proposed for different species are homologous. Nevertheless, a distinction between what might cautiously be termed X-like and Y-like cells now seems at least as clear in monkeys as in cats, both at the retinal level (54, 55, 190) and at the thalamic level, where X and Y cells are apparently segregated into different laminae, X cells comprising the small cell laminae and Y cells comprising the less extensively explored magnocellular laminae (64, 196; see also 36). The X/Y distinction also bears on primate color vision. At the retinal level, the indications are that the X cells are spectrally opponent, while the Y cells have spectrally broad-band receptive field centers (55, 190), but at the lateral geniculate, many X cells are spectrally nonopponent like the Y cells and are found alongside the other, opponent X cells in the parvocellular laminae (64).

Thus far it is a reasonably tidy arrangement; but there are complications. In monkey cortex, the concordance between the opponent/nonopponent classification and receptive field categories seems looser than at lower levels (60, 61, 95), although the reported sparsity of opponent cells in Brodmann's layer 4B does suggest some preservation of the segregation established at the lateral geniculate level. Somewhat similarly, Bartlett & Doty (16) find some units in monkey cortex that are only slightly influenced by the large cell laminae of the lateral geniculate, but nevertheless "a definitive relation between magnocellular input and type of response could not be discerned." Even at the ganglion cell level in the monkey, DeMonasterio & Gouras (54) have refined their X-like/Y-like dichotomy to yield at least 25 different functional types of cell, mostly distinguished by their spectral sensitivities, and certain cells appear to have some X-like as well as some Y-like properties (55). The X cells of the monkey lateral geniculate may be very different from those of the cat

retina—some, for instance, identified as X-like by Dreher, Fukada & Rodieck (64), show spectral opponency but lack the spatial opponency prominent in cat X cells. A distinction between sustained and transient cells has recently been proposed for the parvocellular laminae of the monkey lateral geniculate (151), which, according to other investigators (64), contain only X-like cells. In general it is seldom clear that the classifications of different investigators are the same, especially when based on different tests. Because of this uncertainty, the X-like/Y-like dichotomies reviewed above are usually reported using different labels. The resulting riot of proposed classifications nicely illustrates the shrewd observation that most scientists would rather share another person's toothbrush than adopt his terminology (or his classification criteria).

A final complication is that the X/Y classification is by no means exhaustive. There is also a large and heterogeneous population of slowly conducting cells dubbed W cells, which at first were thought to project only to the midbrain, but which have recently been discovered in the cat's lateral geniculate nucleus (e.g. 36, 227). Often passed over because of their small size, they are probably about as numerous as X cells and much more numerous than Y cells. Their role in vision is still a matter for speculation.

CORTICAL PROCESSING: PARALLEL, SERIAL, OR JUST COMPLICATED? The extension of the X/Y distinction into the brain, together with the somewhat X-like and Y-like qualities of simple and complex cortical cells as described by Hubel & Wiesel (111), led to the suggestion made, for instance, by Stone & Freeman (204) that simple cells are cortical representatives of the X system and complex cells cortical representatives of the Y system. Each cell type would be driven directly by cells of the same type in the lateral geniculate. This parallel processing view of the simple/complex distinction stands in contrast to the hierarchical scheme formerly in vogue (193). The parallel processing view has been favored for several reasons: first, as Stone and Freeman point out, complex cell latencies may be shorter than those of simple cells, so they cannot always be the successors of simple cells in a serial process. Second, the excitatory input to complex cells is not markedly directionally selective, suggesting that it originates in the lateral geniculate rather than from simple cells (198). Third, complex cells may respond well to stimuli such as rapid motion (161) or coherent movement of a field of randomly arranged tiny speckles within a similarly textured background (102), to which simple cells are relatively or absolutely unresponsive. Fourth, complex cells are generally pyramidal cells (127) and these receive a direct input from the lateral geniculate (204). But although the refutation of a simple hierarchical model has put the parallel processing view in the ascendant, there are several indications that the parallel processing view is likewise inadequate. Kelly & van Essen (127) and Ikeda & Wright (113) found both sustained and transient-responding cells within both the simple and the complex groups. The identity of their sustained/transient distinction to the X/Y distinction could be doubted, however, since tests of linearity or conduction velocity were not made. Recent studies of response latency (200) show both X (slow) and Y (fast) inputs to all cortical receptive-field types. In addition, both among simple cells and among complex cells, some are directly excited from the lateral geniculate while others of the same class are only indirectly excited (200). A specific organization scheme capable of accommodating these complexities has not yet been proposed.

Psychophysical Identification of X and Y Systems?

Presumably X and Y cells have more or less distinct roles in vision. What are those roles? The physiological literature is curiously silent on this point, but eager psychophysicists have rushed in where cautious physiologists feared to tread. Several investigators have tried to isolate X or Y systems by using appropriate stimulus conditions so that their stimulus requirements and visual consequences could be demonstrated in human vision, or have ascribed changes of visual performance under different conditions to the use of X cells under some conditions, Y cells under others. The situation could reasonably be summed up with the Scottish verdict "not proven," but some of the suggested correlations between physiology and psychophysics are at least interesting and plausible.

SPACE-TIME INTERACTIONS Grating stimuli have provided a popular tool for preferentially stimulating the X and Y systems: Y cells tend to prefer coarse gratings and fail to resolve fine ones, but the smaller receptive fields and stronger center-surround antagonism of X cells make them relatively responsive to fine gratings (75). Tolhurst (206) examined the loss of sensitivity to moving grating test stimuli incurred by pre-exposure to adapting gratings of the same spatial frequency as the test grating. For coarse gratings, movement made the grating much more visible and the adapting grating had to move to effectively reduce sensitivity, whereas with stationary test stimuli, static adapting gratings are also effective. The inference is that the coarse moving gratings were detected by a system selectively responsive to moving stimuli (like the "transient" Y system) whereas the stationary stimuli were detected by a system poorly responsive to coarse gratings but responsive to static as well as to moving stimuli (like the "sustained" X system). An alternative interpretation, not convincingly excluded, is that transient stimuli are necessary for inducing a loss of sensitivity at any spatial frequency (as might be expected in view of the fading of stabilized images); the necessary change of stimulation with time might require movement of a coarse grating stimulus but could be supplied by small fixational eye movements if the grating were a fine one. A similar eye movement interpretation can be applied to Kulikowski & Tolhurst's (134) demonstration that phase alternation, as compared with exposure in one phase only, improves visibility for coarse gratings but not for fine ones; with fine gratings, eye movements create phase alternations even when the external stimulus is static. Eye movement effects might also account for some other phenomena cited in support of sustained and transient channels in human vision (207, 208), but they were presumably not involved in Keesey's early study (123) using stabilized vision. More recently, King-Smith & Kulikowski's formidable study of summation between subthreshold flickering lines and gratings for detection of pattern or of flicker (129) buttresses the previous psychophysical identifications of X and Y systems by demonstrating rough

correlations with physiological findings in respect to temporal frequency response, nonlinearity, width of receptive field, strength of inhibitory surround, and sensitivity to motion.

