CHAPTER 9

Visual Adaptation and Retinal Gain Controls

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1. INTRODUCTION

Vision is our primary sensory channel for interaction with the outside world. It allows us to recognize familiar faces and creatures, and objects; it allows us to orient ourselves in space and to navigate from place to place. It is a pathway for esthetic enjoyment and for information transmission. The visual system is one of the many miracles of nature.

Intensive study of the visual process has revealed that the retina must perform several operations on the image delivered by the eye's optics in order to make manageable the difficult jobs of the brain : pattern recognition and spatial localization. One of the basic operations the retina performs is the subject of this chapter: retinal adaptation. The retinal neurons adapt to variations in illumination by changing their gain and response time course. The purpose of adaptation is to keep the retinal response to visual objects approximately the same when the level of illumination changes. Thus, central visual processing may proceed without the brain having to attend to changes in the average light level caused by the daily solar cycle, by shading, by artificial illumination, or by other, perhaps unpredictable, events.

We will demonstrate the visual significance of the retinal regulation of contrast sensitivity at different levels of illumination. This basic function of retinal adaptation is so important for vision that there is a hierarchy of retinal adaptation mechanisms at several different sites within the retinal network. There is clear evidence for adaptation in individual photoreceptors, in some species. There is evidence for adaptation at the level of the outer plexiform layer of the retina, in bipolar cells. There is also evidence for another stage of adaptation at the inner plexiform layer, in amacrine cells. Adaptation performed by the retinal network thus appears to involve at least three mechanisms in most retinas. Such evidence leads to the concept of a hierarchy of mechanisms which may be engaged at different background levels and with different time courses. In individual cells or in the retinal network, the neural signals sent on to the next stage in neural processing usually are fed back to regulate the response to new or persistent inputs. The theories for adaptation which we shall discuss require feedback in order to explain the phenomena associated with visual adaptation. One can therefore state as an overview that visual adaptation is achieved by a hierarchy of feedbacks designed to regulate contrast sensitivity.

It is impossible within the space of a review chapter to deal with visual adaptation to changes in illumination and also to do justice to the subject of recovery of sensitivity in the dark after all light has been turned off. The latter phenomenon usually goes by the name *dark adaptation*. Dark adaptation is in some ways similar to light adaptation but is different in such significant other ways that it deserves a chapter all its own. It is not covered in the following pages.

1.1. Terminology

Because the facts and theories of visual adaptation are complicated enough, one ought to be clear about the meanings of words which are used to describe the facts, and so we will define several words which are critical for the ensuing discussion. It is most important to define what *adaptation* means, but some preliminary terms require definition first.

1.1.1. SENSITIVITY AND GAIN

Unfortunately, "sensitivity" has different meanings in different fields. In psychophysics it means 1/threshold or, in other words, the reciprocal of the stimulus strength required for the stimulus to be perceived reliably. According to this meaning, "sensitivity" is related to the signal/noise ratio inside the psychophysical observer (cf. for example, Barlow and Levick, 1969; or Rose, 1948, 1973). The "noise" in this case is caused by all the physiological fluctuations in the retina and brain, fluctuations which make it difficult for an observer to be certain that a stimulus has been presented. This "noise" is caused, in the dark, by thermal breakdown of photopigment in photoreceptors, spontaneous random release of neurotransmitters, and fluctuations in the physiological state of the retina and brain. When the retina is illuminated, additional noise is caused by the retinal response to the randomly arriving stream of light quanta. Psychophysical "sensitivity" can be influenced by

processes which change the magnitude of the noise from which the signal must be picked out as well as by those which affect the size of the signal.

In physiological experiments, the word "sensitivity" is usually used to mean the reciprocal of the stimulus required to produce a neural response of a criterion size. In this meaning of the word, noise is ignored because neural responses are usually averaged over a number of identical stimulus cycles to eliminate noise as much as possible. There has been confusion in the literature when a result on psychophysical sensitivity has been taken to imply something definite about physiological sensitivity or vice versa, because in one case noise has an effect and in the other it does not.

We will try to avoid confusion by calling psychophysical sensitivity, *"sensitivity"*, and physiological sensitivity, *"gain"*. Gain we define as the ratio of the magnitude of the physiological response to the stimulus magnitude, in the small-signal range in which response is proportional to the stimulus. Gain thus has units like mV/quantum in photoreceptors, or impulse/quantum in retinal ganglion cells. Although we distinguish physiological gain from psychophysical sensitivity, the two are related. If the internal noise were more or less unaffected by background light, a reduction in retinal gain as a consequence of an increase in background would produce a corresponding reduction in visual sensitivity.

1.1.2. ADAPTATION

Considered in terms of sensitivity and gain, the one unambiguous traditional term which describes the adaptation state is *total dark adaptation,* the state of highest sensitivity reached by an observer and highest retinal gain reached by a living retina that has been left in total darkness for a few hours. We define *light adaptation* as those variations in the properties of the visual system from the totally dark adapted state which are produced by variation in the level of light. For instance, light may change the gain of the retina, or its time course of response, or its spectral sensitivity, or its spatial summation properties. However, an increase of retinal noise due to the random times of arrivals of light quanta is not strictly speaking a property of the retina which is changed by light. But it is a factor which could lead to an increase of the psychophysical threshold. Therefore, in order to understand how light adaptation contributes to vision, specifically to the variation of visual sensitivity with mean level of illumination, one must distinguish between adaptation and the effects of increased noise caused by light. Similarly, as we will show, gain can be reduced by saturation, e.g. the limitation on the amplitude of response imposed by a response ceiling. We wish to distinguish this kind of gain reduction due to saturation, or as it has been called "response compression", and the gain control of adaptation which involves a change in the properties of the retina with time during illumination.

1.1.3. BRIGHTNESS, LUMINANCE, AND RETINAL ILLUMINATION

Throughout our discussion, *brightness* means apparent brightness, the subjective sensation of how light or dark an object is. The objective measure of the amount of light emanating from a luminous source or reflecting object, weighted by the observer's spectral sensitivity function, is called *luminance* (Wyszecki and Stiles, 1967, p. 372). The *illumination* falling onto a surface from a distant luminous source is proportional to the luminance of the source multiplied by the square of the numerical aperture of the optical system between source and surface. The numerical aperture of an optical system is the reciprocal of its *f*-number. For the eye, the numerical aperture is proportional to the pupil diameter, and the retinal illumination is therefore proportional to the area of the pupil. Luminance can be expressed in terms of effective quanta of light per unit solid angle per unit time per unit area of the source. Illumination can be expressed in terms of effective quanta of light per unit time per unit area of the surface on which the light is falling. We will write L for stimulus luminance and I for retinal illumination, with the understanding that in most experiments the pupil area is fixed and therefore the two quantities are simply proportional. Evidence will be presented below about the importance of the luminous *flux* of light falling on a receptive field. Flux is illumination multiplied by area and can be expressed in units of effective quanta per unit time.

1.1.4. CONTRAST, CONTRAST SENSITIVITY, AND CONTRAST GAIN

Contrast is a physical property of the visual stimulus; it is the magnitude of luminance variation in the stimulus relative to the average luminance. We will show that the perception of contrast depends upon retinal adaptation. There is a problem with defining contrast precisely because there are two obvious definitions which differ approximately by a factor of two. In studying the visibility of aperiodic objects like uniform disks or bars or rectangles on a background, the natural definition of contrast is

$$C = (L_O - L_B)/(L_B) \tag{1a}$$

where L_O is the luminance of the object and L_B the luminance of the background, as indicated in Fig. 1(a). $L_O - L_B$ is usually called ΔL and so equation (1) is usually written as:

$$C = \Delta L/L_B. \tag{1b}$$

It is well known that for test stimuli of large area the psychophysical sensitivity follows Weber's Law

$$\Delta L_T/L_B = k \tag{2a}$$

$$C_T = k \tag{2b}$$

where ΔL_T is the threshold luminance increment, and k is a constant, the threshold contrast. C is referred to in the psychophysical literature as the Weber fraction, but we prefer to call it the *Weber contrast*, C_W.

There is a second definition of contrast which is used for periodic spatial patterns like sine gratings. This is the definition used implicitly by Rayleigh (1889) and more explicitly by Michelson (1927) to express the visibility of interference fringes:

$$C_R = (L_{max} - L_{min})/(L_{max} + L_{min}) = (L_{max} - L_{min})/(2\ L_{mean}). \tag{3}$$

As seen in Fig. 1(b), L_{max} is the maximum luminance and L_{min} the minimum luminance in the spatially periodic pattern. We call C_R the *Rayleigh contrast*. Both definitions of contrast have been used in the literature of adaptation, so we make them explicit here. It is important to realize that though there are these two different definitions of contrast, which are each appropriate for a particular kind of stimulus, the two different definitions are related because they refer to a single physical reality, namely the relative variation of a modulated component referred to a steady state, or average, component.

Contrast sensitivity we define as one divided by the psychophysical threshold contrast, either Weber or Rayleigh contrast as the case may be. *Contrast gain* is neural response divided by stimulus contrast (Weber or Rayleigh) and will have units mV per unit contrast, or (impulses/s) per unit contrast.

At low contrast, the Rayleigh contrast of a grating is approximately *one half* the Weber contrast, as can be seen by comparing equation (1) with equation (3) as applied to Fig. 1. The Rayleigh

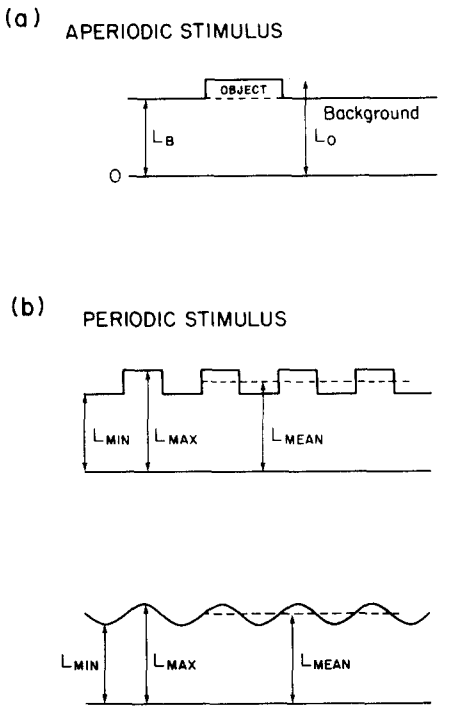

FIG. 1. The two kinds of contrast.
(a). Weber contrast is illustrated by a one-dimensional plot of the luminance profile of a bright object on a background. The Weber contrast is defined as $(L_O - L_B)/L_B$.
(b). Rayleigh contrast is illustrated with two different luminance profiles of grating patterns: the upper profile is of a square wave grating, the lower is the profile of a sine grating. For each grating, the Rayleigh contrast is defined as $(L_{max} - L_{min})/(L_{max} + L_{min})$. This is equivalent to the amplitude of the grating divided by the mean level, i.e. $(L_{max} - L_{mean})/L_{mean}$.

contrast is the mean-to-peak amplitude of the grating divided by the mean; the Weber contrast is the peak-to-peak amplitude divided by the luminance at the trough of the luminance profile, which for low contrasts is approximately the same as the mean luminance. One may therefore compare measurements of Rayleigh and Weber contrast sensitivities when the stimuli are at low contrast, as in most psychophysical experiments. One should expect the Rayleigh contrast sensitivity to be about twice that of the Weber contrast sensitivity, because of the approximate factor of two difference in the definition of the two kinds of contrast. Therefore, when we write about (Weber) contrast sensitivity or (Rayleigh) contrast sensitivity, we distinguish them because of the way the measurements were made, but we conceive of them as two different measures of one underlying property of the visual system, its sensitivity for contrast.

1.2. The Purpose of Adaptation

1.2.1. To make the magnitude of the retinal response dependent on contrast

In science as in other activities it is a good strategy to ask "Why?" This question puts problems in perspective, and the attempts to answer "Why?" are nearly always fruitful. So one ought to ask, "Why is there light adaptation?" We believe the answer is that adaptation keeps the retinal response to contrast invariant with changes of illumination, and thereby achieves one major goal of vision: constancy of the visual perception of reflecting objects.

Animals including man have evolved in a world of reflecting surfaces: water, earth, leaves, flowers, and other animals. What characterizes a reflecting object optically is its reflectance, since the reflectance is determined by the physical characteristics of the surface of the object. The reflectance is invariant with respect to illumination, within reasonable limits. The luminance of an object, L_O, is proportional to the product of the object's reflectance, R_O, and illumination, I.

$$L_O = K \cdot R_O \cdot I \tag{4}$$

We know from experience and experiment (e.g. Land and McCann, 1971) that over a wide range of illumination the brightness of a reflecting object does not change and that the brightnesses of an array of reflecting surfaces are perceived in the order of their reflectances. This leads to the (correct) inference that the visual system must have a method for estimating something about the reflectance of reflecting objects (cf. Land and McCann, 1971; Marr, 1982). We believe this "something" is the contrast (either Weber or Rayleigh contrast), which depends only on the reflectance, as shown below. Furthermore, one may infer that the constancy of perception of an object as its luminance varies indicates an underlying constancy of retinal response to the contrast of the object in spite of the luminance variation. For instance, the brightness of this print does not appear to vary as the page is brought closer to or further away from a light source, even though the luminance of the print may vary by factors of ten. We believe the reason the print appears the same brightness is that the magnitude of our retinal responses is the same when our eyes sweep across the print at each of the different levels of illumination, because the retinal response magnitude depends on the contrast of the print.

We will now present the argument that the constancy of retinal response is achieved by means of the mechanisms of retinal adaptation which produce a dependence of that response on contrast (Robson, 1975). This concept arises naturally out of a critique of a statement of the problem by Ernst Mach, the great philosopher-physicist (Mach, 1865, translated in Ratliff, 1965).

At the end of his first paper on the visual illusion now called "Mach Bands," Mach proposed a new principle for psychology: unique psychological events must correspond to unique physical processes inside the brain. As an illustration of this new principle, he offered the following example:

> "Let us examine another phenomenon with the help of our principle, which to my knowledge no one has yet discussed. White of a lesser intensity appears gray next to a brighter white. On the other hand, we are never in doubt whether we have before us a white or gray paper even under quite different conditions of illumination: in bright sunshine, overcast sky, in twilight, or by candle light, we have always almost the same sensation. What might be the cause of this? If the light intensity is 2-,3-,

> or *n*-fold brighter, so then is the retinal image of the white paper 2-,3-, or *n*-fold brighter, but so also is the rest of the visual field and the entire retina receives the 2-,3-, or *n*-fold illumination. The ratio of the quantity of light on the entire retina and the image of the paper remains constant under otherwise equal conditions. I think, therefore, that a process is initiated whose intensity depends on this ratio, and which causes the sensation of white for the retinal image. The brightness of the retinal image is, so to speak, being evaluated in terms of the total excitation. This is a judgement, the psychological side of the matter. The physical side is the process mentioned. It has not yet been discovered."

Mach's example shows a deep insight into the perception of brightness and the purpose of visual adaptation. However, there are some details of his brief analysis which are not quite right. In appreciating what is still valid and what needs correction, one may begin to see the point of light adaptation.

We will discuss two related but distinct comments in the quoted paragraph. The first is, "White of a lesser intensity appears gray next to a brighter white". This observation can be explained by modern psychophysics which has established that the brightness of an object is determined, to a great extent, by the Weber contrast between the object and its surroundings (Heinemann, 1955, 1972; Whittle and Challands, 1969). However, it is also known that brightness is mainly determined by *the contrast near the border* between an object and its surroundings. This surprising conclusion is forced by two experiments. First, the brightness difference between the two regions of unequal luminance varies directly with the sharpness of the border between them, being maximal for the steepest border (Thomas and Kovar, 1965; Shapley and Tolhurst, 1973). Second, two regions of *equal* luminance appear of *unequal* brightness when a local luminance difference is introduced as a border between them (Ratliff, 1965; Craik, 1966; Cornsweet, 1970; Land and McCann, 1971; Shapley and Tolhurst, 1973). See Fig. 2 for an illustration of this effect. Thus, the dependence of brightness on contrast, which Mach referred to in his 1865 paper, is now known to be mainly a dependence on border contrast.

The relation between brightness, contrast, and light adaptation emerges from a critical examination of a second remark made by Mach, which concerns brightness constancy, " . . . we are never in doubt whether we have before us a white or gray paper even under quite different conditions of illumination . . .". This observation can be explained by the fact that the contrast of an object on a background is not changed by variation in the level of illumination. Thus, if the visual system has the ability to derive brightness from contrast, it will thereby achieve brightness constancy. We will at this point demonstrate that contrast is invariant with illumination, and then show how the calculation of contrast by the visual system can be explained in terms of retinal adaptation.

The invariance of contrast with changes in the level of illumination can be demonstrated by an example. Consider as the simplest case a uniformly illuminated scene with an object on a background. The situation is illustrated in Fig. 3. The light coming from the object side of the border is proportional to $I \cdot R_O$ where I is the light falling on the scene from the source of illumination, and R_O is the reflectance of the object. The light reflected from the background side of the border is $I \cdot R_B$. The Weber contrast is $(IR_O - IR_B)/IR_B$. Dividing numerator and denominator by I yields $(R_O - R_B)/R_B$ as the contrast of an object of reflectance R_O upon a background of reflectance R_B. Thus, the contrast is *independent of the level of illumination* I and depends only on the reflectances of object and background.

Now we must show that retinal adaptation provides the mechanism by which the visual system responds to contrast. Consider what happens when the receptive field of a retinal cell (see Appendix 2) crosses the border between an object and a background, as in the example of Fig. 3. Suppose that the receptive field is "looking" at the background just before an eye movement occurs [Fig. 3(a)], and that the eye movement causes the receptive field to cross the border [Fig. 3(b)]. The *change* in the amount of light falling on the receptive field of the neural unit is the stimulus which elicits a neural signal which identifies the border. The stimulus is thus $IR_O - IR_B$. Now we must consider the role of adaptation. A neuron crossing the border only has been "looking" at the background side of the border. So the neuron is adapted to IR_B. As a reasonable hypothesis about what adaptation does, to be justified by data later, we propose it adjusts the gain of the neuron to be

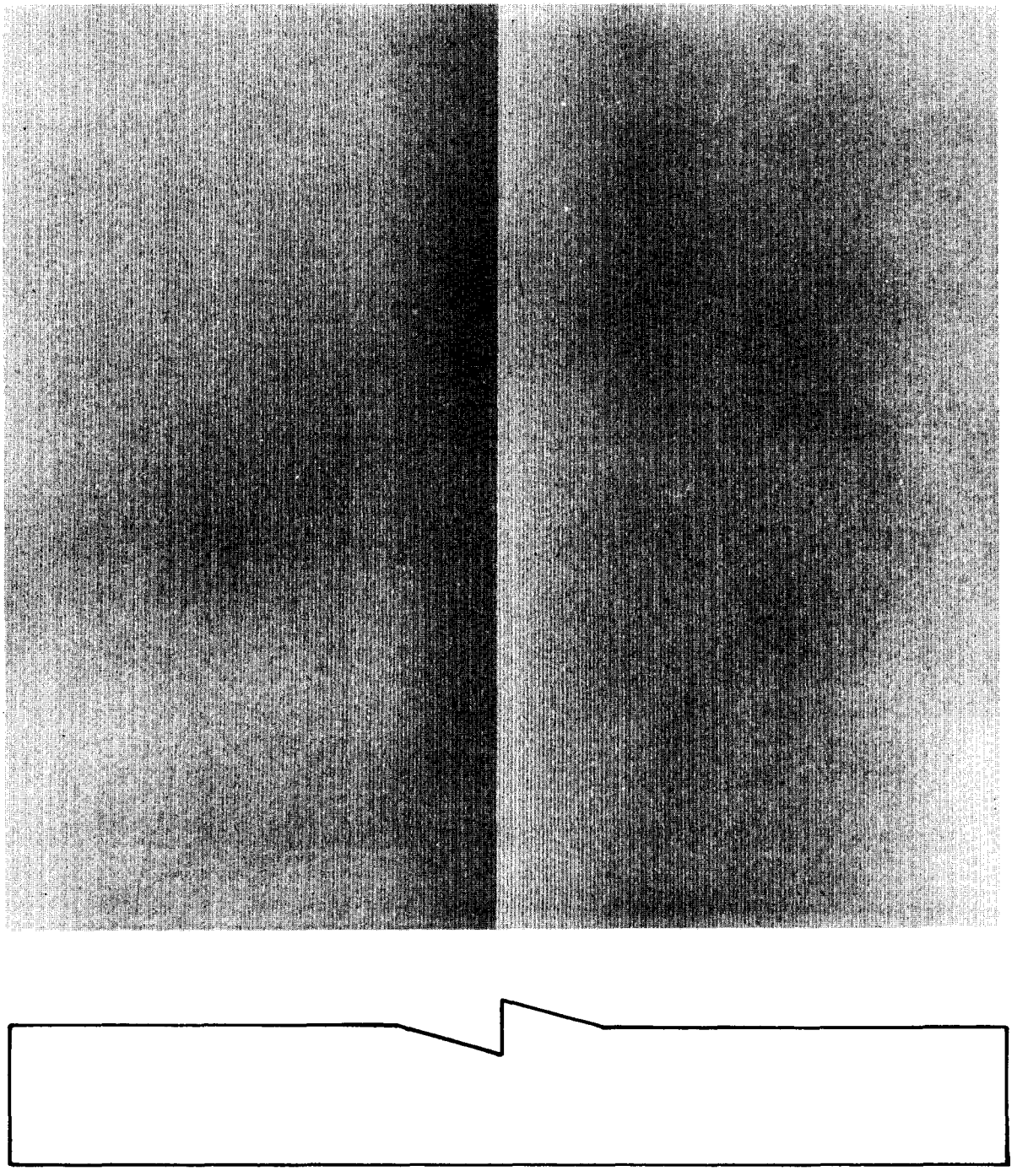

FIG. 2. Brightness depends on border contrast. This is an illustration of the Craik – O'Brien – Cornsweet illusion (cf. Ratliff, 1965; Cornsweet, 1970). The entire right half of the field is apparently brighter than the left half, yet the luminances of the two half fields are equal away from the border between them, as can be seen by covering the border with an opaque strip. Near the border, the luminance is steeply *decreasing* towards the border on the dark side, and it is steeply *increasing* towards the border on the bright side, as can be seen in the luminance profile drawn underneath the photograph.

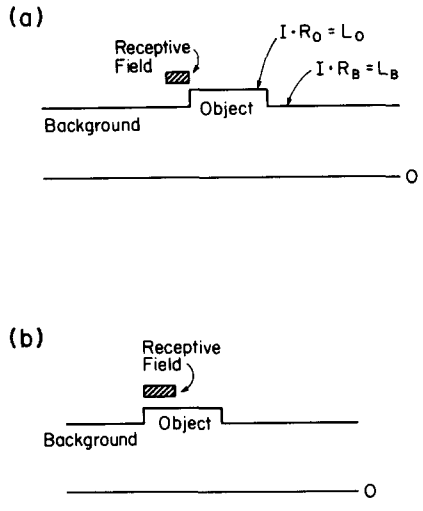

FIG. 3. The invariance of contrast with changes in level of illumination, and how this is sensed by a visual cell's receptive field. This picture shows the luminance profile of an object (with reflectance R_O) on a background (with reflectance R_B) when both are illuminated uniformly, with illumination I. The position of the receptive field of an individual retinal cell is indicated by the shaded rectangle. In (a), the visual cell is "looking" at the background. In (b), an eye movement has carried the receptive field of the cell onto the object. Equation (5) in the text demonstrates that this cell will respond to the Weber contrast of the object on the background, i.e. $(R_O\text{-}R_B)/R_B$, which is independent of illumination I. As explained in the text, in order for this calculation to work the cell must be influenced by a gain control which adjusts the cell's gain to be reciprocal with background illumination.

proportional to the reciprocal of the value of the illumination at which the cell's receptive field has been "looking" up to the moment it crosses the border. This inference is suggested by Weber's Law, equation (2). Thus the signal in our hypothetical neuron will equal the stimulus $I\cdot R_O\text{-}I\cdot R_B$ multiplied by the gain, $K/(I\cdot R_B)$, where K is a proportionality constant. The conclusion is that what the visual system uses to characterize an object is a signal

$$S = K\cdot(I\cdot R_O\text{-}I\cdot R_B)/I\cdot R_B \qquad (5)$$

which is proportional to the Weber contrast. In this case the neural response to the object is invariant with respect to changes in the illumination, I. This chain of reasoning only holds if the gain of the retina is reciprocal with background illumination, in analogy with Weber's Law. When retinal gain is not reciprocal with background, one should expect deviations from brightness constancy.

This line of reasoning leads to the conclusion that the detection of contrast depends on light adaptation. A different view is that the dependence of retinal responses on contrast is a consequence of the spatially antagonistic center – surround organization of receptive fields (discussed below in Appendix 2), but we believe this view to be incorrect. If there are eye movements, spatial contrast will be "seen" by the highly localized receptive fields of retinal neurons as successive contrast, or in other words as temporal modulation of the amount of light falling on those receptive fields (Helmholtz, 1909). As shown above, in this situation, the mechanisms of adaptation together with the compact nature of the receptive fields produce a response dependent on contrast without any requirement for a spatially antagonistic surround. The purpose of having receptive field surrounds probably is to make retinal neurons more sensitive to patterns with narrow spatial gradients, or high spatial frequencies, than to coarse patterns or diffuse light. Put another way, with light adaptation but without receptive field surrounds, retinal neurons would respond to contrast. However, with receptive field surrounds but without adaptation, the retina would be unable to respond to contrast; it would saturate quickly (see Section 1.2.2.). This point of view is similar to that put forward by Whittle and Challands (1969).

Consideration of Mach's statement together with more recent work on brightness constancy under conditions of non-uniform illumination leads to an interesting conclusion about the spatial spread of adaptation. Mach, and others after him (Hering, 1920; Helson, 1964), have supposed that the eye compared, "the quantity of light on the entire retina and the image of the paper", in order to calculate the brightness of the paper. This is not correct. The first correction is that brightness depends on a local phenomenon, the contrast near the border of an object, not on the quantity of light in the image of the object (see Fig. 2). Second, the retina does not compare the quantity of light "on the entire retina" with the amount of light in the object, as Mach suggested. As Davidson and Freeman (1965) and Land and McCann (1971) have shown, brightness is constant under conditions of spatially non-uniform illumination. We have argued that retinal

adaptation is the basis of brightness constancy. If retinal adaptation averaged light "on the entire retina" as Mach supposed, it could not produce brightness constancy when the illumination was spatially non-uniform. Retinal gain would be the same at all points on the retina. Neural signals at one point on the retina would be attenuated to the same extent as neural signals from another region of unequal local illumination. The correct calculation of the border contrast by the retina would break down. This implies that the retinal gain control mechanism must be somewhat localized, as indeed physiological evidence demonstrates (Cleland and Enroth-Cugell, 1968 among others).

Mach and many others later, including Land and McCann (1971), proposed that the visual system calculated the ratio $IR_O/IR_B = R_O/R_B$ directly, but this is incorrect, probably for the following reason. The ratio R_O/R_B is nearly always close to 1 in nature because the (achromatic) reflectances of most natural objects are so nearly the same. If R_O/R_B were the quantity being measured, correct identification of whether an object is brighter or darker than the background would become a problem of accurate measurement and comparison of the ratio with 1. The Weber contrast as defined earlier is the difference between the reflectance ratio and 1. That is, $(R_O\text{-}R_B)/R_B = (R_O/R_B)\text{-}1$. The Weber contrast, $(R_O\text{-}R_B)/R_B$, changes sign for objects brighter than the background (positive contrast) compared to objects darker than the background (negative contrast). The change of sign leads to a much easier, less error-prone discrimination of dark from bright objects than would computation of the reflectance ratio R_O/R_B. Furthermore, the visual systems of vertebrates put the basic measurement of the sign of the contrast into the functional architecture of the retina (cf. Hering, 1920). Cells which are excited by positive contrast (the "on" cells of Hartline, 1938; and the "on-center" cells of Kuffler, 1953) are segregated from the cells which are excited by negative contrast ("off" cells of Hartline and "off-center" cells of Kuffler). As discussed in Appendix 1, the elaboration of these "on" and "off" pathways involves separation of retinal synaptic connections in the inner plexiform layer of the retina, as suggested by Famiglietti and Kolb (1976), and then proven by Famiglietti *et al.* (1977) and Nelson *et al.* (1978). These physiological and anatomical observations reinforce the purely functional hypothesis that the retina is designed to measure the contrast of objects in order to provide to the brain an illumination-invariant description of the world of objects. Spatially localized light adaptation is a crucial factor in this retinal function.

1.2.2. To handle the large range of illumination levels

A related reason for the necessity of light adaptation is the very extensive range of average light levels presented to the eye by nature. A white paper (reflectance = 1) in moonlight has a luminance of about 3×10^{-2} cd m^{-2}. A white paper in sunlight is six orders of magnitude brighter, about 3×10^{4} cd m^{-2}. Backgrounds which affect vision extend three log units below reflected moonlight and one log unit above reflected sunlight, a total range of about 10^{10}. Part of this enormous range is taken care of by parallel processing in separate rod and cone pathways. In humans for example, the highly sensitive rod system handles the three lowest decades of backgrounds. The less sensitive cone system handles the upper six log units, and the decade of order 0.1 cd m^{-2} is shared (Riggs, 1965). However, in other animals the overlap of the ranges of background handled by rod and cone systems may differ because of a different rod – cone weighting. For instance, in the cat in which the rod – cone ratio is about one hundred times greater than in man (Steinberg *et al.*, 1973), the rod system handles five log units rather than three and the cones only dominate visual responses above 10 cd m^{-2} in background. A table which expresses the luminances in standard units and in terms of quanta of light per second per degree squared in area, is offered as Table 1.

A human is so sensitive when dark adapted that he can detect (without any false positive responses) that a light flash has been presented when only about one hundred quanta of light are incident on his cornea (Nagel, 1909; Hecht *et al.*, 1942; cf. Cornsweet, 1970). When one takes into account losses in the eye and the inefficiency of transduction, this implies that about twenty retinal responses to quanta of light are required for such ultra-reliable visual performance (this is a higher number than estimated by Hecht *et al.*, 1942, and

TABLE 1. *Quantal Equivalents of Photometric Units and some Other Useful Equivalences*

Photometric Unit	Equivalent in Quanta
1 cd m^{-2} through 1 mm^2 pupil, scotopic (1 scotopic td, human)	$4.46 \cdot 10^5$ quanta(507 nm) $(deg^2\ s)^{-1}$
1 cd m^{-2} through 1 mm^2 pupil, photopic (1 photopic td, human)	$1.26 \cdot 10^6$ quanta(560 nm) $(deg^2\ s)^{-1}$
1 lumen (scotopic)	$1.4 \cdot 10^{15}$ quanta(507 nm) s^{-1}
1 lumen (photopic)	$4.2 \cdot 10^{15}$ quanta(560 nm) s^{-1}
Other Useful Equivalences	
Unit	**Useful Equivalent**
1 quantum(507 nm) s^{-1}	$4 \cdot 10^{-19}$ watts
1 quantum(560 nm) s^{-1}	$3.5 \cdot 10^{-19}$ watts
1 deg^2 on human retina	$8.5 \cdot 10^{-4}$ cm^2
1 deg^2 cat retina	$4.8 \cdot 10^{-4}$ cm^2

is based on more recent estimates of quantum efficiency by Barlow, 1977). However, Sakitt (1972) has shown that if one relaxes the stringent requirement of no false positives, human observers can do better than chance when on the average only a single quantum of light excites the retina. This must mean that a single quantum response is comparable in magnitude to the intra-retinal dark noise. Comparable sensitivity is possessed by other animals.

The high sensitivity of a dark adapted eye poses a problem when the observer moves into brighter surroundings. Almost all neurons have a limited response range, from small signals to the peak levels set by biological constraints such as ionic equilibrium potentials. The range of *responses* is no greater than a factor of one hundred from noise to ceiling. The problem is obvious. How can the rod pathway in the retina encode three to five log units of stimulus level when it only has a factor of one hundred in response to work with? The answer is it cannot, and it does not. The retina adapts (reduces its gain) in the presence of large average inputs in order to represent only modulations around the average level when the average becomes too large to handle with the high dark adapted gain.

This problem of saturation of neural responses is related to the need for stable contrast sensitivity, which we have argued is the main purpose of adaptation. If the retina did not adapt, the contrast gain and contrast sensitivity would plummet at high light levels. Thus we would become blind to reflecting objects in bright daylight. This would not be a stable survival strategy, and therefore there is a biological need for adaptation. In fact, as our review of the psychophysical and physiological results will show, the contrast sensitivity and contrast gain of humans and animals generally increase as the illumination increases, finally levelling off to asymptotic values in bright light. At this point we will demonstrate how the saturation of neural response would lead to a decline in contrast gain. Then we will present a suggestion for an adaptation mechanism which offers an escape from the "saturation catastrophe".

First let us consider how the Naka – Rushton equation, which approximately describes the intensity – response function of distal retinal neurons, would lead to a "saturation – catastrophe" if the retina did not adapt. This is the Naka – Rushton equation:

$$R = (I/(I + I_S))\ R_{max} \tag{6}$$

where R is the response of the neuron measured as the change in membrane potential from its totally dark adapted level, I is the illumination of the stimulus. I_S is the semi-saturation constant also equal to the illumination at which R reaches its half-maximal value. This equation (Naka and Rushton, 1966) is called the Michaelis – Menten equation by Baylor and colleagues (Baylor and Hodgkin, 1973;

Baylor *et al.*, 1974; Baylor *et al.*, 1979) after a similar equation which arises in the theory of enzyme kinetics. The equation describes a system which saturates. Figure 4 shows a graph of R/R_{max} vs I plotted on linear – linear coordinates, and it can be seen that R is a saturating function of I: above the value I_S, the light can increase by several orders of magnitude but the response, R, can increase at most by a factor of two.

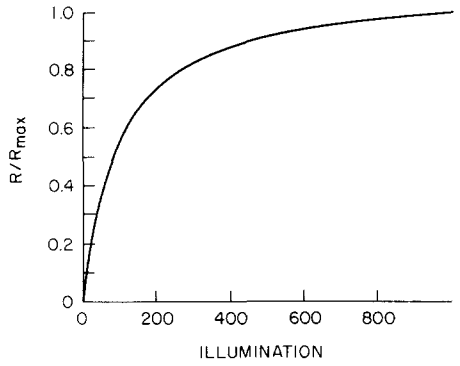

FIG. 4. The Naka – Rushton relation. This is a plot of $(R/R_{max}) = I/(I+I_S)$, the Naka – Rushton relation, on linear – linear coordinates. As can be seen, the relation produces a compressive nonlinear curve; the response saturates when $I > I_S$. In this example, the semi-saturation illumination, I_S, was chosen to be 100 in arbitrary units.

As argued by Williams and Gale (1977), Naka *et al.* (1979), Normann and Perlman (1979c), and Valeton and van Norren (1983), the Naka – Rushton equation can be used to describe gain and contrast gain in experiments in which the stimulus is an increment on a background, by examining how an incremental response ΔR depends on an incremental stimulus ΔI superimposed on a background I_B. In this case the I in the Naka – Rushton equation is $I_B + \Delta I$, the response is $R_B + \Delta R$, and the gain is $dR/dI \cong \Delta R/\Delta I$, when ΔI is small. The gain can be calculated by differentiating equation (6) (Williams and Gale, 1977; Naka *et al.*, 1979):

$$dR/dI = [1/(I_B + I_S)]^2 \, I_S R_{max} \rightarrow \{1/(I_B^2)\} \cdot (I_S R_{max}) \text{ for } I_B >> I_S \qquad (7)$$

The Weber contrast gain G_{con} can be defined as the amount of response per amount of contrast, thus:

$$G_{con} = dR/(dI/I_B) = dR/d\log I \cong \Delta R/(\Delta I/I_B) \qquad (8)$$

and to calculate the contrast gain for a neuron which obeys the Naka – Rushton relation we substitute equation (7) into equation (8) to obtain:

$$G_{con} = I_S R_{max} \cdot I_B/(I_B + I_S)^2 \rightarrow (I_S R_{max}) \cdot 1/(I_B) \text{ as } I_B >> I_S. \qquad (9)$$

What this means is that on account of saturation the Weber contrast gain would decline as the amount of steady background light increased, in inverse proportion to the average light level. Thus the "saturation – catastrophe" is implicit in equation (9). Figure 5 illustrates the gain and the contrast gain (equations 7 and 9) for a neuron which obeys the Naka – Rushton relation. If the retina would not adapt, the (Weber) contrast gain would drop as the light level increased.

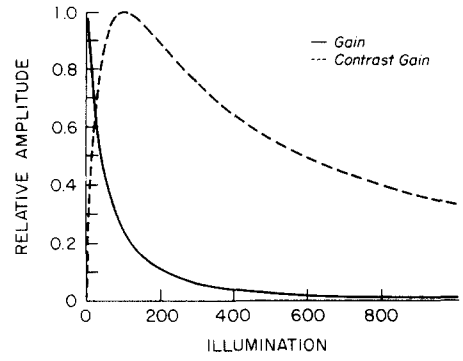

FIG. 5. The gain and (Weber) contrast gain of the Naka – Rushton relation. Gain is drawn as the continuous curve and contrast gain as the dashed curve. The saturating nature of the Naka – Rushton relation causes the gain to be a monotonically decreasing function of illumination [cf. equation (7)], while the contrast gain has a peak at $I = I_S$ (which was chosen to be 100 as in Fig. 4).

The retina adapts to avoid saturation by having an "automatic gain control" (Rose, 1948); the gain of the retina is reduced after the reception of enough light, so that the neural response usually doesn't saturate in the physiological range of illumination. We will have a lot to say about the mechanism of this automatic gain control, when we discuss the

experimental psychophysical and physiological data. It is worth observing how an automatic gain control solves the problem of the "saturation catastrophe" formally. One requires that R is not simply a function of the illumination I but depends on I times a gain factor, g, which depends on the value of I at present and in the recent past. Thus g is a "functional" of $I(t)$, $g = g\{t, I(t)\}$, but we will write it simply as $g\{I_B\}$ to simplify the algebra. This simplification is reasonable since we are only analyzing the case of an increment on a steady background, in which case g would be fixed at a steady state level set by the background. The Naka–Rushton equation modified to include adaptation becomes:

$$R/R_{max} = g\{I_B\} \cdot I/(g\{I_B\} \cdot I + I_S) \qquad (10a)$$

which can be expressed another way by dividing numerator and denominator by $g\{I_B\}$, as follows:

$$R/R_{max} = I/[I + (I_S/g\{I_B\})] = I/(I + I_S') \qquad (10b)$$

in which $I_S' = I_S/g\{I_B\}$.

The effect of adaptation can therefore be thought of, to a first approximation, as changing the value of the semi-saturation constant in the Naka–Rushton equation, I_S (Dawis and Purple, 1982). This change in the semi-saturation constant has been observed in retinal neurons; often it is referred to as "curve-shifting" because when equation (10b) is graphed on linear–log coordinates, the curve translates to the right as I_S' increases (Normann and Werblin, 1974; Werblin, 1977; Normann and Perlman, 1979c among others). Figure 6 illustrates the phenomenon of "curve-shifting" in cat retinal ganglion cells, from the work of Sakmann and Creutzfeldt (1969). Saturation is postponed by this strategy. With the appropriate choice of the g functional, one can obtain Weber's Law with such a modified Naka–Rushton model (Dawis and Purple, 1982; Valeton and van Norren, 1983).

