The high-frequency cutoff of the semicircular canals is unknown but probably also lies above 5 Hz. The high-frequency cutoff of the extraocular muscles is 1 Hz, so that central mechanisms (that introduce phase lead) must compensate from 1 to 5 Hz. The pursuit system begins to cut off between 0.5 to 1 Hz, so that it can compensate for slow head movements while the vestibular system compensates for rapid head movements.

Ratliff: In some cases your integrator seemed to be ideal and in other cases leaky.

Robinson: The integrator is not likely to be ideal. Its time constant (of the leak) need only be larger than a typical slow phase duration of nystagmus, say 2 to 5 sec. Its time constant may be revealed by the fact that, on eccentric fixation in the dark, the eyes tend to drift back toward the primary position at a velocity which suggests a leak time constant of 10 sec.

Roddick: Isn't it curious that in an animal with a fovea one finds less directional selectivity in colliculus and lower visual centers than one finds in afoveate animals?

Robinson: Yes. In lower animals it may be that directionally selective units form a special subset of ganglion cells that are used to stabilize the eye with respect to the visual world but do not participate in form discrimination. Lack of such cells in foveate animals would suggest that this system has been abandoned in favor of an equivalent system which utilizes the more complex processing of the visual cortex. This observation is a serious problem in trying to draw an analogy between Collewijn's model for the rabbit's ocular stabilizing system and the smooth pursuit system of man.

Levick: The output of these directional selective units is a signal which corresponds to the velocity of the retinal image. So that if you really want the system to stay put you can't use these units because they don't go down to dc. Couldn't you consider the concentric units which do give you absolute position?

Robinson: The directionally selective on-units in the rabbit detect velocities of 0.01 degrees per second and stabilize the eye drift to comparable values. If the drift disturbance were unidirectional (it usually isn't) the eye would travel six minutes of arc in ten seconds or about 0.5 degree in a minute. Since the drift disturbance is not unidirectional, the long-term drift of mean eye position is so slow that it would be difficult to distinguish it from a true dc mechanism. All one can say from Collewijn's experiments is that the directionally selective units appear to be capable of explaining the observed behavior. This, of course, does not exclude the possibility of an absolute position system based on concentric units.

Light and dark adaptation

W. A. H. Rushton

The nature of visual adaptation is an extremely complex question and our understanding of it received a mighty setback when Hecht and Wald used their outstanding force and eloquence to insist that everything depended simply upon the level of rhodopsin in the rods and the photopic pigments in the cones. Thus all desensitization by light was called 'bleaching' and all recovery 'regeneration.' I am sorry to note that American psychologists and others still use this misleading terminology and there has been widespread belief that it rested upon experimental proof, though in fact it was a lively speculation which not only was unsupported by any measurements of pigment kinetics, but which resolutely ignored the work of Dartnall, Goodeve, and Lythgoe (1938) who had made those kinetic measurements in the '30s and proved the photochemical theory of visual performance to be untenable.

In fact the vision of everyday life is
very little affected by the pigment level in the rods and cones; it depends in the main upon an adaptive nerve organization in the retina which we shall now consider.

Part I: Steady backgrounds

If the eye is adapted to a luminance $I$ and must discriminate a region of luminance $(I + \Delta I)$, the just noticeable difference $\Delta I$ has long been known to be given approximately by the relation $\Delta I/I = K$. This was studied by Bouguer (1760) in a bipartite field (Fig. 1), and by Fechner (1860) a century later using a superposed flash. These conditions are often treated as though essentially identical, but physiologically they are entirely distinct. The Fechner relation can be considered physiologically as detection of the increased output of the receptor or the receptor organization when the steady light $I$ is suddenly increased by $\Delta I$. In principle the discrimination could be made by a single receptor. In the Bouguer relation, it must be the separate outputs from receptors in different parts of the retina that are compared. To be sure, when we examine the relative brightness of the two halves of a bipartite field, we often scan across the boundary line and thus discriminate partly by Fechner’s successive contrast and partly by Bouguer’s simultaneous contrast. However, the Bouguer situation may be ob-
Fig. 2. (a) Geometry of contrast flash presentation. Test flash of intensity $\lambda$ falls upon a $2^\circ$ circle $6^\circ$ from fixation point (F.P.). The contrast flash of intensity $\phi$ falls on the $8^\circ$ surround annulus with $2^\circ$ black center. These flashes may fall upon steady backgrounds $\lambda$ on $\mu$ and $\phi$ on $\theta$. (b) Schema of neural interaction. Each region generates from the flash ($\lambda$ or $\phi$) a pulse which is attenuated by the $G_{-}$-box in proportion to the total steady background ($\mu + \phi$), ($\theta + \phi$) (where $\mu_{0}$, $\phi_{0}$ is Fechner's eigengrau). If $\lambda$ and $\phi$ are kept fixed, then the inhibitory signal $N$ must remain fixed when the $\lambda$ flash is reduced just to threshold. In this way we may vary $\phi$ and $\theta$ for a constant $N$ and establish the relation between them. This interaction presumably all takes place in the amplitude-coded range of signals before the difficult transformation into spike frequency in ganglion cells.

mission line to anticipate overloading. For eight years I have hoped that the horizontal cells are Fechner scalors, and I still hope so.