A marked increase of reaction time with increasing spatial frequency of a grating test object has been noted independently in three laboratories (29, 144, 215), and in each case the interpretation has been suggested that the coarser gratings are detected mainly by the relatively quick responding Y cells, the finer ones by X cells. The effect has also been detected in the magnetic evoked response (226), and the latency difference between X-like and Y-like cells may approach the required magnitude (60), even though the characteristic difference in conduction velocity could account for only a tiny fraction of it. However, the way that reaction time (or neuromagnetic latency) varies with frequency seems difficult to reconcile with the X/Y interpretation, for instead of approaching an asymptote characteristic of the X system at high spatial frequencies, it continues to increase without obvious limit as the gratings become finer (29, 144). Watson & Nachmias (219), in their study of temporal summation as a function of spatial frequency, account for their similar observations by proposing that observers have available an array of spatial frequency-selective channels, the temporal properties of which vary progressively with their characteristic spatial frequencies. This view need not invoke any X/Y distinction but is not incompatible with it; the postulated diversity might, for instance, be characteristic of the X system alone (31), or of both systems (206).

Another consequence of the X/Y difference in receptive field organization is that X cells, while useful for fine resolution, are less tolerant of image blurring than Y cells, as confirmed by Ikeda & Wright (112). A possibly parallel psychophysical result is Hood's (109) observation that blurring reduces both sensitivity and perceived distinctness for stimuli presented under long exposure (a condition relatively favorable to the X cells) whereas with short exposures, presumably favorable to the Y cells, appearance and visibility were hardly affected by blurring; somewhat similarly, long (say, 400 msec) exposure benefits acuity (presumably an X cell function) more than simple detection (for which the observer might avail himself of transient cell signals) (15, 35). But observations like these can be equally explained without postulating two channels, by appeal to the interplay of excitation and inhibition in a homogeneous array of cells (109, 126).

If only one of the parallel rod or cone receptor systems is activated by a test stimulus, visual sensitivity reflects the properties of that mechanism alone, and simplifying principles like Stiles' displacement rules (203) can be applied to predict how sensitivity will vary with changing conditions. Successful prediction helps both to establish the existence of the postulated mechanism and to define its characteristics. This level of analysis has yet to be attained in the study of sustained and transient afferent systems. Kulikowski & Tolhurst come close when they show that the temporal frequency response of each of the two hypothetical systems may be unaltered by changing spatial frequency (134, Figures 7 and 8). However, Breitmeyer & Ganz (31) postulate different temporal properties for "sustained" signals evoked by different spatial frequencies, and King-Smith & Kulikowski (129) have to let the spatial frequency response of their flicker detecting system change with temporal frequency. This is not unreasonable but it does slightly weaken the explanatory power and empirical foundation of the two-channel scheme.

Other time-space interactions are positively embarrassing for the two channel view: Arend (3) finds that blurring abolishes the Broca-Sulzer brightness overshoot (commonly regarded as an index of the transience of the neural response) instead of increasing it, as might be expected if blurring selectively reduces X cell signals; and Barlow (7) reported that in the fading of stabilized images, details disappear before large blobs. Barlow's observation led him to formulate the first psychophysical theory involving sustained and transient channels, but in Barlow's scheme it was the transient channels that have small receptive fields! Curiously enough the first physiological reports on primates (94) proposed a similar arrangement. Will the different scheme currently in vogue prove equally transient, or can it be sustained?

COLOR CORRELATES Since the distinction between spectrally opponent and nonopponent cells has a clear subjective correlate in the well-documented distinction between processing of luminance differences and purely chromatic differences (57), the suggestion that all opponent cells are X cells (64) could aid in the psychophysical identification of X and Y systems. The proposal that opponent cells are sustained (X-like) in character is supported by the now familiar observation (125) that a sinusoidal alternation of equally luminous colors (unlike a luminance fluctuation) loses nothing in visibility if it is slowed down to very low alternation rates. Awareness of luminance fluctuations dominates over awareness of chromatic fluctuations as temporal frequency increases, just as in Kulikowski & Tolhurst's experiments (134), awareness of flicker dominates over awareness of pattern. Observations of spatial resolution, however, are harder to accommodate within this framework. Experiments with gratings suggest that the color-opponent system has a wider range of spatial integration than the nonopponent system, with little evidence of lateral inhibition (218). Yet if one could generalize from cat retina, the small, spatially antagonistic receptive fields of the sustained system ought to provide superior resolution and more prominent lateral inhibition. Perhaps the limits on spatial resolution, for achromatic as well as chromatic stimuli, are set by X-like cells. In the primate lateral geniculate, even the spectrally opponent X-like cells respond much better to a fine grating with light and dark bars than to an equiluminous chromatic pattern, thus providing a possible basis for our good resolution of luminance contrasts (57).

SUBJECTIVE SIGNS AND VISUAL FUNCTIONS In the classical duplicity theory, the roles of the rod and cone systems in vision could easily be appreciated by setting up conditions under which vision depends on rods alone or on cones alone. But there is at present no clear consensus on what a pure X system signal or a pure Y signal would actually look like and no agreed technique for producing such signals. The contribution of the Y system is particularly obscure: if "X marks the spot" (a mnemonic suggested by D. A. Norman), what does Y do? One currently favored view is that X cells subserve inspection of static or tracked objects, while the Y system alerts the observer to changes in the scene. According to Kulikowski &

Tolhurst (134), when a sinusoidal grating stimulus oscillates in polarity, it is the "sustained" X cells that report the spatial pattern, whereas the sensation associated with excitation of "transient" Y cells is one of flicker. Rapid oscillation and coarse grating patterns are conditions relatively favorable for the flicker sensation. More recently, however, the general validity of the assumption that we see pattern with sustained channels and flicker with transient channels has been called in question by the results of Watson & Nachmias (219; see also 4), who find similar temporal summation characteristics for seeing flicker and for seeing pattern. King-Smith & Kulikowski (129) note that subjective motion is the usual consequence of activating the presumed Y-like system with the flickering patterns they used, and they point out that nonlinearity, the defining characteristic of the Y system, is a prerequisite for a useful motion detector. E. Matin (153) also associates the Y system with detection of motion but suggests that it may also serve to report the occurrence of saccadic eye movements even though retinal velocities during saccades may be too high to generate a subjective impression of motion. Sekuler (194, p. 405) and Nelson (163) "second the motion" and point out that it is consistent with a concentration of Y cells in the extrafoveal retina. Breitmeyer & Ganz (31) agree that Y cells serve to detect rather than identify a stimulus, but do not associate them with a sensation of motion. Breitmeyer & Ganz propose that the test flash in a metacontrast experiment, often unmistakably detected but not identifiable, is being detected by the Y system. The other "pure" case, seeing with X cells, may be occurring in the rotating windmill experiments of Johnson & Enoch (119), where subjects were easily able to perceive the test spot but could not detect that it was being briefly interrupted.