It can also be shown that such a model can avoid the problem of contrast gain falling at high light levels, as follows. In analogy with equation (9) we can derive the contrast gain of the modified

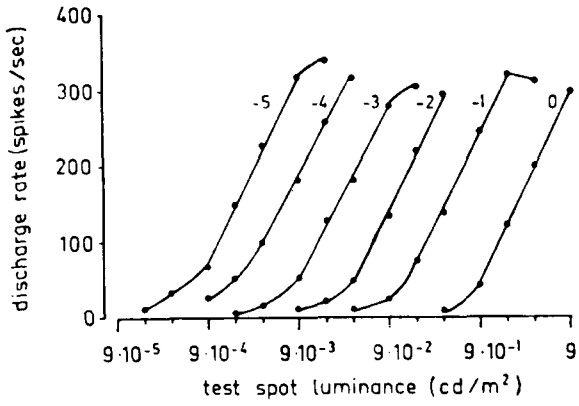

FIG. 6. "Curve-shifting" in cat retinal ganglion cell responses. Peak response of a cat retinal ganglion cell (in spikes s^{-1}) to a test spot flashed on a background, as a function of test spot luminance, at six different background luminances. The background luminances are indicated in the figure above and to the left of each curve, in units of log cd m^{-2} (dilated pupil). The test spot was 0.5 deg in diameter and was directed to the receptive field center. The background was 30 deg in diameter, and was on continuously. The stimulus spot was on for 500 ms, and was presented repetitively at 0.2 Hz. The response measure was the number of impulses in the 50 ms after stimulus onset minus the number of impulses in a comparable control period. Twenty responses were averaged for each point. From Sakmann and Creutzfeldt (1969).

Naka–Rushton model:

$$G_{con}' = I_S' \cdot R_{max} \cdot I_B/(I_B + I_S')^2$$

and since adaptation keeps $I_B \ll I_S'$, the contrast gain is approximately:

$$G_{con}' \sim R_{max} \cdot I_B/I_S' = R_{max} \cdot g\{I_B\} I_B/I_S \qquad (11)$$

which can be kept always increasing and approximately constant at high backgrounds by the correct choice of the dependence of the g functional, and therefore I_S', on the background. As will be seen, the retina usually makes this correct choice.

Summing up, we may say that adaptation is necessary to prevent saturation which would otherwise depress the contrast sensitivity (cf. Adelson, 1982). In discussing psychophysical and electrophysiological results on sensitivity and gain, we will show how the processes of light adaptation counteract the tendency of visual neurons to saturate.

It may be instructive to consider Fechner's integration of Weber's Law (equation 2) as another proposed answer to the problem of providing

constant contrast sensitivity (Boring, 1950). Fechner proposed that a certain fixed amount of change in internal sensation was required for threshold. We can call this value Δr_T. He then proposed that Δr_T was proportional to $\Delta I/I_B$ which is also fixed at threshold in the Weber's Law range. The crucial assumption was that r, the internal amount of sensation, was simply a function of the amount of light I; thus $r = r(I)$. With this assumption, Fechner could then treat Weber's Law as an approximation to a differential equation for r. Thus, $\Delta I/I$ could be approximated by a differential dI/I, while the increment in sensation Δr could be approximated by the differential dr. This led to Fechner's differential equation:

$$dr = a \cdot dI/I \tag{12a}$$

where a is a proportionality constant. Integrating this equation, he obtained,

$$r = a \cdot \log(I) + r_1 \tag{12b}$$

where r_1 is the response at $I = 1$. Fechner's Law (12b) is not right, as can be seen both theoretically and empirically. First, the theoretical objection is that r may not be viewed, without adequate justification, as a simple function of the value of I, the light, independent of the past history of illumination. Because it is now well known that the retina and brain have a finite time course of response, and response amplitude and time course are modified by the previous history of illumination, we now must consider r to be a transformation of I, or, to be technical, a *functional*. Therefore, the mathematical forms of equations (12a, b) are not justified. Furthermore, there are empirical consequences of Fechner's theory. Fechner's reasoning leads us to believe that Weber's Law implies that r must be proportional to $\log I$. This is a very shallow saturating function of I. The much steeper Naka–Rushton equation is actually observed. In essence, Fechner's explanation of Weber's Law is in terms of response compression on a very gently saturating intensity–response function, rather than the correct explanation in terms of automatic control of gain on a much steeper intensity–response curve. Combining theoretical and empirical objections, one may say that Fechner arrived at too shallow an intensity–response curve because his theory was too simple (see Sperling and Sondhi, 1968 for a similar point of view).

Having discussed the functional role of the retinal gain control in staving off saturation and maintaining high contrast sensitivity, we will now consider psychophysical results on the dependence of visual sensitivity on the background or mean level of illumination. A critical issue is, to what extent is sensitivity determined by noise and to what extent by retinal adaptation? Then we will consider the properties and mechanisms of retinal adaptation.

2. PSYCHOPHYSICAL LAWS OF LIGHT ADAPTATION

As proposed in the *Introduction,* we believe light adaptation to be an essential retinal mechanism for allowing effortless, illumination–invariant evaluation of the optical characteristics of *reflecting objects.* One hardly ever is aware of adaptation when it is performing this function. However, the effects of adaptation become noticeable when one observes *self-luminous sources of light*: stars, lamps, candles, and televisions. These visual objects change in brightness when the ambient level of illumination changes. When the light level is high enough, they disappear from view completely; they fall below the perceptual threshold for increments. Thus the action of light adaptation is exposed by the daily cycle of the fading and reappearance of the stars, though adaptation is working unobtrusively all the time to keep the perception of reflecting objects unaffected by that same cycle.

The influence of ambient illumination on the sensitivity for luminous sources like stars and candles has been noticed since antiquity. A poetic description and psychological explanation was offered by Shakespeare in *The Merchant of Venice.* As Portia and Nerissa return to Portia's villa Belmont at night after the triumph over Shylock in Venice, they speak about sensation and perception:

PORTIA
That light we see is burning in my hall.
How far that little candle throws his beams,
So shines a good deed in a naughty world.

NERISSA
When the moon shone we did not see the candle.

PORTIA
So doth the greater glory dim the less.
A substitute shines brightly as a king
Until a king be by, and then his state
Empties itself, as doth an inland brook
Into the main of waters. . . .
(Act V, Scene 1, II. 89–97).

The basic phenomenon is accurately described here, the fading to disappearance of a weak increment on a background. Since the background was moonlight in Shakespeare's "experiment", Nerissa must have been observing light adaptation of the rod system. This we now consider more quantitatively.

2.1. The Rod System

2.1.1. Sensitivity as a function of background

2.1.1.1. *Aguilar and Stiles' experiment.* A basic fact of visual experience is that the sensitivity for increments of light is reduced by steady background illumination. We will review the psychophysical investigations of this phenomenon in order to answer the question, "to what extent is the loss of visual sensitivity during illumination caused by true light adaptation of the visual system, and to what extent is it caused by increased "noise" in the stimulus itself?"

The canonical data concerning the dependence of the psychophysical threshold on the level of background illumination are those of Aguilar and Stiles (1954; Fig. 7). The stimuli were chosen to stimulate the rod system most effectively. The stimulus was large (9° diameter disk), presented in the periphery of the visual field, and was of a blue–green color on a red background. The red background desensitized the cone system relatively more than would a white background, and the blue–green test disk stimulated the rods more effectively compared to cones than would a white stimulus. The stimulus was presented for 0.2 s which allowed the rods to summate their signals temporally. In this way, Aguilar and Stiles were able

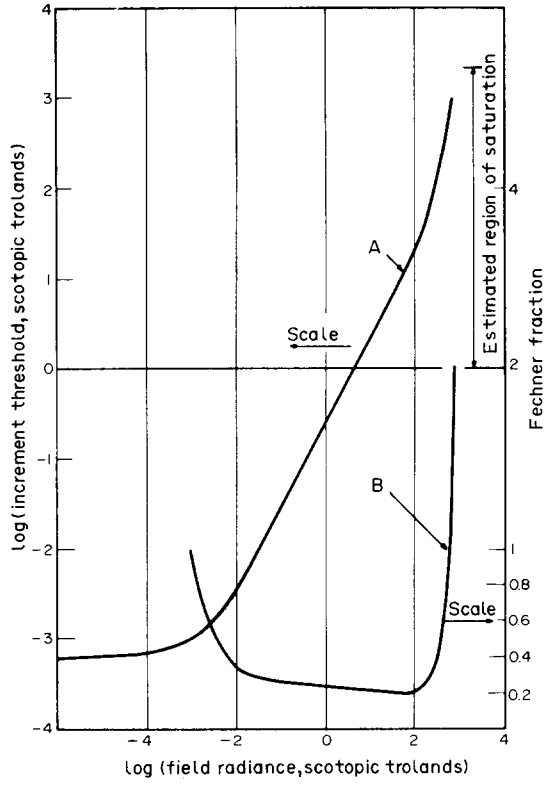

FIG. 7. Human "rod" threshold as a function of background illumination. The stimulus was a 9 deg disk on a 20 deg background presented 9 deg from the fovea. The disk was blue–green and the background was red in color, in order to enhance rod responses compared to cone responses. The stimulus was presented for 200 ms. The background was continuous. Curve A is the threshold illumination vs background. Curve B is what we would call the Weber contrast at threshold, and is labeled the "Fechner fraction" in this graph. From Wyszecki and Stiles (1967); original data replotted from Aguilar and Stiles (1954).

to isolate the rod system and study its properties over a wider range of backgrounds than has usually been possible. One unit must be defined in order to understand their results, the unit of retinal illumination, the troland (abbreviated td). In humans, a luminance of 1 cd m^{-2} viewed through a pupil with an area of 1 mm^2 is said to generate a retinal illumination of 1 td. The values of trolands in more physical units is given in great detail by Wyszecki and Stiles (1967).

As Aguilar and Stiles described their data, the first major feature shared by several observers was that whether there was zero background or a background below $2 \cdot 10^{-3}$ td, the threshold

remained constant and equal to the absolute threshold [Fig. 7 (curve A)]. In the human, $2 \cdot 10^{-3}$ td of retinal illumination is equivalent to about 1000 quanta(507 nm) $(\text{deg}^2\ \text{s})^{-1}$ incident on the cornea (Wyszecki and Stiles, 1967). This is the range of backgrounds in which the sensitivity is limited by internal noise in the observer, the so-called "dark – light" (Barlow, 1957, 1965). Then Aguilar and Stiles observed that the threshold began to rise and the sensitivity began to fall when the background exceeded $2 \cdot 10^{-3}$ td. At backgrounds above 10^{-2} td (equivalent to roughly the order of 10^{-3} cd m^{-2} with a 3 mm pupil), the threshold was equal to a constant times the background: Weber's Law.

$$\Delta I_T = k_W I_B \tag{13}$$

Thus the *sensitivity* $S = 1/(\Delta I_T)$ is inversely proportional to background in the Weber's Law range:

$$S = 1/(k_W \cdot I_B) = k'_W \cdot 1/(I_B). \tag{14}$$

Above 10^2 td (equivalent to $5 \cdot 10^7$ q(507 nm) $(\text{deg}^2\ \text{s})^{-1}$ incident on the cornea), the increment threshold rose more steeply than Weber's Law. This is what would be expected for a system which saturates. For example, a system which obeyed the Naka – Rushton equation (6), which is one kind of saturation, ultimately has a threshold which depends on the square of the background level, as shown in our equation (7) above. Therefore, the sensitivity above 10^2 td in background illumination can be explained by saturation in one of the neural elements in the visual pathway leading from rods to the central nervous system. This phenomenon is often called "rod saturation", but that is a shorthand description. The psychophysical data do not establish that the responses of rod photoreceptors themselves are saturating during "rod saturation".

In humans, the rods normally yield the increment threshold to the cones at a background around 10^{-1} td, as shown in Fig. 8 (Wyszecki and Stiles, 1967). Here backgrounds are given in log erg $(\text{deg}^2\ \text{s})^{-1}$, and the rod – cone break occurs at -6.7 log erg $(\text{deg}^2\ \text{s})^{-1}$. This corresponds to approximately $6 \cdot 10^4$ quanta$(\text{deg}^2\ \text{s})^{-1}$ which is just a little greater than 10^{-1} td, consistent with the value reported by Fuortes *et al.* (1961) and slightly higher but close to Blackwell's (1946) value. The rod – cone break depends on retinal locus since, for example, there can be no rod – cone break in the all-cone fovea. The numbers cited above have been taken from psychophysical investigation of the near periphery, $5 - 10°$ from fovea.

2.1.1.2. *The square root law and "noise".* As was first made clear by Rose (1948), the rod-driven visual system is often starved for light quanta and works right up against the limit imposed by quantal noise in the stimulus and the background. Under such conditions, the ultimate limitation on visual performance will be neural noise caused either by fluctuations in the light, or by fluctuations in the sensory properties of the observer, or both. This has definite implications about dependence of sensitivity on background luminance. The form of the human sensitivity versus background curve depends on experimental conditions (e.g. stimulus size, duration, wavelength) and does not always look like the curve obtained by Aguilar and Stiles (1954; Fig. 7, curve A). It is likely that over much of the visual range in the real world of white stimuli on a white background, the rod-driven visual system does not attain the ideal condition of adaptation, Weber's Law.

The effects of quantal noise and internal noise on the dependence of sensitivity on background are illustrated especially dramatically in Fig. 9 from Barlow (1965). He arranged a rod-isolation increment threshold experiment to illustrate the four different regimes which can be seen in the human sensitivity's dependence on background. The stimulus was a blue green spot 0.75° in diameter, of 8 ms duration, presented 10° from the fovea on a 10° orange background. If the stimulus had been smaller in area, the sensitivity would have been fluctuation-limited from the region labeled "dark light" to "rod saturation". If it had been of longer duration and larger in area, the curve would have looked like the results of Aguilar and Stiles (1954).

The limitation on sensitivity at the low end of the background scale is usually attributed to "dark light" rather than to the quantal fluctuations from the background (Barlow, 1957, 1964, 1977). In this region of backgrounds it is supposed that quantal fluctuations from the background light are small

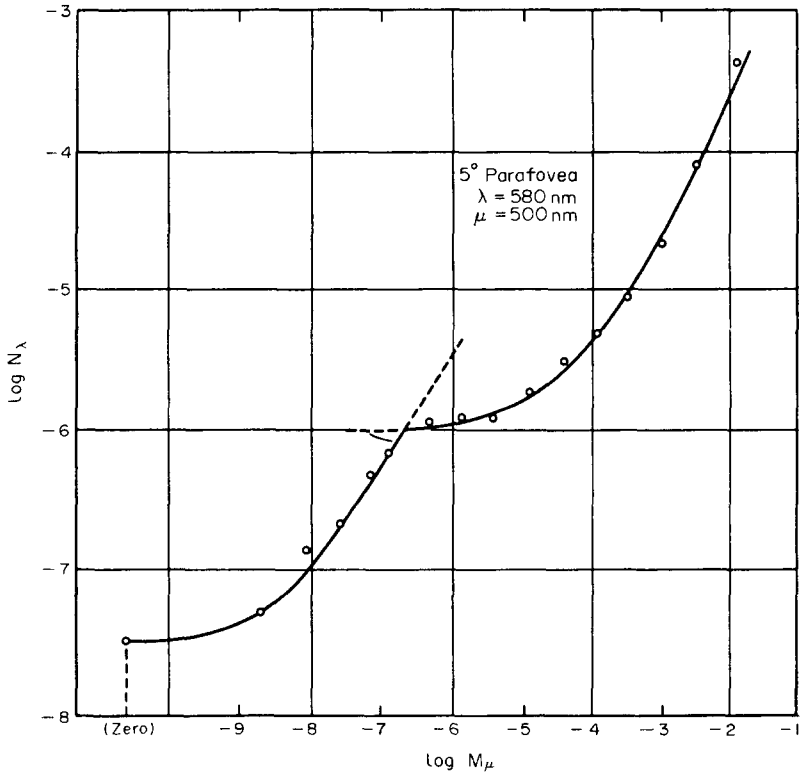

FIG. 8. The rod – cone transition in human vision. The log of the energy per unit time per unit area of a test spot is plotted on the ordinate. The abscissa is the log energy per unit time per unit area of a concentric background disk. The units for both are ergs $(deg^2\ s)^{-1}$ measured at the cornea (before losses in the eye and retina). These can be expressed equivalently as quanta(507 nm) $(deg^2\ s)^{-1}$ and therefore compared to retinal illuminations in trolands, td (cf. Table 1). The rod – cone break takes place at about -6.7 log ergs $(deg^2\ s)^{-1}$ which is equal to $6 \cdot 10^4$ quanta(507 nm) $(deg^2\ s)^{-1}$, and approximately equivalent to 0.1 td. In this experiment the test stimulus was a 1 deg spot presented for 60 ms. The test wavelength was 580 nm. The background wavelength was 500 nm, presented continuously. Background size was not given but was probably about 10 deg in diameter, concentric with the test spot. Test and background spots were placed 5 deg from the fovea. From Wyszecki and Stiles (1967).

compared to the variance which is internal to the retina caused by thermal isomerization of photopigment, spontaneous opening and closing of photoreceptor membrane channels, or spontaneous neurotransmitter release. These sources of noise have been shown to act in a way comparable to illumination, summing over distance and time, and therefore are conceived of as an equivalent "dark light" (Barlow, 1957).

The value of "dark light" was estimated by Barlow (1957) as the retinal illumination at the intersection of a line drawn through the initial flat portion of the increment – threshold curve with a line extrapolated down from the straight sloping portion (on log – log coordinates). Let us make explicit why this intersection gives an estimate of the "dark light". The increment – threshold curve may be expressed in an equation: $\Delta I_T = (I_D + I_B)^P$. P is the slope of the rising portion of the curve on log – log coordinates, and varies from 1 for Weber's Law to 0.5 for the square root law. The initial flat portion of the curve is where I_D is much larger than I_B and therefore in this range $\Delta I_T = (I_D)^P$. The straight sloping portion is where I_B is much larger than I_D, and there $\Delta I_T = (I_B)^P$. At the intersection of the two lines (on log – log coordinates), the two expressions for the threshold illumination are equal, and thus at this point the background illumination I_B equals the "dark light" I_D.

It appears that the "dark light" limits visual sensitivity by being the dominant term in the retinal noise rather than by setting the value of the retinal gain control. Barlow (1957) surveyed the literature on measurements of "dark light" and found that

most investigators agreed that the "dark light" was represented by an equivalent retinal illumination of $2 \cdot 10^{-3}$ td. This is equivalent to 1000 quanta(507 nm) $(\text{deg}^2\ \text{s})^{-1}$ incident on the cornea. As a source of noise, such a value of "dark light" is enough to explain the magnitude of the absolute threshold of about one hundred quanta at the cornea (Hecht *et al.*, 1942), with reasonable assumptions about integration time, area of summation, and quantum efficiency of the eye, as the following calculation demonstrates (cf. Barlow, 1957). If the integration time is about 0.1 s, and the integration area is about 1 deg^2, and the eye's quantum efficiency is about 0.25, then one can calculate that the "dark light" has a mean and variance of 25 events per integration time per integration area, and therefore a standard deviation of 5 events per integration time per integration area. If one assumes a threshold signal to noise ratio of 4 (Rose, 1973), the absolute threshold predicted from the "dark noise" caused by "dark light" would be about 20 quanta effectively absorbed by the rods, which is close to the observed 25 (the measured 100 at the cornea multiplied by the quantum efficiency of 0.25). Thus, "dark light" appears to limit absolute sensitivity by causing "dark noise".

Ascending the scale of backgrounds in Fig. 9, we next encounter the region in which the increment threshold increases like the square root of the background. This is the range in which Rose (1948) proposed that the visual threshold would be limited by quantal fluctuations. In this range one observes the square root law;

$$\Delta I_T = k_Q\, I_B^{1/2} \tag{15a}$$

$$S = k_Q'\, I_B^{-1/2}. \tag{15b}$$

The square root law follows from Rose's explanation because the standard deviation of a neural shot-noise (Dodge *et al.*, 1968), resulting from the temporal summation of the neural responses to randomly arriving quanta, would grow like the square root of the background light level. The response to the increment on this background would have to be picked out in the presence of the neural shot noise induced by the background, and the stimulus strength would have to be increased in proportion with the standard deviation of that noise. This argument is valid with the assumption that the signal/noise ratio at threshold is kept

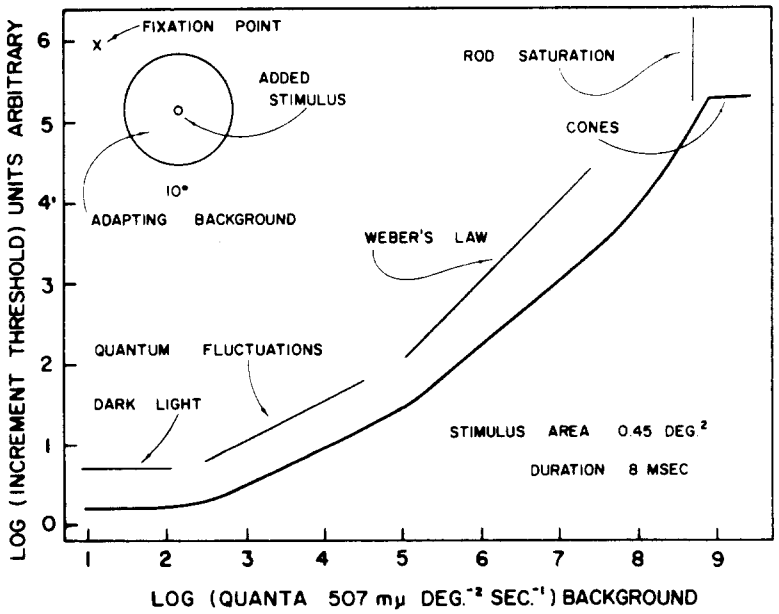

FIG. 9. Sensitivity vs background: dark light, quantal fluctuations, Weber's Law, and rod saturation. The full repertoire of psychophysical laws is illustrated in this experimental curve which is the graph of sensitivity for a blue – green test spot, 0.75 deg in diameter, 8 ms in duration, presented 10 deg from the fovea on an orange colored background which was 10 deg in diameter. The different regimes are labeled in the figure. From Barlow(1965).

constant (Rose, 1948). There are good reasons to believe in this assumption, because human subjects can keep their reliability (e.g. percentage of false positive responses) pretty constant at threshold across backgrounds, and this is consistent with a constant signal/noise ratio across backgrounds (Rose, 1973). The explanation of the square root law as a result of quantal fluctuations and the resulting neural shot noise seems intuitively implausible to some people because we do not "see" the quantal fluctuations which limit our performance as we can see suprathreshold visual noise, e.g. the "snow" on a poor television picture. However, if one computes the number of quanta available in contrast detection tasks, as Rose did originally, one is forced to the conclusion that quantal noise is the limiting factor in such performance. We will do this calculation below in the context of the dependence of contrast sensitivity on mean level of illumination.

Note that the decrease in sensitivity caused by quantal noise is not strictly speaking light adaptation. Rather, it is a loss in sensitivity caused by the properties of the stimulus. The visual system would not have to adapt in order to follow the square root law.

Next in Fig. 9 are the transition to Weber's Law, and finally rod saturation, both of which we considered when we discussed the results of Aguilar and Stiles.

2.1.1.3. *Square root to Weber transition.* The transition from the square root law to Weber's Law deserves special consideration. This occurs at the background illumination at which the visual system switches from being quantum-limited to being gain-control limited. As such, it is the first psychophysical indication of real light adaptation. Crawford's (1947) results were the first to indicate that the transition occurred at different backgrounds for test targets of different size. Barlow (1957) explored the conditions which influenced the transition. The conclusions he reached were that brief, small test spots produced increment thresholds which rose like the square root of the background over the entire scotopic range. The increment threshold curves for spots of large area, presented for longer times, had slopes close to 1, i.e. Weber's Law, over the whole rod range as in Aguilar and Stiles' work. Figure 9 illustrates that increment threshold curves for spots of intermediate area, presented briefly, have a transition from square root to Weber laws. It would be interesting to know exactly how this transition depends on the spatial and temporal factors, but Barlow's data for the rod system are incomplete on this point.

2.1.2. CONTRAST SENSITIVITY AS A FUNCTION OF ILLUMINATION LEVEL

Under the conditions that Weber's Law holds, the threshold increment of illumination is a constant times the background, i.e. $(\Delta I_T) = k_w\ I_B$. This implies that the threshold contrast and its inverse, the contrast sensitivity, are constant in the Weber's Law regime, as can be seen in the following equation

$$S_{con} = 1/(\Delta I_T/I_B)$$

$$S_{con} = 1/k_w = k'_w. \qquad (16)$$

In Fig. 7 this is the range from 10^{-2} to 10^{2} td. Remember that constant contrast sensitivity is the ideal which light adaptation must strive for if it is to achieve its main goal: perceptual invariance of reflecting surfaces with changes in background. Therefore, to the extent the psychophysics approaches Weber's Law, this major goal is met.

The threshold Weber contrast shows a clear break between rod and cone function as can be seen in Fig. 10, from Blackwell's (1946) data (see also Steinhardt, 1936; Craik, 1938). The minimal (Weber) contrast threshold for the rods is about 0.08 corresponding approximately to a contrast sensitivity, as we have defined it above, of 12. However, smaller targets require higher contrast; an 18′ spot has as its least rod contrast threshold a (Weber) contrast of 0.67 with a corresponding (Weber) contrast sensitivity of only 1.5. There is a clear jump in performance when the cones come in, with Weber contrast thresholds declining asymptotically to 0.008 and contrast sensitivities climbing to 125 or more.

At the transition from square root to Weber's Law, the contrast threshold undergoes a transition from declining like the inverse square root of background to becoming a constant independent of background. This can be seen from the expressions

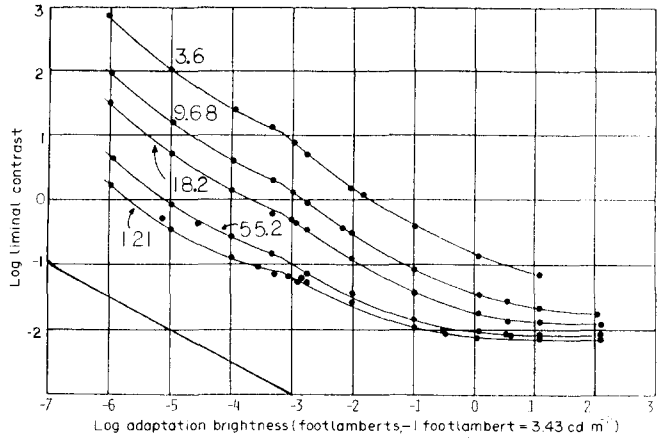

FIG. 10. The dependence of the Weber contrast threshold on background illumination. The test targets were circular disks on a large concentric background, viewed continuously. The test and background were neutral in color, and thresholds were determined by the method of forced choice. The points are the average thresholds of ten subjects. Different sized test targets generated the different curves, and the size of the target, in minutes of visual angle, is indicated for each curve. The straight line drawn in at the lower left corner of the graph has a slope of $-1/2$, which is the predicted slope of the contrast threshold against background for the square root law. Modified from Blackwell (1946).

for the square root law equation (15) and Weber's Law equation (14). The contrast threshold, $\Delta I_T/I_B$, becomes

$$\text{Square root law} \quad (\Delta I_T/I_B) = k_Q I_B^{-1/2} \tag{17a}$$

$$\text{Weber's Law} \quad (\Delta I_T/I_B) = k_w. \tag{17b}$$

Behavior of this sort can be seen in Blackwell's data Fig. 10. The contrast threshold falls roughly like the inverse square root over most of the scotopic range, though it is beginning to level off towards Weber's Law [equation (17b)] for the largest targets. Under the conditions of Blackwell's experiments, the rod system does not enter into the Weber Law regime before the cones take over. However, the results of Koenderink *et al.* (1978) on contrast sensitivity as a function of retinal eccentricity and mean level suggest that, in the far periphery of the retina, Weber's Law is achieved in the scotopic range even without the two-color procedure of Aguilar and Stiles (1954).

More recent studies of threshold contrast have used a stimulus with a sinusoidal luminance profile [Fig. 1(B)]. There are three extensive studies on the (Rayleigh) contrast sensitivity of the rod system, by van Nes and Bouman (1967), by Daitch and Green (1969), and by Smith (1973). These are consistent in finding a maximal (Rayleigh) contrast sensitivity of between 20 and 30 (which is equivalent to a Weber contrast sensitivity of $10-15$, in agreement with Blackwell). Figure 11 is a plot of some of Daitch and Green's data showing the dependence of scotopic contrast sensitivity on mean level.

The dependence on mean level of the (Rayleigh) contrast sensitivity for gratings confirms the conclusion that the square root law rather than Weber's Law dominates rod vision in humans (Daitch and Green, 1969; Smith, 1973). The contrast sensitivities for very low spatial frequency gratings (less than 0.5 c/deg) level off to a Weber Law limit, but contrast sensitivities for higher spatial frequencies increase with the average level of illumination, following the prediction of the square root law, equation (17a). Therefore, the shape of the "contrast sensitivity vs spatial frequency" curves for steady viewing change with the average level. At low levels they are "low-pass" functions which have their peak sensitivity at low spatial frequency. At higher levels of illumination, there is a definite peak at an intermediate spatial frequency, and a low spatial frequency "cutoff" (Daitch and Green, 1969; Smith, 1973). An analogous effect is seen in photopic human vision (van Nes and Bouman, 1967).

If one calculates how many quanta are available to detect threshold contrast, one is forced to admit that quantal fluctuations must play a significant

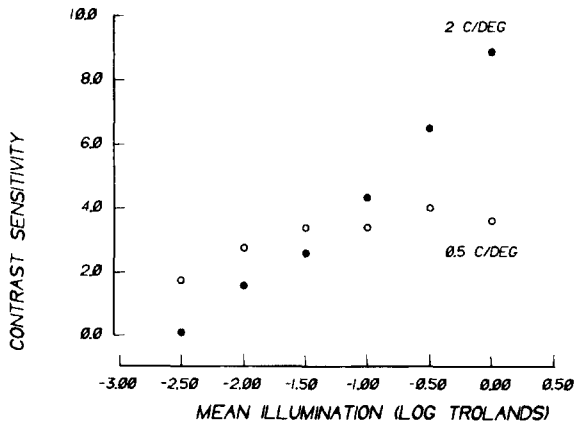

FIG. 11. The dependence of the Rayleigh contrast sensitivity on mean level. Results for two spatial frequencies of test target are shown: 0.5 c deg^{-1} as empty circles, and 2 c deg^{-1} as filled circles. An artificial pupil of 1 mm diameter was used. The test targets were sinusoidal grating patterns generated on the face of a cathode ray tube (CRT) display which subtended 8 deg by 3 deg, centered 12 deg from the fovea. The phosphor of the CRT was P31(yellow – green) but a Wratten 65 filter was interposed between subject and screen to shift the dominant wavelength of the phosphor towards the peak of the rod spectral sensitivity function. The target was presented for 200 ms every second. Control experiments established that the rods were the photoreceptors which determined threshold up to 0 log td. Plotted from Daitch and Green (1969).

part in limiting contrast sensitivity in the scotopic range. Suppose we make the conservative assumption that no less than one quarter of the quanta incident on the cornea may be used for contrast detection. Further we assume that the integration time over which quanta may be counted is about 0.1 s. Finally, and most crucially, we assume that the integration area for the neurons which are involved in the detection task is at most 1 deg^2. This last assumption is based on the measurements of receptive field center sizes of retinal ganglion cells in cats and monkeys at comparable retinal loci to the ones used in the human psychophysical experiments (Hubel and Wiesel, 1960; de Monasterio, 1978; Cleland *et al.*, 1979), and on the notions of the Channel Hypothesis expanded in Section 2.1.3. Given these assumptions, one may calculate what the highest contrast sensitivity might be in the scotopic range, at the highest background in this range which is about 10^{-1} td. This background retinal illumination corresponds to $5 \cdot 10^4$ quanta(deg^2 s)$^{-1}$ incident on the cornea. If one quarter of these quanta may be used by the retina, in an area of 1 deg^2, over a time of 0.1 s, then the total available quanta from such a background are 1250. If quantal fluctuations are the limiting factor, then the standard deviation of the quantal count with this mean number of 1250 must be the limiting source of noise. The standard deviation of the quantal noise must be the square root of 1250, which is about 35. In most detection experiments the signal – noise ratio is taken to be about 4, so that the threshold amount of quanta would have to be about $4 \cdot 35 = 140$. This is 140 neurally triggered quantal events; we must multiply by 40 (the reciprocal of 1/40 which is the product of the quantal efficiency of 1/4 times the integration time of 0.1 s) to obtain the stimulus illumination, measured at the cornea, to produce this many neural events. Thus we get that the stimulus illumination must be equivalent to $5.6 \cdot 10^3$ quanta(deg^2 s)$^{-1}$. The best quantum-limited (Weber) contrast sensitivity one could expect would therefore be $(I_B/\Delta I_T) = (5 \cdot 10^4)/(5.6 \cdot 10^3)$ or about 9, which is about as high as the human (Weber) contrast sensitivity reaches in the scotopic range. It is interesting that Aguilar and Stiles (1954) went through an analogous calculation in the Discussion of their famous paper, and concluded that they had discredited the idea that quantal fluctuations limited detection of contrast. However, in their calculation they made the assumption that, because they used a disk 9 deg in diameter as a stimulus, all the quanta in the 64 deg^2 area of the disk were available for the detection task. We now know this assumption is unreasonable. Presently available knowledge about the distribution of receptive field sizes supports Rose's (1948) hypothesis about the importance of quantum fluctuations in scotopic vision.

The great improvement in contrast sensitivity associated with the shift from rod to cone pathways is complicated by the effects of retinal inhomogeneity studied by Koenderink *et al.* (1978). They measured contrast sensitivity functions with sine gratings which subtended a fixed area of 4 deg by 4 deg, at several locations in the visual field. Figure 12 shows their finding that in the far periphery there is no increase of contrast sensitivity between scotopic and photopic background levels, and in the near periphery there is only an increase of about a factor of two in the photopic contrast

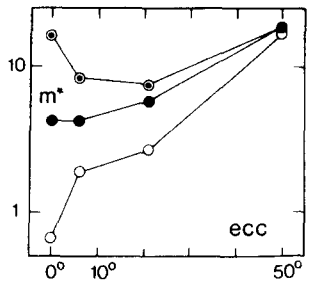

FIG. 12. Lowest threshold modulation depth as a function of retinal eccentricity and mean level of illumination. These data are all from contrast threshold measurements for sine grating patterns which subtended 4 deg by 4 deg and were drifting at a temporal frequency of 4 Hz. The stimuli were presented on a Hewlett – Packard 1310A display with a P15 phosphor (dominant wavelength at 510 nm), and were viewed by the subject through a 2 mm diameter artificial pupil. Contrast sensitivity curves were measured at each eccentricity and illumination, and the *minimum* threshold modulation depth (the reciprocal of the peak contrast sensitivity across spatial frequency) for each condition was denoted m*, which is the quantity plotted in the figure. The different symbols are for different mean illuminations. Empty circles are for 10 td; filled circles are for 1 td; circles with dots in the center are for 0.1 td. From Koenderink *et al.* (1978).

sensitivity over the scotopic. It seems that the foveal photopic contrast sensitivity is especially high when compared with the photopic (and scotopic) contrast sensitivity of the peripheral retina. They also found that, for a stimulus of a given size and spatial frequency, the transition from square root law to Weber's law depended on the retinal eccentricity; the transition occurred at lower luminances the more peripheral was the stimulus.

2.1.3. THE CHANNEL HYPOTHESIS

There exists a working hypothesis one can use to interpret Barlow's, Blackwell's, Daitch and Green's, and Koenderink *et al.*'s, findings about the influence of size or spatial frequency or retinal eccentricity on the transition from square root law to Weber's Law. This hypothesis is that the visual system is composed of a set of size or spatial frequency channels (Campbell and Robson, 1968; Enroth-Cugell and Robson, 1966; Robson, 1975; Graham, 1980). It is supposed that a test stimulus sifts through the channels and excites only those channels which are "tuned" to the stimulus. Such an hypothesis can be used in conjunction with research findings on the adaptation properties of retinal ganglion cells with different-sized receptive fields to make sense out of the transition from square root law to Weber's law (Enroth-Cugell and Shapley, 1973b). In essence, the explanation is that the channels which respond to low spatial frequencies suffer more loss of sensitivity with increase in background than do the channels which respond to high spatial frequencies because retinal gain controls depend on areal summation of background illumination. The channels which are most sensitive to low spatial frequencies, according to this view, sum adaptive signals over a larger area than do the channels most sensitive to high spatial frequencies. A more detailed explanation of this point is offered in the Theory section.

2.1.4. HUMAN AND FELINE CONTRAST SENSITIVITIES COMPARED

Since most of the electrophysiological results on retinal adaptation and gain control come from experiments on animals, it is relevant to compare human psychophysical and cat behavioral measurements of contrast sensitivity at different mean levels of illumination. Recently, this has become possible because of the results of Pasternak and Merigan (1981). Their results show that there are noticeable differences between the contrast sensitivities of cat and man. As can be seen in Fig. 13, the cat's contrast sensitivity improves with mean level in a way analogous to man, but at all mean levels the cat's peak contrast sensitivity is lower. This may be because the high mean luminances were photopic for the human, but all mean luminances were probably scotopic for the cat. The rod – cone transition in cats is at a much higher mean luminance than in man, presumably because of the much higher rod – cone ratio in the cat (Lennie *et al.*, 1976). Contrast sensitivity grows much more steeply for high spatial frequencies than for low, in cat as in man. However, the acuity of the cat is much worse than that of man, as can be seen in Fig. 13 by the separation between the high spatial frequency cutoffs of the contrast sensitivity curves at all mean levels except the lowest. However, the basic similarities between the dependences of feline and human contrast sensitivities on mean level of illumination encourage us to attempt a synthesis of human psychophysical studies of light adaptation with the body of

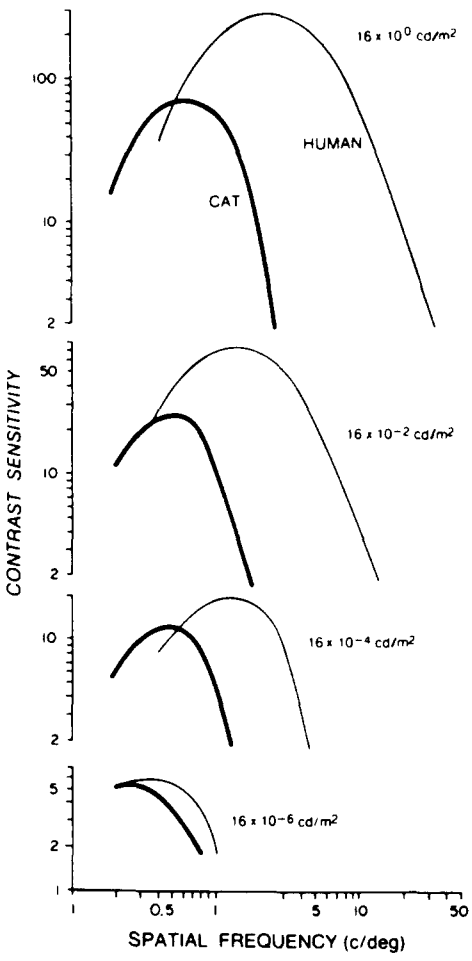

FIG. 13. The dependence of contrast sensitivity on mean luminance in man and cat. The cat results were obtained with a two-alternative forced choice paradigm and threshold was taken as 75% correct. The human results were obtained in a similar manner. For both human and cat, the natural pupil opening determined retinal illumination. The grating patterns were displayed on two CRTs with P31(yellow – green) phosphors. The mean luminance was controlled with neutral density filters placed in front of the CRT screen. From Pasternak and Merigan (1981).

knowledge about retinal adaptation in the cat.