**Retinal nerve signals.** The astonishing range and accuracy of Fechner scaling can be shown by the technique of contrast-flash analysis (Alpern, Rushton, and Torii 1970a,b,c,d). A surround flash $\phi$ falling upon an annulus, Fig. 2(a), inhibits the signal from a flash $\lambda$ falling upon the dark center so that its intensity must be increased if it is still to be visible. Whatever the class of receptor excited by $\lambda$ at threshold (e.g., rod, red cones, etc.), it is only the quantum catch from $\phi$ by that receptor which raises the threshold (Alpern and Rushton 1965). Thus if $\lambda$ lies below cone threshold, the $\lambda$ threshold will be raised only by the excitation of rods in the surround no matter how strong $\phi$ may be. This allows us to measure rod excitability far above cone threshold and to determine the course of rod dark adaptation in a normal subject over 6 log units range of sensitivity change, as will be mentioned later (Fig. 5). If the surround flash does not fall upon the entire annulus but upon four sectors of $111/2^\circ$ (like windmill sails) so that only $1/4$ of the area of the annulus is illuminated, then the inhibition is reduced to $1/4$. That allows us to measure $N$, the size of the nerve signal as a function of the intensity of flash. The argument given in Alpern, Rushton, and Torii (1970a) is too long to be repeated here and we simply state the relation found.

When in 1968 we first determined this relation the result surprised us, but it fits well with recent electrophysiology. If $N$ is the size of nerve signal and $I$ the intensity of flash in the absence of steady background we found experimentally that

$$N = \frac{I}{I + \sigma}$$

From this formula it is clear that when $I = \infty$, $N = 1$ hence $N$ is expressed as a fraction of its maximum value. When $I = \sigma$, $N = \frac{1}{2}$ and thus $\sigma$ is called the "semi-
Fig. 3. Amplitude of turtle cone potentials intracellularly recorded as a function of log light intensity. Dotted curve plots the relation \( V/V_{\text{max}} = 1/(1 + \sigma) \), where \( \log \sigma = -3.8 \). (From Baylor and Fuortes 1970.)

saturating constant,” i.e., the light flash that raises \( N \) to half its maximum. For human rods \( \sigma \) is about 1000 quanta absorbed per rod per flash. This flash bleaches about 1/10,000 of the rhodopsin.

The absolute threshold for human rods is about \( 10^4 \) of the \( \sigma \) value (i.e., 1 quantum caught per 100 rods) and thus the rod nerve signal, \( N \), increases in direct proportion to the flash from threshold to 4 log units above it: at higher levels it begins to saturate. Cones behave like rods, but thresholds and semisaturation are both about 2 log units higher in light level.

The hyperbolic formula (eqn 2) which may be called an H-function, was put forward by Naka and Rushton (1966a, b) to describe the relation between light intensity and S-potentials. It has since been shown to underlie many of the “amplitude-coded” nerve signals of the retina (e.g., Baylor and Fuortes 1970) for turtle cones intracellularly recorded, Fig. 3). If we regard eqn (2) as the synaptic transform function for these amplitude-coded signals, a simple expectation follows from the mathematics.

If the “H curve” output of one synapse (with some fixed attenuation) is the input to the next synapse, then its output is easily computed and found also to be an H curve. Consequently with a chain of such synapses there will be an H relation between the input at one end and the output at the other.

Effect of background on \( N \). This was investigated over a very great range by allowing the inhibitory flash \( \phi \) to fall upon a steady background \( \theta \). To inhibit a test flash \( \lambda \) of some fixed size, the relation found was

\[
N = \frac{\phi}{\phi + \sigma} \cdot \frac{\theta_0}{\theta_0 + \theta}
\]

where \( \theta_0 \) is the same as \( I_0 \) eqn (1) and is Fechner’s eigengrau. The formula held good over the whole available useful range of \( \theta \) intensities (about 5 log units) and for a large range of N-values obtained by using different intensities of \( \lambda \), the test flash to be inhibited.