X/Y inhibition and backward masking Singer and Bedworth (199; see also 87, 200) noted an inhibitory effect of the Y system on the X system in the cat that may be of cardinal importance for theories of masking. The inhibition may persist for a considerable fraction of a second, and because the Y system responds with a shorter latency than the X system, the response to the test flash may be affected even when the test flash precedes the mask by tens of milliseconds. Breitmeyer (30), E. Matin (153), Weisstein, Ozog & Szoc (220), and (most explicitly) Breitmeyer & Ganz (31) have greeted this as the long-sought-after "fast inhibition" required for explaining backward masking. (Ordinarily, lateral inhibition is delayed (73, 145), presumably because it has to be relayed through an interneuron.) Breitmeyer and Ganz show how the inhibition of X by Y can explain not only the superiority of backward masking over simultaneous masking (the "U-shaped function") but also many other aspects of masking that have previously been held to require an "interruption of processing" interpretation too sophisticated to be compatible with a primitive neural mechanism. Matin's theory of metacontrast includes the interesting suggestion that the inhibition is caused by the Y system's response, not to the masking flash itself but rather to the successive paired presentation of test and mask. The motion-detecting role of the Y cells is thought to make them particularly susceptible to such a stimulus, and the theory is thus able to account for the connection between metacontrast and apparent motion, discussed by Matin. Bridgeman's physiological observations in a metacontrast situation (32) seem partly (but not entirely) consistent with these proposals.

Since Y cells are spectrally nonopponent (54, 64, 190) they should not respond to a chromatic substitution at constant luminance (57), so according to the above theories, a chromatic substitution might not be expected to exert any backward masking. Bowen, Pokorny & Cacciato (24) have verified this prediction in a metacontrast situation; Glass & Sternheim (89) did find substantial transient threshold elevations with chromatic substitution of uniform fields, but their criterion for equality of luminance may not have been the appropriate one.

These developments seem likely to inaugurate a new generation of masking studies, both theoretical and experimental.

Saccadic suppression According to E. Matin (153) and Breitmeyer & Ganz (31), inhibition of X cells by Y cells could be important in suppressing vision when a saccadic eye movement occurs. The movement of the scene across the retina during a saccade would powerfully excite the Y system. This assumption is supported by the physiological observation (10, 83) that all Y cells in the retina, both on- and off-center, may be strongly excited by synchronously shifting the parts of the visual field outside the classical receptive field, an effect believed due to the amacrine cells. This unique response (anomalous because on- and off-cells are made to fire together, the opposite of what happens with stimuli delivered within the receptive field) could have the specific function of "wiping the slate clean for the next image" (10), and the mechanism by which this is accomplished could be the inhibition of the X cells by the Y cells (31, 153). It may at first seem unsatisfactory to invoke the same inhibitory process both for metacontrast effects, which require local contour (31), and for saccadic suppression, which operates on a more global basis, but since under saccadic conditions the Y cells have been shown to be excitable from a region much larger than the normal receptive field (10, 83), whereas in a metacontrast situation they would be excitable only within the receptive field, both the global and the local effects could be predicted if X cells are inhibited only by neighboring Y cells.

A central process triggered by oculomotor events rather than by image motion also contributes to saccadic suppression, but its effects are generally weak, both psychophysically and in the cortex (17, 121, 152).

Inhibition of the X system by the Y system might yield a plausible account of other perceptual phenomena, such as the Crawford masking and sensitization effects, but perhaps the further development of such notions should be postponed in view of the recent report (64) of no obvious X/Y inhibition in monkey lateral geniculate. If confirmed and extended to monkey cortex, this observation could make a striking object lesson on the need for caution in extrapolating to man from his more or less distant relatives.

TWO VISUAL SYSTEMS The function ascribed to Y cells, of allowing initial orientation to new or moving stimuli, has also often been associated with the "second visual system" centered in the midbrain (91, 114). This may not be coincidence,

because Y ganglion cell axons (but not those of X cells) bifurcate to form a pathway to the midbrain as well as to the cortex (e.g. 190).

CONCLUSION

As this review closes, a persevering reader may share the reaction of King Alphonso X (The Wise) of Castile, who on being introduced to the then prevailing Ptolemaic cosmology of wheels within wheels pronounced: "If the Lord Almighty had consulted me before embarking upon the Creation, I should have recommended something simpler." When seen in contrast to the Galilean simplicity of Hecht's conception of the visual system, the events outlined here (and particularly the multiple channels developments) may seem a backward step, a Ptolemaic revolution in vision. But the analogy is misleading, for the complexity of structure revealed by physiological and anatomical probes is undeniable, and no intellectual conjury will make it vanish. What does remain open to discussion and investigation is the role of that complexity in seeing.