2.1.5. THE TIME COURSE OF ADAPTATION

The change in sensitivity after a background has been turned on is not immediate. The time course of this change has been studied using the technique of "Crawford masking", in which a background is flashed on for a finite time, and a brief test flash is superimposed on the background at various times after the onset of the background. The most useful results obtained with this method on the rod visual pathways are those of Adelson (1982). Figure 14 summarizes his main findings. It can be seen that when the test flash is superimposed on a steady background, the threshold for the test follows a curve like that obtained by Aguilar and Stiles (1954; Fig. 7A). However, when the test is applied just at the onset of the background, the data show evidence for saturation, a steep upturn in the threshold vs background curve, at a background illumination more than two log units lower than the steady-state curve. One can therefore infer that the process of adaptation takes time to rescue the response from this saturation. How much time can be inferred from the other curves in Fig. 14. At test-background offsets of 200 – 1000 ms the saturation occurs at about one log unit higher than at zero offset. This means that a first stage of adaptation is basically complete within 200 ms. Then between 1 s and steady state a second slower process moves the saturation out another log unit.

Adelson (1982) interprets these results in terms of a cascade of processing, in which an initial linear stage is followed by a saturating transducer. If adaptation acts at or before the saturation, saturation of the response of the cascade can be avoided. This is a similar scheme to the one we outlined in Section 1.2.2. However, Adelson (1982) considers two different types of adaptation which he calls "subtractive" and "multiplicative". In experiments in which he used pre-adaptation before the "Crawford masking", he was able to show that the rapid adaptation, which has a time constant of about 100 ms, is multiplicative, i.e. it acts like a gain control. However, the slow adaptation, which has a time constant of about 30 s under the conditions of Adelson's experiments, appeared to be subtractive. The nature of the slow subtractive adaptation discovered in these experiments remains to be determined. The rapid gain control has been observed in physiological experiments to be described below. It is of theoretical importance that the gain control mechanism, while fast, is not turned on instantaneously when the background goes on.

2.1.6. WHAT PSYCHOPHYSICS IMPLIES ABOUT RETINAL MECHANISMS

2.1.6.1. *Psychophysical sensitivity loss: gain*

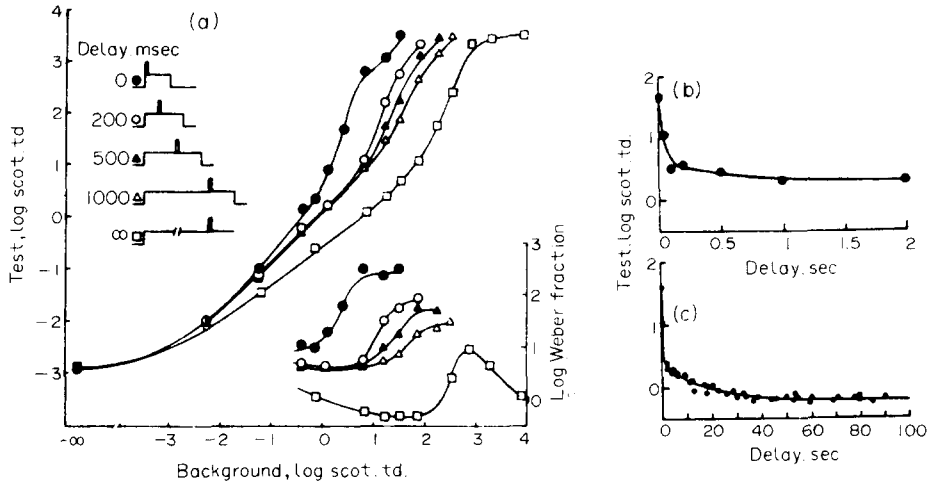

FIG. 14. Transient saturation and adaptation in the human rod system. A square test spot, 4.5 deg on a side and of 480 nm wavelength, was superimposed on an 11 deg red background centered 12 deg from the fovea in the nasal direction of the visual field (temporal retina).

In panel (a) are shown increment threshold curves taken at different times after the onset of the background flash. The test was 30 ms in duration, and the background was either continuous (empty squares) or was flashed on at various times before the test and was left on for 400 ms after the test. The delay between background onset and test onset is shown in (a) as an inset. Also graphed there (lower right) are the Weber contrasts at threshold for the different conditions, though they are labeled Weber fractions here. In panels (b) and (c) the time course of adaptation is shown by plotting the value of the threshold for the test spot presented with different delays after the onset of the background (3 scotopic td) which remains on. Panels (b) and (c) are the same data plotted on two different time scales, to illustrate the rapid and slow phases of adaptation. From Adelson (1982).

reduction or noise increase? What do the psychophysical results tell us about the underlying mechanisms of the control of sensitivity in human rod vision? In experiments which simulated our everyday visual perception of objects (Blackwell, 1946), the rod-driven human visual system did not quite reach the goal of Weber's Law. Up to the transition from the square root law to Weber's Law, the theoretical explanation required is only in terms of changes in the neural noise level caused by quantum fluctuations in the background light. At higher backgrounds than the levels required for the rod – cone transition in the white-on-white stimulus situation, the rod system must adapt, for its sensitivity drops more than can be accounted for simply in terms of an increase of noise due to quantum fluctuations. Also, the neurons which are the medium for perceiving gratings of very low spatial frequency must have their sensitivities controlled by more than quantal noise, because the sensitivity drops more steeply than the square root law. Thus noise and gain control must be involved in the variation of visual sensitivity with changes in background or average level of illumination.

2.1.6.2. *The site of adaptation: the retina.* One question we have not asked or answered is, what do the psychophysical results imply about the site of adaptation? It could be in the retina or in the brain or both. Several psychophysical experiments imply that it is retinal. For example, Battersby and Wagman (1962) demonstrated that there is little interocular transfer of steady light adaptation. If there were a large central component of light adaptation, one might expect a large amount of interocular transfer. Other similar experiments are those of Heinemann (1955) and Whittle and Challands (1969) both of which involved interocular brightness matching. In both sets of experiments, a dim matching light on an even darker background was matched to much brighter lights on bright backgrounds. The two lights were presented to different eyes and there was evidently little or no interocular transfer of the adapting effect of the bright background.

The gain of the human electroretinogram (ERG) in response to a large test target changes with background illumination in a way very similar to the psychophysical sensitivity for targets of the same

size (Biersdorf *et al.*, 1965). This experiment indicates that the gain of the retina is affected at the same light levels as the visual threshold for large targets. A further question one might raise is, where in the retina is the site of this gain change? Much of the physiological work we present below was motivated by this question. Prior to the physiological investigation, this question of the site in the retina of gain control was investigated psychophysically and this work led to the two concepts of gain control by pigment depletion, which has been shown to be only a minor factor in the control of retinal gain, and the adaptation pool, which still motivates research.

2.1.6.3. *Pigment depletion.* Vertebrate visual pigments are bleached by light into non-receptive intermediates which must be regenerated to a receptive state enzymatically. It was thought for some time that visual pigment depletion, resulting from bleaching by the background light, was the main cause of light adaptation (Hecht, 1924). This would put the site of adaptation right at the initial transduction stage in vision. However, it is now known that the pigment regenerates so quickly that there is a rather small amount of pigment bleached for either rods or cones over much of the visual range of backgrounds (Rushton, 1962). This can be seen in Figs. 15 and 16. Figure 15 is from Alpern and Pugh (1974) and shows the fraction of human rhodopsin, the rod pigment, bleached as a function of a steady background. Even up to rod saturation ($5 \cdot 10^8$ quanta(507 nm) (deg^2 s)$^{-1}$) the rod pigment has been bleached away less than 2%, but the visual threshold would have risen by a factor of more than 10^7, a discrepancy in prediction. Figure 16 shows a similar result for human cones, from Alpern *et al.* (1971). The cone pigment has been only bleached by half at about $10^{4.5}$ trolands which is near the top of the photopic range. So we can rule out pigment depletion as playing a major role in human light adaptation, though it has some effect on photopic (cone-driven) sensitivity (Boynton and Whitten, 1970; Valeton and van Norren, 1983). It is interesting that photopigment depletion began to be doubted as an explanation of light adaptation quite a long time ago, from experiments on increment threshold and photopigment concentration in the frog retina conducted by Granit *et al.* (1939). In those experiments ERG thresholds were increased by log units by backgrounds which had no

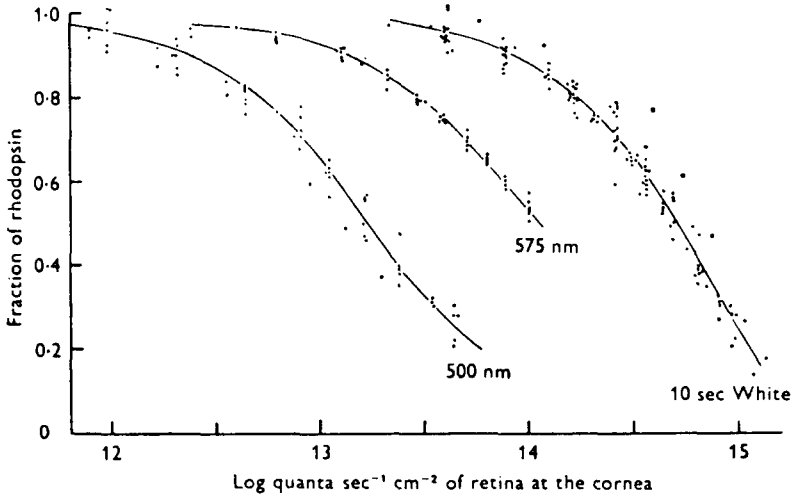

FIG. 15. Pigment depletion in rods caused by steady illumination. The amount of the rhodopsin in a 5 deg field located 18 deg peripheral to the fovea in the temporal retina was measured with a retinal densitometer (described in Alpern *et al.*, 1971 and in Alpern and Pugh, 1974). Plotted vertically is the fractional amount of rhodopsin left in the steady state at the background illumination plotted on the horizontal scale. The three curves are for steady lights of 500 nm, 575 nm, and white light. The 500 and 575 nm lights were obtained with the white source and interference filters. The results to concentrate on are the 500 nm results, since 500 nm is near the peak wavelength of absorption of rhodopsin and so will give the greatest pigment depletion at the lowest retinal illumination. The half bleaching point for the 500 nm curve is at about 13.2 log quanta(cm^2 s)$^{-1}$ which is equivalent to about 10.2 log quanta(deg^2 s)$^{-1}$, the more familiar unit of retinal illumination [also equivalent to about 4.3 log (scotopic) td]. These values of quanta are referred to the cornea and do not take into account losses in the eye. From Alpern and Pugh (1974).

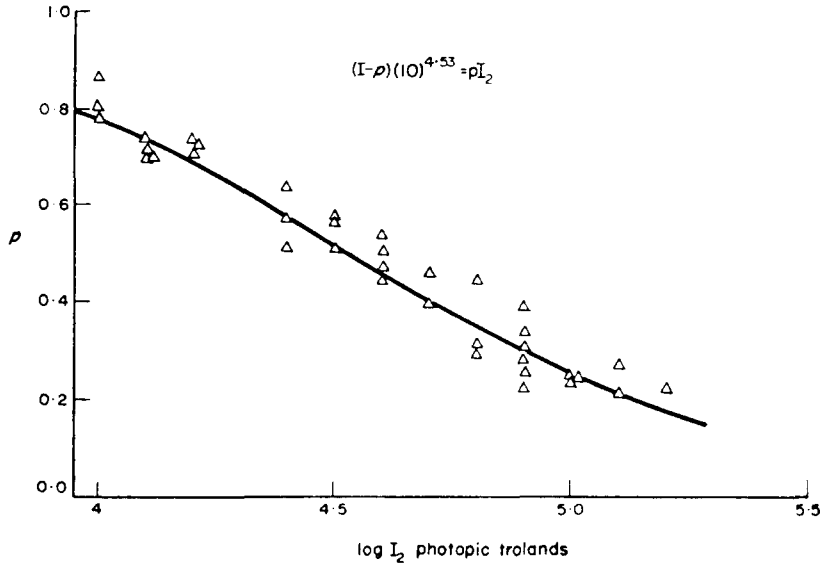

FIG. 16. Pigment depletion in cones caused by steady illumination. Plotted in the graph are retinal densitometry measurements of the human fovea at different levels of steady illumination of a yellow – green bleaching light. P is the fraction of cone pigment remaining in the steady state. The half-bleaching illumination is about 4.5 log photopic td, which is equivalent to approximately 10.6 log quanta(deg^2 s)$^{-1}$ of quanta at 560 nm, the peak wavelength for the long-wavelength cones. Note that the half-bleaching point for the cones is within half a log unit of the half bleaching point for rods, when expressed in units of quanta of the optimal wavelength for the pigment. From Alpern *et al.* (1971).

measurable effect on the extractable rhodopsin concentration. Furthermore, Rose (1948, 1973) has presented an elegant argument which disposed of pigment depletion as the sole explanation of light adaptation. If pigment depletion were significant in the scotopic range, the quantal-noise limited contrast sensitivity would be held down to a very low level. This would be the consequence of depleted pigment; light would be absorbed less efficiently. Since the contrast sensitivity does rise in the quantal-noise limited range, and it rises to a degree consistent with a relatively constant quantum-efficiency, there cannot be a significant amount of pigment depletion in the scotopic range of vision. Figure 15 proves the correctness of this argument.

2.1.6.4. *Adaptation pools.* Since one of the critical issues in this field is the actual site in the retina of the process of light adaptation, a natural question to ask is, how far does adaptation spread in space? We have already argued in the Introduction that light adaptation has to be localized to some extent, for functional reasons. But how localized? Perhaps the photoreceptor is the site of adaptation, or perhaps the retinal ganglion cell, or perhaps one of the retinal interneurons.

William Rushton, in his well known Ferrier lecture, summarized his years of work on this problem (Rushton, 1965). He concluded that there must be adaptation pools more proximal in the retina than the photoreceptors, and that within these pools light evoked signals were added up to set the gain of the retina by negative feedback. Rushton used two pieces of psychophysical evidence to support his contention that there were adaptation pools. His arguments from these two bits of evidence are vulnerable to criticism, and yet the idea of adaptation pools has been extremely fruitful and provocative.

Rushton's first bit of evidence for the concept of adaptation pools was the extremely low level of light required to raise the psychophysical threshold by 0.5 log units above the absolute threshold. According to Rushton, the number of light quanta which adapted the retina was only one tenth of the number of rods upon which the background fell, i.e. about $5 \cdot 10^4$ quanta s^{-1} over a circular background 10 deg in diameter. Thus, he concluded rod

signals were reduced in magnitude, even those from rods which had never seen the adapting light. However, as we have stated repeatedly, a decline in psychophysical sensitivity does not necessarily mean a decline in retinal gain but may simply be a rise in the noise level against which the signal must be discriminated. There would be no change in retinal gain in this latter case, even though the psychophysical sensitivity had declined. When we repeated this experiment of Rushton's on retinal ganglion cells, and found a significant effect on retinal gain at such low backgrounds that only a fraction of the rods received a photon each second, Rushton's argument was made much tighter (Enroth-Cugell and Shapley, 1973a).

Rushton's second argument was based on an experiment with gratings. The idea was to present a background grating and then a test grating in phase, and another test grating 180 deg out of phase, with the background grating. All gratings were square waves of 2 c deg^{-1} spatial frequency. Rushton reported no difference in threshold for the in-phase and out-of-phase condition, and then argued this meant that the background signals were pooled in an adaptation pool which he supposed was larger in diameter than the period of the background grating, 0.5 deg. There is something wrong with the reasoning behind this experiment, and with the actual result, which was not replicated by Barlow and Andrews (1967). The weakness of the argument is interesting because it relates to the degree of localization of adaptive effects, to the concept of spatial channels, and to results on adaptation pools in retinal neurons.

Adaptation pools ought to be roughly the same size as the receptive fields of the retinal neurons they adapt. This is consistent with our argument in the Introduction (Section 1) about the function of adaptation in providing brightness constancy when there are gradients of illumination, as was observed by Davidson and Freeman (1965) and Land and McCann (1971). Electrophysiology indicates that the size of an adaptation pool is equal to (Cleland and Enroth-Cugell, 1968; Enroth-Cugell and Shapley, 1973b) or less than (Green *et al.*, 1977; Harding, 1977) the size of the receptive field of a retinal ganglion cell it regulates. Therefore, functional reasoning and electrophysiological evidence support the inference that if a retinal cell can resolve a grating pattern, the adaptation pool which controls that cell's adaptation state should also be able to resolve the grating. Now the channel hypothesis implies that the 2 c deg^{-1} grating that Rushton (1965) used stimulated just those cells whose receptive fields could resolve 2 c deg^{-1}. Daitch and Green's (1969) data indicate that these cells are towards the high end of the spatial frequency range for rod-driven visual cells in the peripheral retina. We must conclude that if these cells resolved the 2 c deg^{-1} test pattern their adaptation pools must also have been able to resolve the pattern. Therefore, one ought to have observed that the threshold for a superimposed test pattern should have depended on its position relative to the adapting grating. In-phase with the adapting grating should have been the position of highest threshold. This is just what Barlow and Andrews (1967) found, and the failure of Rushton (1965) to obtain this result probably was a consequence of image blur or light scatter. Barlow and Andrews concluded that their results cast doubt on the existence of adaptation pools. However, by the argument above, their result does not disprove the existence of adaptation pools at all. It just confirms what is now known from other experiments, that the adaptation pools "see", i.e. resolve patterns, as well as the receptive fields they serve.

A different kind of experiment on adaptive summation was performed by Westheimer (1965). The test spot was small (0.1 deg) and the adapting field was a circular disk concentric with the stimulus. The adapting disk was constant in luminance but varied in diameter. Under these conditions, threshold increased as a function of adapting disk diameter up to a diameter of about 0.75 deg. In this range, threshold was approximately proportional to the area of the adapting disk. Surprisingly, for disk diameters larger than 0.75 deg the threshold dropped, suggesting that light falling beyond 0.375 deg from the test spot acted to "sensitize" the response to the test.

At first the "sensitization" discovered by Westheimer was thought to be due to adaptation produced by a neural element which had a spatially – opponent center – surround receptive field. Such a neuron might have a steady state response to the large adapting disk which was much smaller than its steady response to the small disk.

If the gain of the cell were related to this steady state response, the gain reduction would be less for the large disk. However, physiological results on scotopic adaptation do not support this interpretation of the psychophysical result (Enroth-Cugell *et al.*, 1975; Barlow and Levick, 1976). That is, the gain of the receptive field center in cat retinal ganglion cells in the scotopic range decreases and then levels off as background diameter is increased, and is not a decreasing and then increasing function as would be supposed from the conventional interpretation of sensitization. Furthermore, psychophysical sensitization in the scotopic range has not been convincingly demonstrated under stabilized image conditions (MacLeod, 1978). This suggests that retinal or central stimulation by the moving border of the background disk may be needed for sensitization to be observed (MacLeod, 1978). Moreover, the relation of sensitization to retinal adaptation has been questioned by Lennie and MacLeod (1973) who showed that the key to sensitization was the *uniformity* in luminance of the 0.75 deg diameter desensitizing disk and the outer annulus. If the outer annulus were either lower *or higher* in luminance than the central disk it would reduce sensitization.

2.2. The Cone System

2.2.1. Sensitivity as a function of background

The cone system (Photopic system) is like the rod system in yielding Weber's Law under some circumstances and the square root law under others. As is evident from Fig. 8 from Wyszecki and Stiles (1967), in human vision the cones take over at threshold from the rods above 0.1 to 1 td in background retinal illumination. The increment threshold curves in the literature tend to all show a cone plateau (cone "dark light"; Barlow, 1958) from 0.1 up to about 10 td. The photopic Weber's Law for a moderate-sized test spot (diameter > 0.5 deg) takes over from the cone "dark light" limited behavior at 10 td very reliably.

Although the photopic thresholds for spots follow Weber's Law up to the point where the pigment is bleached away, it has recently been shown that saturation can be demonstrated in the cone system with adapting backgrounds which are themselves small in area (Buss *et al.*, 1982). This saturation can begin at as low a background as 10 td, in the low photopic range. On a small diameter (0.25 deg) background the threshold of a small spot climbs much more steeply than Weber's Law (obtained with a large background) would predict. This effect may be observed under stabilized image conditions (Tulunay-Keesey and Vassilev, 1974). The photopic saturation is thought to be due to saturation of a cone-driven interneuron in the retina, one which has a center-surround interaction so that a background of large area will release the putative interneuron from saturation. Electrophysiological evidence on sensitization in the photopic range is presently scant and equivocal, but there are some indications it may exist (see Section 3.7.).

2.2.2. Contrast sensitivity and mean level

For most targets, and in particular moderate-sized spots with a sharp edge on a large background, Weber's Law holds from 10 td to 10^5 td, i.e. throughout the photopic range of backgrounds. This is shown very clearly in the data of Whittle and Challands (1969), Fig. 17. The lower curve for each subject is the increment threshold curve; above it are curves of constant brightness as matched to a test flash in the contralateral eye. It is interesting that the constant brightness curves are approximately parallel to the increment threshold curve. This means that the apparent brightness of two different increments was proportional to their (Weber) contrasts. Thus, Whittle and Challands' results imply that (Weber) contrast rather than luminance determined the apparent brightness. They also point out that other, central, factors may contribute to apparent brightness. Under the conditions of their experiments, the mechanisms which determined brightness were purely monocular and therefore probably retinal — as evidenced by the fact that stimulus contrast against a fairly high background in one eye was needed to match a much weaker flash luminance against zero background in the other eye. These data of Whittle and Challands support the main hypotheses we started out with about the functional significance of adaptation in establishing brightness constancy contingent on contrast, which contrast depends on the reflectances

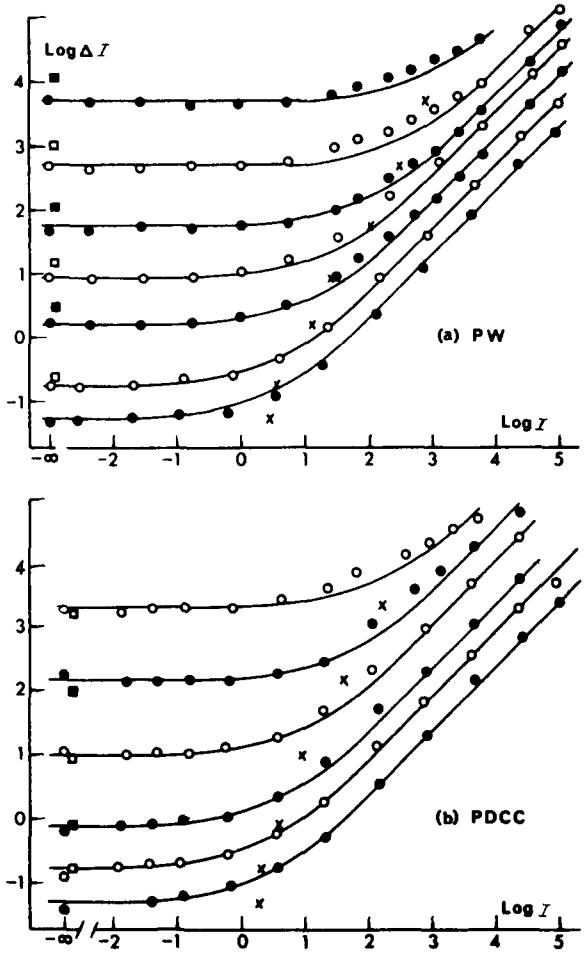

FIG. 17. Increment threshold and constant brightness curves for two human subjects. A test patch subtending 56′ by 28′ on a uniform background was presented to one eye, and a comparison patch, on zero background, was presented to the other eye, except for the bottom curve in each panel, which is a monocular increment threshold curve for each subject. For each of the constant brightness curves, the luminance of the comparison patch was held fixed, and the luminances of the background and test patch were varied so that the test matched the comparison in brightness. Therefore, each such curve represents test and comparisons matched at a constant fixed (subjective) brightness. The test and comparison patches were flashed for 200 ms, every 1.5 s at the low backgrounds up to every 8 s at the high background levels. The curves are similar at all brightness levels, having a flat portion and then a sloping portion with slope = 1. At the higher brightnesses, the transition from flat to sloping portions was somewhat broader than at low brightness or at threshold brightness. Each cross (×) marks the value of I at the intersection of the flat and sloping portions of the curve above it. Units are in log (photopic) td. From Whittle and Challands (1969).

of objects.

There has been a lot of work on spatial vision of the cone system, in particular foveal cone vision. The idea of spatial channels is derived from this work. The outstanding research on the effect of mean luminances on spatial contrast sensitivity has been done by van Nes and Bouman (1967). Their work was presented both as contrast threshold vs spatial frequency parametric in mean level shown in Fig. 18, and also as threshold contrast vs mean level, parametric in spatial frequency, as in Fig. 19. What can be seen, especially from Fig. 19, is that the transition from square root law behavior to Weber Law behavior depends a lot on spatial frequency. If we call the spatial frequency k, then their results imply that the transition illumination I_{BT} is proportional to the square of the spatial frequency

$$I_{BT} = A \cdot k^2 \tag{18}$$

where A is simply a proportionality constant. This

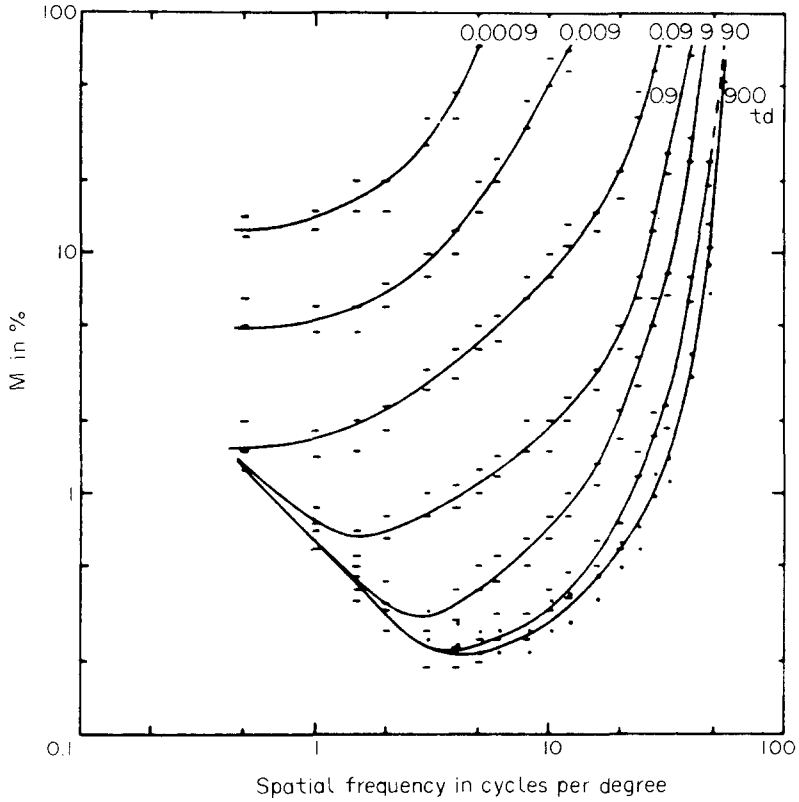

FIG. 18. The dependence of contrast sensitivity on spatial frequency at different mean retinal illuminations in the photopic range. Actually what is plotted is threshold modulation M (what we would term the threshold Rayleigh contrast) of a sine grating as a function of spatial frequency at each of several levels of mean illumination, indicated at the top of the figure. Light from a monochromator was transmitted through a photographic transparency of a sine grating which was imaged by a Maxwellian view system on the subject's retina. The grating subtended 8.25 deg by 4.5 deg and was fixated, therefore centered on the fovea. The contrast of the pattern was reduced by adding a constant level of light from another monochromator. The threshold modulation is the reciprocal of what we have called the (Rayleigh) contrast sensitivity. In this figure mean illuminations are indicated in photopic td. The wavelength was 525 nm. Compare these data with those of Pasternak and Merigan (1981), Fig. 13. From van Nes and Bouman (1967).

has interesting consequences for models of photopic light adaptation and in particular for a model which uses the ideas of spatial frequency channels and adaptation pools, presented later on.

An interesting sidelight on Fig. 18 is that it indicates that contrast sensitivity continues to improve for gratings of high spatial frequency until well into the photopic range, and Fig. 19 confirms that fine gratings obey the square root law even up to quite high levels of illumination. If one accepts the quantum fluctuation explanation for this behavior, one must conclude that vision of fine patterns is quantum limited up to quite high light levels. As before, we must add the qualification that this result does not indicate definitely whether or not the retinal gain is affected at these light levels.

It is important that the cones "see" so much better than the rods when the criterion is threshold contrast. This can be seen in Figs. 8–10, and especially by comparing Fig. 11 from Daitch and Green with Fig. 18 from van Nes and Bouman. There's a jump of about a factor of 10 or so from the optimal rod-driven contrast sensitivity of Daitch and Green, at about 1 td background, to the best contrast sensitivity of van Nes and Bouman with peak contrast sensitivities above 200. This corresponds to a Weber fraction ($\Delta I_T/I_B$) of less than 0.01, in agreement with the sensitivities of Blackwell's subjects who had an asymptotically low Weber fraction of 0.008 (Blackwell, 1946). However, the situation may be complicated by retinal inhomogeneity. Koenderink *et al.* (1978)

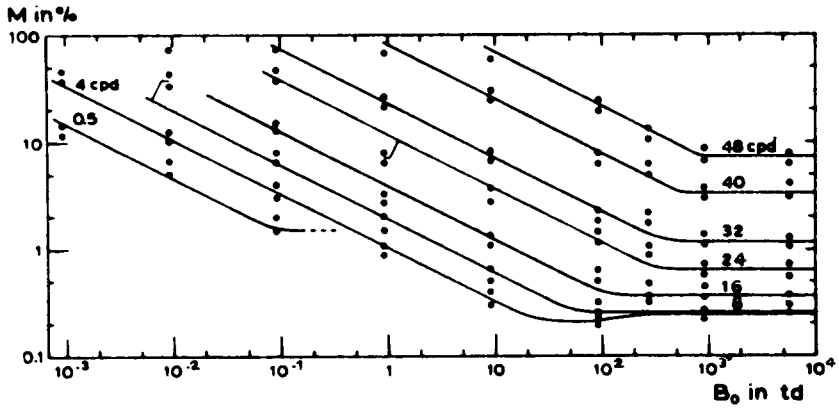

FIG. 19. The dependence of threshold modulation on retinal illumination for different spatial frequencies. Wavelength was 525 nm in the same experiments as in Fig. 18. B_0 is what van Nes and Bouman called mean retinal illumination, and it is given in td. M is what we would call threshold (Rayleigh) contrast. The sloping portions of these curves are therefore roughly consistent with the square root law, and the flat portions, where contrast sensitivity is constant as mean level changes, are consistent with Weber's Law. The transition between these two parts of the curve appears to shift to the right, to higher illuminations, for higher spatial frequencies. From van Nes and Bouman (1967).

measured contrast sensitivity in central and peripheral retina (Fig. 12) as a function of mean level. They found much less improvement in contrast sensitivity with the shift from rod to cone vision in the peripheral retina. This result suggests there may be something special about foveal cone vision which allows especially high contrast sensitivities.

2.2.3. TEMPORAL FREQUENCY RESPONSES AND MEAN ILLUMINATION

As with spatial frequency, the temporal frequency of a stimulus influences the dependence of sensitivity on mean level. This has been shown by Kelly (1972). Figure 20 from his work demonstrates the sensitivity for gratings of different spatial frequencies over a range of temporal frequencies at several mean levels. At low spatial frequency and low to intermediate temporal frequency, he obtained Weber's Law. At high spatial frequency and low to intermediate temporal frequencies he observed the square root law (indicated as D – R for deVries – Rose). He discovered that, at very high temporal frequencies and low spatial frequencies, sensitivity was more or less independent of mean level. This is the "linear" region, so-called because the visual system appears to be behaving in a linear manner in that the sensitivity for a modulated stimulus is not affected by the presence of different steady levels. Figure 21 also from Kelly (1972) illustrates these results in terms of two spatio – temporal adaptation maps, one at a mean illumination of 50 td and the other at 200 td. A complete theoretical explanation for all these results of Kelly is not available, but we can indicate some basic ideas which may account for them. The effects of spatial frequency have been discussed above. The tendency towards Weber Law behavior of lower temporal frequencies is comparable to Barlow's (1957) results on steeper increment threshold curves with longer duration stimuli. Both results suggest that the adaptation mechanisms in the retina are somewhat sluggish in time course and fail to be as effective on the responses to stimuli which are higher in temporal frequency than 8 Hz as they are on responses to stimuli lower in frequency. This is consistent with Adelson's (1982) observations about the onset of light adaptation and with electrophysiological measurements (Enroth-Cugell and Shapley, 1973a; Baylor and Hodgkin, 1974; Derrington and Lennie, 1982).

The "linear" range observed by Kelly (1972) is somewhat harder to understand. Suppose the gain of the retina for high frequency stimuli is independent of mean level. Still, one would expect that the greater quantal fluctuations at high light levels would cause a reduction in sensitivity. This leads to the inference that the noise which limits performance at high temporal frequencies must not

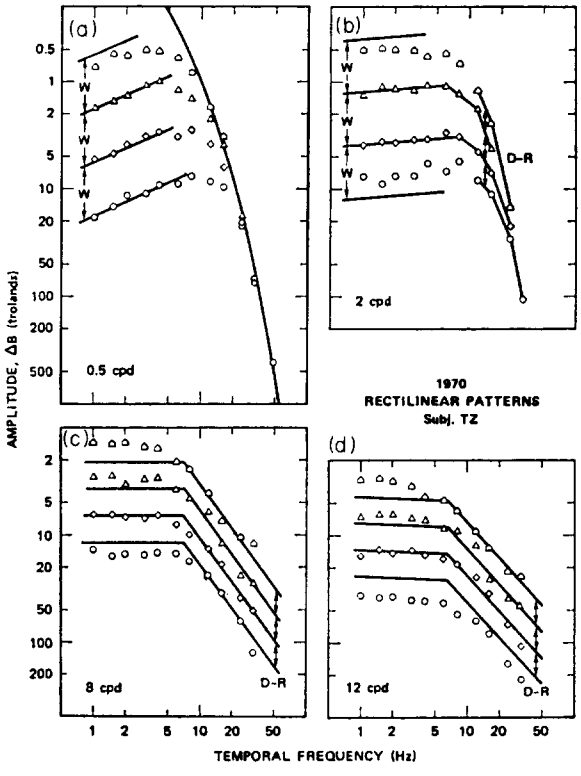

FIG. 20. Threshold illumination as a function of temporal frequency at different spatial frequencies. The threshold illumination ΔB was the amplitude of sine grating which could just be seen on a mean illumination of B td. In each of the four panels of the figure, the four different curves are from measurements at the following mean retinal illuminations: 36 td (arrowheads), 114 td (triangles), 360 td (diamonds), and 1140 td (circles). The test target was a pale-green CRT which subtended 7 deg; it was viewed monocularly and fixated. The gratings were modulated in time with a sinusoidal waveform; the temporal frequency of the modulation is plotted on the horizontal axis. Measurements from four spatial frequencies are shown: 0.5 c deg^{-1}, 2 c deg^{-1}, 8 c deg^{-1}, and 12 c deg^{-1}. Thresholds, which are separated from those at other mean illuminations by a factor which is equal to the ratio of the mean levels, conform to Weber's Law, and have a W written next to the curve. Thresholds which rise like the square root of the mean level are labeled $D-R$ for the *de Vries-Rose* law, synonymous with the square root law. From Kelly (1972).

be quantal noise and must be independent of mean light level. This is not implausible. Most sources of noise in the visual system, e.g. channel opening and closing in neuronal membranes, or spontaneous transmitter release, should have wide-band components. These components may be relatively larger at high temporal frequencies than at low, compared to the light evoked neural shot noise caused by quantal fluctuations. One must suppose that this high frequency noise is immune to the adaptational effects of the mean level of illumination. Perhaps the noise which limits detection of high frequency responses is post-retinal. In any case, a complete and adequate explanation for Kelly's "linear range" requires future research.

3. GAIN AND CONTRAST GAIN IN RETINAL GANGLION CELLS

At the outset of this section on the physiology of retinal adaptation we concentrate on retinal ganglion cells, the output stage of the retina. All information which flows from the retina to the brain about the visual appearance of the outside world passes along the axons of these ganglion cells. The evidence of retinal adaptation in the activity of these neurons allows us to establish a link between the visual, perceptual function of light adaptation and the underlying retinal mechanisms. We will further concentrate our attention on two kinds of retinal ganglion cells in the cat's retina, the X and Y cells (see Appendix 2), because most is known about them. Comparison with the retinas of other species and with human vision will be made frequently. As in the Introduction, we stress the importance of a hierarchy of gain control mechanisms at different sites in the retina. Furthermore, the role of retinal gain controls in making the retina respond to contrast will be made evident.

One can speak about the *gain* of retinal ganglion cells because their impulse rate variation caused by increments (or decrements) of illumination are proportional to the magnitude of the increment (or decrement) over a considerable range of response amplitude. This is illustrated by Fig. 22 (Shapley and Kaplan, unpublished). The stimuli were fine gratings which stimulated the center of the receptive field. (In this initial discussion we will be dealing with the gain of the center only, but will consider the gain of the surround below.)

The ratio of the change in impulse rate with change in stimulus magnitude is the gain, G

$$G = \mathrm{d}R/\mathrm{d}I. \tag{19a}$$

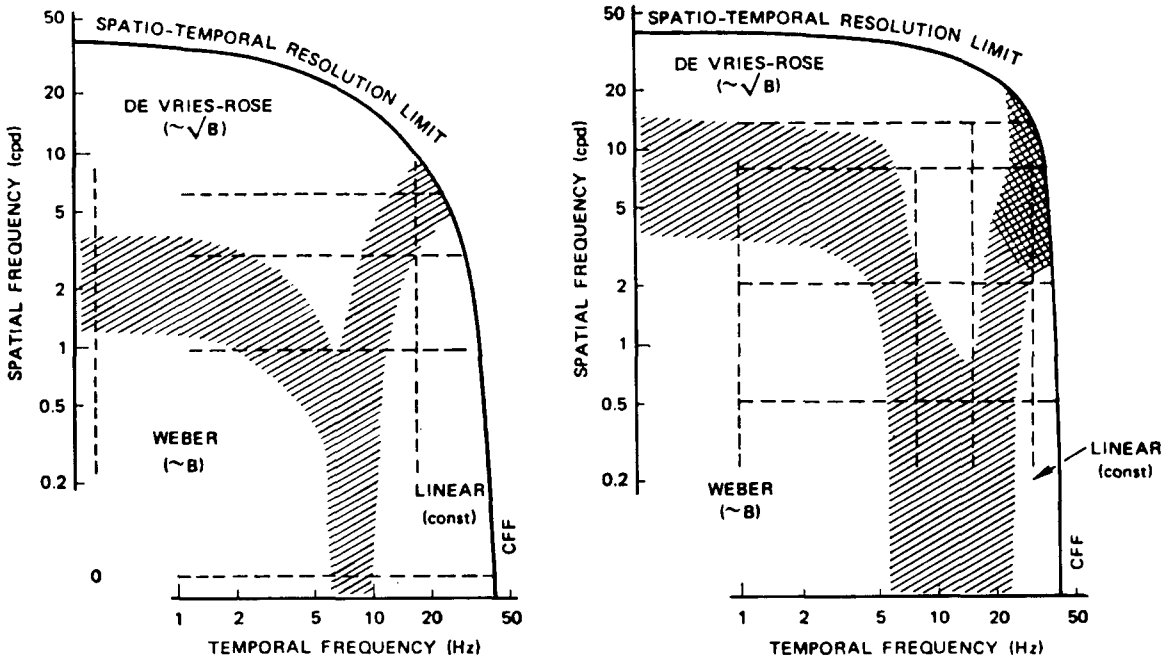

FIG. 21. So-called Spatio – Temporal Adaptation Maps. The adaptation behavior of a human observer is mapped on the plane defined by spatial frequency and temporal frequency, as vertical and horizontal coordinates. Each of these maps only applies to one mean retinal illumination: 50 td for the one on the left, and 200 td on the right. They indicate the slopes of the curves which relate increment threshold to mean illumination, at the different points in the spatio – temporal frequency plane. *de Vries-Rose* is equivalent to the square root law, as explained in connection with Fig. 20. *Weber* means that, in this spatio – temporal region, increase of mean illumination will cause a corresponding and proportional increase in the luminance required to reach threshold: in other words, contrast sensitivity will be constant in the region marked *Weber*. *Linear* means that mean illumination does not affect the modulation threshold; in other words, in the *Linear* region, contrast sensitivity grows in proportion with mean illumination. The shaded areas are transition zones between the other regions. From Kelly (1972).