Fig. 4 shows the size of \( N \) plotted against log \( \phi \) the inhibiting flash, which in the upper curve fell on a dark background (log \( \theta = -\infty \)) and in the others the log
light and dark adaptation

Fig. 4. Nerve signal \( N \) plotted experimentally as function of inhibitory flash intensity \( \phi \), and steady background intensity \( \theta \) upon which it falls. \( H_2 \) curves on left plot \( \log N \) (scale on left); \( H_1 \) are the same curves plotted simply as \( N \) (scale on right). All curves are the theoretical expectations of eqn. (3). (From Alpern, Rushton, and Torii 1970b.)

...background was as marked. In the upper left (\( H_2 \)) curves \( \log N \) is plotted, in the lower right (\( H_1 \)), \( N \) itself. All the curves are \( H \) curves with \( \sigma \) unaltered by background adaptation. In the \( H_2 \) plot, the curve is displaced vertically downward by backgrounds. In other experiments pure bleaching was found to displace the curve not downward but horizontally to the right.

In many cases it is not easy to tell whether adaptation is in effect by background or by bleaching (possibly what is called 'nervous' or 'chemical'). It looks as though the direction of shift of the \( H_2 \) curve of the nerve signal resulting should tell us; the vertical shift is the 'nervous' part, the lateral shift is the chemical.

A pleasing result from eqn (3) is that it provides a quantitative explanation of the famous 'rod saturation' of Aguilar and Stiles (1954). For any fixed small value of \( N \) say \( 10^{-3} \) (= 100 times the dark threshold) \( \phi \) will be negligible compared with \( \sigma \) unless \( \theta \) is very large, and eqn (3) will become

\[
\phi = \left( \frac{\theta_D}{\theta} \right) \cdot \frac{N\sigma}{\theta_D}
\]

which is identically Fechner's formula (1) with \( K \) evaluated as \( N\sigma/\theta_D \). But as \( \theta \) continues to increase \( \phi \) must also increase until it no longer is negligible compared with \( \sigma \).

From our fundamental equation

\[
N = \frac{\phi}{\phi + \sigma} \cdot \frac{\theta_D}{\theta_D + \theta}
\]

it is clear that however great \( \phi \) becomes, \( N \) can never exceed \( \theta_D/(\theta_D + \theta) \) and when, with increasing \( \theta \), this falls below the \( N \) criterion (10^{-5} in the present case), obviously no increase in \( \phi \) can restore it. On the right of Fig. 5 are displayed log incre-
Fig. 5. Left, 3 dark adaptation curves, right, 3 increment threshold curves for normal human rods determined by contrast flash technique. For each curve some fixed supra-threshold flash $\lambda$, applied to the center (see Fig. 2) was inhibited by the surround flash $\phi$. Ordinates plot the least value of $\log \phi$ which just inhibits $\lambda$, when $\phi$ falls on various steady backgrounds $\theta$ (curves on the right). The relation between $\log \phi$ and $\log \theta$ corresponds to Aguilar and Stiles' saturation, and the curves plot exactly the relation of eqn (3) with $\log \theta_0 = -1.9$ (td) and $\log \sigma = 3.3$ (td. sec). Curves on the left plot similar results where there is no steady $\theta$ background, but the region had been 90% bleached at time zero. Contrast flash technique when $\lambda$ lies below cone threshold measures only rod excitation by $\phi$, thus we obtain a pure rod dark adaptation curve over a range of about 6 log units without cone interference (From Alpern, Rushton, and Torii, 1970c.)

Theoretical curves where $\phi$ falls on various backgrounds $\theta$ and the criterion for $\phi$ 'threshold' is that the $\phi$ signal after attenuation by the $\theta$ background should just extinguish the perception of the fixed $\lambda$ flash (at the center of the annulus). The three curves were obtained with 3 different levels of $\lambda$. What the experimental points show (right half of the figure) is first the familiar Fechner log increment threshold curve, but at high values the Aguilar and Stiles saturation develops. The theoretical curves of the figure are simply the plotted expectation of the fundamental formula (3). All the curves have the same eigengrau $-1.9 \log$ td.) and the same semisaturation $\sigma$ (3.3 log td. sec), and indeed the whole family is simply a replot of the results of Fig. 4, which also displays the fundamental relation of eqn. (3).