Literature Cited

1. Afanador, A. J., Adams, A. J. 1974. Early visual adaptation in goldfish retinal ganglion cells. *Nature* 250:347–48
2. Alexander, K. R. 1974. Sensitization by annular surrounds: The effect of test stimulus size. *Vision Res.* 14:1107–13
3. Arend, L. E. 1973. Spatial factors in the Broca-Sulzer phenomenon. *J. Opt. Soc. Am.* 63:879–83
4. Arend, L. E. 1976. Temporal determinants of the form of the spatial contrast threshold MTF. *Vision Res.* 16:1035–42
5. Armington, J. C. 1974. *The Electroretinogram.* New York: Academic
6. Augenstein, E. J., Pugh, E. N. 1977. The dynamics of the π_1 color mechanism: Further evidence for two sites of adaptation. *J. Physiol.* In press
7. Barlow, H. B. 1963. Slippage of contact lenses and other artifacts in relation to fading and regeneration of supposedly stable retinal images. *Q. J. Exp. Psychol.* 15:36–51
8. Barlow, H. B. 1972. Single units and sensation: A neuron doctrine for perceptual psychology? *Perception* 1: 371–94
9. Barlow, H. B. 1975. Visual experience and cortical development. *Nature* 258:199–204
10. Barlow, H. B., Derrington, A. M., Harris, L. R., Lennie, P. 1977. The effects of remote retinal stimulation on the responses of cat retinal ganglion cells. *J. Physiol.* 269:177–94
11. Barlow, H. B., Fatt, P., eds. 1978. *Photoreception.* New York: Academic. In preparation
12. Barlow, H. B., Levick, W. R. 1976. Threshold setting by the surround of cat retinal ganglion cells. *J. Physiol.* 259:737–57
13. Barlow, H. B., Sakitt, B. 1973. Doubts about scotopic interactions in stabilized vision. *Vision Res.* 13:523–24
14. Baron, W. S., Boynton, R. M. 1975. Response of primate cones to sinusoidally flickering homochromatic stimuli. *J. Physiol.* 246:311–31
15. Baron, W. S., Westheimer, G. 1973. Visual acuity as a function of exposure duration. *J. Opt. Soc. Am.* 63:212–19
16. Bartlett, J. R., Doty, R. W. Sr. 1974. Responses of units in striate cortex of squirrel monkeys to visual and electrical stimuli. *J. Neurophysiol.* 37: 621–41
17. Bartlett, J. R., Doty, R. W., Lee, B. B., Sakakura, H. 1976. Influence of saccadic eye movements on geniculostriate excitability in normal monkeys. *Exp. Brain Res.* 25:487–509
18. Baylor, D. A., Fettiplace, R. 1976. Transmission of signals from photoreceptors to ganglion cells in the eye of the turtle. *Cold Spring Harbor Symp. Quant. Biol.* 40:529–36
19. Baylor, D. A., Fuortes, M. G. F., O'Bryan, P. M. 1971. Receptive fields of single cones in the retina of the turtle. *J. Physiol.* 214:265–94
20. Baylor, D. A., Hodgkin, A. L. 1974. Changes in time scale and sensitivity in turtle photoreceptors. *J. Physiol.* 242:729–58
21. Baylor, D. A., Hodgkin, A. L., Lamb, T. 1974. The electrical response of turtle cones to flashes and steps of light. *J. Physiol.* 242:685–727
22. Baylor, D. A., Hodgkin, A. L., Lamb, T. 1974. Reconstruction of the electrical responses of turtle cones to flashes and steps of light. *J. Physiol.* 242: 759–91
23. Bonting, S. L., ed. 1976. *Transmitters in the Visual Process.* Oxford: Pergamon
24. Bowen, R. W., Pokorny, J., Cacciato, D. 1977. Metacontrast masking depends on luminance transients. *Vision Res.* 17:971–76
25. Boycott, B. B. 1974. Aspects of the comparative anatomy and physiology of the vertebrate retina. In *Essays on the Nervous System,* ed. R. Bellairs, E. G. Gray. Oxford: Clarendon
26. Boynton, R. M., Baron, W. S. 1975. Sinusoidal flicker characteristics of primate cones in response to heterochromatic stimuli. *J. Opt. Soc. Am.* 65:1091–1100
27. Boynton, R. M., Whitten, D. N. 1970. Visual adaptation in monkey cones: Recordings of late receptor potentials. *Science* 170:1423–26
28. Braddick, O. J., Campbell, F. W., Atkinson, J. 1978. Channels in vision: Basic aspects. In *Handbook of Sensory Physiology,* Vol. VIII: *Perception,* ed. R. Held, H. Leibowitz, H. L. Teuber. Heidelberg: Springer-Verlag. In press
29. Breitmeyer, B. G. 1975. Simple reaction time as a measure of the temporal response properties of transient and sustained channels. *Vision Res.* 15: 1411–12
30. Breitmeyer, B. G. 1975. Predictions of U-shaped backward pattern masking from considerations of the spatio-temporal frequency response. *Perception* 4:297–304
31. Breitmeyer, B. G., Ganz, L. 1976. Implications of sustained and transient channels for theories of visual pattern masking, saccadic suppression, and information processing. *Psychol. Rev.* 83:1–36
32. Bridgeman, B. 1975. Correlates of metacontrast in single cells of the cat visual system. *Vision Res.* 15:91–99
33. Brown, J. E., Coles, J. A., Pinto, L. H. 1977. Effects of injections of calcium and EGTA into the outer segments of retinal rods of Bufo marinus. *J. Physiol.* 269:707–22
34. Brown, J. L. 1973. Visual sensitivity. *Ann. Rev. Psychol.* 24:151–86
35. Brown, J. L., Black, J. E. 1976. Critical duration for resolution of acuity targets. *Vision Res.* 16:309–15
36. Bunt, A. H., Hendrickson, A. E., Lund, J. S., Lund, R. D., Fuchs, A. F. 1975. Monkey retinal ganglion cells: Morphometric analysis and tracing of axonal projections, with a consideration of the peroxidase technique. *J. Comp. Neurol.* 164:265–86
37. Burkhardt, D. A. 1974. Sensitization and center-surround antagonism in *Necturus* retina. *J. Physiol.* 236:593–610
38. Burkhardt, D. A. 1975. A nascent electrophysiology of amacrine cells. *Invest. Ophthalmol.* 14:171–73
39. Burkhardt, D. A., Berntson, G. G. 1972. Light adaptation and excitation: Lateral spread of signals within the frog retina. *Vision Res.* 12:1095–1112
40. Büttner, U., Grüsser, O. -J., Schwanz, E. 1975. The effect of area and intensity on the response of cat retinal ganglion cells to brief light flashes. *Exp. Brain Res.* 23:259–78
41. Carterette, E. C., Friedman, M. P., eds. 1975. *Handbook of Perception,* Vol. V: *Seeing.* New York: Academic
42. Chan, R. Y., Naka, K. -I. 1976. The amacrine cell. *Vision Res.* 16:1119–29
43. Cleland, B. G., Levick, W. R., Sanderson, K. J. 1973. Properties of sustained and transient cells in the cat retina. *J. Physiol.* 228:649–80
44. Cleland, B. G., Levick, W. R., Wassle, H. 1975. Physiological identification of a morphological class of cat retinal ganglion cells. *J. Physiol.* 248:151–71
45. Coenen, A. M. L., Gerrits, H. J. M., Vendrik, A. J. H. 1972. Analysis of the response characteristics of optic tract and geniculate units and their mutual relationship. *Exp. Brain Res.* 15:452–71
46. Cohen, L. B., Salapatek, P. 1975. *Infant Perception. From Sensation to Cognition.* New York: Academic. 2 vols.
47. Conner, J. D., MacLeod, D. I. A. 1977. Rod photoreceptors detect rapid flicker. *Science* 195:698–99
48. Copenhagen, D. R. 1975. Time course of threshold elevation in on-off ganglion cells of *Necturus* retina: Effects of lateral interactions. *Vision Res.* 15:573–81
49. Copenhagen, D. R., Owen, W. G. 1976. Functional characteristics of lateral interactions between rods in the retina of the snapping turtle. *J. Physiol.* 259: 251–82