As Cleland and Enroth-Cugell (1968) showed, the response of the center mechanism of ganglion cells actually depends on the luminous flux falling on the center of the receptive field (see Section 3.5.1.). Therefore, the gain is more properly expressed as,

$$G = \mathrm{d}R/\mathrm{d}F \qquad (19b)$$

and has units impulses/quantum (i/q) of light. The flux F is the stimulus illumination times the stimulus area, or, if the stimulus is larger than the receptive field center, stimulus illumination times the summing area of the center.

The link between visual sensitivity and retinal gain is very strong. Barlow and Levick (1969) presented the argument that three factors determine the ability of retinal ganglion cells to detect a stimulus: (i) the variance in the discharge of nerve impulses; (ii) the time course of the response which determines the optimal integration time over which nerve impulses should be counted by the nervous system in order to determine if a stimulus is present; and (iii) the retinal gain. The gain will be discussed at length subsequently.

The first of Barlow and Levick's three factors, the variance of the discharge of nerve impulses, could conceivably be influenced by photoreceptor "noise", or by fluctuations in neural response due to fluctuations in the number of quanta arriving from the background, or by post – receptoral, intraretinal "noise", e.g. synaptic "noise". The second factor, the optimal integration time in Barlow and Levick's scheme, is the duration of time over which nerve impulses should be counted to obtain the best separation of signal from noise. It is greater than zero because the photoreceptors and

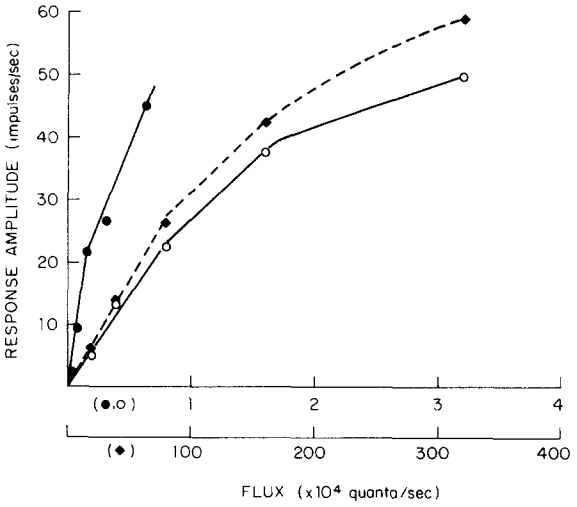

FIG. 22. The proportionality of response with stimulus flux at different mean levels of illumination in cat retinal ganglion cells. These data were taken from an X on-center retinal ganglion cell in a decerebrate, paralyzed cat. The stimuli were drifting sine grating patterns. The response measure was the amplitude of the response at 4 Hz, the temporal frequency of the drift. The spatial frequency was chosen to stimulate the center of the receptive field only; it was 1 c deg^{-1} (cf. Linsenmeier *et al.*, 1982, p. 1175 for a discussion of how gratings can pick out the center). The stimulus was produced on a CRT with a white P4 phosphor and subtended 8 deg by 10 deg on the retina. The center of the receptive field had a summing area of 0.02 deg^2, approximately, and the stimulus display was centered on the middle of the receptive field with a mirror. The mean retinal illumination was controlled with neutral density filters interposed between the CRT and the cat. The responses plotted as filled circles were obtained with a mean retinal illumination of approximately $5 \cdot 10^5$ quanta(507 nm) $(deg^2\ s)^{-1}$. The other two curves were obtained at ten (open circles), and one thousand (diamonds) times higher mean retinal illuminations. The stimulus contrast ranged from 0.02 up to 0.64, but only part of this range is shown for each background level. The stimulus flux was estimated by multiplying the amplitude of the stimulus retinal illumination by the central summing area of the cell. At the highest mean retinal illumination, the stimulus fluxes would be off the scale for the two lower means, and so the data are plotted on a reduced horizontal scale, as indicated in the figure. The two scales were chosen so that equal horizontal distance is equal contrast for the open circles and diamonds, the results from the experiments with the two higher mean illuminations. The approximate equality of the slopes of the response curves for these two sets of results implies equal contrast gain for these two mean levels. However, the main point of the figure is the linear range of response. Shapley and Kaplan, unpublished results.

the following retinal stages have a prolonged response to each quantum of light (see for example Fig. 55), and thus integrating over some finite time allows one to add up the neural consequences of each quantum absorption. But the optimal integration time is less than infinity because the retina's response to each quantum eventually dies away, so there is no point integrating the neural response beyond the time at which the signal outweighs the noise. Therefore, there is some optimum time, about 100 ms, over which the nervous system should count nerve impulses in order to detect that a stimulus has been presented to the ganglion cell.

Barlow and Levick measured how these three factors depended on background light in order to discover which was most important in controlling the ability of ganglion cells to detect a stimulus-initiated signal. The motivation for this analysis was that if these three factors controlled ganglion cell performance they should also contribute to behavioral sensitivity. They demonstrated that the ganglion cell gain was reduced dramatically by background illumination even though the variance of the impulse discharge and the optimal integration time for retinal responses changed rather little with background. This is illustrated in Fig. 23. They therefore found compelling evidence for the proposition that the main link between visual adaptation and retinal adaptation is the control of retinal gain by steady background illumination. Further work by Derrington and Lennie (1982), on the relative constancy of the variability of the maintained discharge with mean level, has strengthened this conclusion.

3.1. Gain Control in the Scotopic Range

3.1.1. GAIN AND BACKGROUND

All of the X and Y cat ganglion cells studied so far have received input from retinal rod and cone pathways (Daw and Pearlman, 1969). This gives an experimenter the opportunity to study the gain controls for the rod and cone pathways in an individual cell. Representative results on the gain of the receptive field center as a function of background in the scotopic range are given in Fig. 24 from Enroth-Cugell and Shapley (1973a). The stimuli were small spots placed in the center of the receptive field and modulated with a slow square wave time course. The gain [the i/q ratio equivalent to the gain as in equation (19)] is plotted on double-logarithmic coordinates vs background

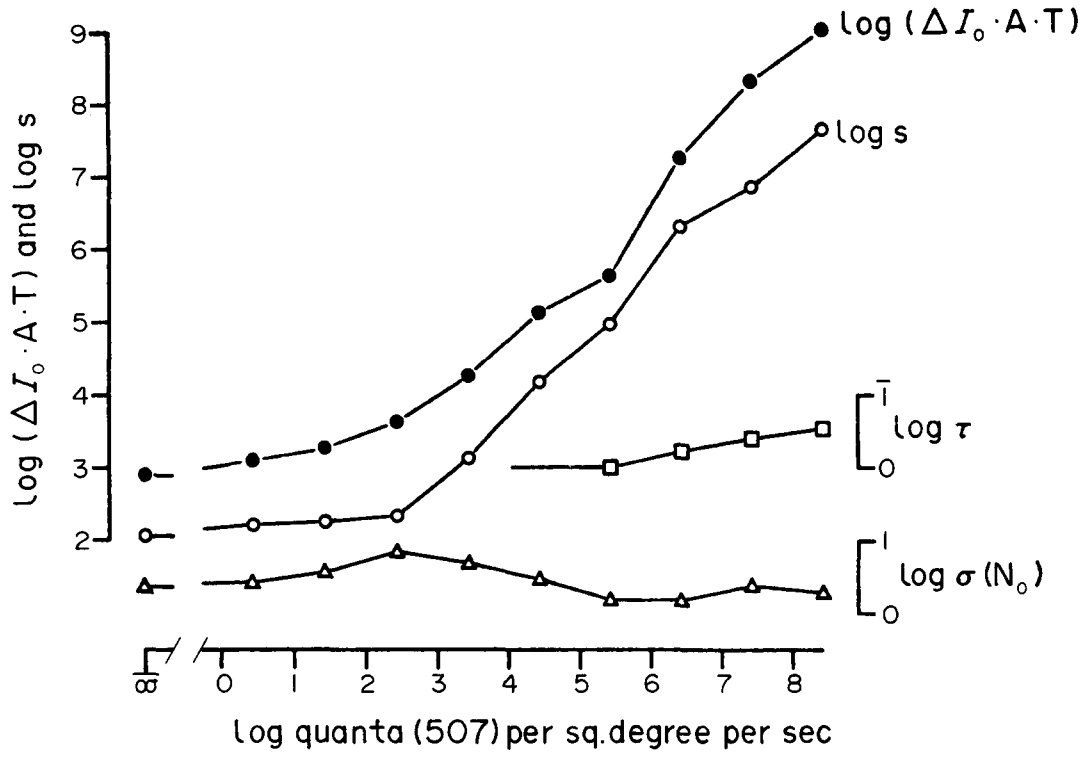

FIG. 23. Three factors limit the reliable detection of light by retinal ganglion cells in the cat, and the dominant factor is the gain. The incremental "threshold" (labeled $\Delta I_0 \cdot A \cdot T$, and having units of quanta) of a ganglion cell is plotted here as filled circles against the background retinal illumination. The open circles are the quantum – spike ratios at different backgrounds. The quantum – spike ratio is the reciprocal of what we have called *gain*. The empty squares are the estimated optimal averaging time; the empty triangles are the standard deviations of the impulse number distributions. The vertical coordinates were chosen so that changes in log "threshold" are the sum of the changes in the logarithms of the underlying factors. Clearly the quantum – spike ratio, the reciprocal of the gain, is the dominant factor. From Barlow and Levick (1969).

illumination. It is seen that the gain is constant for backgrounds below a critical level, and that above this level the gain declines almost inversely with background. Actually, the typical behavior of retinal ganglion cells is described by the following equation:

$$G_R = G_{RO} / (1 + I_B / I_{RO})^P \qquad (20)$$

G_R is the gain for rod-driven ganglion cell activity. G_{RO} is the dark-adapted gain for the rod pathway. I_{RO} is the illumination at which the gain has dropped by 2^{-P} and is referred to as the "transition illumination". P is the exponent of the term in the denominator which depends on the action of the gain control; P is also the slope of the gain vs background curve on log – log cooordinates. For the cells in the Enroth-Cugell and Shapley (1973a) study, the average value of P was 0.9. This is very close to Weber's Law which would have an exponent of 1 instead of 0.9 in the denominator. At mean retinal illuminations above $5 \cdot 10^8$ quanta(507 nm) $(deg^2\ s)^{-1}$ the gain of the rod pathway declines more steeply than equation 20 would indicate, because of "rod saturation" (Lennie *et al.*, 1976).

The value of the transition illumination I_{RO}, varied over a range of two log units, from about 300 quanta(507 nm) $(deg^2\ s)^{-1}$ up to $3 \cdot 10^4$ quanta$(deg^2\ s)^{-1}$. The ganglion cells with the largest centers, presumably large peripherally located Y cells, had the lowest values of the transition illumination.

Barlow and Levick (1976) and Barlow (1977) have suggested that the transition illumination, I_{RO}, may be analogous to the "dark light" inferred from the plateau in the human psychophysical increment sensitivity at low backgrounds. There is a similarity,

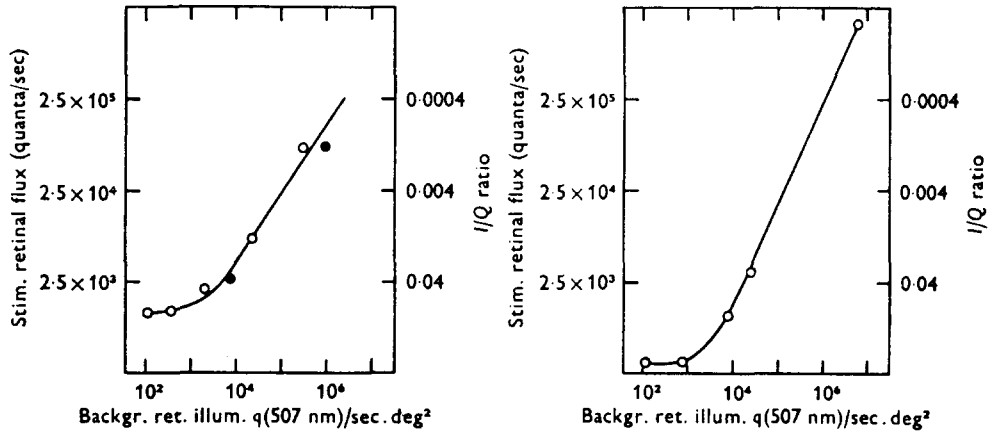

FIG. 24. Gain vs background illumination, in the scotopic range, for cat retinal ganglion cell centers. Retinal flux [in units of quanta(507 nm) s^{-1}] required for a small central stimulus to elicit a criterion response (30 impulses s^{-1}) is plotted vs the retinal illumination of a 12 deg concentrically located background. The gain in impulses/quantum (i/q) is indicated also on the right hand vertical scales. The latter quantity was calculated by multiplying the stimulus retinal illumination by the area of the stimulus, 0.03 deg^2, and by a factor of 1/3, the estimated fraction of quanta incident on the retina which were absorbed. In the left hand panel, the filled circles are for white stimuli on a white background; the open circles are for blue – green stimuli on a red background, to demonstrate rod isolation. In the right panel all the points are for white on white. The results in the left panel are from an on-center ganglion cell; the results in the right panel are from an off-center cell. From Enroth-Cugell and Shapley (1973a).

because both the "dark light" and the transition illumination are needed to account for observed plateaus: the background illumination must exceed I_{RO} for the gain to drop from its dark adapted value in ganglion cells, while the background illumination must exceed I_D, the "dark light", for the psychophysical sensitivity to drop from its dark adapted value. However, the functional difference outweighs the apparent similarity. The transition illumination is involved in gain control; the "dark light" (either estimated from psychophysics or from physiological experiments) sets the noise level of the retina in the dark. This argument is supported by the estimated values of "dark light" and the transition illumination, which are quite different. The "dark light" of cat ganglion cells was estimated by Barlow *et al.* (1971), as follows. Based on the value of the maintained discharge in the dark, and the slope of the stimulus – response curve obtained in the dark, these authors estimated the magnitude of the light flux which would have been required to generate the maintained discharge in the dark, and called this value the "dark light". They found a wide variation in this estimate of the "dark light". However, taking their highest value, the "dark light" was equivalent to about 100 quanta s^{-1} retinal flux. For a cell with a small center, say about 0.1 deg^2 in area, this would be produced by 10^3 quanta$(deg^2\ s)^{-1}$ retinal illumination; for the largest cells it would be produced by about 3 $q(deg^2\ s)^{-1}$. These values for the feline "dark light" are too low, by at least a factor of ten, for the "dark light" to be equivalent to the transition illumination in cat retinal ganglion cells. Rather, some criterion amount of voltage or current or substance in a retinal cell, much larger than that caused by "dark light", must be exceeded, and then the gain control of adaptation begins to act. As argued in Section 2.1.1.2., "dark light" probably limits sensitivity by providing a noise, the "dark noise", against which a signal must be picked out, rather than by setting the gain.

3.1.2. GAIN AND DYNAMICS

There are dynamic consequences of adaptation which are hidden in the simple picture of Fig. 24. As shown in Fig. 25, the time course of response of the receptive field center varies with adaptation level, as found both by Yoon (1972) and by Enroth-Cugell and Shapley (1973a). The nature of the change is that the response of the center to an incremental step of illumination becomes more transient, the more light adapted the cell is in the

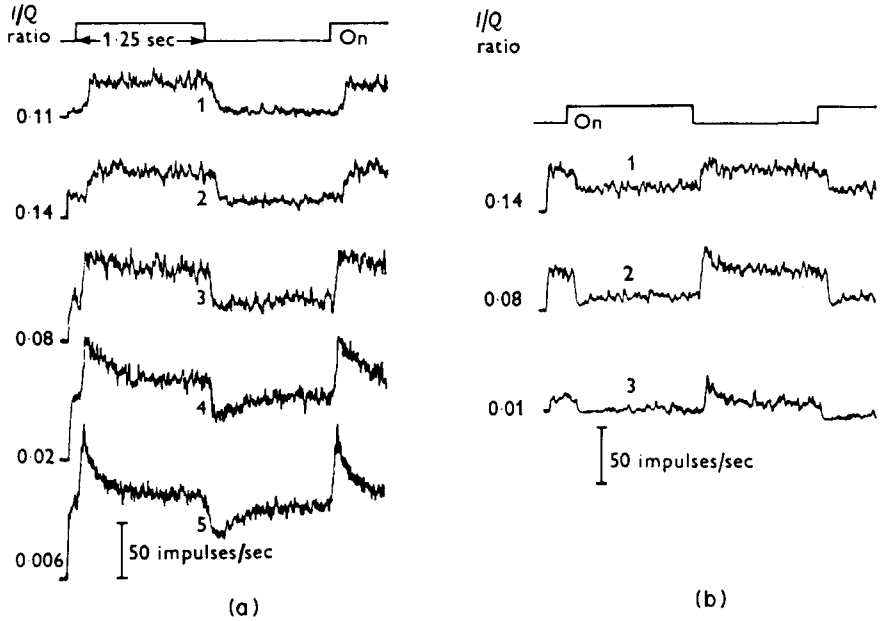

FIG. 25. Time course of (rod-driven) square wave responses at different adaptation levels in cat retinal ganglion cells.
(a) The responses (averaged over many stimulus cycles and smoothed) of the receptive field center of an on-center cell at five different levels of background retinal illumination: (a)1—50 quanta(deg^2 s)$^{-1}$, (a)2—$6 \cdot 10^2$ quanta(deg^2 s)$^{-1}$, (a)—$6 \cdot 10^3$ quanta(deg^2 s)$^{-1}$, (a)4—$9 \cdot 10^4$ quanta(deg^2 s)$^{-1}$, and (a)5—$5 \cdot 10^5$ quanta (deg^2 s)$^{-1}$.
(b) A similar result is shown for an off-center cell at three background illuminations: (b)1—100 quanta (deg^2 s)$^{-1}$, (b)2—$3 \cdot 10^2$ quanta(deg^2 s)$^{-1}$, (b)3—10^3 quanta (deg^2 s)$^{-1}$. Note that in the cat 1 deg^2 = 0.048 mm^2, approximately.
For each response the *i/q* ratio, the gain, is shown at the left of the averaged response histogram. The time courses of the responses and the *i/q* ratios at the top of each column were the same as those obtained in total dark adaptation. The drop in gain with increase in background illumination goes hand in hand with the change in time course observed in the histograms. From Enroth-Cugell and Shapley (1973a).

scotopic range. This finding applies to off-center as well as to on-center cells, and to X as well as Y cells (Jakiela *et al.*, 1976).

This change in time course of response with adaptation is related to another observation: the steepness of the decline in gain with background depends on the temporal pattern of the stimulus (and response). For example, the results in Fig. 24 were derived from measurements of the peak response to an incremental step of illumination on a background. One would obtain rather similar data from measurements of the gain of the response to a sinusoidally modulated small spot for temporal frequencies of 2 Hz or less. In this case, either peak-to-peak impulse rate modulation or the amplitude of the sinusoid which is the best approximation to the neural response are two response measures which would give the same dependence on background. Similar measurements at 8 Hz or above have a shallower dependence on background, i.e. the exponent P in equation (20) would be around 0.6 for intermediate temporal frequencies of modulation (between 3 and 10 Hz; Enroth-Cugell and Shapley, 1973a; Derrington and Lennie, 1982). The responses to temporal frequencies above 16 Hz suffer almost no attenuation in amplitude with increases in mean level; for such high temporal frequencies, the exponent P in equation (20) is near zero (Shapley *et al.*, 1983). Previously, Sakmann and Creutzfeldt (1969) and Barlow and Levick (1976) also observed shallow slopes of gain vs background curves with brief incremental stimuli. The responses to such brief pulses of light, which contain a wide range of temporal frequency components, follow a gain vs background curve which obeys equation (20) with an exponent of about 0.6 on the average. However, as presented earlier, the gain of the peak of the response to a prolonged flash has a steeper dropoff of gain with background; the exponent in this case is about 0.8, according to Barlow and

Levick (1976; however, cf. Cleland and Enroth-Cugell, 1970; and Enroth-Cugell and Shapley, 1973a in which $P = 0.9$). The steeper slope for the longer flash makes sense because the prolonged flash can be viewed as the sum of predominantly low frequency components, which tend to be attenuated more by increases in background (adapting) light.

There is a good reason to consider the effects of adaptation on different temporal frequency components. As mentioned in Section 1.2.2., the response of the retina to stimulation by light should be considered to be a *functional,* or *transformation,* of the stimulus. If $L(t)$ is the stimulus, then the response is $R = R\{(t,L(t)\}$. That is, the response R depends on time and, furthermore, the response at time t depends on the value of the stimulus at time t and also the values of the stimulus in the past. This is a formal way of expressing what is well known about the retina: it has a finite integration time, and there are sluggish gain controls, in the receptors and the network, which modify the retina's response contingent on the past history of illumination. There is a standard mathematical apparatus for analyzing functionals; it is called systems analysis. A particularly useful subset of this apparatus is linear systems analysis, a mathematical technique for analyzing functionals which are linear, i.e. systems in which the response to two separate inputs is simply the sum of the responses to each of the inputs presented alone. In analyzing a linear system, sinusoids are the stimulus of choice because they pass through such a system unchanged in waveform, though scaled in amplitude and shifted in phase. While the retina is decidedly not linear under all conditions, it may behave like a linear system around an operating point set by the mean level of illumination, and therefore the retinal responses to sinusoids of different temporal frequency serve to provide a good quantitative description of how the retinal functional behaves at different mean levels of illumination.

The different effects of background on the responses of ganglion cells to different temporal frequencies can be explained by a theory in which the centerpiece is a gain control which is a nonlinear negative feedback (Enroth-Cugell and Shapley, 1973a). In order to account for the increased transience of square wave responses in the light adapted state (Fig. 25), one must assume in this theory that the gain control has a somewhat more prolonged integration time than the photoreceptors. According to this view, the decay seen in the ganglion cell's step response is due to the incremental increase in the value of the gain control signal due to the step stimulus.

3.1.3. Adaptation of the surround mechanism

The surround of the receptive field also adapts to background light in the scotopic range. Figure 26

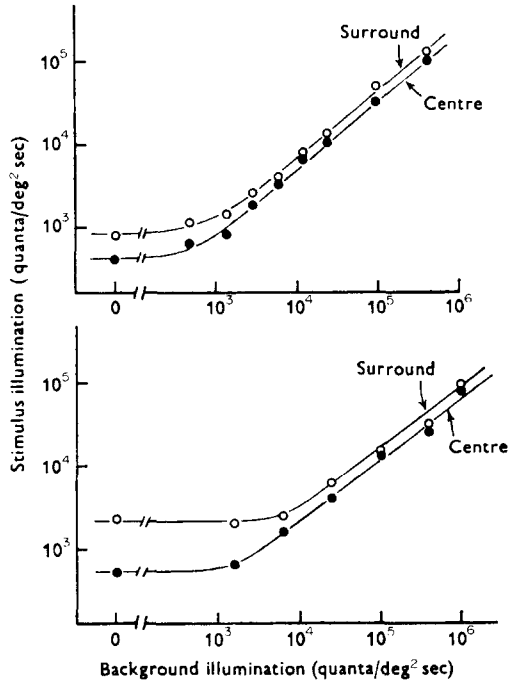

FIG. 26. Comparison of center and surround gains in cat retinal ganglion cells. Plotted vertically is the stimulus retinal illumination required to evoke criterion responses as a function of the background retinal illumination plotted on the horizontal axis. Upper and lower panels are from two different on-center X cells. For the centre responses (filled symbols) the criterion was ten extra impulses evoked during a one second presentation of an increment of the illumination of an optimum spot which just filled the center. For the surrounds (empty symbols) the criterion was determined from stimulus – response curves for optimum spots and diffuse illumination. The criterion illumination for the surround was such that the response to diffuse illumination of this amount was ten impulses less than the response to an optimum spot of the same retinal illumination. Retinal illuminations are given as quanta (507 nm) $(\text{deg}^2\ \text{s})^{-1}$ incident on the retina, under the assumption that 75% of the quanta incident on the cornea reach the retina. Rod isolation was insured by the use of blue – green stimuli on red backgrounds. From Enroth-Cugell and Lennie (1975).

from Enroth-Cugell and Lennie (1975) reveals the kinds of surround adaptation which are seen. In these experiments, the surround response was estimated by subtracting the center response from that obtained by illumination of both center and surround. The data are the incremental illumination required to elicit a criterion surround response; thus these curves may be interpreted as indications of changes in surround gain with background illumination. In the upper panel, the surround and center begin to reduce their gains together at roughly the same level of background. In the lower panel the surround starts off with a lower dark-adapted gain, but reduces its gain with background at a higher level than the center. The result is that in both cases the center and surround have roughly the same gains (integrated over their respective total summing areas) in the Weber-Law, light adapted scotopic range. But there are differences across the population of retinal ganglion cells in the relative gain of center and surround in the low scotopic range (see also Kaplan *et al.*, 1979).

The results of the well-known investigation of Barlow *et al.* (1957) are sometimes interpreted to mean that the gain of the surround goes to zero in total dark adaptation. This, however, is not precisely what Barlow *et al.* found. Their results on the dependence of gain on area at different backgrounds implied that the ratio of the gain of the center to the gain of the surround increased in total dark adaptation. However, the extent of the increase in the center – surround ratio was not determined in their study. It was later shown by Enroth-Cugell and Lennie (1975) and by Kaplan *et al.* (1979) that this ratio may increase from a value of 1.2 in the light adapted state to as much as 3 in the dark adapted state. That is, the surround is relatively weaker compared to the center in the dark, but it is not gone. In fact, the gain of the surround is always maximal when the retina is dark adapted (see Fig. 26). In other words, as the background level is increased from total darkness, the center gain usually is reduced at a lower level of background than is the surround's gain.

There is a methodological reason for the differences in conclusions about the strength of the surround in the dark. Barlow *et al.* (1957) used auditory threshold for an "off-response" to measure the magnitude of the response of the surround in on-center cells. This method has the disadvantage that, in these cells, the surround response consists of sustained inhibition when the surround mechanism is dark adapted (Enroth-Cugell and Lennie, 1975). The presence and magnitude of the "off-response" is associated with the adaptation level, just as the magnitude of the center's transient overshoot depends on the center being somewhat light-adapted (see Fig. 25). Enroth-Cugell and Lennie (1975) and Kaplan *et al.* (1979) measured magnitude of inhibition as an indicator of surround response strength, and they did it with objective averaging techniques. Their work reveals that there are marked variations across the population of ganglion cells in the degree to which background illumination affects the ratio of the center and surround gains (see Fig. 26). The finding of variability in the degree of center – surround balance in the dark has also been reported by Barlow and Levick (1976).

The fact that background light in the scotopic range can affect the gains of center and surround differently implies that, in this range, the gains of these receptive field mechanisms are controlled at a site (or sites) in the retina more proximal than the photoreceptors. If the only site of gain reduction were the photoreceptors, one would observe that the gains of center and surround would begin to drop at the same background level. There is good evidence that at higher backgrounds some of the reduction in gain in the mammalian retina is due to photoreceptor adaptation (Sakmann and Filion, 1972; Valeton and van Norren, 1983). These observations are consistent with our assertion in the Introduction that there is a hierarchy of gain controls.

The ratio of the total integrated gains of center and surround in the light-adapted state is approximately 1.2 for a large population of retinal ganglion cells (Linsenmeier *et al.*, 1982). There is considerable variance in this ratio among the ganglion cell population. In any one cell, the ratio is approximately constant from $10^{-2.5}$cd m^{-2} background luminance on up (Enroth-Cugell and Lennie, 1975). Thus, in cat ganglion cells the balance between center and surround is established in the low- to mid- scotopic range and is invariant with adaptation level throughout the mid- to high-scotopic and photopic ranges.

3.2. Gain Control in the Photopic Range

The inputs from cones to cat ganglion cells can be isolated from those from rods by means of Stiles' two-color technique (see Wyszecki and Stiles, 1967, p. 572). When this is done, it is found that cone signals have a constant gain over the range that the gain of rod signals drops by a factor of one thousand or more (Enroth-Cugell *et al.*, 1977a), from total dark adaptation to the high scotopic range. This can be seen in Fig. 27. Plotted there are the threshold illuminations for criterion cone-driven and rod-driven responses to be elicited by a test stimulus on a blue background, as a function of the level of background. For a single ganglion cell one obtains a two-branched gain vs background curve which is reminiscent of the two-branched, psychophysical sensitivity vs background curves (Fig. 8). This indicates that the separate gain control of rod and cone signals is achieved by the retina prior to the ganglion cells, and that rod and cone signals are kept segregated at least up to the points at which the gains are set in the parallel "rod" and "cone" pathways through the retina. These results are found for receptive field centers of both on- and off-center cells, and for both X and Y cells. This has the added implication that rod and cone signals are segregated in both the X and Y pathways until the gain is set.

The results in Fig. 27 imply independence of adaptation mechanisms for the rod and cone pathways to the ganglion cells in the cat. However, Nelson (1977) found that rod signals are coupled into cones. His work led him to the conclusion that the main pathway of rod signals to horizontal cells was through the cones. Presumably a similar conclusion would apply to the bipolar cells. That is, rod signals should travel through cone bipolar cells because of the large amount of coupling of rod signals into cones. This poses a problem, namely how can the cone and rod signals adapt separately when they are carried by the same interneurons? One possible explanation is that all adaptation may take place in receptors, but that explanation has

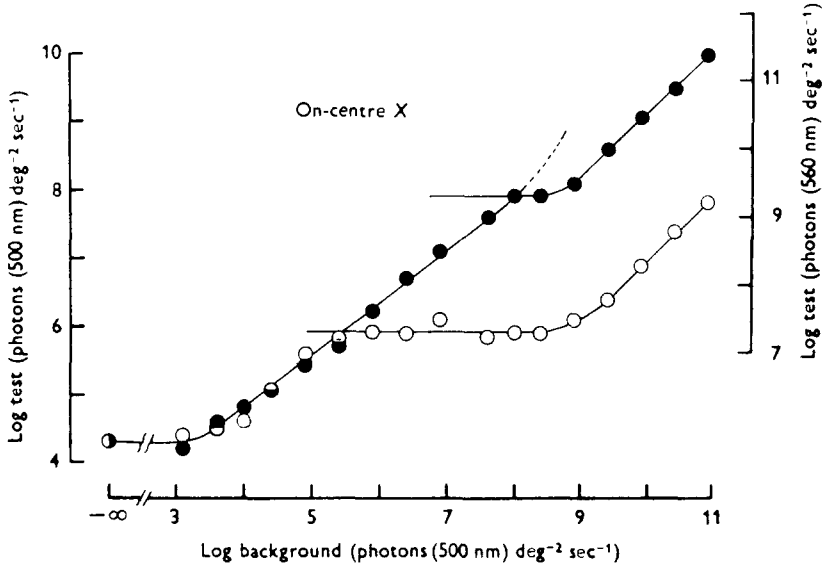

FIG. 27. Gain for the rod and cone systems in a cat retinal ganglion cell. The illumination required to evoke a criterion response is graphed vs the background illumination. The points plotted as filled symbols were obtained with a blue – green stimulus light on a blue – green background, and were taken to represent the gain of the rod pathway. Their values are given by their heights on the left vertical axis, in equivalent quanta of a monochromatic 507 nm light. The empty symbols are the criterion illuminations for a red spot of light on the same blue – green background, and are interpreted as indicating the cone pathway's gain. The response criterion was just-audible synchrony of the cell's firing rate with the 4 Hz square wave modulation of the small stimulus spot (0.2 deg diameter), which was located in the middle of the receptive field. In this figure, retinal illuminations are referred to quanta at the cornea, before losses in the eye. From Enroth-Cugell *et al.* (1977a).

other problems with the spatial interactions which are involved in adaptation. At present we can only raise this issue as one which must be resolved by future research.

The gain in the photopic range is proportional to $1/I_B$, on the average, above a critical background illumination I_{C0} (Daw and Pearlman, 1969; Enroth-Cugell *et al.*, 1977a). Thus a description of the gain for the cone system in Fig. 27 is (with G_{C0} the dark adapted gain of the cone signals, I_{C0} the illumination at which the gain is halved):

$$G_C = G_{C0}/(1 + I_B/I_{C0}) \tag{21}$$

and the gain of the ganglion cell would be

$$G = G_C + G_R \tag{22}$$

if we assume that rod and cone signals are simply added (Enroth-Cugell *et al.*, 1977b).

The time course of the ganglion cell's response undergoes another change at the rod – cone break. This is illustrated in Fig. 28 from Enroth-Cugell *et al.* (1977a). When the background puts the cell into the mesopic range, but is less than I_{C0}, the response to a small increment of illumination which only stimulates the cones is sustained, like the response to a stimulus which only stimulates the rods in the dark adapted state. As the background is increased above I_{C0}, the step response becomes more transient, recapitulating the rod results. Thus, the response to a step of light in a cat ganglion cell is sustained in total dark adaptation, becomes more transient throughout the scotopic range, becomes sustained again just above the rod – cone transition, then becomes more transient again in the mid- to high-photopic range. The remarks above about the explanation of the effects of adaptation on response time course in the scotopic range should also apply to the photopic range. One should expect that a nonlinear feedback is the mechanism which links gain reduction with the change in response time course.

Both in the scotopic and photopic ranges, the incremental step responses of Y cells decay at a faster rate than X cells when both cell types are in the Weber Law regions of the gain vs. background curve and have suffered the same drop in gain with respect to their dark adapted values (Jakiela *et al.*, 1976; Enroth-Cugell *et al.* 1977a). If the time course of the step responses are a result of the action of retinal gain controls, these results indicate that the gain control mechanism for X cells is not the same as for Y cells. It suggests that the X cell's gain is controlled by a mechanism with a longer time constant than the gain control for the Y cell. Such a concept of two gain controls, one for X and one for Y, arises also in the consideration of the spatial properties of retinal gain controls, and will be discussed below.

3.3. Contrast Gain

We have presented the argument earlier that one purpose, perhaps the most important purpose, of light adaptation is to maximize the visual contrast sensitivity and to keep it constant as the background or mean level varies. One of the determinants of contrast sensitivity is contrast gain, or how large a response is produced by a given amount of contrast. The contrast gain G_{con} is the background illumination I_B times the Gain G as defined in equation (19a), so

$$G_{con} = I_B \cdot (G_R + G_C) \tag{23a}$$

$$G_{con} = G_{R0} \cdot I_B/(1 + I_B/I_{R0})^P + G_{C0} \cdot I_B/(1 + I_B/I_{C0}). \tag{23b}$$

If the gain is expressed as impulses/quantum as in equation (19b), then the contrast gain is equal to gain · background flux. In either case, the contrast gain has units of [impulses s^{-1}][contrast]$^{-1}$, or may sometimes be expressed as [impulses s^{-1}][percent contrast]$^{-1}$ if contrast is given in percentage rather than as a fraction.

A graph of the contrast gain in single ganglion cells is shown in Fig. 29 (Shapley *et al.*, 1983). The contrast gain increases steadily in the scotopic range, then levels off (or sometimes may even drop somewhat) as the cell enters the photopic range. X and Y cells have a similar dependence of contrast gain on mean level.

The dependence of contrast gain on mean level is related to the dependence of gain on mean level. In the range of backgrounds in which I_B is much

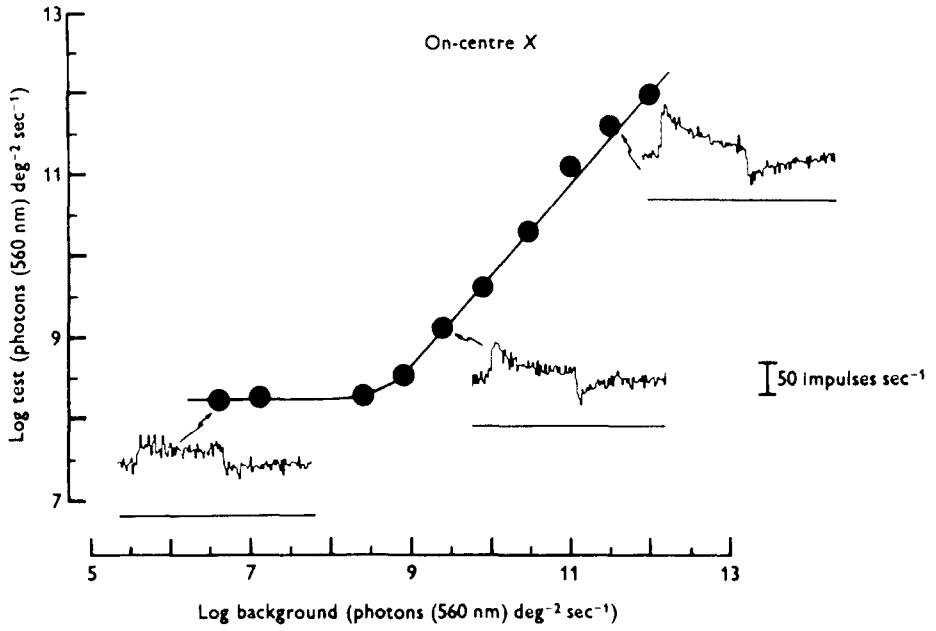

FIG. 28. Change of time course and gain in cone-driven X ganglion cell responses as a function of increasing background illumination. The curve plots the retinal illumination (referred to the cornea) required to evoke a criterion peak response of 35 impulses s^{-1} above the mean impulse rate. Stimuli were 0.2° diameter red spots located at the midpoint of the receptive field center, and were modulated in a square wave manner at 0.5 Hz. The background was blue – green, in order to suppress the rods and yield an isolated cone-driven response. From Enroth-Cugell *et al.* (1977a).

larger than the transition illumination I_{RO}, and the response is driven by rods, the slope of the contrast gain curve on log – log coordinates is $1-P$, when the slope of the gain curve is $-P$. Thus, Weber's Law, when the slope of the gain curve is -1, implies a slope of the contrast gain curve of zero. When P is greater than 1, in the region of rod saturation, the slope of the contrast gain curve becomes negative, and the contrast gain actually drops.

3.4. Effect of Adaptation on the Size of the Receptive Field Center

There is evidence that the size of the receptive field center in cat retinal ganglion cells is practically constant over wide ranges of mean level or background level of illumination. Smaller and larger spots which fall completely within the central-summing area of a receptive field have almost identical gain vs background curves, as implied in Fig. 30 (Cleland and Enroth-Cugell, 1968). In the figure, gain vs area was measured for a single ganglion cell at several different scotopic backgrounds. The parallelism of the curves suggests that all these spots of different sizes were affected to the same extent by the increase in background illumination. Cleland and Enroth-Cugell (1968) also showed that the distribution of luminous flux among several spots produced exactly the same response in magnitude and time course as the same luminous flux concentrated in a single spot, as long as all stimulus spots were placed at equally sensitive points in the receptive field center. This led to the concept of a single center-mechanism or central summation pool within which neural signals are added; the evidence of Fig. 30 suggests that, at least under some experimental conditions, the receptive field center adapts as a unit at a site in the retina at which the center's signals have been pooled. This finding applies to the receptive field in the photopic as well as the scotopic range (Enroth-Cugell *et al.*, 1977a).