Now equation (3) which implies for rods the saturation which Aguilar and Stiles saw was there, equally implies it for cones where they saw that it was not there. Fortunately the explanation is simple. With cones the $\sigma$ level is some 2 log units above the $\sigma$ level for rods, moreover with the same illumination cones bleach much faster than rods do. Consequently, in attempting to reach the cone $\sigma$ level of quantum catching from a steady background, so much pigment is bleached away that insufficient remains to achieve the required quantum catch. Alpern, Rushton, and Torii (1970d)
not only examined this quantitatively and explained how in these conditions no steady background could saturate the cones, they also showed how cone saturation could nevertheless be displayed. For, if the background I instead of being continuous, shines only for a second or so at a time, just while the increment ΔI is flashed upon it, the bleaching produced will be slight and the Fechner threshold rise can be observed nearly unimpoverished by any impoverishment of quantum catching through bleaching. When this experiment was done the cone saturation was seen to occur as expected and eqn (3) was followed for cones, as in Fig. 5 it is for rods, but naturally the constants θ₀ and σ now had values appropriate to cones not rods (Alpern, Rushton, and Torii, 1970d).

**Brightness adaptation.** The N-signal which we have been considering is an inhibitory signal—one that is just insufficient to reduce to invisibility a fixed strong test flash λ. Now Alpern and Rushton (1967) showed that nerve signals were adapted by steady backgrounds in the same way whether their destination was to inhibit or to excite. This suggests the question, “Do equal N signals look equally bright?” Suppose upon the retina there are two steady backgrounds of luminances θ₁, θ₂, what will be the energies φ₁, φ₂ flashed upon them when those flashes appear equally bright? Will they accord with eqn (3) so that brightness is equal when N is equal?

There is a difficulty in making this kind of observation because it is hard to judge the equality of flashes when seen in contrast to their unequal backgrounds. But this difficulty can be overcome if the background is stabilized on the retina. As has been recognized since the pioneer experiments of Ditchburn and Ginsborg (1952) and Biggs, Ratliffe, Cornsweet, and Cornsweet (1953), images that are stabilized and thus remain fixed on the retina despite eye movements, quickly fade away and are not seen at all. Flashes applied to the two regions in this condition are therefore projected upon an empty field and thus may be directly compared in brightness without contamination from background contrast. This experiment has recently been performed by Mrs. Cynthia Gosline and me (unpublished) in the University of Cambridge. The subject (C. G.) wore a specially worked tight contact lens that carried on a stalk the target and a lens to focus it on the retina.

The principle of the experiment is indicated in Fig. 6. The 4° stabilized field consists of an upper half of luminance I₁ and a lower half J₁. After stabilized fading has occurred and the field is empty, the lever is pressed and suddenly I₁ is replaced by the brighter I₂ and J₁ by the brighter J₂. The aim of the experiment is to find what is the luminance ratio I₂/J₂ that makes I₂ and J₂ look equally bright when they suddenly appear in the empty field.

We shall not digress here to describe the optics in detail, how the change is made from I₁, J₁ to I₂, J₂ and how the ratio I₂/J₂ is adjusted. In principle, light falls on the glass plate at the polarizing angle thus pressing the level and rotating through 90° the attached sheet polarizer switches the light from beam 1 to beam 2. The lower aspect of Fig. 6 shows the eye, with tight contact lens holding on the stalk a target θ with the lens L₁ to focus it onto the retina at U₁. The target consists of λ/2, a strip of half-wave cellophane that covers half of the target circle with horizontal edge sharply in focus. In conjunction with P, the sheet polarizer with axis at 45° to that of the cellophane we have in effect two polarizers with axes at right angles. Horizontally polarized light falling on the target illuminates only the upper half I₁, vertically polarized light illuminates only the lower half J₁. Light polarized at an angle α to the horizontal illuminates I in proportion to cos² α, J in proportion to sin² α. Thus by adjusting α, the plane of polarization of beams 1 and 2 we may set the ratio I/J as required.

Fig. 7 displays the results of one experiment out of many performed by Mrs.
Fig. 6. Optical equipment to replace the 4° bipartite field I₁/J₁ by I₂/J₂. When the lever is depressed it rotates the sheet polarizer P through 90° so that light falling on the glass plate at the polarizing angle, instead of being reflected as beam 1 is transmitted as beam 2. The lower part of the figure shows the observer’s eye wearing a tight contact lens with a stalk that holds the target λ/2, a half-wave cellophane strip whose horizontal edge is focussed sharply by L₁ onto the retina at U₂. In conjunction with the sheet polarizer P, λ/2 accepts only horizontally polarized light in the upper half-field I and only vertically polarized light in the lower half-field J. Thus in changing from beam 1 to 2 we can alter the total intensity (I + J) by adjusting the neutral filters F₁, F₂, and the ratio I/J can be altered by adjusting the plane in which the beams are polarized, easily set by rotating the half-wave plates \( \lambda \)₁ and \( \lambda \)₂, which causes the polarization plane falling upon the stabilized target to rotate through twice that angle.