50. Creutzfeldt, O. D., Kuhnt, U., Benevento, L. A. 1974. An intracellular analysis of visual cortical neurones to moving stimuli: responses in a co-operative neuronal network. *Exp. Brain Res.* 21:251–74
51. Davson, H., ed. 1976. *The Eye.* Vol. 2A: *Visual Function in Man.* New York: Academic. 2nd ed.
52. Davson, H., Graham, L. T., eds. 1974. *The Eye.* Vol. 6: *Comparative Physiology.* New York: Academic. 2nd ed.
53. Dealy, R. S., Tolhurst, D. J. 1974. Is spatial adaptation an after-effect of prolonged inhibition? *J. Physiol.* 241: 261–70
54. DeMonasterio, F. M., Gouras, P. 1975. Functional properties of ganglion cells of the rhesus monkey retina. *J. Physiol.* 251:167–95
55. DeMonasterio, F. M., Gouras, P., Tolhurst, D. J. 1976. Spatial summation, response pattern and conduction velocity of ganglion cells of the rhesus monkey retina. *Vision Res.* 16:674–78
56. Desmedt, J. E., ed. 1977. *Visual Evoked Potentials in Man.* New York: Oxford Univ. Press
57. DeValois, R. L., DeValois, K. K. 1975. Neural coding of color. See Ref. 41, pp. 117–66
58. Ditchburn, R. W. 1973. *Eye-movements and Visual Perception.* Oxford: Clarendon
59. Doty, R. W., Wilson, P. D., Bartlett, J. R., Pecci-Saavedra, J. 1973. Mesencephalic control of lateral geniculate nucleus in primates. I. Electrophysiology. *Exp. Brain Res.* 18:189–203
60. Dow, B. M. 1974. Functional classes of cells and their laminar distribution in monkey visual cortex. *J. Neurophysiol.* 37:927–46
61. Dow, B. M. 1976. Central mechanisms of vision: Parallel processing. *Fed. Proc.* 35:54–59
62. Dowling, J. E., Ehinger, B., Hedden, W. L. 1976. The interplexiform cell: A new type of retinal neuron. *Invest. Ophthalmol.* 15:916–26
63. Dowling, J. E., Ripps, H. 1976. Potassium and retinal sensitivity. *Brain Res.* 107:617–22
64. Dreher, B., Fukada, Y., Rodieck, R. W. 1976. Identification, classification and anatomical segregation of cells with X- and Y-like properties in the lateral geniculate nucleus of old-world primates. *J. Physiol.* 258:433–52
65. Dubin, M. W., Cleland, B. G. 1977. Organization of visual inputs to interneurons of lateral geniculate nucleus of the cat. *J. Neurophysiol.* 40:410–27
66. Duffy, F. H., Snodgrass, S. R., Burchfiel, J. L., Conway, J. L. 1976. Bicuculline reversal of deprivation amblyopia in the cat. *Nature* 260:256–57
67. Enoch, J. M. 1976. Vertebrate photoreceptor orientation. *Int. J. Quantum Chem. Symp.* 3:65–88
68. Enoch, J. M., Horowitz, B. R. 1975. The vertebrate retinal receptor as a waveguide. In *Optical and Acoustical Micro-Electronics,* ed. J. Fox. *Microwave Res. Inst. Symp. Ser.* 23:133–59. New York: Wiley
69. Enoch, J. M., Johnson, C. A. 1976. Additivity of effects within sectors of the sensitization zone of the Westheimer function. *Am. J. Optom. Physiol. Optics* 53:350–58
70. Enoch, J. M., Lazarus, J., Johnson, C. A. 1976. Human psychophysical analysis of receptive field-like properties-I. A new transient-like visual response using a moving windmill (Werblin-type) target. *Sens. Proc.* 1:14–32
71. Enroth-Cugell, C., Hertz, B. G., Lennie, P. 1977. Cone signals in the cat's retina. *J. Physiol.* 269:273–96
72. Enroth-Cugell, C., Hertz, B. G., Lennie, P. 1977. Convergence of rod and cone signals in the cat's retina. *J. Physiol.* 269:297–318
73. Enroth-Cugell, C., Lennie, P. 1975. Control of retinal ganglion cell discharge by receptive field surrounds. *J. Physiol.* 247:551–78
74. Enroth-Cugell, C., Lennie, P., Shapley, R. M. 1975. Surround contribution to light adaptation in cat retinal ganglion cells. *J. Physiol.* 245:579–88
75. Enroth-Cugell, C., Robson, J. G. 1966. The contrast sensitivity of retinal ganglion cells of the cat. *J. Physiol.* 187:517–52
76. Enroth-Cugell, C., Shapley, R. M. 1973. Adaptation and dynamics of cat retinal ganglion cells. *J. Physiol.* 233: 271–309
77. Enroth-Cugell, C., Shapley, R. M. 1973. Flux, not retinal illumination, is what cat retinal ganglion cells really care about. *J. Physiol.* 233:311–26
78. Fain, G. L. 1976. Sensitivity of toad rods: Dependence on wave-length and background illumination. *J. Physiol.* 261:71–101
79. Fain, G. L., Dowling, J. E. 1973. Intracellular recordings from single rods and cones in the mudpuppy retina. *Science* 180:1178–81

80. Fain, G. L., Gold, G. H., Dowling, J. E. 1976. Receptor coupling in the toad retina. *Cold Spring Harbor Symp. Quant. Biol.* 60:547–61
81. Famiglietti, E. V., Kolb, H. 1976. Structural basis for ON- and OFF-center responses in retinal ganglion cells. *Science* 194:193–95
82. Fischer, B. 1973. Overlap of receptive field centers and representation of the visual field in the cat's optic tract. *Vision Res.* 13:2113–20
83. Fischer, B., Kruger, J., Droll, W. 1975. Quantitative aspects of the shift-effect in cat retinal ganglion cells. *Brain Res.* 83:391–403
84. Fite, K. V., ed. 1976. *The Amphibian Visual System: A Multidisciplinary Approach.* New York: Academic
85. Frumkes, T. E., Temme, L. A. 1977. Rod-cone interaction in human scotopic vision-II. Cones influence increment thresholds detected by rods. *Vision Res.* 17:673–79
86. Fry, G. A. 1973. Response of the eye to a flash of light. *Am. J. Optom.* 50: 355–75
87. Fukada, Y., Stone, J. 1976. Evidence of differential inhibitory influences on X- and Y-type relay cells in the cat's lateral geniculate nucleus. *Brain Res.* 113: 188–96
88. Gazzaniga, M. S., Blakemore, C. B. 1975. *Handbook of Psychobiology.* New York: Academic
89. Glass, R. A., Sternheim, C. E. 1973. Visual sensitivity in the presence of alternating monochromatic fields of light. *Vision Res.* 13:689–99
90. Glezer, V. D., ed. 1976. *Information processing in the visual system. Proc. 4th Symp. Sens. Syst. Physiol.* Leningrad: USSR Acad. Sci.
91. Gordon, B. 1975. Superior colliculus: Structure, physiology and possible functions. In *Neurophysiology,* ed. C. C. Hunt, 3:185–230. MTP Rev. Sci. Physiol., Ser. 1. Baltimore: Univ. Park Press
92. Gosline, C. J., MacLeod, D. I. A., Rushton, W. A. H. 1976. The dark adaptation curve of rods measured by their afterimage. *J. Physiol.* 259: 491–99
93. Gottlieb, G., ed. 1976. Studies on the development of behavior and the nervous system. *Neural and Behavioral Specificity,* Vol. 3. New York: Academic
94. Gouras, P. 1968. Identification of cone mechanisms in monkey ganglion cells. *J. Physiol.* 199:533–47
95. Gouras, P. 1974. Opponent-color cells in different layers of foveal striate cortex. *J. Physiol.* 238:583–602
96. Graham, N. 1978. Spatial frequency channels in human vision: Detecting edges without edge detectors. In *Visual Coding and Adaptability,* ed. C. S. Harris. Hillsdale, NJ: Erlbaum
97. Green, D. G. 1973. Scotopic and photopic components of the rat electroretinogram. *J. Physiol.* 228:781–97
98. Green, D. G., Dowling, J. E., Siegel, I. M., Ripps, H. 1975. Retinal mechanisms of visual adaptation in the skate. *J. Gen. Physiol.* 65:483–502
99. Green, D. G., Siegel, I. M. 1975. Double branched flicker fusion curves from the all-rod skate retina. *Science* 188: 1120–22
100. Green, D. G., Tong, L., Cicerone, C. M. 1977. Lateral spread of light adaptation in the rat retina. *Vision Res.* 17:479–86
101. Hallett, P. E. 1971. Rapid changes and hysteresis in spatial integration for human rod vision. *J. Physiol.* 215:433–47
102. Hammond, P., MacKay, D. M. 1975. Differential responses of cat visual cortical cells to textured stimuli. *Exp. Brain Res.* 22:427–30
103. Hayhoe, M. M., MacLeod, D. I. A., Bruch, T. 1976. Rod-cone independence in dark adaptation. *Vision Res.* 16:591–600
104. Hess, R., Negishi, K., Creutzfeldt, O. D. 1975. The horizontal spread of intracortical inhibition in the visual cortex. *Exp. Brain Res.* 22:415–19
105. Higgins, K. E., Rinalducci, E. J. 1975. The spatial Broca-Sulzer and sensitization effects for foveal viewing. *Vision Res.* 15:423–25
106. Hochstein, S., Shapley, R. M. 1976. Quantitative analysis of retinal ganglion cell classifications. *J. Physiol.* 262: 237–64
107. Hochstein, S., Shapley, R. M. 1976. Linear and nonlinear spatial subunits in Y cat retinal ganglion cells. *J. Physiol.* 262:265–84
108. Hodgkin, A. L. 1971. Recent work on visual mechanisms. *Proc. R. Soc. London Ser. A* 326:v–xx
109. Hood, D. C. 1973. The effects of edge sharpness and exposure duration on detection threshold. *Vision Res.* 13: 759–66
110. Hood, D. C., Hock, P. A. 1975. Light adaptation of the receptors: Increment threshold functions for the frog's rods and cones. *Vision Res.* 15:545–53