However, there are some data indicating some variation of receptive field center size with mean level. All these results have been obtained from experiments which used sinusoidal grating stimuli to estimate the size of the center. The first result

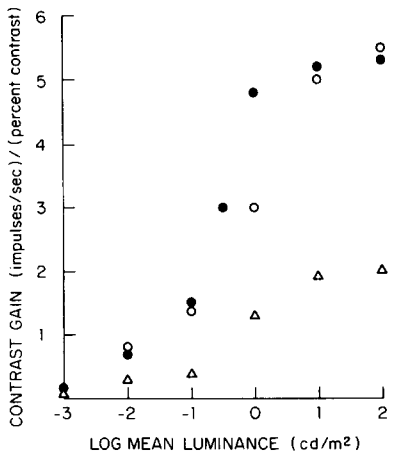

FIG. 29. Contrast gain in cat retinal ganglion cells as a function of mean luminance. The stimuli were drifting sine gratings, presented at (Rayleigh) contrasts from 0.02 (2%) up to 0.64 (64%). The temporal frequency of the drift was 4 Hz. Mean stimulus luminance was varied over a four to five log unit range with neutral density filters. The CRT screen had a white P4 phosphor. The photopic luminance of the screen was measured; the scotopic equivalent luminance is approximately twice the photopic for this light. Artificial pupils, 3 mm in diameter, were used. With such a pupil, 1 cd m^{-2} produces a retinal illumination of approximately $4 \cdot 10^6$ quanta(507 nm) $(deg^2\ s)^{-1}$ on the cat's retina. The slope of the linear portion of the response – contrast curve provided an estimate of the contrast gain in impulses s^{-1} $[contrast]^{-1}$. These are the results from three different on-centre X cells; off-center and Y cells gave essentially similar results. The responses at the highest mean luminance used, 100 cd m^{-2}, were cone-driven, but responses at lower mean levels were rod-driven. From Shapley *et al.* (1983).

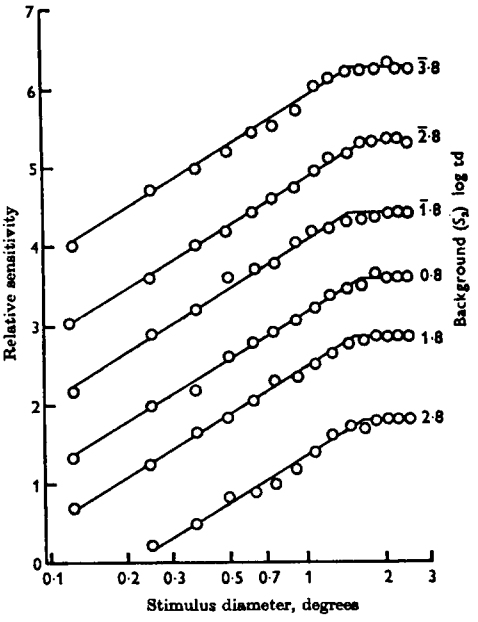

FIG. 30. Area – sensitivity curves at several background luminances in cat ganglion cells. In this figure the label on the ordinate, "sensitivity", is used to mean what we have called "gain". The diameter of a circular spot centered on the receptive field was varied in steps and the luminance required to produce a criterion response was measured at each value of the diameter. The temporal modulation was a 4 Hz sinewave. The criterion response was just-audible synchronization of the cell's discharge with the stimulus modulation. The sloping and flat portions of these curves intersect at a diameter denoted D_t which is taken to be the diameter of the central summing area of the receptive field center. In these experiments the value of D_t was approximately constant across background level. Zero on the "Relative sensitivity" scale corresponds to a stimulus amplitude modulation of approximately 10^9 quanta(507 nm) $(deg^2\ s)^{-1}$ retinal illumination. The stimulus depth of modulation was 0.6. The background was a circular spot with a diameter of 8.5 deg. The retinal illuminations of the background are given in log td, but these are "cat td", and 1 "cat td" is equivalent to approximately $6 \cdot 10^5$ quanta(507 nm) $(deg^2\ s)^{-1}$ on the retina. Thus the lowest background illumination used in these experiments was approximately $3.6 \cdot 10^3$ quanta$(deg^2\ s)^{-1}$ on the retina. The luminance required, through the 4 mm diam artificial pupil used, to produce this lowest background illumination was $5 \cdot 10^{-4}$ cd m^2. The brightest background was 10^5 brighter. This was an on-center cell, presumably a Y-cell. From Cleland and Enroth-Cugell (1968).

is that of Enroth-Cugell and Robson (1966) who found a two-fold reduction in the receptive field center diameter of an X cell when the mean level was varied from $5 \cdot 10^{-4}$ to 16 cd m^{-2}, apparently most of the change taking place at the low end of the range of mean levels, as seen in Fig. 31. Similar results have recently been obtained by Derrington and Lennie (1982) who report a 32% reduction in center diameter when the average illumination was increased from $2 \cdot 10^{-3}$ to 200 cd m^{-2}. Since these mean levels span the rod-cone transition in the cat which occurs around 10 – 100 cd m^{-2} in white light (when a 3 mm pupil diameter is used; cf. Enroth-Cugell *et al.*, 1977a), one would wish to have more detailed knowledge about how the center size varied with average light level before formulating an explanation of the center's contraction in the light. However, the results of Enroth-Cugell *et al.* (1977b) on the relative sizes of the receptive field center when it is driven by rods or driven by cones suggest that the transition from rods to cones does not produce the center's contraction. Rather, it appears more likely that the center size is relatively constant from mid-scotopic to mid-photopic levels, and only becomes larger in the low scotopic range. This inference should be tested further since it may be

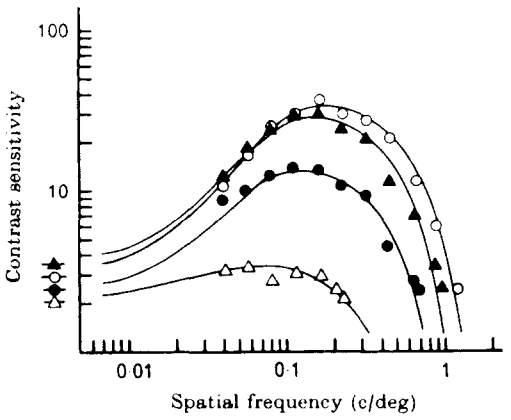

FIG. 31. The effect of mean luminance on the dependence of a ganglion cell's contrast gain on spatial frequency. Four mean luminance levels were used: 16 (empty circles), 0.5 (filled triangles), $1.6 \cdot 10^{-2}$ (filled circles), and $5 \cdot 10^{-4}$ cd m^{-2} (empty triangles). The highest mean luminance corresponds to approximately 10^8 quanta(507 nm) (deg^2 s)$^{-1}$ retinal illumination. What we have called "contrast gain" is labeled "contrast sensitivity" in the figure. A 3.5 mm diameter artificial pupil was used. The criterion response was audible impulse rate modulation at the drift rate of the grating, 4 Hz. The data were fit with smooth curves calculated from a Difference of Gaussians model, where the smaller Gaussian represents the receptive field center. The estimated diameter of the center changed by about a factor of two over the range of mean luminances studied. The cell studied here was an on-center X cell. From Enroth-Cugell and Robson (1966).

a way to forge a stronger link between the microcircuitry of the retina and its function. In any case, the data on the relatively small changes of receptive field center size with background or mean level serve to reinforce even more strongly the conclusion that signals from different parts of the receptive field center adapt together and with approximately the same slope on the gain vs background curve.

3.5. Adaptational Pooling and Receptive Field Size

3.5.1. SIGNAL POOLS AND ADAPTATION POOLS

Investigation of the spatial summation of desensitization by adapting lights is important for an understanding of the functions and mechanisms of light adaptation. As we discussed in Sections 1.2.1. and 2.1.6. in connection with the ideas of Whittle and Challands, Land and McCann, Rushton, and Westheimer, the spatial summation of retinal adaptive signals has been inferred from psychophysical experiments. Furthermore, questions about the influence of background light at one place in the visual field on the response to a test light at another place have been raised in theories of vision by (among others) Hering (1920), Helson (1964), Sperling (1970), and Grossberg (1981). A wealth of physiological results supports the concept of adaptational pooling, but indicates that the pools are smaller than most theorists have expected. Furthermore, there is evidence for pooling of adaptive signals over the entire center of the receptive field, and also more localized adaptive pooling in sub-regions of the receptive field center and surround. However, in the cat, steady illumination of the surround has little or no effect on the gain of the center.

In the cat retina, the gain of the center mechanism of the receptive field of a retinal ganglion cell is determined by the sum of all the steady light falling on the center, and only on the center. This has been proven by a number of different experiments which are consistent with each other. The first was the experiment of Cleland and Enroth-Cugell (1968), the results of which are shown in Fig. 32. In this experiment, the *signal summation area* was measured with stimulus disks of various areas. Illumination was adjusted to give a constant criterion response. Ricco's Law held approximately for disk areas less than the signal summation area, i.e. $I \cdot A = k_{RS}$, where k_{RS} is the constant for Ricco signal summation. For areas larger than the signal summation area, Ricco's Law no longer held and a constant illumination was required to elicit a criterion response. The area over which Ricco's Law held was equated with the center's area of signal summation. For the same cell the *adaptive summation area* was determined, again with a constant response criterion. In this case, area and illumination of an adapting disk were varied reciprocally in order that the ganglion cell would produce a constant response to the test spot. The test spot was constant in area and illumination; it was placed in the center of the receptive field. Adaptive summation followed Ricco's Law also for disks with areas less than the adaptive summation area. That is, for adaptation, $I \cdot A = k_{RA}$, where k_{RA} is the constant for Ricco adaptive summation. For larger disks, summation of adaptive sensitivity

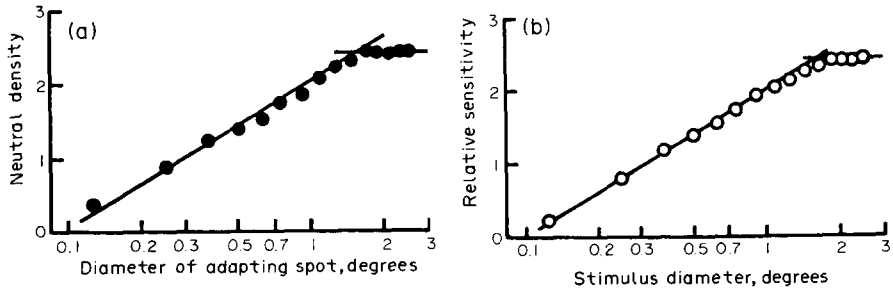

FIG. 32. Comparison of adaptive (a) and signal (b) summation areas of an on-center cat ganglion cell.
(a) The horizontal axis gives the log of the diameter of unmodulated *adapting* spots. The vertical axis gives the log of the reciprocal of the adapting spot illumination in relative units. The adapting illumination was set so that the cell produced a criterion response to a small (0.13 deg) centrally located spot of fixed luminance, which was sinusoidally modulated (4 Hz, 0.6 contrast).
(b) The horizontal axis gives the log of the diameter of the *stimulus,* which was again a spot modulated at 4 Hz, sinusoidally, at 0.6 contrast. But in this experiment the stimulus diameter was varied. The vertical axis in (b) gives the log of the relative gain for the spots of different size; it is also the log of the reciprocal of the illumination required to elicit a constant response. In (b), the spots were presented on a steady background of $6 \cdot 10^5$ quanta(deg^2 s)$^{-1}$. The criterion response in both (a) and (b) was just audible synchronization of the cell's discharge with the stimulus modulation. From Cleland and Enroth-Cugell (1968).

stopped. The important result of this experiment is that the signal summation area and the adaptive summation area were the same. Probably most if not all of these experiments were on Y retinal ganglion cells, but later work indicates similar results hold for X cells (Harding, 1977). The results are the same for on- and off-cells. Very similar results have been obtained with this experimental design on rat optic tract fibers by Green *et al.* (1977) and Tong and Green (1977). Results of this kind have also been obtained in lower vertebrates: in frog ganglion cells (Reuter, 1969; Burkhardt and Berntson, 1972), and in goldfish ganglion cells (Schellart and Spekreijse, 1972).

Related experiments by Shapley *et al.* (1972) and by Enroth-Cugell and Shapley (1973b) indicate that, as the area of an adapting spot of *fixed* luminance is increased, the gain declines. This is illustrated in Fig. 33. The (fixed) luminance of the adapting spot was chosen so that for the smallest adapting spot the gain had not been reduced from its dark adapted maximum. As the area of the adapting spot was increased, gain declined and the response to the test stimulus became more transient. Results similar to these were obtained by Schellart and Spekreijse (1972) in the goldfish retina. They found that the temporal impulse response was speeded up and made more diphasic by increasing the area of a background spot of constant luminance.

Another experiment which demonstrates the significance of the area as well as the luminance of the background is illustrated in Fig. 34. The figure shows the actual responses of a ganglion cell to turning on *adapting* spots of quite different area and luminances. The luminance of the larger one had been adjusted until it produced the same gain reduction as the smaller adapting spot, as indicated by the response to the superimposed brief test flash. Equal adaptive effect is associated with equal responses of the ganglion cell to the "adapting" spots. The conclusion from all these experiments is that the gain of the ganglion cell center mechanism is set by the sum of all the steady state input to the center. This in turn implies that the adaptive effect of a background on the center mechanism of a ganglion cell is, under the conditions of these experiments, determined by the *total effective flux*: the sum of all the light per unit time which falls on the center from the background, weighted by the spatial "sensitivity profile" (Cleland and Enroth-Cugell, 1968; Enroth-Cugell and Shapley, 1973b).

The meaning of these results in a psychophysical context is that the summation pool of a ganglion cell is the same size as the adaptation pool. The meaning in a neuroanatomical context is that whatever interneuron determines the size of the receptive field center is also implicated in the gain control process.

In the light of these results on the dependence of

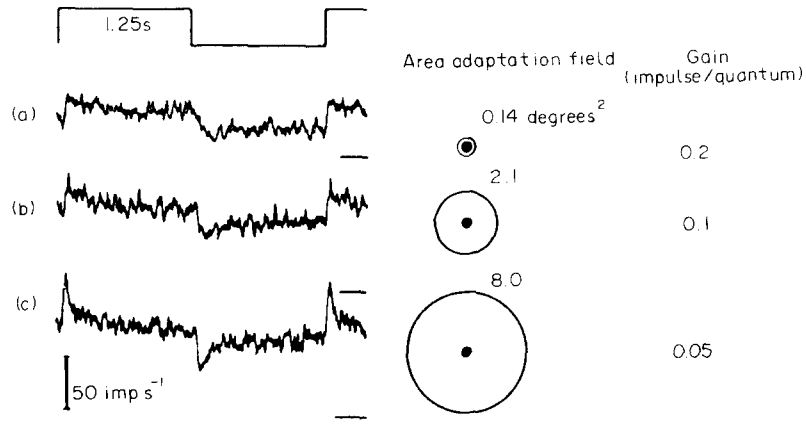

FIG. 33. Reduction in gain with increase in area of an adapting spot of constant retinal illumination. Three averaged responses were elicited by a small (0.18 deg) centrally located spot (a,b,c). In each run, the area of an adapting disk of constant retinal illumination—$6 \cdot 10^3$ quanta(507 nm) $(deg^2\ s)^{-1}$ was varied; the area in deg^2 is given in the figure. Gain was calculated by dividing response in impulses s^{-1} by the retinal stimulus flux in quanta s^{-1}. This experiment was done on an on-center cat ganglion cell, probably a Y cell. From Shapley *et al.* (1972).

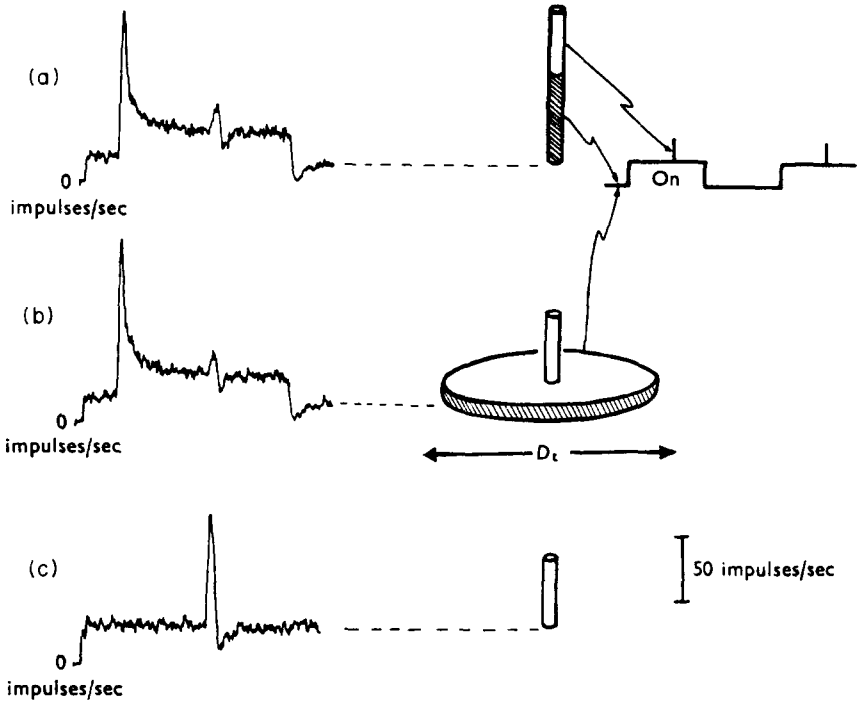

FIG. 34. Gain reduction with two adapting spots of different area but the same effective flux. The test stimulus was a 50 ms pulse of light, 0.1 deg in diameter, $2 \cdot 10^7$ quanta$(deg^2\ s)^{-1}$ retinal illumination.

(a) The conditioning stimulus (hatched) was 0.1 deg spot superimposed on the test spot, modulated in a square wave manner at 0.4 Hz. The illumination in the light phase was $3 \cdot 10^7$ quanta$(deg^2\ s)^{-1}$.

(b) The conditioning stimulus was a 1.57 deg diameter disk, concentric with the test spot. Its illumination was $2.5 \cdot 10^4$ quanta$(deg^2\ s)^{-1}$, and also 0.4 Hz.

(c) Test spot was presented without any conditioning stimulus. The two conditioning stimuli must have had the same effective flux, by definition, because they produced identical responses. These results are from an on-center ganglion cell, presumably a Y cell. From Enroth-Cugell and Shapley (1973b).

gain control on the product of illumination and area, we must revise equations (20) and (21) which expressed the gain in terms of illumination. The earlier equations were correct under the conditions of full field illumination when the entire receptive field would be covered by the uniform background. In this case the gain would just be scaled down by a factor equal to the area of the center of the receptive field. But a more general equation can be written which applies to backgrounds of any spatial configuration. For the rod pathway:

$$G_R = G_{R0}/(1 + F_B/F_{R0})^P \qquad (24)$$

and a similar equation describes the gain of the cone pathway, G_C. Flux is illumination multiplied by area, in this case the total summing area of the center of the receptive field. For step responses, the exponent P has the value 0.9 for the rod pathway and is somewhat higher for the cone pathway. What equation (24) means is that the gain depends on background flux, F_B, not retinal illumination, flux added up over the entire center of the receptive field and weighted by the distribution of sensitivity of the receptive field center. Also, the adapting flux has to exceed a critical value, denoted F_{R0} for the scotopic system, in order for the cell to undergo the transition from dark adaptation to light adaptation. It may help to conceive of this critical flux as the background flux required to produce a critical level of D.C. neural signal which, when exceeded, turns on the retinal gain control. The value of the critical flux, F_{R0}, is about 10^4 quanta s^{-1} at the retina on the average (Enroth-Cugell and Shapley, 1973b).

3.5.2. Local and global gain controls which depend on flux

Further investigation of the spatial summation of adaption in cat ganglion cells was performed by Harding (1977). One of his major results is illustrated in Fig. 35. The experiment was designed to measure the spatial weighting of adaptation with a two-spot paradigm: one test and one adapting spot. The test spot was fixed in position in the middle of the receptive field. Then the position of the adapting spot was varied and its luminance adjusted so that the gain of the response to the test was reduced by a criterion amount. In the same cells the sensitivity profile for eliciting a response was also measured. This latter profile is called the signal sensitivity profile. As can be seen from the results in Fig. 35, the signal sensitivity profile and the adaptation profile were approximately the same in X cells. In some Y cells the gain reduction for a test spot near the adapting spot was greater than for a test spot farther from the adapting spot, by more than would be predicted from the Y cell's signal sensitivity profile. That is, there were indications of local adaptation in the Y cell center. A similar sort of effect was seen in an investigation of light adaptation of the receptive field surround of Y cells (Cleland *et al.*, 1973). There is thus evidence for two different gain controls in the Y cell center: one local, one more global. There is also evidence for local adaptation in some X cells (Harding, 1978). A discussion of the implications of these experiments for X/Y receptive field organization would carry us too far from the central issues of this paper. But a brief comment about these different adaptation profiles in X and Y cells may provoke some thought about the retinal microcircuitry underlying adaptation.

It is known that at any retinal locus the receptive field centers of X cells are about ten times smaller in area than those of Y cells (Hochstein and Shapley, 1976b; Cleland *et al.*, 1979; So and Shapley, 1979; Linsenmeier *et al.*, 1982). There exist subunits of the Y cell's receptive field which are roughly the same size as X cell centers at the same retinal eccentricity (Hochstein and Shapley, 1976b; So and Shapley, 1979; cf. Appendix 2). It has been suggested that X cell centers and Y cell subunits are approximately determined by the spatial summing areas of bipolar cells (Hochstein and Shapley, 1976b; Victor and Shapley, 1979). While recent neuroanatomical investigation of the retina suggests this is only an approximation to the actual situation, it seems now to be an approximation rather than mere speculation (see Sterling, 1983). Thus, if one accepts our previous assertion that the interneuron which determines the size of the X cell's center must set the gain of the center, then the bipolar cells must control the gain of the X center, in some way. If these same bipolar cells feed into the Y cells' subunits, one must suppose they control the gain of the subunits. The local effects of gain reduction in Y cells seen in Fig. 35 could be explained by this "bipolar" gain control, which we infer to be

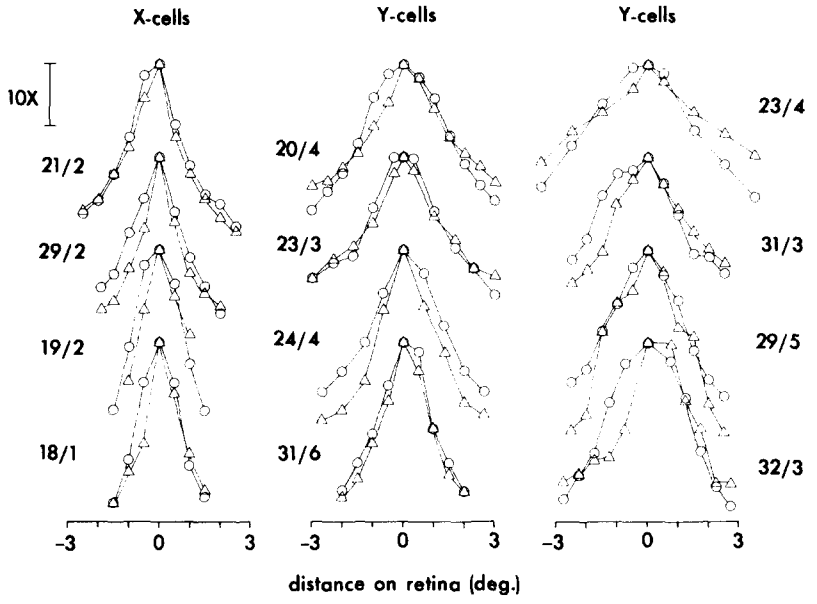

FIG. 35. Adaptive and signal gain profiles from four X and eight Y cells. The stimulus used to obtain each profile was a spot 0.2 deg in diameter, modulated by a 2 Hz squarewave. The response criterion was 3 extra impulses over 100 ms at the beginning of each response. *Signal gain:* the circles indicate the relative gain at each position with respect to the peak gain at position zero, the middle of the receptive field center. The vertical scale is logarithmic and a vertical calibration for 10x is given in the figure. *Adaptive effectiveness:* The horizontal position of the triangles indicates the location within the receptive field center of a steady *adapting* spot of 0.2 deg diameter. The modulated test stimulus was fixed *in the middle of the receptive field center.* The test stimulus was set to be 3x brighter than required to reach criterion. Then the luminance of the adapting spot was adjusted to reduce the response to the criterion level, at each position of the adapting spot. The log, of the ratio of the adapting luminance at position zero divided by the adapting luminance at each of the other positions tested, is given by the vertical position of the triangles. Signal and adaptive profiles have been superimposed at position zero to allow comparison of their respective spread. From Harding (1977).

common to X cell centers and Y cell subunits. There must also be a *second* gain control for the much larger Y cell center; a likely candidate is one of the large field amacrine cells which should have a dendritic spread roughly ten times larger in area than the dendritic spread of an X ganglion cell.

Local adaptation effects like those in Fig. 35 have been seen previously in ganglion cells from other species: rat (Green *et al.*, 1977; Tong and Green, 1977), frog (Burkhardt and Berntson, 1972), goldfish (Easter, 1968). The functional analogies of these cells with cat X or Y cells is obscure, but in every case the cells studied had relatively large receptive fields compared to the size of dendritic spreads of bipolar cells. Moreover, evidence for a second gain control with a large summing area comparable to the center size has been found in each of these retinas: in rat (Green *et al.*, 1977), in frog (Reuter, 1969; Burkhardt and Berntson, 1972), and in goldfish (Schellart and Spekreijse, 1972). Thus, local adaptation effects in each of these cases might be due to localized "bipolar" gain controls which are present in addition to a second, "amacrine", gain control which sums light evoked neural signals over a larger area. This should be clearly labelled as a chain of inference rather than established fact. Yet it is interesting because it serves to reinforce the idea that there is a hierarchy of gain controls, a "bipolar" gain control and an "amacrine" gain control as well as photoreceptor gain controls which will be discussed below.

3.5.3. Optical and neural factors in adaptational pooling

One must consider the proposition that all retinal gain control by light is a purely local, receptoral phenomenon. It may seem an absurd proposition in the face of all the evidence cited above about the adaptive summing area being equal in size to the receptive field center, but it deserves serious

consideration because of the problem of light scattering within the eye and of optical aberrations in the lens and cornea. If the measured size of the receptive field center and adaptive summing area were greatly influenced by such imperfections in physiological optics, one would have to discount the conclusions about the site or sites of gain control(s) which were based on the correlation of the spatial extents of these two different mechanisms. However, direct measurements of the physiological optics of the cat's eye (Bonds, 1974; Robson and Enroth-Cugell, 1978) indicate that, except for rare cells with the smallest receptive fields, the optical effect on the measured size of the receptive field and adaptive summing area is small. It is interesting to note that the existence of the postulated "amacrine" gain control of the Y cells is not subject to any doubts based on optical blur or scatter, because the adaptive summing area and receptive field center of Y cells are so large. The presence of neighboring X cells with fields ten times smaller in area serves as a control on the optical contribution to the size of the Y cells' fields. However, there could be some question about whether the size of the receptive fields of X cells, which can be quite small in area, might be due to optical blur or scatter. It becomes a quantitative question in the case of the X cells. However, even for X cells, the optical blur seems to be less than the neural summation area (Robson and Enroth-Cugell, 1978) when the physiological optics are optimized with best refraction and a small artificial pupil. Throughout this paper, arguments based on receptive fields' sizes have only cited as evidence the results of experiments in which the physiological optics were optimized.

That some of the retinal gain — setting mechanisms must involve pooling of signals from many receptors is the conclusion of a physiological extension of Rushton's reasoning about the low level of backgrounds which produce light adaptation (Enroth-Cugell and Shapley, 1973a). As discussed above, Rushton found that the psychophysical scotopic threshold was raised by a factor of two from its dark adapted value when only one rod in a hundred actually caught a quantum of light. However, as stated before, this result implies nothing about gain control in the retina. It could be explained in terms of an increase in "noise" from the background. Applying Rushton's reasoning to retinal gain, we measured how much background light is required to reduce the *gain* of a ganglion cell by a factor of two. In several cells with large receptive fields, the required background light yields one quantum absorbed per second per hundred rods. The integration time of feline rods is almost certainly shorter than a second; indeed, other work indicates a maximum integration time of 0.1 s. Thus, one rod in a thousand per integration time receiving a quantum of background light is enough to reduce the gain by half a log unit. This is solid support for Rushton's conclusion that signals from a pool of rods must set the gain for signals from rods which have not themselves received light quanta from the background.

In cells with smaller receptive fields, the luminance required to reduce gain was higher, so that in the worst case approximately one rod in ten received a quantum of light per integration time. We now presume that these ganglion cells with small fields were X cells, on the basis of the receptive field center size. So again the very low level of background luminance which is required to reduce the gain may suggest that photoreceptor signals must be pooled to set the gain in the cat retina. Note that light scatter would not affect these measurements because they were made with large uniform backgrounds which were at least one thousand times larger in area than the optical point spread function at half height. Furthermore, the force of these arguments is not affected by the fact that not all photoreceptors which lie within the spread of the dendritic tree of a ganglion cell project to that ganglion cell. The statistical randomness of the quantum catch makes the fraction of receptors hit by quanta the same whether one considers the entire population of photoreceptors, or only that population which projects to the ganglion cell under study, as long as the background is truly uniform.

That there are different transition levels, from dark to light adapted, for scotopic receptive field center and surround mechanisms (Fig. 26) also supports the idea of a gain control proximal to the photoreceptors. The same rods must drive the center and surround. If only the rods adapted, the center and surround would have to lose their gain in parallel. Since this is not observed, we must conclude that gain must be controlled at a site in

the retina after center and surround have been segregated. The neuroanatomy of the cat retina tells us that all post-receptoral interneurons pool the activity of many photoreceptors. Therefore, the post-receptoral gain control must pool photoreceptor signals.

3.6. Gain Control and Receptive Field Size Across the Population of Ganglion Cells

Since light – evoked neural signals are summed over the receptive field center to set the gain of the center of a particular ganglion cell, one naturally would guess that ganglion cells with centers of different sizes would be light adapted to different extents by a large uniform background. The results on the spatial summation of adaptive effect suggest that gain depends on the total, steady state, effective flux, i.e. illumination multiplied by area weighted by the center's gain per unit area. The total effective flux falling on the center of a cell with a small receptive field will be less than the flux falling on a large receptive field center; the gain should be reduced less in the small receptive field center.

The initial test of this idea is offered in Fig. 36, from Enroth-Cugell and Shapley (1973b). What is plotted in Fig. 36 is the transition illumination at the knee of the curve relating gain and background illumination. The transition illumination is defined here empirically as the illumination at which the gain has dropped by a factor of two from the dark adapted gain (Note the slight difference between this definition and the more rigorous definition of transition illumination in connection with equation (20)). Because the data in Fig. 36 were collected from many different cats, possibly in different physiological states, the transition illumination was multiplied by the dark adapted gain to obtain a corrected transition illumination. This corrected transition illumination is plotted against center summing area, determined from an area – threshold curve (Cleland and Enroth-Cugell, 1968). It can be seen in Fig. 36 that the cells with larger centers have a lower effective transition level, and that the transition level is approximately proportional to the center summing area. These data might be compatible with other functions of center size besides area, because of the large variance. The cells

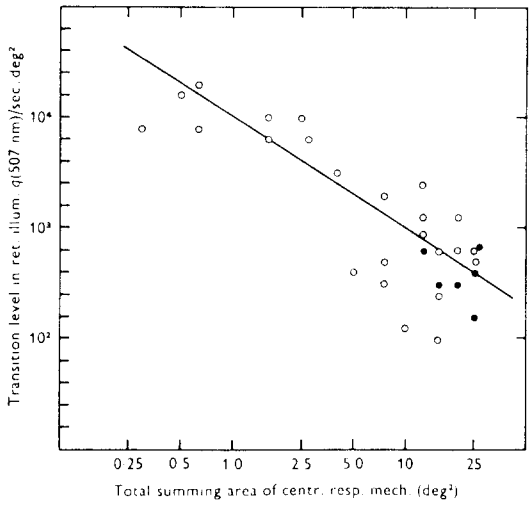

FIG. 36. Transition level as a function of center area for a population of cat retinal ganglion cells. The value of the transition level from the horizontal to the sloping portions of the gain vs background illumination curves (as in Fig. 24) is plotted against center summing area. Empty circles are from on-center cells, while filled circles are from off-center cells. The cells were not grouped into X and Y classes. From Enroth-Cugell and Shapley (1973b).

in this study were not classified as X or Y. Therefore, a question unresolved by these results is whether X and Y cells have the same dependence of effective transition level on center area.

Further evidence on the dependence of gain setting on receptive field center size across the population of ganglion cells comes from the concordant studies of Fischer and May (1970) and Cleland *et al.* (1973). The results of both studies implied that the center's gain, defined as $G = dR/dF$ where F is luminous flux (illumination times area), is inversely proportional to the center's summing area when the ganglion cell is well light adapted in the mid-scotopic to mesopic range by large uniform backgrounds. This is consistent with the result of Enroth-Cugell and Shapley (1973b) on the "effective transition level" and with the approximately inverse relationship between gain and background above the transition level, equation (21). Thus, three studies seem consistent in supporting the hypothesis that ganglion cells with larger centers are more light-adapted than those with smaller centers under the same fixed uniform background conditions, because of spatial summation of adapting signals.

Recently, Linsenmeier *et al.* (1982) have taken a fresh look at this question by measuring the gain of X and Y ganglion cells in the cat in response to drifting gratings on high scotopic or mesopic backgrounds. They also gauged the size of the center of each ganglion cell by fitting the observed dependence of contrast gain on spatial frequency with a "Difference of Gaussians" model. The spatial sensitivity profiles of receptive field center and surround are approximated by Gaussian functions in this model. The spatial spread of the center's Gaussian is a measure of the effective radius of the center's distribution of sensitivity (or, more precisely, gain). Figure 37 is their graph of the peak gain of the center plotted vs the center's effective radius, for a large population of cat retinal ganglion cells. The figure demonstrates that the cells with the largest centers had the lowest peak gain, and that the gain was approximately the inverse of the center's *radius*. While this result is qualitatively like the earlier results of Enroth-Cugell and Shapley, Fischer and May, and Cleland *et al.*, it is quantitatively different in that gain in the light adapted state is inverse to the radius and not the area of the center. However, there is quite a lot of variance of gain across the population of ganglion cells, so much so that Linsenmeier *et al.*'s results do not conclusively disprove the area – gain relation. Furthermore, since their measurements were made at backgrounds which might be in the high scotopic or in the mesopic range, the precise value of the slope of the gain vs area line might be influenced by the degree to which rods or cones are the predominant photoreceptor input for cells of different sizes. These qualifying remarks suggest that the book is not closed on the dependence of gain on area of the receptive field center. As suggested below, the hypothesis of Enroth-Cugell and Shapley (1973b), that gain varies inversely with center *area* in the light-adapted state, is useful in rationalizing psychophysical results on the dependence of sensitivity vs background curves on target size.

The interpretation of these area effects in adaptation must be modified by the discovery that there is not a wide variation in receptive field center size among ganglion cells of one type, X or Y, at a given retinal locus (Cleland *et al.*, 1979; So and Shapley, 1979). The coefficient of variation of the

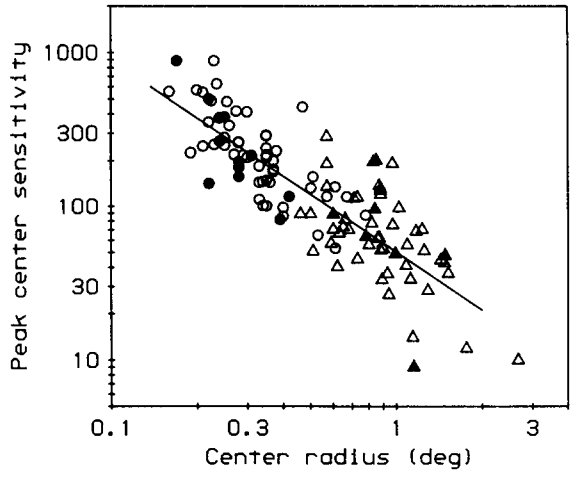

FIG. 37. Relation between the center's size and its peak gain in the middle of the receptive field. The dependence of (Rayleigh) contrast gain on spatial frequency was determined for X and Y cells by adjustment of contrast to reach a constant response criterion. The experimental curves were fit with a Difference of Gaussians model as in Fig. 31, from which both the center's radius and its gain at the peak of its sensitivity profile could be determined. These two values are plotted against each other to show that, at the same mean luminance, cells with larger centers have lower gain. The empty symbols are for on-center cells, the filled symbols are for off-center cells. Circles denote X cells; triangles denote Y cells. The pupillary area was 16 mm^2. The mean luminance was around 14 cd m^{-2}. From Linsenmeier *et al.* (1982).

center-diameter distribution at any one retinal locus is at most 0.25 (So and Shapley, 1979) and is probably less in an individual animal. There is a marked increase in receptive field center-diameter at retinal loci away from the *area centralis*; the diameter of the center is approximately proportional to the distance from *area centralis*. This is true for both X and Y cells, and for both on- and off-center cells. On- and off-cells have approximately the same size at any one locus on the retina. As stated above, Y cells have an approximately ten times larger area than X cells at each locus. The combination of these facts with the preceding results on the effects of area on adaptation leads to the following conclusions. First, cells with larger receptive fields in the periphery of the retina ought to be more light-adapted than central ganglion cells with smaller centers, under conditions of uniform constant background illumination. Second, Y ganglion cells ought to be more light-adapted than X ganglion cells at the same retinal locus. By the degree of light-adaptation we mean the degree to which gain has been reduced

relative to its dark-adapted value. These conclusions have to be qualified to include the proviso that the background conditions have to be such that all the ganglion cells are driven by the rod pathway. At present there are no firm data about the effect of the size of centers on the degree of adaptation of the ganglion cell population in the photopic range.

3.7. The Effect of the Receptive Field Surround on the Gain of the Center

The surround of the receptive field plays no role, or at most a minor role, in setting the gain of the center. This is the conclusion which is implicit in the profusion of results presented above which demonstrate that the summation area for adaptation is either equal to or smaller than the signal summation area of the receptive field center itself. However, this conclusion is counter-intuitive to a number of theorists who have proposed that the center's gain ought to be regulated by the surround (Helson, 1964; Sperling, 1970; Grossberg, 1981). Such an important point deserves direct scrutiny.

The influence of the surround on the center in the scotopic range was measured by Enroth-Cugell *et al.* (1975). Their results are graphed in Fig. 38. The gain and the mean impulse rate as a function of background area are shown for two cells, one X on-center cell, and one Y on-center cell. The gain drops as the area of a background spot, of constant luminance, is increased so that it just fills the center. Increase of area beyond this value produces no further increase or decrease in gain. However, there is steady state input from the surround caused by the large backgrounds because the mean impulse rate does decline when the background spot grows larger in size than the ganglion cell's center and intrudes into the surround. Thus steady surround input does not regulate center gain in this experiment.