Cynthia Gosline (subject) and me in the Zoological Laboratory of the University of Cambridge. After proper alignment of the target on the eye’s optic axis and of the eye in the equipment, the subject relaxed and the two fields I₁, J₁ in Fig. 6 faded away and left emptiness. Then the subject pressed the lever (which replaced I₁ by I₂ and J₁ by J₂) and quickly released it again. This caused the empty field to be replaced by two contiguous fields, and the subject said which was brighter. The operator then altered the polarization plane in beam 2 (by rotating the half-wave plate \( \lambda \)₂ to give a new I₂/J₂ ratio and the procedure was repeated. The subject never knew what changes were being made and generally was consistent in log \( (I₂/J₂) \) judgments correct to ± 0.1 in a total log luminance change of 24 or more. The judgments were always made for an increase in brightness of the fields. With a decrease, the new fields both looked black and their equality in blackness was hard to judge.

Fig. 7 shows one critical experiment repeated on 4 days. A 0.8 neutral density covered the J half of the target and in conjunction with suitable polarization of beam 1, the adaptation ratio I₁/J₁ was always maintained so that log \( (I₁/J₁) = \)
1.4. Though the ratio \( I_1/J_1 \) was always the same (about 24) the actual luminance for adaptation varied between experiments as shown by abscissae in Fig. 7 from complete darkness (\(-\infty\)) to about 1000 td. for \( I_1 \). The changeover was always made to full brightness where \( I_2 \) was about 10,000 td. and \( J_2 \) was adjusted to look equally bright.

The results of the repeated experiments of Fig. 7 (four different symbols) are reasonably consistent and run an expected course. When the adapting light is blackness (\(-\infty\)) no differential adaptation to \( I_1/J_1 \) could be expected and \( I_2 \) will appear as bright as \( J_2 \) when it has the same luminance. Consequently when \( \log I_1 = -\infty \) the ordinate \( \log (I_2/J_2) \) should be zero, which it is. On the other hand, if the adapting brightness is so high as to be well above the eigengrau, we should expect \( I_2 \) to be scaled down in proportion to \( I_1 \) in proportion to \( J_1 \) so that at top right of the curve the ordinate, \( \log (I_2/J_2) \) should approach \( \log (I_1/J_1) = 1.4 \). This also is fulfilled.

Both these results are consistent with the statement “equal N values are seen as equally bright.” In fact the theoretical curve (no arbitrary constants) that is drawn through the points of Fig. 7 is the mathematic expression of that statement.

In the application of the fundamental formula (3)

\[
N = \frac{\phi}{\theta + \sigma} \cdot \frac{\theta_0}{\theta_0 + \sigma}
\]

to the half-field \( I_1 \) (or the half-field \( J_1 \)) we put \( \theta = I_1 \), \( \theta_D = I_D \), \( \phi = I_1 - I_1 \ll \sigma \). This gives

\[
N_1 = \frac{I_1 - I_1}{\sigma} \cdot \frac{I_0}{I_0 + I_1}
\]

with a similar expression for \( N_J \) in terms of \( J \). If the condition that flashes appear equally bright is that \( N_1 = N_J \), we may equate the expressions (4) and since \( I_D = J_0 \)

\[
\frac{I_0 + J_0}{I_1 + J_0} = \frac{J_1 + J_0}{I_1 + J_0}
\]

This gives

\[
\log (I_1/J_1) = \log (I_1 + I_0) - \log (J_1 + J_0)
\]

This is a very simple extension of Fechner’s concept. The eigengrau \( I_D \) must always be added to the real light \( I \) or \( J \) to which the eye is adapting, and with this concept of total light, the \( N \) signal is proportional to the change in total divided by the old total. Equal \( N \) signals look equally bright.