111. Hubel, D. H., Wiesel, T. N. 1968. Receptive fields and functional architecture of monkey striate cortex. *J. Physiol.* 195:215–43
112. Ikeda, H., Wright, M. J. 1972. Differential effects of refractive errors and receptive field organization of central and peripheral ganglion cells. *Vision Res.* 12:1465–76
113. Ikeda, H., Wright, M. J. 1975. Spatial and temporal properties of "sustained" and "transient" neurons in Area 17 of the cat's visual cortex. *Exp. Brain Res.* 22:363–83
114. Ingle, D., Sprague, J. M. 1975. Sensorimotor function of the midbrain tectum. *Neurosci. Res. Program Bull.* 13:173–288
115. Ingling, C. R., Lewis, A. L., Loose, D. R., Myers, K. J. 1977. Cones change rod sensitivity. *Vision Res.* 17:555–64
116. Jacobs, G. H. 1976. Color vision. *Ann. Rev. Psychol.* 27:63–89
117. Jakiela, H. G., Enroth-Cugell, C., Shapley, R. M. 1976. Adaptation and dynamics in X-cells and Y-cells of the cat retina. *Exp. Brain Res.* 24:335–42
118. Johnson, C. A., Enoch, J. M. 1976. Human psychophysical analysis of receptive field-like properties-II. Dichoptic properties of the Westheimer function. *Vision Res.* 16:1455–62
119. Johnson, C. A., Enoch, J. M. 1976. Human psychophysical analysis of receptive field-like properties-III. Dichoptic properties of a new transient-like psychophysical function. *Vision Res.* 16:1463–70
120. Karwoski, C. J., Burkhardt, D. A. 1976. Ganglion cell responses of the mudpuppy retina to flashing and moving stimuli. *Vision Res.* 16:1483–95
121. Kasamatsu, T. 1976. Visual cortical neurons influenced by the oculomotor input: Characterization of their receptive field properties. *Brain Res.* 113: 271–92
122. Kaufman, L. 1974. *Sight and Mind.* New York: Oxford Univ. Press
123. Keesey, U. T. 1972. Flicker and pattern detection: A comparison of thresholds. *J. Opt. Soc. Am.* 62:446–48
124. Kelly, D. H. 1971. Theory of flicker and transient responses, I. Uniform fields. *J. Opt. Soc. Am.* 61:537–46
125. Kelly, D. H. 1975. Luminous and chromatic flickering patterns have opposite effects. *Science* 188:371–72
126. Kelly, D. H. 1977. Visual contrast sensitivity. *Opt. Acta* 24:107–29
127. Kelly, J. P., van Essen, D. C. 1974. Cell structure and function in the visual cortex of the cat. *J. Physiol.* 238:515–47
128. King-Smith, P. E., Carden, D. 1976. Luminance and opponent-color contributions to visual detection and adaptation and to temporal and spatial integration. *J. Opt. Soc. Am.* 66:709–17
129. King-Smith, P. E., Kulikowski, J. J. 1975. Pattern and flicker detection analyzed by subthreshold summation. *J. Physiol.* 249:519–48
130. Kirby, A. W., Enroth-Cugell, C. 1976. The involvement of gamma-aminobutyric acid in the organization of cat retinal ganglion cell receptive fields. A study with picrotoxin and bicuculline. *J. Gen. Physiol.* 68:465–84
131. Kleinschmidt, J., Dowling, J. E. 1975. Intracellular recordings from gecko photoreceptors during light and dark adaptation. *J. Gen. Physiol.* 66:617–48
132. Kolb, H., Famiglietti, E. V. 1976. Rod and cone pathways in the retina of the cat. *Invest. Ophthalmol.* 15:935–46
133. Kuffler, S. W., Nicholls, J. G. 1976. *From Neuron to Brain. A Cellular Approach to the Function of the Nervous System.* Sunderland: Sinauer
134. Kulikowski, J. J., Tolhurst, D. J. 1973. Psychophysical evidence for sustained and transient detectors in human vision. *J. Physiol.* 232:149–62
135. Lamb, T. D., Simon, E. J. 1976. The relation between intercellular coupling and electrical noise in turtle photoreceptors. *J. Physiol.* 263:257–86
136. Langer, H., ed. 1973. *Biochemistry and Physiology of Visual Pigments.* Heidelberg: Springer-Verlag
137. Lasansky, A. 1976. Interactions between horizontal cells of the salamander retina. *Invest. Ophthalmol.* 15:909–16
138. Latch, M., Lennie, P. 1977. Rod-cone interaction in light adaptation. *J. Physiol.* 269:517–34
139. Lennie, P., Hertz, B. G., Enroth-Cugell, C. 1976. Saturation of rod pools in cat. *Vision Res.* 16:935–40
140. Lennie, P., MacLeod, D. I. A. 1973. Background configuration and rod threshold. *J. Physiol.* 233:143–56
141. LeVay, S., Ferster, D. 1977. Relay cell classes in the lateral geniculate nucleus of the cat and the effects of visual deprivation. *J. Comp. Neurol.* 172:563–84
142. Levick, W. R. 1975. Form and function of cat retinal ganglion cells. *Nature* 254:659–62
143. Levinson, E., Sekuler, R. W. 1975. Inhibition and disinhibition of direction-specific mechanisms in human vision. *Nature* 254:692–94
143a. Loomis, J. M. 1977. Complementary afterimages and the unequal adapting effects of steady and flickering lights. *J. Opt. Soc. Am.* 67. In press
144. Lupp, U., Hauske, G., Wolf, W. 1976. Perceptual latencies to sinusoidal gratings. *Vision Res.* 16:969–72
145. Magnussen, S., Glad, A. 1975. Temporal frequency characteristics of spatial interaction in human vision. *Exp. Brain Res.* 23:519–28
146. Mansfield, R. J. W. 1973. Brightness function: Effect of area and duration. *J. Opt. Soc. Am.* 63:913–20
147. Mansfield, R. J. W. 1973. Latency functions in human vision. *Vision Res.* 13:2219–34
148. Mansfield, R. J. W. 1976. Visual adaptation: Retinal transduction, brightness and sensitivity. *Vision Res.* 16:679–90
149. Marr, D. 1974. The computation of lightness by the primate retina. *Vision Res.* 14:1377–88
150. Marrocco, R. T. 1972. Responses of monkey optic tract fibers to monochromatic lights. *Vision Res.* 12: 1167–74
151. Marrocco, R. T. 1976. Sustained and transient cells in monkey lateral geniculate nucleus: Conduction velocities and response properties. *J. Neurophysiol.* 39:340–53
152. Matin, E. 1974. Saccadic suppression: A review and an analysis. *Psychol. Bull.* 81:899–917
153. Matin, E. 1975. The two-transient (masking) paradigm. *Psychol. Rev.* 82:451–61
154. Matin, L., Kornheiser, A. 1976. Linked changes in spatial integration, size discrimination and increment threshold with change in background diameter. *Vision Res.* 16:847–60
155. McIlwain, J. T. 1976. Large receptive fields and spatial transformations in the visual system. In *International Review of Psychology.* Neurophysiology II, Vol. 10, ed. R. Porter. Baltimore: Univ. Park Press
156. Miller, R. F., Dacheux, R. F. 1976. Synaptic organization and ionic basis of on and off channels in mudpuppy retina. III. A model of ganglion cell receptive field organization based on chloride-free experiments. *J. Gen. Physiol.* 67:679–90
157. Mollon, J. D., Polden, P. G. 1977. Saturation of a retinal cone mechanism. *Nature* 265:243–46
158. Mollon, J. D., Polden, P. G. 1977. An anomaly in the response of the eye to light of short wavelengths. *Philos. Trans. R. Soc. London Ser. B* 278: 207–40
159. Monty, R. A., Senders, J. W. 1976. *Eye Movements and Psychological Processes* New Jersey: Erlbaum
160. Moore, R. Y. 1977. Central neural control of circadian rhythms. *Front. Neuroendocrinol.* 5. In press
161. Movshon, J. A. 1975. The velocity tuning of single units in cat striate cortex *J. Physiol.* 249:445–68
162. Naka, K.-I. 1976. Neuronal circuitry in the catfish retina. *Invest. Ophthalmol.* 15:926–35
163. Nelson, J. I. 1974. Motion sensitivity in peripheral vision. *Perception* 3:151–52
164. Nelson, J. I. 1975. Globality and stereoscopic fusion in binocular vision. *J. Theor. Biol.* 49:1–88
165. Nelson, R., Kolb, H., Famiglietti, E. V., Gouras, P. 1976. Neural responses in the rod and cone systems of the cat retina: Intracellular records and Procion stains. *Invest. Ophthalmol.* 15: 946–53
166. Nelson, R., Lutzow, A. v., Kolb, H., Gouras, P. 1975. Horizontal cells in cat retina with independent dendritic systems. *Science* 189:137–39
167. Newsome, D. A. 1971. Afterimage and pupillary activity following strong light exposure. *Vision Res.* 11:275–88
168. Normann, R. A., Werblin, F. S. 1974. Control of retinal sensitivity I. Light and dark adaptation of vertebrate rods and cones. *J. Gen. Physiol.* 63:37–61
169. Ono, T., Noell, W. K. 1973. Characteristics of P- and I-cells of the cat's lateral geniculate body. *Vision Res.* 13:639–46
170. Penn, R., Hagins, W. A. 1972. Kinetics of the photocurrent of retinal rods. *Biophysics J.* 12:1073–94
171. Pepperberg, D. R., Lurie, M., Brown, P. K., Dowling, J. E. 1976. Visual adaptation: Effects of externally applied retinal on the light-adapted, isolated skate retina. *Science* 191:394–96
172. Poppel, E., Held, R., Dowling, J. E. 1977. The visual field: Psychophysics and neurophysiology. *Neurosci. Res. Program Bull.* 15(3):In press
173. Pribram, K. H. 1975. *Central Processing of Sensory Input.* Cambridge: MIT Press
174. Pugh, E. N. 1975. Rushton's paradox: Rod dark adaptation after flash photolysis. *J. Physiol.* 248:413–31