The question of whether the surround might regulate the gain of the center in the photopic range cannot be answered so definitively in the negative. Enroth-Cugell *et al.* (1977a) found that Y cells behaved the same way in the photopic and scotopic ranges, i.e. there was no change in gain when the area of a background spot of fixed luminance was

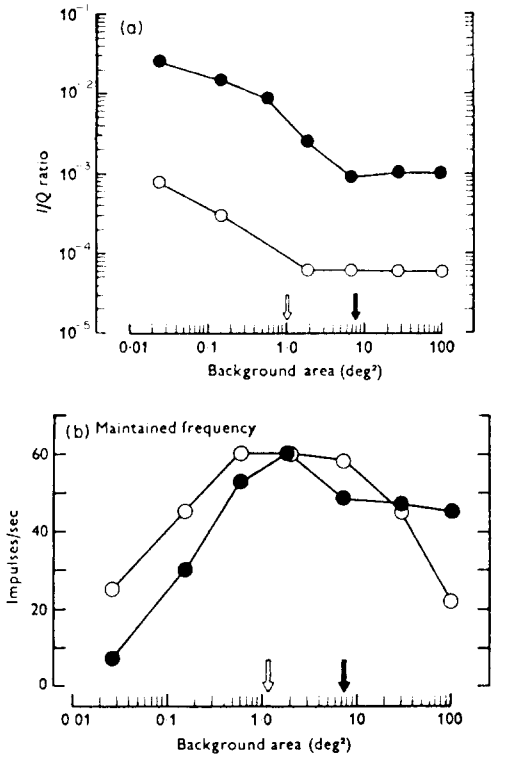

FIG. 38. No effect of the surround on the gain of the center in cat retinal ganglion cells in the scotopic range. Red steady spots, centered on the receptive field middle, and of diameters indicated by their horizontal coordinates, were used as backgrounds. Their illuminations were $4 \cdot 10^4$ quanta(507 nm) $(deg^2\ s)^{-1}$ for the Y cell (filled symbols) and $2.5 \cdot 10^5$ for the X cell (empty symbols). For both cells the stimulus was a blue – green test spot (0.18 deg diameter for the Y cell, 1 deg diameter for the X cell). The stimulus illumination was adjusted to produce an approximately constant peak response, and the gain calculated from response magnitude and stimulus flux. The gain (i/q ratio) fell as the background diameter was increased so long as the background diameter was less than the diameter of the center (marked with the empty arrow for the X cell, with the filled arrow for the Y cell). To determine whether light falling outside the center did indeed activate the surround, the maintained firing was measured and is displayed in the lower panel. The cells' maintained firing rates were reduced by the light falling beyond the center, indicating sustained surround antagonism of the center, even though the gains of the centers were not affected by this same light. From Enroth-Cugell *et al.* (1975).

increased beyond that of the central summing area, as illustrated in Fig. 39 (Note that in Figs 39 and 40 the vertical axis is log threshold for a criterion response, which is equivalent to the log of the *reciprocal* of the gain). However, the data on X cells were incomplete. Only one X cell was studied in the photopic range, and it showed a small but significant increase of center gain when the adapting

spot was enlarged to cover the surround, as shown in Fig. 40(a) from unpublished results of Lennie, Hertz and Enroth-Cugell. The fractional recovery of center gain when the background was enlarged to include the surround was smaller than the fractional loss of gain when a stimulus spot was enlarged to include the surround as is indicated in Fig. 40(b). This could be because the surround

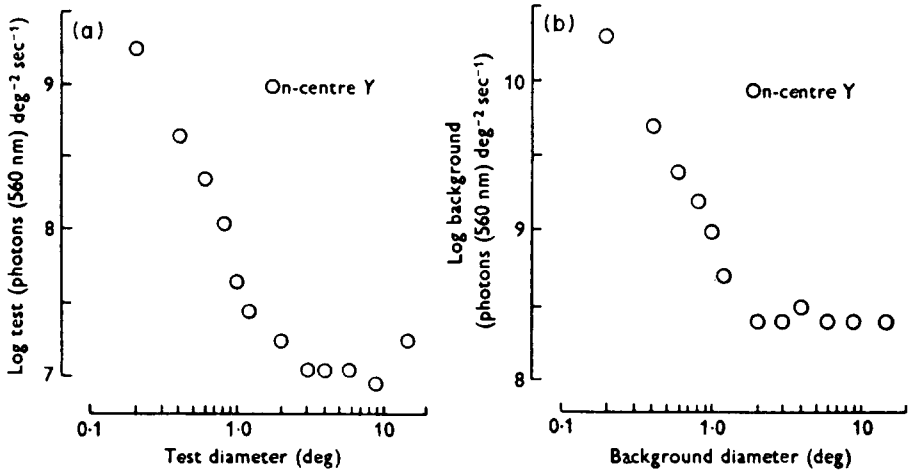

FIG. 39. Signal and adaptive summation in a Y ganglion cell in the photopic range: no effect of surround on adaptive summation.

(a) The photopic center's size was determined as in Figs 30 and 32, by obtaining threshold illumination for a constant response, as a function of area. This was done with red spots (modulated at 4 Hz) on a steady blue – green background which had a retinal illumination of $8 \cdot 10^8$ quanta(507 nm) $(deg^2\ s)^{-1}$.

(b) The size of the "adaptation pool" of the center was determined with an area – adaptation curve as in Fig. 32(b). A fixed 0.2 deg diameter stimulus was set to be at threshold on a 15 deg background. The background was reduced in area, in discrete steps, and the illumination of the background was adjusted to keep the response to the central test spot at criterion. The required illumination of the background is plotted against its diameter. From Enroth-Cugell *et al.* (1977a).

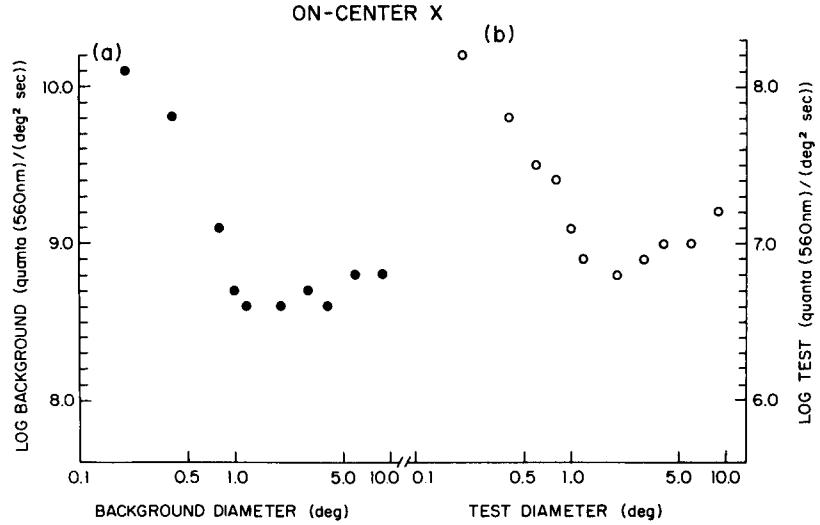

FIG. 40. Signal and adaptive summation in an individual X ganglion cell in the photopic range.

(a) The left panel shows the background illumination required to keep a 0.2 deg diameter red test stimulus (modulated at 4 hz) at auditory threshold for modulation, as the (blue – green) background diameter was varied from 0.2 to 10 deg.

(b) The right panel shows the test illumination required for a 4 Hz modulated, red, 0.2 deg diameter, spot to produce a threshold response, on a blue – green 15 deg background of retinal illumination $8 \cdot 10^8$ quanta(507 nm) $(deg^2\ s)^{-1}$. From Lennie, Hertz and Enroth-Cugell, unpublished results.

might be relatively less sensitive to steady state illumination than is the center; the measurement of surround – center gain in (b) was made at a stimulus modulation rate of 4 Hz, while the illumination of center and surround in the adaptation experiment, plotted in (a), was constant in time. This experiment needs to be replicated on a larger population of X cells than one, in order to be able to evaluate the surround's control of center gain. However, it is an indication that the surround may play a different role in X cells in the photopic range from its ineffectual performance in the scotopic range. This result is tantalizing from the standpoint of structure – function correlations. The absence of either sensitization or desensitization (in the cat retina) caused by the surround in the scotopic range implies that the interneuron which controls the gain of the ganglion cell center has a receptive field the size of the center, with no spatially antagonistic input. The absence of surround gain control of the center in the photopic range for Y cells carries the same implication. The presence of a small but significant sensitization of the center by the surround in the photopic range in X cells suggests that the gain of the X center in the photopic range might be set with an interneuron with a center the size of the X cell's photopic center but with an additional surround. One possible speculation is that X cells always have their gain set by the bipolar cells from which they receive direct input, and that the rod bipolar cells have a weak or non-existent surround mechanism while the cone bipolar cells do possess a surround (cf. Nelson *et al.*, 1981). The Y cells which receive most of their input via the amacrine route would always have their gain set by interneurons which do not have surround antagonism.

There is a completely different explanation for sensitization which should be considered. Suppose that the sensitization experiment is done against a low diffuse background. Scattered light *from the test spot* may excite the surround when the adapting spot is small, and not when the adapting spot is large, and effective in desensitizing the surround. Thus, the "control" response to a test spot on the small background may be a "mixed" response from center and surround, which is cleansed of surround contamination by the enlargement of the background. Occasionally, such mixed responses to test stimuli on small adapting spots were seen, both in the Enroth-Cugell *et al.* (1975) study (Figs 2, 3 and 4 in their paper) and in later work on sensitization of the cone pathway by Lennie, Hertz and Enroth-Cugell (unpublished). Removal of surround contamination produces a larger peak response, and a much larger sustained response, to a test spot placed in the center of the receptive field. The large sensitization effects observed in psychophysical experiments may be due more to this "release from surround contamination" than from the possible but probably weaker influences of surround signals on the gain of the center.

3.8. A Different Kind of Gain Control: The Contrast Gain Control

Up to this point we have only considered the effect of steady illumination on the control of retinal gain and dynamics. However, there is another gain control which depends not on the steady light level but rather on the average modulation of optical stimuli over a wide region of visual space. This is what Shapley and Victor (1978, 1979, 1980, 1981) have termed the *contrast gain control.* It is probably equivalent to the "silent surround" discovered by H. B. Barlow (1953), and to the "suppressive surround" found in the retina by Cleland and Levick (1974). Analogous retinal mechanisms have been found in pre-ganglionic interneurons in the mudpuppy retina by Werblin and Copenhagen (1974) and Thibos and Werblin (1978b). Please note that the terminology may be confusing in this case; the contrast gain control adjusts the gain of the retina contingent on contrast rather than flux. Perhaps we should call it the *contrast gaincontrol*, to distinguish it from the flux gaincontrols.

This new kind of gain control was originally discovered in experiments in which the temporal frequency response of cat retinal ganglion cells was measured as a function of contrast. The results of such an experiment are shown in Fig. 41. The data are displayed in a Bode plot, with log amplitude vs log temporal frequency in the upper graph and with linear phase vs log temporal frequency in the lower panel. If there were no contrast gaincontrol, the amplitude curves would be the same shape, i.e. they could be superimposed by means of a vertical shift.

Furthermore, the phase curves at different contrasts would be superimposable. This is not the case. The amplitude curve is shifted towards higher temporal frequencies at higher contrasts, and the phases of intermediate frequencies are advanced at higher contrasts. At an r.m.s. average contrast of 0.2, the amplitude of the response to 0.5 Hz may be reduced by 50% on account of the action of the contrast gaincontrol. The phase may be shifted at 8 Hz by as much as 60 deg. Another way to look at the contrast gaincontrol is that it produces what appears to be a frequency – dependent saturation. The amplitudes of responses to higher temporal frequencies grow approximately proportionally with contrast. The amplitudes of responses to lower temporal frequencies grow less than proportionally with contrast, and appear to saturate at a lower contrast. That this effect is not simple saturation is proven by the temporal frequency dependence.

The spatial and temporal dependence of the contrast gaincontrol's action indicate that it has the same characteristics as the nonlinear receptive field subunits which feed excitation to Y cells (Shapley and Victor, 1978; cf. Appendix 2). It appears that the subunits precede the contrast gaincontrol in retinal processing; the subunits compute the total average contrast which is then used to control the retina's dynamic responses. The way that the contrast gaincontrol modifies the time course of retinal responses appears to be by modulation of the strength of pre-existing feedback pathways. That is, increase of contrast tends to turn on the contrast gaincontrol which then increases the strength of negative feedback in both X and Y retinal pathways (Shapley and Victor, 1981).

The contrast gaincontrol calculates a contrast signal by averaging the contrast modulation over a wide expanse of retina. The spatial extent of the area of contrast averaging has not been determined precisely, but it must be considerably larger than the extent of the center mechanism of Y ganglion cells. Rough estimates of its spatial extent may be based on the area over which the product, contrast times area, produces a given amount of low frequency suppression or mid-frequency phase advance. This area is on the order of ten degrees, i.e. about 2 mm on the retina. Furthermore, the contrast gaincontrol may receive weaker but still significant input from still further reaches of the retina, in this way also resembling the excitatory nonlinear subunits of Y cells. Thus the contrast gaincontrol must involve a third type of interneuron different from the previously inferred interneurons which are needed to account for the flux gaincontrols of the X and Y cell center mechanisms.

Because of its wide summing area, the contrast gaincontrol can account for nonlinear spatial summation of responses produced by contrast modulation in the center and periphery of X and Y ganglion cell receptive fields (Shapley and Victor, 1979). This has been observed with sinusoidal test stimuli by Shapley and Victor and with square wave

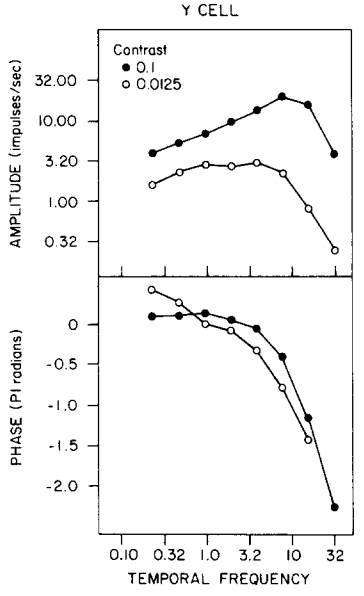

FIG. 41. The contrast gaincontrol revealed in temporal frequency responses of retinal ganglion cells. These are data from an on-center Y cell. The mean luminance was 20 cd m^{-2}, and the pupil diameter was 3 mm. The stimulus was a 0.25 c deg^{-1} sine grating which was modulated in amplitude by a sum of sinusoids, with temporal frequencies from 0.2 up to 32 Hz. The empty circles were obtained when the contrast was 0.0125 per sinusoid, i.e. if any one of the eight sinusoids had been presented alone, the peak contrast of the modulated grating would have been 0.0125. The filled circles were obtained with 0.1/sinusoid contrast. The amplitudes plotted are the Fourier amplitudes in the cell's impulse train which were at the temporal frequencies present in the stimulus; the phases are the phase shifts of those Fourier components in the impulse train with respect to the corresponding component in the input signal. If the retina were linear, or if the nonlinearity were a simple saturation, the two amplitude curves should be parallel, and the phase curves should be parallel, and the phase curves should superimpose. The accentuation of responses at high frequencies, and the phase advance at mid-range frequencies, as contrast increases, is the signature of the contrast gaincontrol. From Shapley and Victor (1979).

test stimuli by Enroth-Cugell and Jakiela (1980). The effect of the contrast gaincontrol on square wave responses is to make them smaller and more transient, as can be seen in Fig. 42 from Enroth-Cugell and Jakiela (1980). This is an example of nonlinear spatial summation because the peripheral stimulus generates no response when presented alone but suppresses the response to the centrally placed bar stimulus when both are presented together. This kind of suppressive effect from peripheral stimulation has been observed in many ganglion cell types in several different species.

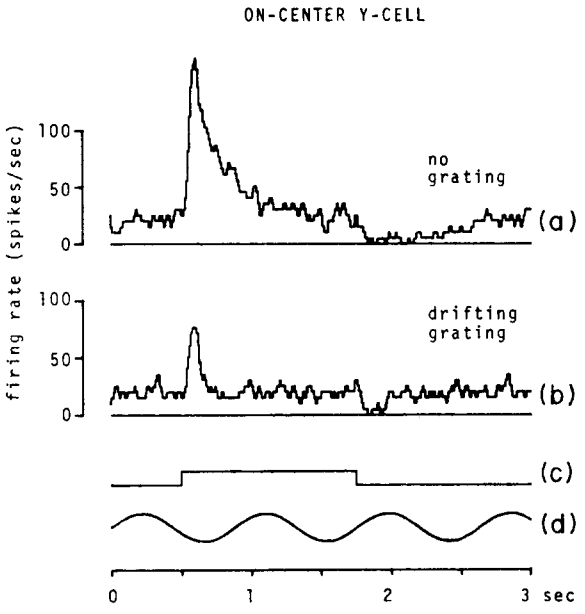

FIG. 42. Reduction of the center's gain by a moving pattern in the periphery of the receptive field in a cat retinal ganglion cell. Responses of an on-center Y cell to a centered bar, 1 deg wide, 13 deg high, of luminance 0.25 cd m^{-2} flashing at 0.4 Hz.

(a) The bar was superimposed on a background of luminance 5 cd m^{-2}. Both bar and background were generated on a CRT which subtended 13 deg by 16 deg.

(b) A drifting sine grating with a contrast of 0.5, and spatial frequency 1.25 c deg^{-1} covered the CRT screen except for a 2 deg wide band down the center of the screen where the stimulus bar was presented. The grating was drifting with a temporal frequency of 1.14 Hz.

(c) Shows the time course of the bar stimulus.

(d) Indicates the time course of the luminance change at a point in the field over which the grating was drifting. From Enroth-Cugell and Jakiela (1980).

3.9. Time Course of Gain Adjustment in Retinal Ganglion Cells

The retinal gain as measured in ganglion cells is readjusted rapidly but not instantaneously by sudden changes in background level. This has been determined in physiological experiments which are analogous to Crawford's psychophysical experiment on the time course of light adaptation in humans (Crawford, 1947).

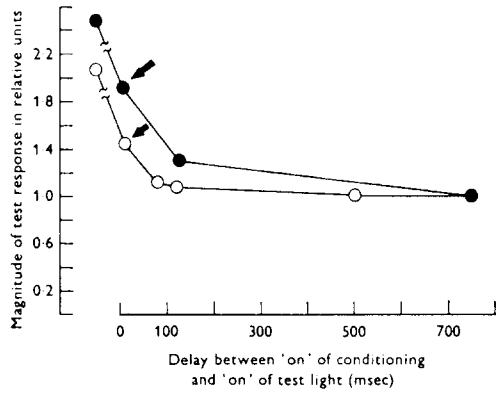

FIG. 43. Time course of gain change in cat ganglion cells: modified Crawford experiment. On a 12 deg steady background of retinal illumination $5 \cdot 10^3$ quanta(507 nm) $(deg^2\ s)^{-1}$ were centered: (a) a square wave modulated conditioning spot (1.5 s on, 3.5 s off), diam. 0.57 deg, retinal illumination $2.5 \cdot 10^5$ quanta$(deg^2\ s)^{-1}$; (b) a test spot, flashed on for 20 ms in one cell (filled circles) and for 50 ms in another cell (empty circles), of diam. 0.1 deg, with retinal illumination $3 \cdot 10^8$ quanta(507 nm) $(deg^2\ s)^{-1}$. The test stimuli were presented with varying delays with respect to the onset of the conditioning light. Plotted in the figure for two different on-center cells are the magnitudes of the responses to the test pulses of light at different delay times between onset of conditioning light and onset of test pulse. The arrows point to the responses to the test stimuli which generated responses which coincided in time with the peak of the response to the conditioning light. From Enroth-Cugell and Shapley (1973a).

In this experiment retinal gain is probed by measurement of the magnitude of the response to a brief test pulse of light. The test pulse is applied in the dark and at various times after a conditioning light is turned on. The results of an experiment of this sort are shown in Fig. 43 from Enroth-Cugell and Shapley (1973a). The response to the test pulse of light is the same a half second after the conditioning light is off as it is when the conditioning light has been off for several minutes. The response is definitely reduced within 200 ms from the time the conditioning light is turned on.

Between zero and 200 ms, the response declines uniformly in magnitude. In related experiments, it was shown that the gain stayed at the same new value from 200 ms until 5 s after the conditioning light was turned on. These experiments were done in Y cells under low scotopic conditions.

Similar results have been obtained in X cells of the cat, by Saito and Fukada (1975), who studied the time course of gain adjustment in X and Y cells under mesopic or low photopic conditions. There is a gap in our knowledge about the time course of gain adjustment in X cells under scotopic conditions, and a question about whether the time course of adaptation changes markedly between scotopic and photopic levels.

Saito and Fukada (1975) found that Y cells showed a much sharper, more transient, gain reduction immediately after the conditioning light was turned on, and then again a reduction immediately after it was extinguished, as indicated in Fig. 44. This was at higher levels of background illumination than the earlier experiments of Enroth-Cugell and Shapley (1973a), which may explain the differences in findings. The gain reduction at both "on" and "off" of the conditioning light seen by Saito and Fukada may possibly have been due to one of the "amacrine" gain controls we have postulated, either the contrast gaincontrol or the steady-state flux gaincontrol of the Y cell center. It seems more likely that the contrast gaincontrol is involved in this phenomenon. The contrast gaincontrol contains the kind of nonlinearity which would cause reduction at both "on" and "off"; it is an even-order nonlinearity which generates responses of the same sign at "on" and "off", responses like those seen in amacrine cells and Y cells. It is interesting that similar gain reductions at "on" and "off" are seen psychophysically in the original Crawford (1947) experiment and in intraretinal recording from the amacrine cell layer (Gordon and Graham, 1973; also unpublished results). It may be that transient gain reductions in

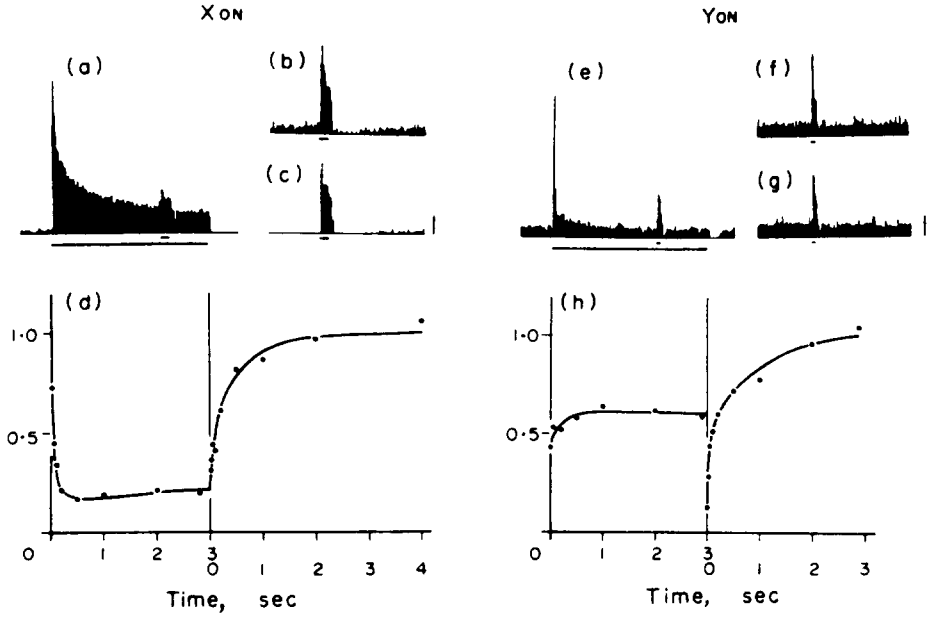

FIG. 44. Crawford experiment for X and Y cells under low photopic or high scotopic conditions. X cell data are in (a) – (d); Y cell data are (e) – (h). PST histograms show averaged responses to the test spot (short bar under histograms indicates stimulus time) and to the conditioning spot (longer bar under histograms). The background luminance was 5.9 cd m^{-2} through a 4 mm diameter artificial pupil. The unattenuated stimuli were : test, $3.9 \cdot 10^3$, and conditioning, $6.8 \cdot 10^3$ cd m^{-2}. They each subtended 5′ diameter. The test spot was placed in the middle of the receptive field center, while the conditioning spot was placed 15′ to the side, still within the center. (a) and (e): the test spot was presented 2 s after onset of the conditioning spot. (b) and (f): test spot alone, as a control. (c) and (g): test spot presented 2 s after offset of the conditioning spot. The duration of the test spot was 200 ms for the X cell, 100 ms for the Y cell. The duration of the conditioning spot was 3 s. The vertical calibration is 50 impulses s^{-1}. (d) and (h): The time courses of the change of magnitude of the response to the test caused by presentation of the conditioning spot. The relative magnitude of the response is plotted against the delay between the onset times of test and conditioning spots. From Saito and Fukada (1975).

Y cells, due to substantial amacrine input to these cells, are the basis for the psychophysical "on – off" transient sensitivity losses.

While the above results indicate the rapidity of some gain control processes in the retina, they do not rule out the existence of slower gain controls. These slower processes have not been studied with the Crawford paradigm, but they should be. Adelson's (1982) results on the time course of human light adaptation indicate the presence of slower adaptation processes than have been studied in ganglion cells up to now. It is well known but poorly documented that when the mean level of illumination is stepped up or down by two or more log units in an adaptation experiment, the gain may take several minutes to settle down or up to its new steady value. Such slow adaptation processes require further research.

4. GAIN AS A FUNCTION OF ILLUMINATION IN AMACRINE CELLS, BIPOLAR CELLS, AND HORIZONTAL CELLS

The phenomena of gain control in ganglion cells require an explanation in terms of the functional connections of the retinal network and/or the intrinsic properties of the receptors and interneurons in the retina. The fundamental question is, where does the control of gain begin? Ideally, one would want to have the answer to this question for the cat retina from which so much of the results on gain control in ganglion cells have been obtained. While there are some fragmentary results on the cat, the technical difficulties of intracellular recording have prevented a comprehensive study of gain control in cat retinal interneurons. We will therefore concentrate on the results from the retinas of two cold-blooded vertebrates which have been studied the most: the mudpuppy (Normann and Werblin, 1974; Werblin, 1974; Werblin and Copenhagen, 1974; Thibos and Werblin, 1978a, b) and the channel catfish (Naka *et al.*, 1979).

4.1. Amacrine Cells

In the mudpuppy the amacrine cells have a steep response versus illumination curve which can be shifted along the log illumination axis by steady background illumination. This is illustrated in Fig. 45 from the work of Werblin and Copenhagen (1974). This "curve shifting" is evidence for a gain control at or prior to the amacrine cell (see Section 1.2.2.).

In the catfish, Naka *et al.* (1975) divided the amacrine cells into two classes, the type N and type C cells. The type C cells correspond to what Werblin and colleagues (Werblin and Dowling, 1969; Werblin and Copenhagen, 1974) call amacrine cells;

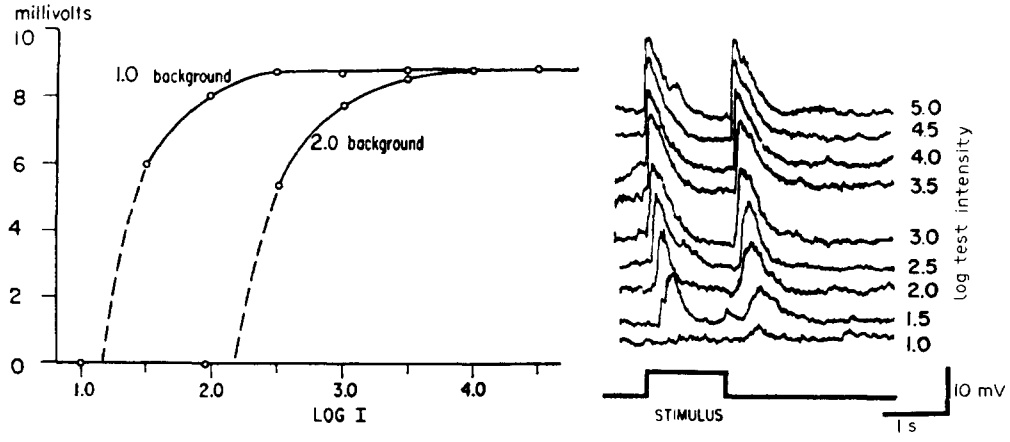

FIG. 45. Curve-shifting in mudpuppy amacrine cells, recorded intracellularly. Diffuse illumination was used as a test stimulus. The dashed curves are inferred in regions where no data were taken. The peak value of the response is plotted against log flash illumination. The retinal illumination from the tungsten source used was about one quantum per rod per second for $\log I = 0$. This works out to roughly $6 \cdot 10^3$ quanta (522 nm) $(mm^2\ s)^{-1}$ for $\log I = 0$. From Werblin and Copenhagen (1974).

they produce "on – off" responses to spots or full-field flashes. The type N cells are like the so-called "sustained" amacrine cells found by Kaneko (1973) and Naka and Ohtsuka (1975). The change of gain of these two types of cell with background is illustrated in Fig. 46 from Naka *et al.* (1979). They clearly are affected similarly by backgrounds and also greatly resemble the ganglion cells in the way they adapt. That is, the gain is approximately the reciprocal of background, approximately Weber's Law.

We have to make a brief digression to make clear that the results in Figs 45 and 46 were obtained in completely different ways. Werblin and Copenhagen (1974) used standard rectangular increments of light on a steady background, and measured the peak of the change in intracellularly recorded membrane potential. Naka *et al.* (1979) measured first-order Wiener kernels by cross-correlating a white-noise modulated light stimulus with the resulting "noisy" modulation of the cells' membrane potential. The gain for the type N cells, and for all the other cells the results of which are shown in Fig. 46, is the gain at the peak of the first order Wiener kernel. The peak value of the kernel (first or second order) at each background level, divided by the standard deviation of the white noise stimulus, may be taken as a measure of gain at that background level.

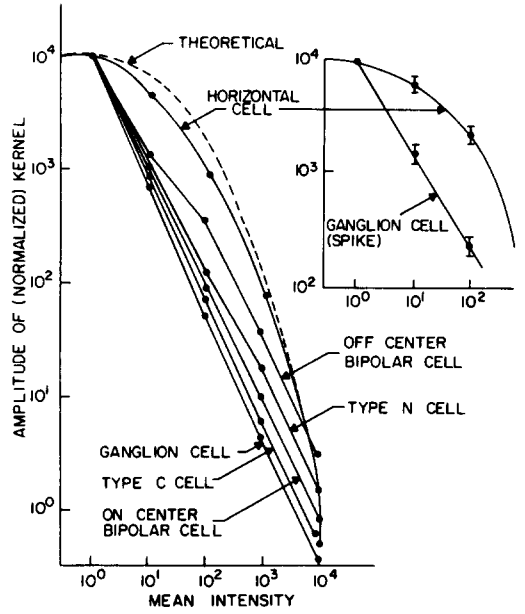

FIG. 46. Incremental gain of catfish neurons, recorded intracellularly. The ordinate is the incremental gain, the amplitude of the first order Wiener kernel normalized by the magnitude of the white-noise modulation. The horizontal coordinate is the mean illumination. The dotted line is a prediction from the Naka – Rushton relation for the gain if there were no adaptation, just saturation. Inset is an average of ten simultaneous recordings from horizontal and ganglion cells. Results for various cell types are labeled in the figure. We believe the lowest mean intensity was a retinal irradiance of 0.1 μ watts cm^{-2} on the retina. The light source was in most cases a glow – modulator tube which was attenuated by neutral density filters. From Naka *et al.* (1979).

4.2. Bipolar Cells

The bipolar cells form the main link between photoreceptors and ganglion cells and thus the dependence of gain on background for these cells is of crucial importance for understanding the site of adaptation.

The available data on gain control in bipolars come mainly from Naka *et al.* (1979) and from Thibos and Werblin (1978a). The results of Naka *et al.* (1979) on catfish bipolars are displayed in Fig. 46, on the same graph as the amacrine cell data. Clearly, the bipolars' gain begins to drop at about the same level of illumination as the amacrines, and falls with a similar but somewhat shallower slope. Since bipolar cells are the input to amacrine cells in catfish (Naka and Ohtsuka, 1975) as in other animals (Dowling and Boycott, 1965), it is reasonable to suppose that the dependence of gain on background observed in the amacrines is already largely determined at the bipolar level.

The results on light adaptation in bipolars in mudpuppy (Thibos and Werblin, 1978a) have concentrated on the way in which the surround of the bipolar receptive field sets the gain of the center. This is illustrated in Fig. 47, which shows response vs log illumination under two conditions: (i) no background illumination; (ii) background illumination falls on the bipolar's surround. In case (i), the intensity – response function is approximately fit by the Naka – Rushton relation, equation (6). In case (ii), the response vs illumination curve of the same form is shifted to

the right as would follow from gain re-adjustment by illumination of the surround (see Section 1.2.2.). Control experiments established that this was not a result of light scatter onto the center (Werblin 1974). This is evidence that, in the mudpuppy retina, the gain of the bipolar cell is set by signals coming from its surround, which in this species is believed to be mediated by horizontal cells (Werblin and Dowling, 1969; Thibos and Werblin, 1978a). Such results suggest that, in the mudpuppy, horizontal cells act as a gain control on bipolar cells. Two points of comparison with previously presented psychophysical and physiological results are needed here, to prevent the (probably erroneous) inference that this conclusion is generally applicable to all vertebrates.

The first comparison of Fig. 47 is with the sensitization phenomenon in human vision discovered by Westheimer (1965). The results in Fig. 47 are the *opposite* of Westheimer sensitization. Illumination in the periphery of the mudpuppy bipolar's receptive field *desensitizes* the center in Werblin's (1974) and Thibos' and Werblin's (1978a) experiments. There is, however, a puzzling and unresolved contradiction with Burkhardt's (1974) report of sensitization in mudpuppy bipolar cells. Perhaps it has to do with different receptor input to the bipolar cells in the two sets of experiments. Thibos and Werblin (1978a) were working at low backgrounds at which rods were the predominant input while Burkhardt was working with a highly light adapted mudpuppy retina in which it is probable that cones were the predominant photoreceptor inputs to the proximal retinal neurons. If this is the explanation for the opposite results, it would be an interesting and unusual example of a reversal in functional characteristics because of the transition from rod to cone pathways.

A second comparison worth pursuing is that between the strong effect on the gain of the center exerted by the surround of mudpuppy bipolar cells (Fig. 47) vis-a-vis negligible or, at most, weak effect of the surround on the gain of the center in X and Y cat retinal ganglion cells (Figs 38 – 40). This difference between results on the spatial extent of "adaptation pools" suggests that quite different mechanisms are involved in the control of gain in the mudpuppy and cat retinas. In a sense such a difference would not be surprising because the mudpuppy and cat have evolved quite differently with widely different visual capacities. The details of exactly how the retinal network is connected spatially to regulate gain might well differ between two such distantly related animals.

Ashmore and Falk (1980) have demonstrated that the gain of bipolar cells, in the almost all-rod retina of the dogfish, begins to drop at extremely low backgrounds because of saturation in the bipolar cell itself. In the dogfish retina, there is a very high amplification of rod signals at the rod-bipolar synapse, and as a result the bipolar cells approach their response ceilings at very low backgrounds. There is no evidence for a gain control, and therefore true light adaptation, in these experiments. However, the results of Werblin (1974) and Naka *et al.* (1979) illustrate how an automatic gain control, acting on signals from photoreceptors to bipolar cells, staves off saturation in mudpuppy and catfish bipolars. In Fig. 46 for example, the catfish bipolar's gain begins to drop at a lower mean level than the horizontal cell's gain, presumably due

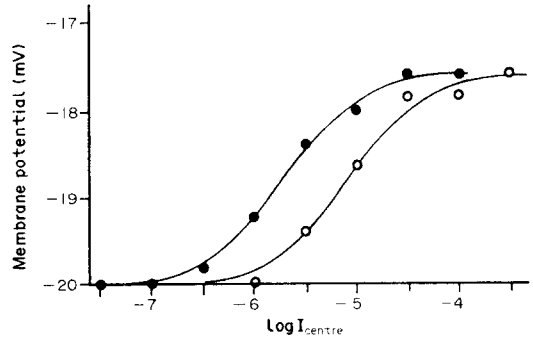

FIG. 47. The effect of a steady illumination in the periphery of the receptive field on the stimulus – response function of mudpuppy bipolar cells; intracellular recording. The stimulus was a flashing spot centered on the receptive field of the bipolar cell. The spot's diameter, 0.4 mm, was chosen to stimulate the center optimally. Stimulus duration was 1 s. The response measure was the steady state plateau of the 1 s response. Filled circles are for no illumination in the periphery of the receptive field; empty circles are response magnitudes when a −4 log unit steadily illuminated annulus was placed in the periphery of the field. A modified Naka – Rushton function was fitted to the data in the condition of no peripheral illumination, and then translated laterally to fit the results obtained with the annulus. The light source was a tungsten lamp, attenuated by neutral filters. The unattenuated retinal illumination was calculated to be about 10^{13} quanta(522 nm) $(cm^2\ s)^{-1}$. Illuminations are given as log attenuation relative to this value. From Thibos and Werblin (1978a).

to a neural gain control acting on the bipolar and not on the horizontal cell. But at higher levels of mean illumination, the horizontal cell potential starts saturating and its gain plummets, falling below the bipolar's gain. Since bipolar cells appear to have steeper intensity – response functions than horizontal cells (Werblin, 1974), they might saturate at even lower mean levels than the horizontal cells, were it not for the saving action of the gain control. The automatic gain control causes the bipolar cells (and the more proximal retinal neurons they feed) to lose some gain at lower levels in order to preserve gain at higher levels which would otherwise be lost because of saturation (see Section 1.2.2.).

4.3. Horizontal Cells

There is quite a lot of information on the control of gain in horizontal cells because they are easier to record from intracellularly than the other retinal interneurons. Since horizontal cells are anatomically one synapse away from photoreceptors, they often mimic the receptors' adaptational properties. One of the significant outcomes of this fact is that the regulation of gain in photoreceptors may be inferred, with caution, from studies of the regulation by light of the gain of horizontal cells. We can begin to see some of the diversity in photoreceptor adaptation in the variety of horizontal cells' adaptational behavior (cf. also Section 5).

Perhaps the best studied receptor – horizontal cell system is in the turtle retina. Results on adaptation in turtle horizontal cells are quite clear, as shown in Fig. 48 (Normann and Perlman, 1979b). The intensity – response curves shift to the right with background, and the gain follows Weber's Law. These horizontal cells are driven exclusively by long-wavelength cones, and their adaptational properties are mainly determined by their cone inputs (Normann and Perlman, 1979a). The curve-shifting in Fig. 48 is clear evidence for adaptation of the type suggested in equation (10), with an automatic gain control located in the cones.