In Fig. 7 the theoretical curve was derived from eqn. 5, the intensities of \( I_2 \) and \( J_2 \) being well above eigengrau level so that we may write

\[
\log (I_2/J_2) = \log (I_2 + I_0) - \log (J_1 + I_0)
\]

In the experiment of Fig. 7 the value of \( I_0 \) was obtained by ordinary increment thresh-
old measurements where an auxiliary test flash was presented superimposed upon either field $I_1$ or $J_1$ at the various levels indicated by abscissae. These increment threshold results for backgrounds $I_1$ are shown by black circles at the bottom right aspect of Fig. 7. The curve shifted 1.4 log units to the right represents results for backgrounds $J_1$. According to Fechner's formula

$$\log \Delta I = \log (I_1 + I_0) + K \quad \text{(upon } I_1)$$

and

$$\log \Delta I = \log (J_1 + I_0) + K \quad \text{(upon } J_1)$$

for the two backgrounds. The ordinate difference between the two curves consequently is

$$\Delta y = \log (I_1 + I_0) - \log (J_1 + I_0)$$

But this from eqn. (6) is precisely $\log (I_1/J_1)$, and the curve through the 4 sets of points in Fig. 7 is in fact simply the ordinate difference $\Delta y$ of the lower curves replotted. The scale of $\Delta y$ is shown on the right. It is enlarged when replotted to match the scale on the left, and with this alteration, the ordinate of the upper scale measured from its zero is equal to the ordinate difference of the two lower curves.

**The size of amplitude-coded signals.** Our fundamental equation (3) that relates $N$, the size of signal with $\phi$ the flash energy, and $\theta$ the steady background upon which it falls appears to be of great importance for vision.

First it is an experimental relation that can be quantitatively checked and has been found to hold over the entire useful range —about 5 log units. It may be proved that at visual threshold $N$ has a fixed size, and this allows various psycho-physical relations to be predicted from the formula, including the Aguillar and Stiles (1954) saturation, with satisfactory agreement. Two $N$ signals calculated to be equal from the formula were found to have these properties. If inhibitory, they inhibited equally so that when impinging upon the empty field of a stabilized image the two flashes appeared equally bright.

The formula of eqn (3) implies a remarkable organization of signals with good accuracy over a great range. It is likely that much of this occurs in the intimate connections between receptor terminals, bipolars, and the processes of the horizontal cells. Expert analysis of this important and complex region is proceeding in many laboratories at present, and we shall soon know how far the transformation of information accords with the expectation of eqn. (3), and what remains to be explained by other mechanisms.

**Part 2: Bleaching**

The slow recovery of visual sensitivity on going from bright sunlight into the darkness of the tomb was no doubt familiar to the builders of the Pyramids. Its explanation began when Kühne showed in the frog's retina that rhodopsin was bleached by light and *in situ* regenerated slowly in the dark. Hecht was correct in his conjecture that the dark adaptation curve was correlated with the regeneration of rhodopsin, but he was never able to decide what the relation was. His insistence upon the hopeless attempt to make a single kinetic system explain two such utterly divergent adaptations as that from bleachings and that from backgrounds has thrown the whole subject into a lasting confusion.

**Bleaching and threshold rise.** When the pigments were objectively measured by retinal densitometry in the eyes of living men, it was found that rhodopsin regenerated along an exponential curve with the same time constant as the rod branch of the dark adaptation curve (Campbell and Rushton 1955), and that cone pigments regenerated similarly along the cone branch (Rushton 1957). Dowling (1960) confirmed this relation more accurately by measuring in the rat the log ERG threshold and correlating it with the extracted rhodopsin content of the rods, reduced either by
Fig. 8. Rod dark adaptation curve (circles) and the regeneration of rhodopsin in the normal human eye (triangles). The pigment was measured by our Florida densitometer (Hood and Rushton 1971) from the 90% bleaching level at zero time, till complete recovery. The dark adaptation log thresholds are a replot from Fig. 5 (left) where the contrast flash technique excludes cones from contaminating the rod threshold curves. Since with suitable scaling both results fall on the same curve the log threshold rise is proportional to the fraction of rhodopsin in the bleached state. (From Alpern, Rushton, and Torii 1970c.)

Fig. 8. Rod dark adaptation curve (circles) and the regeneration of rhodopsin in the normal human eye (triangles). The pigment was measured by our Florida densitometer (Hood and Rushton 1971) from the 90% bleaching level at zero time, till complete recovery. The dark adaptation log thresholds are a replot from Fig. 5 (left) where the contrast flash technique excludes cones from contaminating the rod threshold curves. Since with suitable scaling both results fall on the same curve the log threshold rise is proportional to the fraction of rhodopsin in the bleached state. (From Alpern, Rushton, and Torii 1970c.)