175. Pugh, E. N. 1976. The nature of the π_1 color mechanism of W. S. Stiles. *J. Physiol.* 257:713–47
176. Ratliff, F., ed. 1974. *Studies on Excitation and Inhibition in the Retina.* New York: Rockefeller Univ. Press
177. Raviola, E. 1976. Intercellular junctions in the outer plexiform layer of the retina. *Invest. Ophthalmol.* 15:881–95
178. Regan, D. M. 1972. *Evoked Potentials in Psychology, Sensory Physiology and Clinical Medicine.* London: Chapman & Hall
179. Regan, D. M. 1975. Recent advances in electrical recording from the human brain. *Nature* 253:401–7
180. Richter, A., Simon, E. J. 1975. Properties of center-hyperpolarizing, red-sensitive bipolar cells in the turtle retina. *J. Physiol.* 248:317–34
181. Ripps, H., Shakib, M., MacDonald, E. D. 1976. Peroxidase uptake by photoreceptor terminals of the skate retina. *J. Cell Biol.* 70:86–96
182. Robson, J. G. 1975. Receptive fields: Neural representation of the spatial and intensive attributes of the visual image. See Ref. 41, pp. 81–116
183. Rodieck, R. W. 1973. *The Vertebrate Retina.* San Francisco: Freeman
184. Ronchi, L. 1975. *150 Years of Rods and Cones. An Annotated Bibliography.* Giorgio Ronchi Found. Publ. No. 32. Firenze: Baccini & Chiappi
185. Ronchi, L., Pedata, F. 1974. *An Annotated Bibliography Concerning Human Cortical Visual Evoked Potential (VER) 1970–1973.* Giorgio Ronchi Found. Publ. No. 27. Firenze: Baccini & Chiappi
186. Roufs, J. A. J. 1972. Dynamic properties of vision-I. Experimental relationships between flicker and flash thresholds. *Vision Res.* 12:261–78
187. Roufs, J. A. J. 1974. Dynamic properties of vision-V. Perception lag and reaction time in relation to flicker and flash thresholds. *Vision Res.* 14:853–69
188. Sakitt, B. 1976. Psychophysical correlates of photoreceptor activity. *Vision Res.* 16:129–40
189. Schiller, P. H., Finlay, B. L., Volman, S. F. 1976. Quantitative studies of single-cell properties in monkey striate cortex. V. Multivariate statistical analyses and models. *J. Neurophysiol.* 39:1362–74
190. Schiller, P. H., Malpeli, J. G. 1977. Properties and tectal projections of monkey retinal ganglion cells. *J. Neurophysiol.* 40:428–45
191. Schwartz, E. A. 1975. Rod-rod interaction in the retina of the turtle. *J. Physiol.* 246:617–38
192. Schwartz, E. A. 1975. Cones excite rods in the retina of the turtle. *J. Physiol.* 246:639–51
193. Sekuler, R. W. 1974. Spatial vision. *Ann. Rev. Psychol.* 25:195–232
194. Sekuler, R. W. 1975. Visual motion perception. See Ref. 41, pp. 387–430
195. Shantz, M., Naka, K.-I. 1976. The bipolar cell. *Vision Res.* 16:1517–18
196. Sherman, S. M., Wilson, J. R., Kaas, J. H., Webb, S. V. 1976. X- and Y-cells in the dorsal lateral geniculate nucleus of the owl monkey (*Aotus trivirgatus*). *Science* 192:475–77
197. Shevell, S. K. 1977. Saturation in human cones. *Vision Res.* 17:427–34
198. Sillito, A. M. 1975. The contribution of inhibitory mechanisms to the receptive field properties of neurons in the striate cortex of the cat. *J. Physiol.* 250:305–29
199. Singer, W., Bedworth, N. 1973. Inhibitory interaction between X and Y units in the cat lateral geniculate nucleus. *Brain Res.* 49:291–307
200. Singer, W., Tretter, F., Cynader, M. 1975. Organization of cat striate cortex: A correlation of receptive-field properties with afferent and efferent connections. *J. Neurophysiol.* 38:1080–98
201. Snyder, A. W., Menzel, R. 1975. *Photoreceptor Optics.* Berlin: Springer-Verlag
202. Stevens, J. K., Gerstein, G. L. 1976. Interactions between cat lateral geniculate neurons. *J. Neurophysiol.* 39: 239–56
203. Stiles, W. S. 1977. *Mechanisms of Color Vision.* New York: Academic
204. Stone, J., Freeman, R. B. 1973. Neurophysiological mechanisms in the visual discrimination of form. In R. Jung (Ed.), *Handbook of Sensory Physiology,* Vol. VII(3)part A:153–207. Berlin: Springer-Verlag
205. Sturr, J. F., Teller, D. Y. 1973. Sensitization by annular surrounds: Dichoptic properties. *Vision Res.* 13:909–18
206. Tolhurst, D. J. 1973. Separate channels for the analysis of the shape and of the movement of a moving visual stimulus. *J. Physiol.* 231:385–402
207. Tolhurst, D. J. 1975. Reaction times in the detection of gratings by human observers: A probabilistic mechanism. *Vision Res.* 15:1143–49
208. Tolhurst, D. J. 1975. Sustained and transient channels in human vision. *Vision Res.* 15:1151–55