To illustrate inter-species diversity we compare the turtle horizontal cell with the horizontal cell of the catfish retina, the gain vs background curve of which is illustrated in Fig. 46. Here the horizontal cell curve is not like the curve of Weber's Law. Rather, it follows the dashed curve, which is a prediction based on the Naka – Rushton relation [equations (6) and (7)]. That is, the loss of gain is caused by *saturation*. Under the conditions of these experiments, the catfish horizontal cells were driven exclusively from long-wavelength cones (Naka,

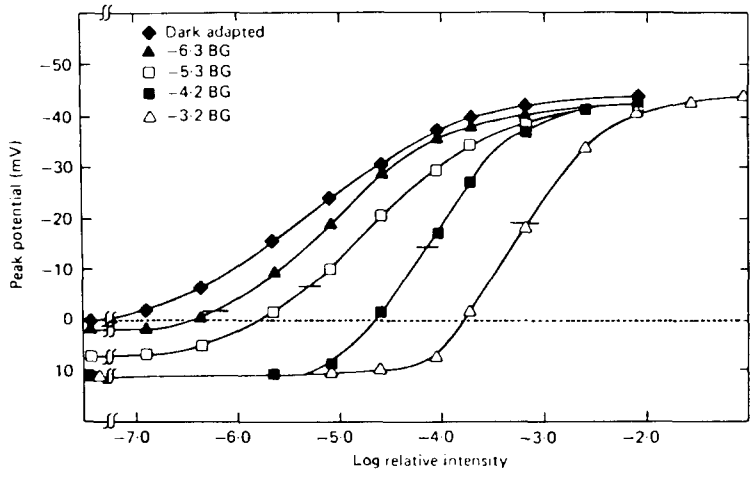

FIG. 48. Adaptation in turtle horizontal cells; intracellular recording. The stimuli were 0.5 s increments or decrements on a background, except for the results on the dark adapted eye, which were increments only. The stimuli were large 3.2 mm diameter spots on the retina. Peak responses measured from the dark-adapted resting potential (dotted line) were plotted as a function of test illumination. The curves were drawn by eye. The horizontal bar through each curve indicates the steady membrane potential measured at least two minutes after background onset. The illuminations are given as log attenuation. The unattenuated test stimulus (0 log) was $6.4 \cdot 10^{15}$ quanta(640 nm) $(cm^2\ s)^{-1}$ on the retina. The unattenuated background illumination was $9.1 \cdot 10^{15}$ quanta(640 nm) $(cm^2\ s)^{-1}$. From Normann and Perlman (1979b).

personal communication). Figure 46 indicates that horizontal cells, and by inference the cones, of the catfish retina *do not* adapt but merely saturate, but that bipolar cells, amacrine, and ganglion cells do adapt. In the catfish, much of the adaptation to steady light must take place between cones and bipolar cells, though Fig. 46 also implies that there are additional stages of gain control in the inner plexiform layer.

There are data on the dependence of gain on background in cat horizontal cells, under conditions such that the responses were due to photoreceptor input from rods only. The graph in Fig. 49 indicates that cat horizontal cells, driven by rods, have a gain vs background dependence which approximately follows Weber's Law above 1 td [Note this is a "cat td" and therefore is equivalent to about $8 \cdot 10^5$ quanta(507 nm) $(\text{deg}^2\ \text{s})^{-1}$; Steinberg, 1971]. Note that these horizontal cells typically receive mixed photoreceptor input (Steinberg, 1971), even though their major direct synaptic contact is with cones (see Appendix 1). This suggests that rod – cone coupling is indeed important for determining the response properties of horizontal cells. In comparing horizontal cell responses with ganglion cell responses (Fig. 24), one notices that horizontal cell gain does not begin to drop until the background is 1 td, which is two to four log units higher than the transition level of illumination for ganglion cells. This suggests that in the cat, as in the catfish, there must be gain control mechanisms more proximal in the retina than the rods or the horizontal cells. However, the Weber Law behavior of horizontal cells in the cat suggests that the rods may also adapt when the level of illumination is high enough (but see Section 5). Gain versus background curves are not available for cone-driven horizontal cell responses in cat.

The diversity of horizontal cell gain changes due to backgrounds indicated so far is representative of that seen generally. For instance, in mudpuppy the horizontal cells behave as if influenced by saturation and adaptation in the scotopic range, but resemble turtle horizontal cells — almost pure adaptation — in the photopic range (Normann and Werblin, 1974). However, carp horizontal cells show only saturation in the photopic range (Witkovsky, 1967). But the rod-driven skate horizontal cells do adapt to light by gain reduction after an initial period of saturation (Dowling and Ripps, 1971). There is not one story for all vertebrates. The proximity of horizontal cells to photoreceptors suggests that the receptors might also be very diverse in how they deal with changes in mean or background illumination. This expected diversity is found.

5. GAIN CONTROL IN PHOTORECEPTORS

Some photoreceptors adjust their gain by adapting in the presence of steady illumination and only saturate in very bright light, and other photoreceptors adapt very little before they saturate. This diversity of receptor function with respect to gain control and saturation has been suggested already in the discussion of gain controls in horizontal cells. In order to organize this diverse material, we will present the data on receptors as follows: (a) photoreceptors which adapt a lot *and* also saturate; and (b) photoreceptors which adapt very little and mainly saturate.

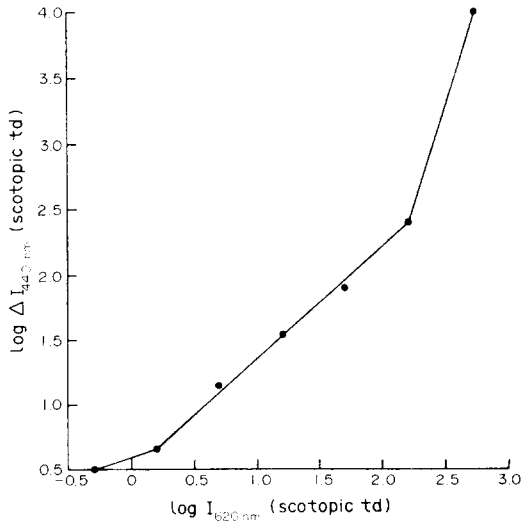

FIG. 49. Log test illumination (at 440 nm) plotted as a function of log background illumination (620 nm) for a constant amplitude peak horizontal cell response intracellularly recorded in the cat retina. The background duration was 5.5 – 6.5 s; the stimulus was 0.5 s in duration applied after the response to the background had settled to a constant value. The stimulus was a spot on a larger background. The criterion response was 2.25 mV, and was rod-driven. From Steinberg (1971).

5.1. Photoreceptors which Adapt a lot and Saturate

The photoreceptors which actually adapt, i.e. more or less rapidly adjust their gain to avoid saturation, include the following *rods*: skate, (Dowling and Ripps, 1972); gecko, (Kleinschmidt and Dowling, 1975); toad (Fain, 1976; Baylor *et al.*, 1979). The adapting *cones* include: turtle (Baylor and Hodgkin, 1974; Normann and Perlman, 1979a); mudpuppy (Normann and Werblin, 1974); frog (Hood and Hock, 1975); ground squirrel (Dawis and Purple, 1982); perhaps monkey (Valeton and van Norren, 1983). The results on cone adaptation in ground squirrel, frog, and monkey are from massed potential recordings which in one way or another isolated the cones.

Figure 50 illustrates photoreceptor adaptation in rods from the toad *Bufo marinus* (Fain, 1976). There is evidence of saturation (in the decline of the peak response on background illumination), but there is also clear curve-shifting of the V-log I curve, consistent with the idea of an automatic gain control, equation (10) (cf. Normann and Werblin, 1974; Dawis and Purple, 1982). Furthermore, the gain of the toad rod follows Weber's Law (Fain, 1976), and this can even be seen in the photocurrent recorded from the outer segment by the suction electrode technique (Baylor *et al.*, 1980) as in Fig. 51. In Fig. 51 the vertical axis is labeled "sensitivity", but it is equivalent to what we have defined as "gain". Log gain vs log background has a flat portion and then a declining portion with a slope about -1. The slope of unity suggests an adaptive gain control rather than the steeper slope associated with pure saturation (see Section 1.2.2.). The transition from flat to sloping is at about an illumination of 0.28 quanta μm^{-2} s^{-1}, which is about forty times greater than the "dark light" of the rods, the spontaneous current fluctuations in the dark, measured independently. Baylor *et al.* (1980) make the point that this implies that the gain control transition level in rods is not determined by the "dark light". Similarly, we have pointed out in connection with gain reduction in ganglion cells, that the "dark light" in the cat retina is too small to be responsible for determining the illumination at which gain starts to fall (see Section 3.1.1.).

The gain reduction of the rod photocurrent is accompanied by a very significant speeding up of the rod response. The gain declines by about a factor of one hundred over a range of backgrounds which speed up the time to peak of the photocurrent (in response to a brief flash) by a factor of seven.

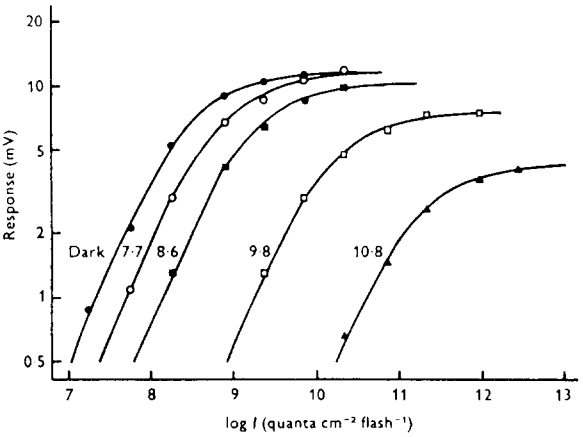

FIG. 50. Effect of background light on the intensity–response curves of toad rods; intracellular recording. Peak response amplitude to a brief (around 100 ms) flash is plotted against log flash energy per unit area (quanta cm^{-2}), for the dark adapted rod and for several background levels of illumination. The stimulus and background were diffuse. The illumination of the backgrounds is given in log units to the left of each curve, and the units are log quanta(505 nm) $(cm^2\ s)^{-1}$. From Fain (1976).

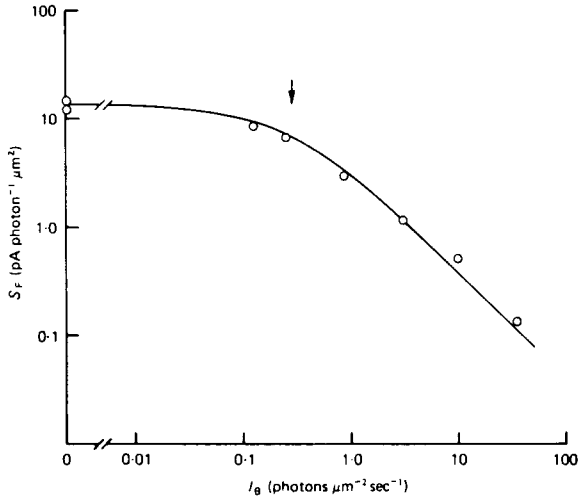

FIG. 51. Gain vs background in the photocurrent of a toad rod outer segment; recorded with suction electrode. The gain is plotted on the vertical axis with units picoamps/(photon μm^{-2}), but could really be given in picoamps/photon since the rods are of fixed cross-sectional area. The background illumination is plotted on the horizontal axis. The transition from horizontal to sloping portions of the curve occurs at 0.28 quanta(507 nm) $(\mu m^2\ s)^{-1}$. The arrow points to this illumination. Background and test were diffuse. From Baylor *et al.* (1980).

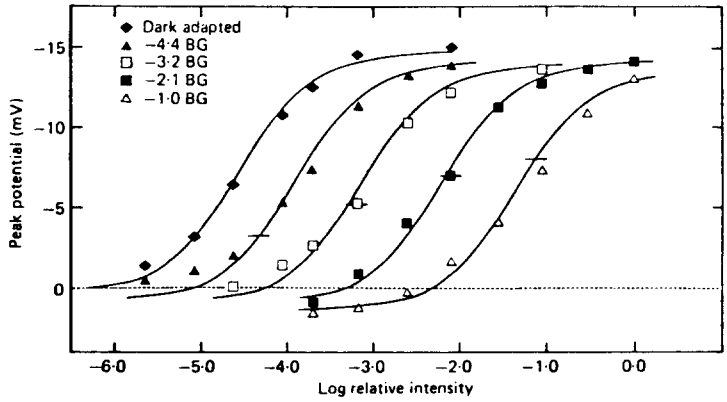

FIG. 52. Gain adjustment in turtle cones (recorded intracellularly) caused by background illumination. The stimuli were 0.5 s increments or decrements on a steady background (except for the curve for the dark adapted cone which only is for increments). The stimulus spot was 3.2 mm in diameter on the retina. Peak responses measured from the dark-adapted resting potential (dotted line) were plotted as a function of test illumination. The curves are Naka – Rushton functions. The horizontal bar through each curve indicates the steady membrane potential measured at least two minutes after background onset. The illuminations are given as log attenuation. The unattenuated test stimulus (0 log) was $6.4 \cdot 10^{15}$ quanta(640 nm) $(cm^2\ s)^{-1}$ on the retina. The unattenuated background illumination was $9.1 \cdot 10^{15}$ quanta(640 nm) $(cm^2\ s)^{-1}$. From Normann and Perlman (1979a).

Baylor *et al.* (1980) found that $G_R \propto (t_{peak})^{2.5}$, where G_R is the gain of the rod in picoamp/quantum and t_{peak} is the time from the onset of the brief flash stimulus to the peak of the response.

Adjustment of gain in cones by adaptation and saturation is represented in Fig. 52 from the data of Normann and Perlman (1979a). The parallel curves are the response template from the dark adapted cone, and are described by the Naka-Rushton relation, equation (6). When the background is raised, the response to increments is somewhat compressed (consistent with saturation) because the steady state response increases towards the maximal potential the cone is capable of producing, as indicated by the short horizontal bars intersecting each operating curve. From the shift of the operating curves, one may infer Weber's Law since there seems to be about a one log unit shift for each log unit increase in background. That turtle cones approximately follow Weber's Law is indicated in Fig. 53 from Baylor *et al.* (1974b). The solid line is Weber's Law up to about $\log I_S = 4.5$, and then rises due to saturation. It is based on a theory for the cone's gain control mechanism by Baylor *et al.* (1974b). Actually the cone's gain follows Weber's Law for about a log unit more than the Baylor *et al.* theory predicts, and saturates rather less than the theory predicts. One possibility not considered by Baylor *et al.* is that some of the

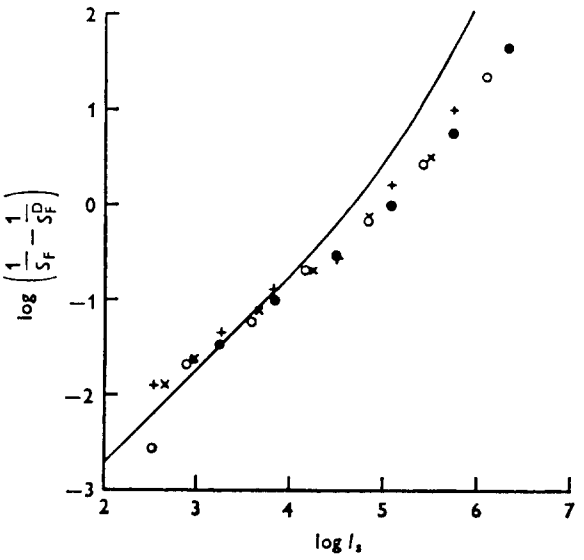

FIG. 53. Turtle cones' gain as a function of background; intracellular recording. The ordinate of this unique plot is the logarithm of the difference between the reciprocal of the gain in the light adapted cone and the reciprocal of the gain in the dark adapted cone, i.e. $\log\{1/S_F - 1/S_F^D\}$. This number unfortunately goes to minus infinity when the background is so low that the gain of the cone is the same as in the dark adapted state, but that region of the curve is not plotted here anyway. The gain S_F is in μV quantum^{-1} effectively absorbed. The horizontal axis is log background flux; the units are quanta/sec effectively absorbed by the cone. The stimuli were 10 ms flashes, and the gain is calculated for the peak of the flash response. These are data from a red turtle cone. The smooth curve is from Baylor *et al.*'s theory for cone adaptation. From Baylor *et al.* (1974).

deviation at the high backgrounds might be due to pigment bleaching, which is not included in their theory. Cones also speed up their response with background and for turtle cones, $G_C \propto (t_{peak})^4$, that is the gain of a cone decreased like the fourth power of its time to peak for the response to a brief flash. The total amount of "speeding up" of the cone response is about a factor of two over a range of backgrounds in which the gain drops by a factor of two hundred. The speeding up of rod and cone responses with the change in gain is illustrated in Fig. 54 (Baylor and Hodgkin, 1974; Baylor *et al.*, 1980).

Steady illumination affects different parts of the cone's response in different ways. This is illustrated in Fig. 55 from Baylor and Hodgkin (1974). The impulse response of the cone, i.e. the response to a brief, weak flash of light in the linear range, changes as the steady background increases. The gain for later parts of the impulse response drops, but the gain for the initial rising phase of the response is relatively unaffected by steady backgrounds. The peak of the response becomes smaller at higher backgrounds and occurs at earlier times after the flash, but the rising phases of impulse responses at several levels of background superimpose. Although this feature may not be completely convincing in Fig. 55, it is true. Recent preliminary experimental results involving the measurements of temporal frequency responses in turtle horizontal cells and cones are consistent with the resistance of the rising phase of the impulse

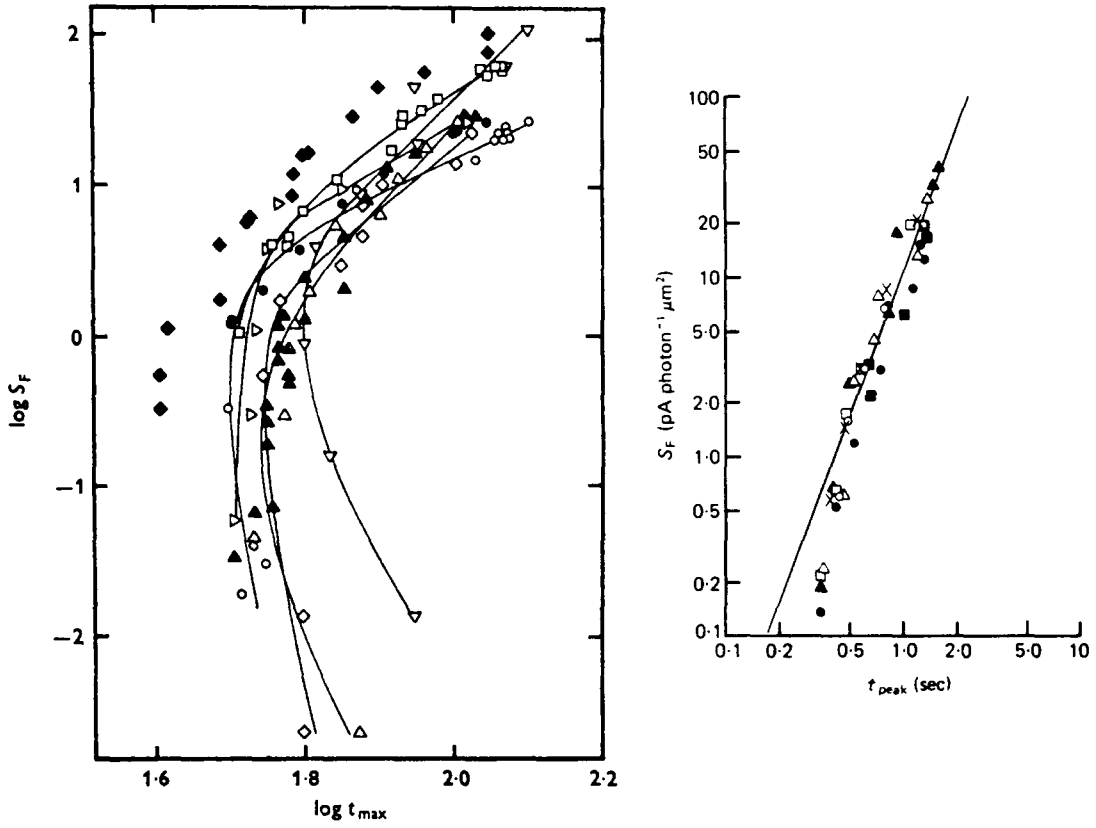

FIG. 54. The speeding up of cone and rod responses in the turtle and toad retinas. As mean illumination increases, the gain and the time to peak decrease. In the left panel, the log of the gain is plotted against the log of the time between the peak response to a brief flash and the onset of the flash, for turtle cones (intracellular recording). The gain is plotted against peak time for toad rods (suction electrode recording) in the right panel. For about two log units log t_{max} is approximately proportional to log gain (here denoted S_F). In the left panel, the different symbols are for different red cones, eight in all. On the right are graphed data points from seven different rod outer segments. Note that the cone time to peak stops decreasing when the gain drops below 0 log. The units of the left ordinate are log[μV (quanta μm^{-2})$^{-1}$]; on the right the units are picoamps (quanta μm^{-2})$^{-1}$. On the left the units of the abscissa are log[ms]; on the right they are simply s. From (left) Baylor and Hodgkin (1974), and (right) Baylor *et al.* (1980).

response to adaptation (Tranchina, Gordon and Shapley, unpublished).

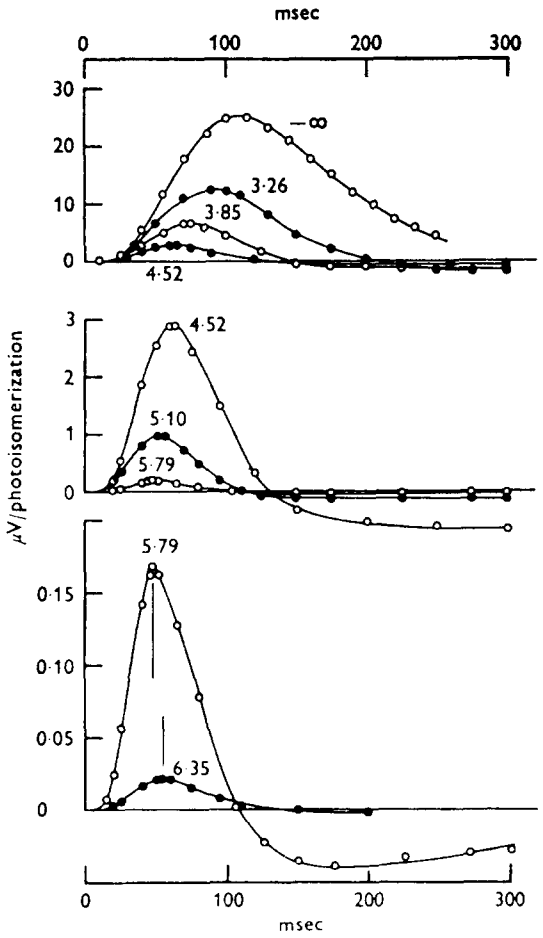

FIG. 55. Effect of increasing illumination of conditioning step on the time course of the flash response in a turtle cone (intracellular). The flash was 11 ms in duration, and was applied 1.1 s after the onset of a conditioning step of illumination. The horizontal axis is the time after flash onset. The vertical axes are all in units of gain: μV/photoisomerization, which is equivalent to μV/quantum effectively absorbed. The numbers near each curve are the illuminations of the conditioning steps, in units of log[effective quanta(cone s)$^{-1}$]. These data are from a red cone. The stimulus was a 150 μm white spot. From Baylor and Hodgkin (1974).

5.2. Photoreceptors which Saturate but do not Adapt

The best studied examples of receptors which saturate without adaptation are the rods of the mudpuppy studied by Normann and Werblin (1974). Figure 56 shows their results. It can be seen that the rod operating curves shift to the right a very little with increasing background, but mainly collapse due to saturation. Similar receptor behavior is implied by the horizontal cell results of Witkovsky (1967) in carp and Naka *et al.* (1979) in catfish. Recently, Nunn and Baylor (1982) have reported that rods in the monkey *Macaca fascicularis* mainly saturate without adapting. There is supportive evidence from mass receptor potential recording in the rat that at least some mammalian rods saturate without adapting (Penn and Hagins, 1972; Green, 1973). This is also consistent with the very small amount of adaptation in the a-wave of the rat's ERG, thought to be determined by receptor and horizontal cell responses (Dowling, 1967), and by mass receptor potential recording in the cat eye in which the inner nuclear layer and the ganglion cell layer were rendered anoxic by occluding the ophthalmic artery (Sakmann and Filion, 1972). Sakmann and Filion also showed that the gain of the isolated rod receptor potential was

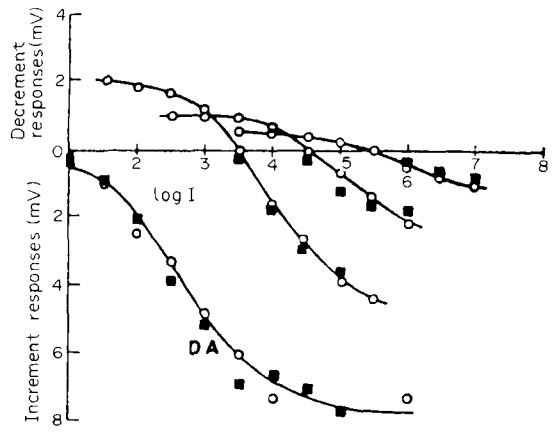

FIG. 56. Rod operating curves on backgrounds. Plotted is the magnitude of the rod response (recorded intracellularly) measured as the difference between the peak of the response and the steady polarization just before the response. The response to test flashes brighter than the background are shown below the horizontal axis, and to test flashes dimmer than the background above the axis. The stimuli were diffuse 2 s flashes. The background illuminations can be read as the test stimuli which produced zero response, i.e. the intersection of the curve with the horizontal axis. Circles are from a rod in a normal retina, and squares are from a rod in a retina treated with aspartate. The units of the horizontal axis, which indicates the test flash illumination, are as follows: $6 \cdot 10^5$ quanta(522 nm) (cm^2 s)$^{-1}$ when log $I = 0$. From Normann and Werblin (1974).

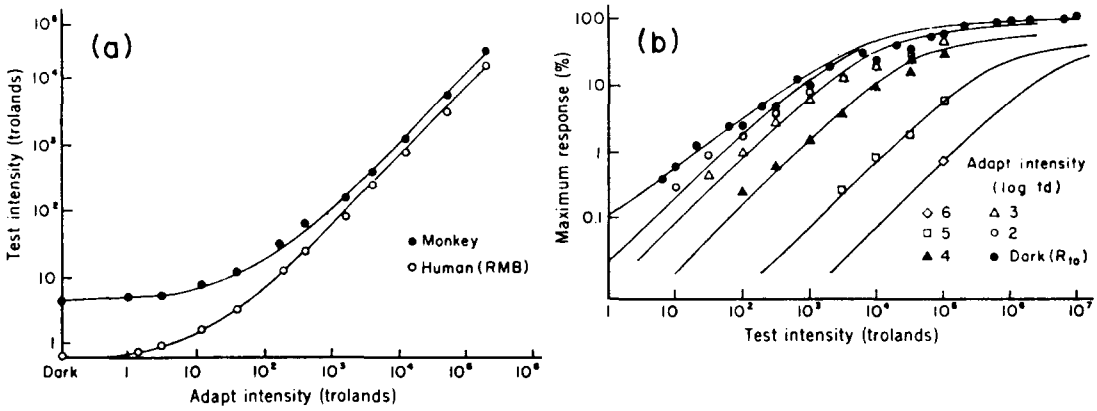

FIG. 57. Adaptation and saturation in the monkey cone receptor potential (extracellular recording of a mass response). (a) The retinal illumination required to reach a criterion response in the monkey's late receptor potential is plotted against background illumination. The criterion was 10 μV. Also shown are human psychophysical thresholds under the same stimulus conditions. In both cases, the stimulus was a yellow (580 nm) light presented on a yellow background. The background was on steadily for at least 5 min before test flashes were presented. The test stimuli were 150 ms in duration, presented in the fovea. The axes are given in photopic td.

(b) Stimulus – response curves under dark adapted conditions and for five background levels for the monkey late receptor potential. The adapting and test fields were both 1 mm spots, centered on the fovea. The adapting illuminations are indicated in the figure in log td(photopic). The data are the mean values from six animals. They have been normalized for between animal comparisons, as per cent of the maximum response for each animal. From Boynton and Whitten (1970).

approximately constant over several log units when ganglion cell gain was dropping like Weber's Law. Thus, mammalian rods and teleost cones seem to saturate without adapting. The anatomically larger rods and cones of amphibians and reptiles do adapt to light. Fain (1976) has suggested the necessity for adaptation in the morphologically larger receptors because of their larger quantum catching areas, and consequent overloading at lower levels of retinal illumination.

We have put off a discussion of mammalian cones to last because the evidence about them is indirect and somewhat equivocal. Figure 57 from Boynton and Whitten (1970) shows, in the right-hand panel, response – log I curves of the isolated cone receptor potential recorded with gross electrodes in the fovea of a monkey's eye in which the ophthalmic artery had been blocked. These curves may be accounted for solely in terms of a modified Naka – Rushton relation. Only saturation, called by Boynton and Whitten "response compression," was used to draw the curves through the points which were averages from results on six monkeys. On the left, data from another monkey are seen to follow Weber's Law. As we know from our previous discussion, in Section 1.2.2., saturation and Weber's Law are incompatible, and in fact upon inspection, Fig. 57(a) is not consistent with Fig. 57(b). If one constructs an incremental gain vs background curve from the data in Fig. 57(b), by choosing a constant response criterion, one obtains a curve quite unlike Fig. 57(a); it is more shallow at low backgrounds and steeper at high backgrounds. It is consistent with an explanation in terms of saturation. So we don't know which cone receptor potential (those in Fig. 57(a or b) is representative of mammalian cones. More recent investigation of the same preparation (Valeton and van Norren, 1983) indicates that an automatic gain control rather than saturation is the predominant factor in controlling gain in primate cones. But this is an important topic which will undoubtedly receive more attention.

6. THEORIES OF RETINAL GAIN CONTROL AND THE DETERMINANTS OF VISUAL SENSITIVITY

We have devoted this chapter up to this point to the questions "why" and "what" concerning the control of retinal gain and visual sensitivity. However, to deal with the question "How" it is done, we will at this point review theories of retinal

and visual function. Theories of light adaptation can be divided into two categories: (a) theories of the retinal gain control and (b) theories of how the gain control and noise combine to determine visual sensitivity in psychophysical or behavioral experiments. These will be discussed in turn.

6.1. Theories of the Retinal Gain Control

All theories of retinal gain control share the common feature that they attempt to account for a basic nonlinearity of vision, namely that the gain and time course of responses to time varying stimuli are dependent on the mean level of illumination, or the background illumination, to which the time varying stimuli are added. The nonlinearity of light adaptation is a gentle one, allowing a linear range of vision around the operating point of the mean level. Another way of saying this is that the mean level must be changed a lot to have a big effect on gain and time course. For instance, in the Weber Law range, in order to reduce the gain to half its value at one mean level, the value of the mean must be doubled. This is a large change in mean level. Thus for normal vision in the real world of (Weber) contrasts, which are in the range $0-0.5$, the response of the visual system will not be thrown into a very nonlinear range by the processes of retinal light adaptation. In the models to be considered, adaptation acts like a nonlinear negative feedback: a negative feedback because increasing the output reduces the gain of earlier stages; nonlinear because the feedback signal is not added or subtracted but is used as a gain control and a controller of the time constants of response.

6.1.1. Fuortes and Hodgkin's Model for Photoreceptors

Fuortes and Hodgkin (1964) originally developed a theoretical model to account for the relation of gain and time course of response in photoreceptors of the horseshoe crab, *Limulus polyphemus*. We have excluded the topic of light adaptation in invertebrates from our review, for obvious reasons of length control. The photoreceptors of invertebrates are quite different from those of vertebrates; most of them depolarize in response to increases in illumination rather than hyperpolarize as do vertebrate rods and cones. However, the Fuortes–Hodgkin (FH) model has crossed phylogenetic boundaries. Suitably modified, it has been used by Baylor *et al.* (1974a, b) and Baylor *et al.* (1980) to analyze the gain and time course of turtle cones and toad rods, respectively. It is also related to models developed for the cat retina by Enroth-Cugell and Shapley (1973a) and Shapley and Victor (1981), and to a model for human vision proposed by Sperling and Sondhi (1968), and therefore is a useful starting point.

The FH model is composed of a sequence of stages of temporal integration connected in series, as diagrammed in Fig. 58. Each stage is represented as an RC circuit in the original model. However, a theory with the same formal structure can be devised for stages which are chemical reaction states reached by repeated catalytic reactions (Borsellino *et al.*, 1965). The time course of the response of stage j in the FH cascade is described by a differential equation which says that the buildup of response (or substance) j is proportional to a reaction rate constant a_1 times the amount of response (or substance) in stage j-1, minus the decay from stage j which is equal to another rate constant a_2 times the amount of substance in state j.

$$dy_j(t)/dt = a_1 y_{j-1}(t) - a_2 y_j(t). \tag{25}$$

In this scheme only the first stage is driven by light,

$$dy_1(t)/dt = Y \cdot I(t) - a_2 y_1(t) \tag{26}$$

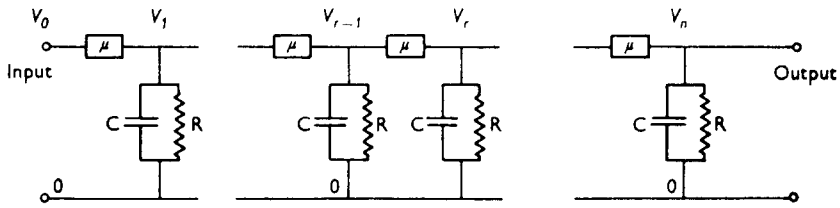

FIG. 58. Diagram of the Fuortes–Hodgkin (FH) model. The elements labeled μ represent isolation stages with gain μ. Light adaptation is assumed to affect only the value of R. From Fuortes and Hodgkin (1964).

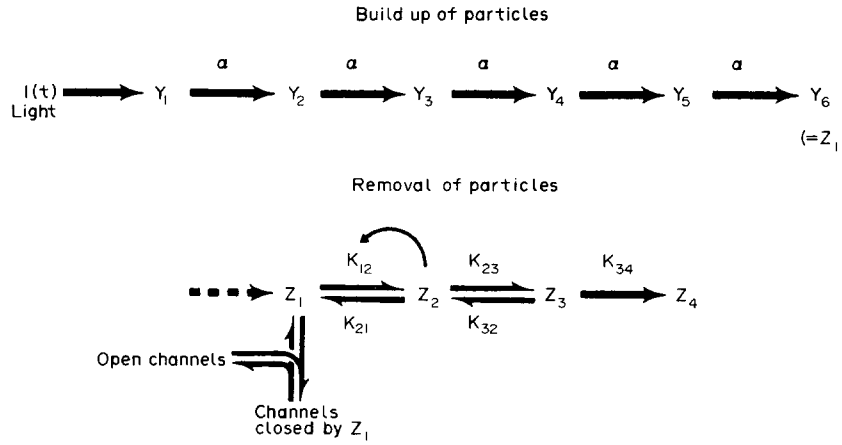

FIG. 59. This is a diagram of the Baylor – Hodgkin – Lamb (BHL) model. The blocking substance is denoted y_6 or z_1. Y is used for build-up, z for destruction of the blocking substance. Note that this scheme is fundamentally different from the FH model (Fig. 58) in that the rate constant for production of each y stage is the same as for its decay, except for the first and last stages. In the FH model, the rate constant for growth of each stage is different from the rate of decay, and only the decay rate is subject to change by adaptation. Here adaptation only works by changing the rate constant of decay from state z_1. From Baylor *et al.* (1974).

where Y is the gain of the initial transduction from light to y and $I(t)$ is the light stimulus. The last stage, the time evolution of which is given by $y_n(t)$, is presumed in the model to control the membrane conductance and thereby the electrophysiological response to light of the photoreceptor. In comparing equations (25) and (26) with Fig. 58, one should make the identification $a_1 = \mu/C$ and $a_2 = 1/\mathrm{RC}$.

This model was developed to explain first of all the many stages of temporal integration in the process of visual transduction, as reflected in the slow rise to the peak of the impulse response of photoreceptors. It does this very well. In turtle cones and toad rods the model indicates that the number of stages is from four to six. From the experimental evidence of the toad rod photocurrent recordings (Baylor *et al.*, 1980) probably all of the temporal integration is in the outer segment of the photoreceptor.

The FH model as represented in equations (25) and (26) is a linear model. That is, the sum of two inputs $I_1(t) + I_2(t)$ yields a response $y_{n1}(t) + y_{n2}(t)$ which is the sum of the responses to the two inputs presented alone — the principle of Superposition. However, Fuortes and Hodgkin (1964) described a modification of the model which would help explain observed departures from linearity, in particular the change of gain with changes in mean level of illumination which is the basis of photoreceptor light adaptation. They suggested that the rate constant of decay, a_2, was increased by increase of steady level of the output stage y_n but that the rate constant for buildup a_1 was independent of illumination. Thus, the scheme they proposed was:

$$a_2 = a_{02}(1 + f(y_n)) \tag{27}$$

where the function $f(y_n)$ is chosen to fit the dependence of gain on mean level.

The point of the FH model for adaptation is that the gain and time course of response are thereby linked, which may be seen as follows. Say $T_2 = 1/a_2$. The impulse response (that is, the response to a brief flash in the linear range) of the simple n-stage FH model is, [cf. equation (7) of Fuortes and Hodgkin, 1964]

$$Q \cdot A \cdot T_2^{n-1} \cdot (t/T_2)^{n-1} e^{-t/T_2} \tag{28}$$

where Q is the amount of light in the flash and A is a constant proportional to $(a_1)^n$. The time to peak of this impulse response is $(n-1)T_2$ and the gain is proportional to T_2^{n-1}. If only T_2 is reduced by adaptation, as assumed by Fuortes and Hodgkin, the time to peak will shorten and the gain will drop, much more steeply than the time to peak.

Therefore, in the original FH model, the relation between gain and time to peak of the impulse response is a power law where the exponent is one less than the number of stages which are affected by adaptation.

6.1.2. THE BAYLOR – HODGKIN – LAMB MODEL

In the treatment of turtle cone responses, Baylor *et al.* (1974b) modified the original FH model by setting

$$a_1 = a_2 = \alpha = 1/T. \tag{29}$$

Several other kinetic schemes were found to fit the waveforms of cone responses better (Baylor *et al.*, 1974a) but the modified FH model was chosen adequate for an expanded theory of adaptation and responses too large to be in the linear range. It also has a much simpler interpretation than the FH model as a chain of chemical reactions (compare Borsellino *et al.*, 1965, with Baylor *et al.*, 1974b). Figure 59 illustrates the model used by Baylor *et al.* (1974b), which we will call the BHL model.