There is little doubt that this holds for human rods for all times longer than 5 minutes in the dark. Fig. 5 shows an unusual example, for it records the dark adaptation following a full rhodopsin bleach in a normal subject with no cone branch to the curve. The dark adaptation curve is displayed on the left of Fig. 5 where the contrast flash technique eliminates the intrusion of cones. Fig. 8 shows the coincidence of these log rod thresholds replotted from Fig. 5 with the rhodopsin level in the rods measured on the same subject by densitometry in a comparable run. Triangles show two runs of pigment regeneration over the whole range; circles show the log threshold over the later half of the range. I do not think it possible to elicit a human rod response with more than 50% of the pigment bleached. If this is due to 'saturation' it is not at all the Aguilar and Stiles saturation as may be seen from comparing the left and right halves of

\[ \log \left( \frac{A_I}{A_{I_0}} \right) = 10 \, H_B \]  

where \( A_I \) is the threshold during dark adaptation when a fraction \( B \) of pigment is in the bleached state, \( A_{I_0} \) is the fully dark value, and \( H \) is a constant being the rise in log threshold when 10% of the pigment is bleached.

There is little doubt that this holds for
Fig. 9. Human rod dark adaptation curves following a 40 sec. exposure that ended at zero time on the figure and left 7%, 2%, 0.5%, and 0.1% of rhodopsin then bleached. The continuous curves show the log threshold rise expected of that amount of bleaching on the basis of Fig. 8. But early after weak bleaches the threshold is always raised much more than this expectation. (From Rushton and Spitzer-Powell 1972b.)

Fig. 5. More likely the sharp threshold rise within the first 5 minutes is related to the rapid adaptation found already by Hecht following weak bleaches. He and especially Wald supposed that this represented a rapid regeneration of bleached rhodopsin. But that misapprehended the problem. The proper question was not why after this weak bleach does the threshold return so quickly toward the base line, but why has it ever left the base line so far?

Early threshold rise. Fig. 9 shows a set of 4 human dark adaptation curves following 40 second exposures to lights that were found to bleach fractions of rhodopsin indicated. The results in this figure show some of a set (Rushton and Spitzer-Powell 1972a, b) where the pigment regeneration and the dark adaptation curves were measured following the whole range of bleaching.

The continuous curves of Fig. 9 show the course of dark adaptation to be expected from the regeneration of rhodopsin following the weak bleaches indicated. With stronger bleaches than these, the thresholds were found to follow these continuous curves exactly, but in Fig. 9 the experimental points start above the curves and only reach them after five minutes. The early rise becomes greater the greater the bleaching light and perhaps drives the early threshold of Fig. 5 above the picture.

This early threshold rise might be background adaptation from the strong background level of the bleaching light, a change in nerve organization so great that it takes a few minutes for complete restoration. Or it might be the result of photoproducts not yet degraded to leave free opsin (which is what the “rhodopsin bleaching” lines of Fig. 9 represent).

Three lines of evidence support the photoproduct explanation. (i) Alpern and Torii in preliminary experiments (unpublished) found with contrast flash technique that the H2 curve (Fig. 4) relating N and
φ was displaced not downward as expected of background adaptation, but laterally as expected of bleaching adaptation. (ii) Rushton and Spitzer-Powell (1972b) found that when the bleaching intensity I and the exposure duration t were varied so that It the total energy was constant the adaptation was the same from t = 1/2 second to t = 1/2 minute. This is what would be expected of a photoproduct, but not of an independent neural reorganization. (iii) The It = k relation held even when I lay far above N saturation. It is impossible that increases in I can produce proportional increases in N many times above the N saturation level. But It will be proportional to the amount of photoproduct produced far above this level. Thus we conclude that the early threshold rise is the result of the presence of a transitory photoproduct, perhaps metarhodopsin II as suggested by the evidence of Donner and Reuter (1968).

According to this idea it is the production of metarhodopsin II which always causes visual excitation. When this is produced in such quantity that it remains undecomposed for some time, a strong afterimage persists and is the cause of the rise of threshold. Certainly the early threshold rise is associated with enhancement in the brightness of the after image. We may unify the concept of threshold rise due to metarhodopsin II with that due to "bleaching" (i.e., unregenerated rhodopsin) if we suppose something like the following kinetics

Meta II ⇌ opsin + all-trans retinal

This reaches equilibrium in about 5 minutes and thereafter the amount of meta II will be proportional to the amount of free opsin, and as that is removed from the arena by a stable combination with 11-cis retinal (regeneration) so will the meta II be removed in proportion, and hence the rise in log threshold that is proportional to meta II will also be proportional to the unregenerated rhodopsin (after 5 minutes), as observed.

The site of bleaching adaptation. An important unification in the effects of bleachings and backgrounds was put forward by Stiles and Crawford (1932) and supported by good evidence. It was that adaptation was only of one kind in its operation despite the many ways in which it could be induced. However brought about, a given state of adaptation would operate in the same way for all input—output transformations. "The equivalent background" of bleaching is an example: if at some stage of bleaching the threshold is raised as much as it is by some particular background, then those two states of adaptation will be found equal as judged by every other test. An example is seen in Fig. 5 for various N-values, where it is seen to break down near saturation.