209. Toyama, K., Matsumani, K., Ohno, T., Tokashiki, S. 1974. An intracellular study of neuronal organization in the visual cortex. *Brain Res.* 21:45–66
210. Toyoda, J., Coles, J. A. 1975. Rod response to sinusoidally flickering light. *Vision Res.* 15:981–83
211. Tulunay-Keesey, U., Vassilev, A. 1974. Foveal spatial sensitization with stabilized vision. *Vision Res.* 14:101–5
212. Uttal, W. R. 1973. *The Psychobiology of Sensory Coding.* New York: Harper
213. van Norren, D., Padmos, P. 1977. Influence of anesthetics, ethyl alcohol, and freon on dark adaptation of monkey cone ERG. *Invest. Ophthalmol. Visual Sci.* 16:80–83
214. Vassilev, A. 1973. Contrast sensitivity near borders: Significance of test stimulus form, size and duration. *Vision Res.* 13:719–30
215. Vassilev, A., Mitov, D. 1976. Perception time and spatial frequency. *Vision Res.* 16:89–92
216. Virsu, V., Laurinen, P. 1977. Long-lasting afterimages caused by neural adaptation. *Vision Res.* 17:In press
217. Virsu, V., Taskinen, H. 1975. Central inhibitory interactions in human vision. *Exp. Brain Res.* 23:65–74
218. Walraven, P. L. 1972. Color vision. *Ann. Rev. Psychol.* 23:347–74
219. Watson, A. B., Nachmias, J. 1977. Patterns of temporal interaction in the detection of gratings. *Vision Res.* 17:893–902
220. Weisstein, N., Ozog, G., Szoc, R. 1975. A comparison and elaboration of two models of metacontrast. *Psychol. Rev.* 82:325–43
221. Werblin, F. S. 1974. Control of retinal sensitivity-II. Lateral interactions at the outer plexiform layer. *J. Gen. Physiol.* 63:62-87
222. Werblin, F. S., Copenhagen, D. R. 1974. Control of retinal sensitivity-III. Lateral interactions at the inner plexiform layer. *J. Gen. Physiol.* 63:88–110
223. Werner, G. 1975. *Feature Extraction by Neurons and Behavior.* Cambridge: MIT Press
224. Whitten, D. N., Brown, K. T. 1973. The time courses of late receptor potentials from monkey cones and rods. *Vision Res.* 13:107–35
225. Whitten, D. N., Brown, K. T. 1973. Photopic suppression on monkey's rod receptor potential, apparently by a cone-initiated lateral inhibition. *Vision Res.* 13:1629–58
226. Williamson, S. J., Kaufman, L., Brenner, D. 1977. Biomagnetism. In *Small Scale Superconducting Devices,* ed. S. Foner, B. Schwartz. New York: Plenum
227. Wilson, P. D., Rowe, M. H., Stone, J. 1976. Properties of relay cells in cat's lateral geniculate nucleus: A comparison of W-cells with X- and Y-cells. *J. Neurophysiol.* 39:1193–1209
228. Wooten, B. R., Butler, T. W. 1976. Possible rod-cone interaction in dark adaptation. *J. Opt. Soc. Am.* 66:1429–30
229. Yamane, S. 1976. A possible model for the electrical responses of frog rods during light and dark adaptation. *Biol. Cybern.* 23:229–39
230. Zettler, F., Weiler, R. 1976. *Neural Principles in Vision.* Berlin: Springer-Verlag