There are important differences between the BHL model and the original FH model. Since, in the BHL model, the rate constants for buildup and decay of the y substance are the same, there is no change in the gain when the value of the rate constants is changed, as there is in the FH model. This can be seen by considering the impulse response of the first n stages of the BHL model:

$$y_n(t) = Q \cdot B \cdot (t/T)^{n-1} e^{-t/T} \tag{30}$$

where $T = 1/\alpha$, and B is a constant.
In this case the peak of y_n is at $(n\text{-}1)T$ and the magnitude of the peak is independent of T. Gain control cannot be achieved in this model simply by an increase of the rate, α. Therefore, the BHL model has to have a new feature, an extra pathway for decay of the final stage of the cascade where the decay rate depends on the level of illumination. This is shown in Fig. 59 as the pathway denoted z_1, z_2, z_3, z_4. z_1 is equivalent to y_n and is assumed to be the concentration of "blocking particles" which lead to closing of the sodium channels in the photoreceptor's membrane. The decay of z_1 to the next state, z_2, is subject to *autocatalysis* by the level of z_2 present (see Fig. 59). Thus, the system of differential equations for the BHL model, up to the z_2 stage is:

$$\frac{dy_1(t)}{dt} = I(t) - \alpha\, y_1(t) \tag{31}$$

$$\frac{dy_j(t)}{dt} = \alpha\,[y_{j-1}(t) - y_j(t)]$$

$$\frac{dy_n(t)}{dt} = \frac{dz_1(t)}{dt} = \alpha\, y_{n-1}(t) -$$

$$\{\bar{K}_{12} + \nu\, z_2(t)\} z_1(t) + \frac{\bar{K}_{21}}{A}\{1 + \nu\, z_2(t)\}\, z_2(t)$$

$$\frac{dz_2(t)}{dt} = \bar{K}_{12}\,\{1 + \nu\, z_2(t)\}\, z_1(t) -$$

$$\{\bar{K}_{12}\,[1 + \nu z_2(t)] + K_{23}\} z_2(t).$$

In these equations ν is the autocatalytic constant implicitly defined by the equation $K_{12} = \bar{K}_{12} + \nu z_2$. A is the equilibrium constant of z_1, z_2 and is equal to K_{12}/K_{21}. This is a complicated set of differential equations and an intuitive feeling of what is going on is difficult. Some insight may be gained by examining a special case of interest: a weak, sinusoidally modulated increment which produces a response in the linear range. In this case,

$$I(t) = I_0 + I_1\, e^{i\omega t}$$

$$y_j(t) = y_{j0} + y_{j1}\, e^{i\omega t}$$

$$z_l(t) = z_{l0} + z_{l1} e^{i\omega t}. \tag{32}$$

We make the approximation that $I_0 >> I_1$ and that I_0, the mean level of illumination which may also be viewed as the mean arrival rate of light quanta, is large compared to the rate constants of the cascade. Then we obtain [cf. equation (40) of Baylor *et al.* (1974b)] for the cone's frequency response in the light adapted range:

$$\frac{z_{11}(\omega)}{I_1(\omega)} = \frac{y_{n1}(\omega)}{I_1(\omega)} = \left[\frac{1}{1 + i\omega T}\right]^{n-1}\left[\frac{1}{1 + i\omega B_1}\right]\left[\frac{1}{1 + \dfrac{A}{1 + i\omega B_2}}\right] \tag{33}$$

where $T = 1/\alpha$

$$B_1 = [\bar{K}_{12} + \bar{K}_{21}]/[\bar{K}_{23}\cdot\bar{K}_{12}]$$
$$B_2 = 1/\{\bar{K}_{21}[1 + \nu I_0/(\bar{K}_{23}\bar{K}_{12})]\}$$
$$A = \bar{K}_{12}/\bar{K}_{21}.$$

Notice that the frequency response of the cone consists of three terms, the first two of which are stages of temporal integration which are independent of the mean level, I_0. The only place where I_0 enters in the expression for the cone's light adapted frequency response is in the time constant B_2 in third term of equation (33). This term has the form of the frequency response of a negative feedback loop which has a gain A and time constant B_2. The time constant of the feedback is what is affected by mean level in the BHL model. As Baylor *et al.* (1974b) demonstrated, this could account qualitatively for the effect of backgrounds on the gain and time course of the cone's impulse response [the Fourier transform of the frequency response in equation (33)]. However, as indicated above in Fig. 53, the predicted gain vs background curve is steeper than the real data, while the dependence of the time to peak of the response on mean level is too shallow. In a later paper, concerning toad rods, Baylor *et al.* (1980) proposed that feedback to earlier stages of processing, rather than to just the last stage as in the BHL model, would provide a better fit to the data. However, the theory of such a system has not been analyzed and would certainly be more complicated than the BHL model.

A very important consequence of the BHL model is that the amplitudes of responses to high temporal frequencies of modulation are unaffected by mean level. This can be seen in equation (33) in the limit as $\omega \to \infty$. Then the only term which contains I_0 approaches the value unity, and it does so at lower values of ω than the other terms approach zero. Thus, the BHL model predicts no effect of light adaptation in the limit of high frequency. This is qualitatively in agreement with the measurements of the cone's impulse response by Baylor and Hodgkin (1974). They found that the rising phase of the impulse response was not affected by background level. This result is also qualitatively in agreement with Kelly's (1972) psychophysical data on the linearity of high frequency response in human vision. Recent direct intracellular measurements of the frequency responses of turtle horizontal cells also confirm this prediction of the BHL theory (Tranchina, Gordon and Shapley unpublished).

6.1.3. ENROTH-CUGELL AND SHAPLEY'S MODEL OF ADAPTATION IN GANGLION CELLS

Another scheme for obtaining Weber's Law behavior linked to dynamic changes is diagrammed in Fig. 60 (Enroth-Cugell and Shapley, 1973a). In this model for the rod-driven retinal network of the cat, the rod signal is subjected to a multiplicative gain control at the level of the rod–bipolar synapse. In the original model it was proposed that the feedback signal which multiplied the rod signal was produced by the horizontal cell. Later work reviewed above indicates rather that the feedback signal may arise in bipolar cells. In any case the formal expression of the model is:

$$r(t) = P(t)/\exp\{H(t)/H_{\text{trig}}\}$$
$$P(t) = \int_0^\infty I(t-t')\, p(t')\, dt'$$
$$p(t') = \bar{g}\,(t'/\tau_p)^3 \exp\{-t'/\tau_p\} \tag{34}$$
$$H(t) = \int_0^\infty r(t-t')\, h\,(t')\, dt'$$
$$h(t') = \exp(-t'/\tau_H).$$

Where $r(t)$ is the rod signal to the bipolars, $H(t)$ is the horizontal cell potential (now thought of as the bipolar cell's potential) and τ_H is the time constant of the feedback neuron. H_{trig} is a critical level this signal must exceed, τ_p is the time constant of the rod, $I(t)$ is the light stimulus, $P(t)$ is the photocurrent of the rod, $\bar{g}$ is the gain in the dark. The frequency response of this model has been worked out and it is (Enroth-Cugell and Shapley, 1973a):

$$\frac{r_1(\omega)}{I_1(\omega)} = \bar{g} \exp\{-r_0/H_{\text{trig}}\} \cdot \left[\frac{1}{1 + i\omega\tau_p}\right]^4 \left[\frac{1}{1 + \dfrac{r_0 H_{\text{trig}}}{1 + i\omega\tau_H}}\right] \quad (35)$$

where r_0 is the steady state value of $r(t)$. In the model, r_0 approximately increases like log I_0 for $\bar{g}I_0 >> H_{\text{trig}}$. Two features are noteworthy. The general "shape" of the frequency response resembles that of the BHL model's frequency response, equation (33). However, the effect of mean level on dynamics is on the gain of the feedback term $r_0 H_{\text{trig}}$, rather than on the time constant τ_H. Moreover, the gain of high frequency responses in this model does drop at high backgrounds by the factor $\exp\{-r_0/H_{trig}\}$, that is approximately reciprocal to I_0 in the high background limit. This prediction is not correct for single cone responses, but it has not been tested in the system for which this model was designed, the cat retina in the scotopic range. Measurement of the temporal frequency response of cat retinal ganglion cells at several different background or mean levels would be a crucial test of the different predictions of the BHL model and the Enroth–Cugell–Shapley model. Preliminary experiments of this type appear to confirm a model of the BHL type and to exclude multiplicative models like the Enroth–Cugell–Shapley model (Shapley *et al.*, 1983).

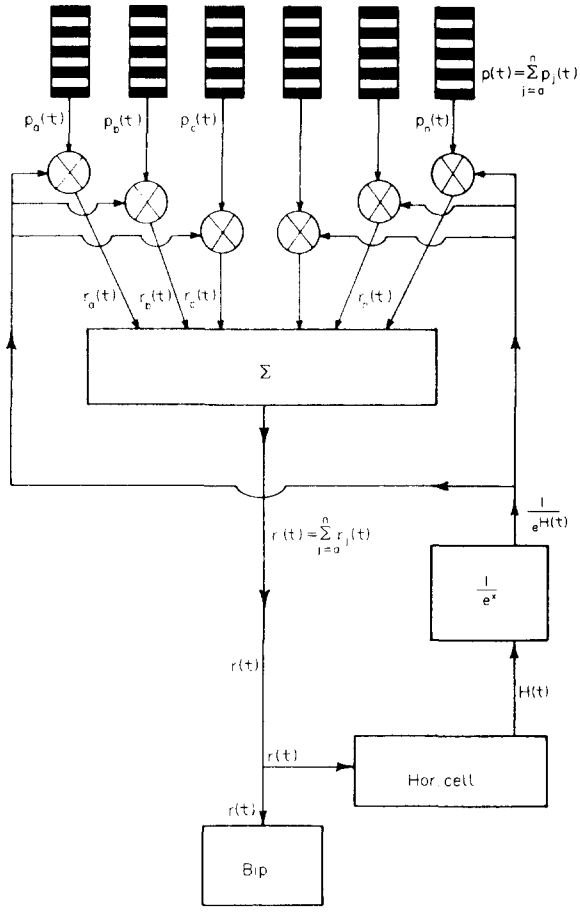

FIG. 60. A model for adaptation in the cat retina. The $p(t)$s are the photocurrents in individual rods. The amount of signal transmission from rods to bipolar and horizontal cells is called $r(t)$, with subscripts for each rod. $H(t)$ is the horizontal cell potential due to the sum of its inputs. An exponential function of the horizontal cell's potential is what controls the sensitivity of receptor transmission. Thus, $r(t) = p(t)/\exp\{H(t)\}$. From Enroth-Cugell and Shapley (1973a).

6.1.4. THE CONTRAST GAINCONTROL MODEL

The contrast gaincontrol adjusts the time course of response contingent on the average level of contrast rather than simply contingent on the mean flux. Shapley and Victor (1981) proposed a theory for the way in which the time course of response was changed by contrast, a theory which resembles the BHL model. Their model is diagrammed in Fig. 61. There are n stages of temporal integration and one negative feedback stage. The temporal frequency response of this system is:

$$\frac{r_1(\omega)}{I_1(\omega)} = A \left[\frac{1}{1 + i\omega\tau_l}\right]^n \left[\frac{1}{1 + \dfrac{K}{l + i\omega\tau_H}}\right] \quad (36)$$

Shapley and Victor (1981) found that increase of contrast affected only the ratio K/t_H and had only negligible affects on A, τ_l or n. Formally, equation (36) resembles equation (33) for the BHL model. The effect of contrast is exerted on the same term, the feedback term, and in a roughly similar way. Whether this similarity of the consequences of these models for incremental responses has any deep

meaning about mechanisms, it does reveal that *functionally* the two processes seem to affect time course of responses in similar ways.

Note that in none of these models is response simply a gain factor times an unadapted signal. Rather, in each case, adjustment of gain and adjustment of time constants, and/or strength of negative feedback, appear to be necessary, in order to explain the association of gain and time course in the data from visual neurons. The response of retinal neurons is thus a *functional* of the light now and the past history of illumination. The retinal functional is under the control of mean level and mean contrast.

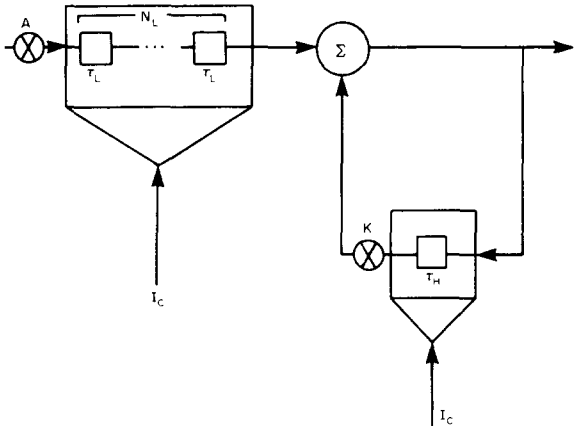

FIG. 61. A diagram of a model for the way the contrast gaincontrol modifies first order responses of ganglion cells. I_C is the contrast signal. The model consists of a gain stage A, unaffected by contrast, N_L stages of low pass filtering, and one stage of high pass filtering. Each low pass stage has a time constant τ_l. The high pass stage is a feedback loop with one stage with a time constant τ_H, and with feedback gain K. Contrast mainly affects K and τ_H. From Shapley and Victor (1981).

6.2. Retinal Gain and Visual Sensitivity

The psychophysical consequences of retinal gain control, and the implications of psychophysical results about retinal gain controls, require for their interpretation a theory linking the psychophysics and neurophysiology of vision. In a previous publication (Enroth-Cugell and Shapley, 1973b) we suggested a sketch of such a theory and here will make it explicit.

The aims of the theory are: (i) to show how gain and noise interact in determining sensitivity in rod vision; (ii) to show how the dependence of gain setting on receptive field size can be used to explain the observed spatial effects on the psychophysical laws of light adaptation (Barlow, 1957, 1965; van Nes and Bouman, 1967; Koenderink *et al.* 1978).

In our proposed model, psychophysical threshold is presumed to be governed by a signal-to-noise ratio. The signal is presumed to be the neural response of a *population* of retinal ganglion cells; the noise is the variability of firing in those cells. The signal is subject to gain control which is supposed, in the theory, to behave in the way that the retinal gain control has been observed to behave in cat retinal ganglion cells. We presume that there are a few (at least) retinal spatial channels at any particular retinal locus. We postulate that rod and cone signals are independent, in their noise generation and in their gain control, until late in the retinal stages of signal processing. Rod and cone pathways must have independent dark noises, and different dark adapted gains. There are two main factors which determine the sensitivity of ganglion cells: gain and noise (Barlow and Levick, 1969; Rose, 1948). From work cited above, the gain of a ganglion cell's response to a step of light on a background in the scotopic range, G_R is:

$$G_R = G_{R0}/(1 + F_B/F_{R0})^P \tag{37}$$

where G_{R0} is the dark adapted gain, F_B is the background flux effectively absorbed, F_{R0} is the criterion amount of flux which must be exceeded to turn on the gain control of the rod pathway, and P is an exponent, usually measured to be 0.9, which we will approximate to 1 (Cleland and Enroth-Cugell, 1970; Enroth-Cugell and Shapley, 1973a).

The noise will be the dark noise summed together with the background flux over the receptive field. Thus the variance σ^2 of the noise from the rod pathway in a ganglion cell's activity will be

$$\sigma_R^2 = [G_R^2 \cdot (F_B + F_{RD})] \tag{38}$$
$$F_{RD} = I_{RD} \cdot A_t$$
$$F_B = I_B \cdot A_t$$

Where I_B is the background retinal illumination, A_t is the summing area of the ganglion cell center, F_{RD} is the dark noise in the rod pathway (in equivalent quanta s^{-1}). It is assumed that the

psychophysical threshold will be reached when the cell's signal – noise ratio reaches 4. Thus, if I_s is the retinal illumination of a light stimulus and A_s is its area in squared degrees, then:

$$4 = I_S \cdot A_S \cdot (G_R)/\sigma_R \tag{39}$$

Solving for the threshold stimulus illumination, one obtains:

$$\begin{aligned} I_S &= 4 \cdot \sigma_R/[A_S \cdot G_R] \\ &= 4 \cdot [G_R^2 \cdot (F_B + F_{RD})]^{1/2}/[A_S\{G_{R0}/(1 + F_B/F_{R0})\}]. \end{aligned} \tag{40}$$

When F_B, the background flux, is small, the threshold is approximately constant depending only on the values of the dark adapted gain G_{R0} and the dark noise F_{RD}. However, as F_B becomes large compared to F_{RD}, the threshold becomes proportional to $F_B^{1/2}$. That is, it follows the square root law. This happens even though the retinal gain is dropping like $1/(1 + F_B/F_{R0})$, because the variance of the noise is dropping also, being proportional to F_B^{-1} when F_B becomes large compared to F_{R0}. In order to obtain Weber's Law one must put a clamp on the noise; it cannot keep declining in variance as the retinal gain declines. Indeed, as Barlow and Levick (1969) and Derrington and Lennie (1982) have found, the noise at the ganglion cell does not decline as the gain declines. How can this be so? One idea is that noise from the *cone pathway* is added into the ganglion cell, and is more or less unaffected by changes of gain in the rod pathway. Thus, if we change equation (39) to include cone pathway noise with variance σ_C^2 we obtain

$$4 = I_S \cdot A_S \cdot G_R/(\sigma_R^2 + \sigma_C^2)^{1/2} \tag{41}$$

where

$$\sigma_C^2 = G_C^2[F_B + F_{CD}] \tag{42}$$

and in analogy with the rod pathway:

$$G_C = G_{C0}\,(1 + F_B/F_{C0}). \tag{43}$$

In the scotopic range we can make the approximation that

$$F_B << F_{CD} < F_{C0} \tag{44}$$

Thus,

$$G_C \cong G_{C0} \tag{45}$$

and, $\sigma_c^2 \cong G_{C0}^2 \cdot F_{CD}$.

In order to obtain the correct value for the (Weber) scotopic contrast sensitivity, which approximately equals 10, one can calculate that

$$\sigma_C = G_{C0}\, F_{CD}^{1/2} = 0.02\, F_{R0} \tag{46}$$

Since we found F_{R0} to be equivalent to 3000 photon absorptions s^{-1}, this would mean the cone dark noise would have to produce a variance of about 60 photon events s^{-1} at the ganglion cell, a

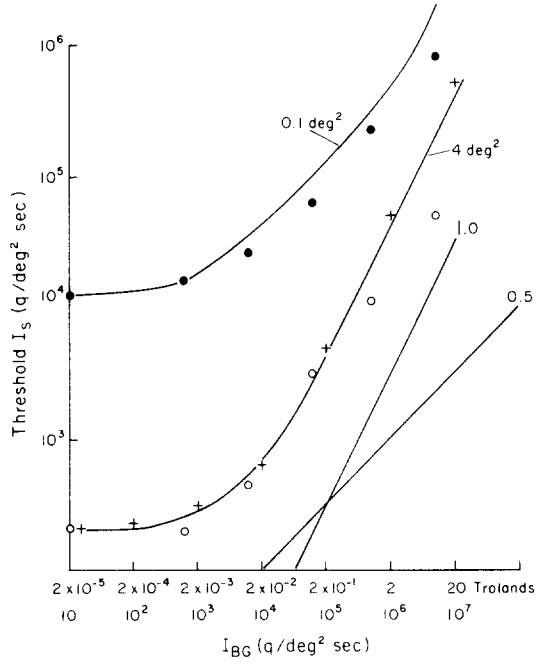

FIG. 62. The predictions of a theory of light adaptation concerning the effects of stimulus area on the slope of the increment threshold curve. The curves are the predicted increment threshold behaviour for retinal ganglion cells which have the areas indicated in the figure. The psychophysical data plotted for comparison are: empty circles from Barlow's (1957) data for a 19 deg^2 target, filled circles for Barlow's (1957) 0.0077 deg^2 target, and crosses for Aguilar and Stiles' (1954) 64 deg^2 target. See text.

not-unreasonable number which must be checked experimentally in the future.

Notice that, in this model, the dark noises and the criterion fluxes for the turning on of the gain control are distinctly different. This is in accord with actual measurements on ganglion cells (Enroth-Cugell and Shapley, 1973a; Barlow, 1977) which show clearly that the dark light is too small to account for the behavior of the gain vs background relationship. A different fundamental constant, ten to one hundred times larger than the dark noise must be hypothesized.

The point of the model is revealed in Fig. 62 which shows predictions of the model for the increment threshold curves of two types of cell, one with a small center and one with a larger center. The cells' sensitivities are plotted on the same coordinates as psychophysical data from Aguilar and Stiles (1954) and Barlow (1957) which exhibit characteristic steep and shallow slopes, respectively. Clearly, the model accounts adequately for the transition from square root to Weber behavior contingent on target size. It provides a way to understand why this transition is a function of the square of the spatial frequency of a sinusoidal target (van Nes and Bouman, 1967). It also provides a framework with which to understand why noise sometimes controls sensitivity and why, under other circumstances, the gain determines sensitivity.

7. RETROSPECTIVE AND CONCLUSION

We have considered the "why", the "what", and the "how" of visual adaptation, and even attempted to say "where" and "when" it takes place. At the outset we demonstrated how automatic adjustment of retinal gain would cause brightness constancy under different conditions of illumination, by making retinal responses dependent on contrast. Then we examined what happens to the visual performance of humans under different conditions of background or mean level of illumination. In this section, we pointed out that "noise" from the stimulus may sometimes limit performance, and that at other times retinal adaptation determines the limits of sensitivity. The visual responses of retinal ganglion cells, particularly cat ganglion cells, as a function of mean or background level, were considered next. In these cells, gain control seems the dominant factor in limiting their capacity to detect stimuli as the mean level of illumination varies. The relation of this work to human vision, where both gain and "noise" seem significant, is an open question for future research. In seeking to answer the question, "where" the gain control is located, we next considered what is known about adaptation in retinal interneurons. Then we reviewed the topic of adaptation in photoreceptors. Finally, we discussed some theories of how the gain of retinal neurons is controlled, and how such a gain control might contribute to visual performance.

There are several neural gain controls in the retina. Surprisingly, what evidence we have about the time course of gain reduction after an increase in mean illumination, together with the change in response time course with change in gain, suggests that all these gain controls share the property of attenuating the components of response to slow variation of the stimulus, and sparing the components of the response to rapid variations of the stimulus light. However, the different gain controls do seem to have different spatial integration areas, and this allows one to distinguish them.

Future work is required to establish the answer to "where" in the retina some of the gain controls live. However, it will be even more important to discover or invent models for "how" the gain controls manage to make the retina exceptionally sensitive to contrast and yet insensitive to large swings in the mean level.

APPENDIX 1 — RETINAL NEURONS

The details of retinal circuitry vary from species to species although many of the general principles, at least in higher mammals, are the same. Much of what we have written about the physiological mechanisms of retinal adaptation stems from experiments on the cat retina and any fruitful consideration of these mechanisms requires some familiarity with retinal functional connectivity. The brief comments about retinal circuitry which follow draw heavily on the detailed studies of the cat retina undertaken over the past ten years or so (for

summary see Sterling, 1983).

Information in the vertebrate retina flows mainly in a *radial* direction from the receptors, in the outer retina, the rod and cone cells, through bipolars towards ganglion cells in the inner retina, with lateral interaction in both plexiform layers, and with feedback within and between layers.

Increasing the amount of light that falls on vertebrate receptors causes the inside of the receptor cell to become more negative. That is, vertebrate receptors respond to light by *hyper*polarizing. Since transmitters are released in response to neuron *de*polarization it must be concluded that vertebrate receptors release the maximum amount of their as yet unidentified transmitter or transmitters in complete darkness. Direct evidence for this has been obtained by Schacher *et al.* (1974) in the frog retina, and by Ripps *et al.* (1976) in the skate retina, using horseradish peroxidase (HRP) uptake as a measure of synaptic vesicle turnover.

Rod signals leave the rod cell's distributing end for one type of bipolar cell while cone signals leave the distributing end of the cone cells for other bipolar cells. The statement that the cat has rod bipolars and cone bipolars means only that at present there is no *anatomical* evidence that both rods and cones contact the same bipolar cell (Kolb *et al.* 1981). This does not necessarily mean that rod and cone signals are strictly separated in the bipolar layer. For example, Nelson (1977) found that mixed rod – cone signals travel in some cone bipolars (see also Nelson, 1980).

Structurally, there is only one kind of *rod* bipolar in the cat, one that receives signals in invaginations of the proximal surface of the rod spherules. There are two structurally different kinds of cone bipolars: the invaginating ones which contact cones in pits in the proximal surface of cone pedicles while other cone bipolars only form superficial contacts with the pedicle bases. These are the "flat" cone bipolars. It seems that all receptor cells in the cat do not "talk" to bipolars in exactly the same language because the ultrastructural synaptic specializations are not the same in the rod and cone pits as in the contacts between the base of the cone pedicles and the flat cone bipolar dendrites (Sterling, 1983), suggesting that the mode of signal transmission is different in the two types of synapse. This is also consistent with the recent physiological work of Saito *et al.* (1979) in the carp retina which suggests that rods and cones affect fundamentally different ionic channels in their respective bipolars.

Although, as far as is known, all vertebrate receptors respond to light by hyperpolarizing, the same does not hold true for all bipolar cells. This has been known since 1969 when Werblin and Dowling showed that some mudpuppy bipolars hyperpolarize while others depolarize when illuminated. Nelson and co-workers (Nelson and Kolb, 1982) have recorded intracellularly from anatomically identified bipolars in the cat retina and they found that in the cat retina too some bipolars hyperpolarize while others depolarize when the light level is increased.

It is in the inner plexiform layer (IPL) that the third order neurons, the ganglion cells, communicate with other retinal cells. Two features of this connectivity are of special interest. First, some ganglion cells' dendritic trees are located within the a-lamina of the IPL where they receive information from those bipolars which extend only as far as the a-lamina. Other ganglion cell dendritic trees branch out in the b-lamina of the IPL and are fed by bipolars whose distributing end extends to the b-lamina. One of the major recent achievements within retinal anatomy – physiology is the demonstration by Famiglietti and co-workers (Famiglietti *et al.*, 1977; Nelson *et al.*, 1978) that when bipolars and ganglion cells talk to each other in lamina-a of the inner plexiform layer, the ganglion cell is of the off-center type, i.e. an increment of illumination within the center of the ganglion receptive field (see Appendix 2) causes it to become hyperpolarized. But when the two talk to each other in lamina-b the ganglion cell is of the on-center type, i.e. it depolarizes in response to increased illumination of the receptive field center. Sterling (1983) and his colleagues have found that at least two different types of bipolar cells converge on each retinal ganglion cell of the morphological class called beta (Boycott and Wässle, 1974), which is equivalent to the functional class, X. Thus each off-center X ganglion cell receives direct synaptic input from one kind of flat cone bipolar and an invaginating cone bipolar, as well as indirect input from rod bipolars. A similar state of affairs applies to on-center X cells: two cone bipolar direct inputs, and an indirect rod bipolar input. There are thus

several cone bipolar types, since on- and off-X cells each receive input from their own special invaginating bipolars, and their own special type of flat bipolar (Sterling, 1983).

Lateral spread of signals within the retina is important for adaptive mechanisms. Lateral interaction is possible already at the receptor cell level. In the cat there are gap junctions between pedicles of individual cone cells and also between rod-spherules and processes extending from cone-pedicles (see e.g. Nelson *et al.*, 1981). Nelson (1977) has demonstrated that individual cat cones receive rod signals which presumably enter via the rod – cone gap junctions. To what extent the cone – cone junctions also represent functional contacts in the cat is not known at present and we are not aware that anybody has shown that the cat retina has any functional or anatomical rod – rod contacts, but, in some cold-blooded vertebrates, functional coupling between rods via gap junctions is a prominent feature (e.g. Detwiler *et al.*, 1980).

The more distal of the two laterally spreading neuron types is the *horizontal cell* of which the cat has two kinds (Boycott, 1974; Boycott *et al.*, 1978). The type A and type B cell both have a soma and a dendritic arborization in which all synaptic contacts are with *cones*. The type B cell in addition has a long, very thin (probably non-impulse carrying) axon which leaves the soma-dendrite complex and at a distance of about half a millimeter "explodes" into an elaborate arborization which contacts only *rods*. In the cat, horizontal cells only hyperpolarize to increases in illumination, and most receive both cone and rod signals from photo-receptors (Steinberg, 1971; Niemeyer and Gouras, 1973; Nelson *et al.*, 1975).

The other interneurons which mediate lateral interaction are the amacrine cells which spread their processes in the inner plexiform layer. The amacrine cell family is made up of at least twenty-two different types in the cat retina, as distinguished by dendritic branching patterns in the IPL (Kolb *et al.*, 1981). The most well studied of these cells is the AII amacrine cell which receives direct synaptic input from rod bipolars in lamina-b of the IPL, and gap-junction contacts from cone-bipolars in lamina-b also. It also receives cone bipolar input from synapses in lamina-a. So far only rod bipolars which hyperpolarize to increments of light have been described (Nelson, 1980) and yet AII amacrines depolarize to increments, suggesting that the rod – bipolar to AII amacrine synapse is sign-inverting. AII amacrines make synaptic contact onto presumed off-center (i.e. decrement – excitatory) ganglion cells, which suggests that the synapse between the AII and the off-center ganglion cell is sign-inverting also. None of the other amacrine cells' connectivity is as well characterized as that of the AII cell, yet certain features of their structure are noteworthy. As in other species, there are numerous amacrine → amacrine synaptic contacts in the IPL of the cat retina. Furthermore, there is a preferential input of amacrine cells to Y ganglion cells, while there is much more direct bipolar input to the X cells (Kolb, 1979; Sterling, 1983). Since most is known about the processes of retinal adaptation in the X and Y ganglion cells in the cat, the similarities and differences in their anatomical connections are important for understanding the site in the retina where retinal adaptation takes place.

One of the curious features of retinal morphology is that there appears to be only one rod bipolar type compared to several cone bipolar types (Sterling, 1983). Since receptive field organization is just as rich in the scotopic range as in the photopic, one might have expected as much anatomical elaboration of the rod bipolar family as of the cone bipolars. Because of the great degree of rod – cone independence, we guess that rod – cone coupling (Nelson, 1977) is of secondary importance. However, Sterling (1983) believes that rod – cone coupling is the main pathway for rod input to ganglion cells over most of the scotopic range. This is not really a controversy, since both our view and Sterling's are equally speculative at present. The AII amacrine, which gets its predominant input from rod bipolar cells, couples into the photopic circuitry when it makes gap junctions with the cone bipolar cells in lamina-b of the IPL. The AII amacrine seems to be a crucial link in scotopic receptive fields.

One of the most interesting interneurons in the retina is the interplexiform cell (Boycott *et al.*, 1975; Kolb and West, 1977; Nakamura *et al.*, 1980). This neuron is perhaps the most likely candidate to be involved in gain control in the retina. It has dendritic arborizations in the IPL, where it receives synapses from amacrine cells of so-far indeter-

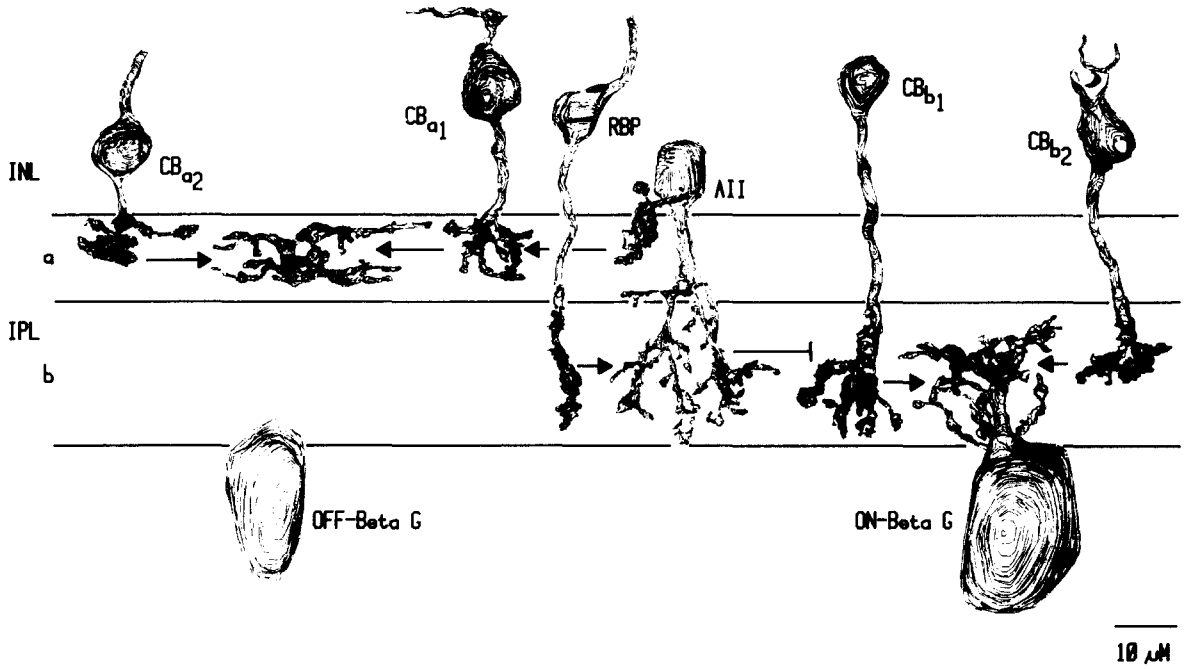

FIG. 63. Diagram of connections in the inner plexiform layer of the cat retina from serial EM reconstructions. Chemical synapses are indicated by the arrows: gap junctions are indicated by the symbol ⊣ . CB indicates a cone bipolar cell; four subtypes are labeled. RBP stands for rod bipolar cell. AII is a subclass of amacrine cells. The Beta ganglion cells are presumed to be the anatomical equivalent of the X cells. The inner plexiform layer is subdivided into sublaminae-a and -b, as indicated in the figure. On ganglion cells receive input in sublamina-b, while off ganglion cells receive input in sublamina-a. From Sterling (1983).

minate type. Then the interplexiform cell makes synapses on all types of bipolar cells at the level of the IPL but more especially at the level of the outer plexiform layer (OPL). It is the perfect example of a feedback neuron. Moreover, it could affect all bipolars which is a property one would want from a gain control since on- and off-pathways must be adapted in the same way in order to keep contrast sensitivity constant. However, the wide terminal branching of the interplexiform cell in the OPL is a problem if this cell is to play a role in regulating gain on steady backgrounds, because, at least for X cells, the area of the retina over which adaptive signals are summed for the center is less than 1 deg^2, i.e. less than 200 μ by 200 μ. However, there is evidence for a different kind of gain control which depends on stimulus contrast rather than average flux (see Section 3.8; Shapley and Victor, 1978; cf. Werblin and Copenhagen, 1974) and perhaps the interplexiform cell's properties may match those of this other gain control.

A schematic diagram of the neuronal circuitry in the cat's IPL, where ganglion cells receive their inputs, is offered in Fig. 63, from a recent review article (Sterling, 1983).

APPENDIX 2 — RECEPTIVE FIELDS

The neural signal which leaves the retina consists of trains of impulses carried by the axons of retinal ganglion cells. The response of these cells to a visual stimulus may be defined as a change in the rate of firing of impulses. An adequate stimulus to cause such a change in firing is some change in the illumination on the retina. The work of Hartline (1940), Barlow (1953), and Kuffler (1952, 1953) showed that each retinal ganglion cell generated responses, as defined above, to stimulation over a limited area of the retina, and this area was defined as the receptive field of that ganglion cell. Working in the cat retina, Kuffler (1953) found that ganglion cell receptive fields consisted of two concentric zones which he called the center and surround. The center and surround were mutually antagonistic. In on-center cells in which the center caused excitatory

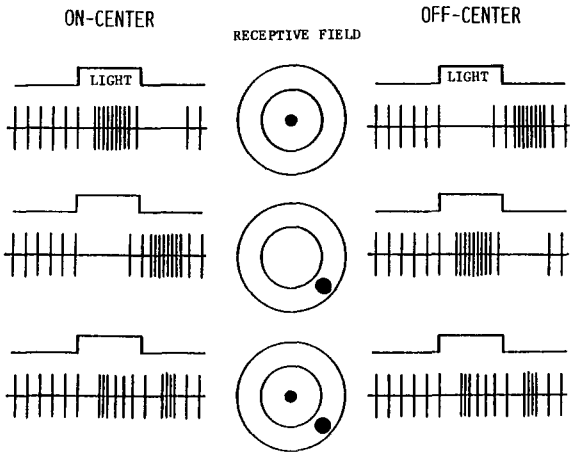

FIG. 64. Schematic representation of the mutual antagonism between center and surround in on and off-center cells. In on-center cells (a,b,c), an increment in the center increases the cell's firing rate while an increment of light in the surround decreases the firing rate. When the two increments are applied together in synchrony, as in (c), the response to light "on" is less than in (a) and the response to light "off" is less than in (b). This means the two regions are mutually antagonistic. In off-center cells (d,e,f), the cells' response pattern at light "on" in the center and in the periphery are reversed, but the mutual antagonism is the same.

responses to increments of light, the surround would cause inhibitory responses to increments. In off-center cells in which the central region was inhibitory during an increment, the surround would be excitatory during an increment. The on- and off-center cells and their center-surround organization are illustrated in Fig. 64.

Rodieck (1965) made a major advance by developing a model for the cat ganglion cell receptive field in terms of overlapping center and surround mechanisms. Each mechanism may be conceived of as the receptors and interneurons, the signals of which are pooled together to influence the firing of the ganglion cell. Within the center mechanism all light evoked signals generated within the pool of these receptors and interneurons are summed, according to the model, and similarly for the surround. Then center and surround signals are summed at the ganglion cell. The signals from different positions within each pool are weighted by what Rodieck called the "sensitivity profile", and which we will refer to as the "spatial distribution of gain". The center has its own narrow spatial distribution of gain, and the surround has a rather broader spatial distribution. Rodieck proposed that these two spatial distributions could be approximated by Gaussian surfaces with different extents of spread. The spatial resolution and optimal spatial tuning of retinal ganglion cells can be rationalized in terms of Rodieck's model (Enroth-Cugell and Robson, 1966). The spatial resolution of the cell is due to the finite size of the center, and in fact can be predicted from knowledge of the magnitude of the spread of the center's Gaussian spatial distribution of gain (Cleland *et al.*, 1979; So and Shapley, 1979; Linsenmeier *et al.*, 1982). Figure 65 shows the Rodieck model for the receptive field of cat retinal ganglion cells.

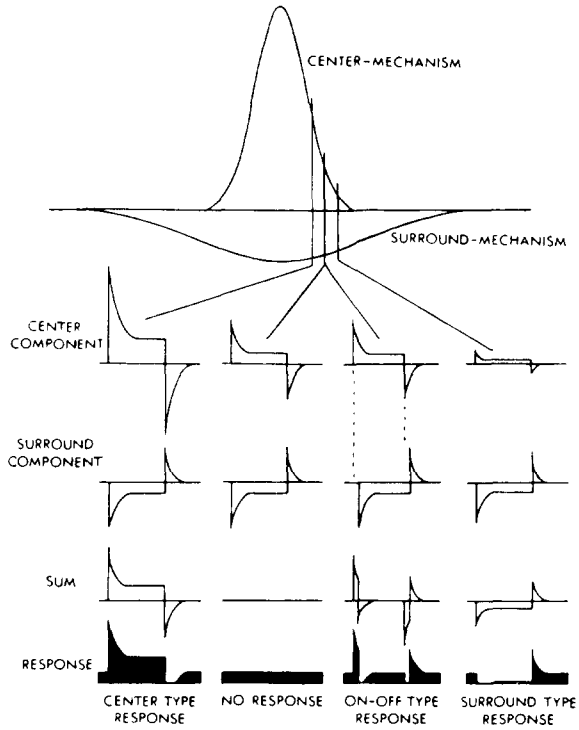

FIG. 65. The two spatially overlapping mechanisms in the Rodieck model of retinal ganglion cell receptive fields. In the sketch at the top, the horizontal axis represents distance on the retina. The heights of the two curves represent the gain of the center and of the surround, as labeled, as a function of position on retina. Both are Gaussian functions of position; the center's Gaussian has a narrower spread than the surround's. The center and surround have opposite sign in this model. This results in mutual antagonism. In this model the center and surround components combine by simple addition, i.e. linearly. Thus, response to stimulation anywhere within the field is, according to the model, simply a sum of the center and surround components in response to the stimulus, as is illustrated in the figure. From Rodieck (1973).

There are several ganglion cell classes in each vertebrate retina (see Rodieck, 1979). This is a significant complication, because not all of these cell types conform to the simple elegance of the Rodieck model. As far as we know, the ganglion cell classes in the cat retina which have the highest contrast gain are the X and Y cells discovered by Enroth-Cugell and Robson (1966). While X cells do behave in a way approximately predictable from the Rodieck model, Y cells exhibit nonlinear summation of visual signals (Enroth-Cugell and Robson, 1966; Hochstein and Shapley, 1976a, b). It seems that while X cells have two pools or mechanisms as in Rodieck's model, Y cells have at least three different types of neuronal mechanism. The new type of mechanism is what Hochstein and Shapley have dubbed the "nonlinear subunits", small spatial pools within which neural signals are summed in a linear manner, but between which a nonlinearity is interposed before signal summation. There have been several excellent reviews of the physiology and anatomy of cat ganglion cells (Robson, 1975; Rodieck, 1979; Lennie, 1980; Wässle, 1982; Levick and Thibos, 1983, among others).

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