The universal truth of the equivalent background concept would be very strong support for the view that bleaching and backgrounds raised thresholds by the same mechanism. However, it now appears that the truth is not so universal, and though part of the adaptation mechanism is probably thrown into play by both backgrounds and bleachings, parts of the mechanisms are certainly distinct. Fig. 4 shows that backgrounds scale down the N signal without affecting σ, the input sensitivity to light. Bleachings on the other hand may act by increasing σ (the light requirement) without affecting the size of saturated signal.

This difference in the mechanism of adaptation by backgrounds and by bleachings was found also by electrophysiology. Naka and Rushton (1968) showed that backgrounds produced a steady hyperpolarization in the S-potential whereas bleachings raised by several log units the threshold for S-potentials without producing any steady change at all. This desensitization therefore appears to occur distal to the horizontal cells, confirmed also by Dowling and Ripps (1970, 1971).

One fact above all we need to know. How far is adaptation found in the rods and cones themselves, intracellularly recorded? Since the pioneer work of Tomita, Kaneko, Murakami, and Pautler (1967) many workers have suddenly developed the skill to do what no one could do before,
Fig. 10. Potential from axolotl rods intracellularly recorded, in response to a 200 msec flash of log I value shown. White circles before bleaching; results fit an H curve with log \( \alpha \) of -4.5. The retina was then 45% bleached and did not regenerate any rhodopsin subsequently. At first the response was abolished but it gradually recovered, the increase in log sensitivity going hand in hand with decay of a photo product absorbing at 400 nm. During this recovery the log \( \alpha \) remains fixed at about -3.5 (where it shifted on bleaching). (From Grabowski, Pinto, and Pak.)

and we shall soon have clear answers to adaptation in the receptors. At present my opinion is that most of background adaptation is signal scaling organized proximal to the receptors, probably by the horizontal cells and their connections. It does not seem as though 1 quantum caught by 1% of the rods will reduce the response of all the other rods by 70% as it does the N-signals in man. On the other hand, much of bleaching adaptation is a change of sensitivity in the receptors themselves. For Fig. 10 I am indebted to Drs. Grabowski, Pinto, and Pak for allowing me to use some of their yet unpublished work. The points plot the height of intracellular potentials from axolotl rods as a function of log intensity of a 200 msec flash. Before bleaching, the expected H curve was obtained (white circles). After bleaching the receptors at first were unresponsive and the black circles show the improvement with time in the dark. The retina was excised and densitometry measurements showed that the exposure bleached 45% and that no rhodopsin regenerated thereafter. The recovery of threshold however was rather well correlated with the decay of metarhodopsin II (or whatever was measured at 400 nm.) over a range of 3 log units. Fig. 10 shows that the bleaching of rhodopsin shifts \( \alpha \) to the right by 1 log unit, and that the decay of meta II at 37 min. half restores the initial Vmax without change of \( \alpha \). Thus within the rod itself there seem to be two mechanisms in action.

(a) Removal of half the rhodopsin from the rods will naturally require twice as much incident light to give the same quantum catch. This would displace log \( \alpha \) 0.3 to the right. In fact it is displaced 1.0, thus the rods are not twice but 10 times reduced in sensitivity by removal of rhodopsin, and signals in addition are attenuated to 60% (perhaps by residual meta II). (b) The main adaptive effect is due not to the absence of rhodopsin but to the presence of meta II. The log threshold falls over 3 log units in proportion to meta II concentration and this is entirely due to attenuation of signals like the "background adaptation" of Fig. 4.

Conclusion

The magnificent achievements of microelectrophysiology and microspectrophotometry are rapidly and expertly taking over the whole experimental field in conjunction with the findings of microanatomy. The psycho-physicist must now gracefully retire from his attempts to explain how the retina works. But perhaps he may still mention what it is that has to be explained, for he alone is professionally concerned with the possibility that the nerves studied by the others may have something to do with visual perception! So let me propose for your solution, O, colleagues of incomparable expertise, the two key problems in
visual adaptation: (i) What organization underlies Fechner’s Law (Fig. 1) and what Bouguer’s? (ii) And when you can explain why a real image raises the threshold in proportion to the light that falls, tell me why an after-image raises it in proportion to the antilogarithm of the light that fell, for the explanation in my Ferrier Lecture (Rushton 1965b) is now rather outdated.

REFERENCES