

ROD-CONE INTERACTION THROUGH FLICKER DETECTION
AND THE COURSE OF CRITICAL FLICKER FREQUENCY
DURING DARK ADAPTATION

by

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INTRODUCTION

Visual sensitivity increases with time in the dark. This simple statement is obvious to anyone who has stepped from the brightly lit theater lobby into the darkened auditorium. This dark adaptation process has been studied formally by preadapting an observer's eye to a bright light of known intensity and duration and then determining the absolute threshold in the dark or an increment threshold against a dim background. The resulting dark adaptation curve exhibits a discontinuity which indicates that sensitivity increases in two steps (see Fig. 2). The upper segment of the dark adaptation curve has been traditionally attributed to the cones which are responsible for high visual acuity, high temporal resolution characteristics, and hue discrimination. The lower segment of the dark adaptation curve represents the activity of rods which have been assumed to function with low visual acuity and not contributing to hue discrimination, but are highly sensitive to low amounts of light and slow flicker rates.

Review of the Literature

The classic view of Hecht [1937] that dark adaptation depends exclusively on bleaching and regeneration of visual pigment can no longer be entertained. This view was discredited when Rushton [1956] measured the actual rate of the bleaching of rhodopsin in man and was

able to show that predictions from Hecht's theory were in error by a factor of a million.

The duplex nature of vision was clearly pointed out over a century ago [Schultze, 1866]. The "duplicity theory" as it is now called, states that cone vision provides daytime and color (photopic) levels of luminance whereas rod vision provides a high degree of sensitivity to light for seeing at night when light levels are low and achromatic (scotopic). This duplicity model is widely accepted and maintains that rods and cones are highly independent in their functions, the only interaction being the inhibition of rods by cones as intensity is increased to photopic levels due to the faster cone signal and saturation of the rods.

Flicker is a useful tool in the methodology of psychophysics. The ultimate purpose of using flicker in this capacity is to account for the psychophysical relations in terms of physiological units. Waveform is a dimension of flicker that is troublesome to the experimenters. Many choose to use sine waves since a linear system is less variant in its response to sine waves. However, the optical system has components of linearity as well as nonlinearity.

The frequency of intermittent light at which flicker just disappears is called the critical flicker frequency (CFF). The CFF varies widely with stimulus intensity, retinal area stimulated, state of adaptation, nature of surround illumination, depth of modulation, and other factors. In foveal vision, the CFF varies from about 5 up to 55 cycles per second (Hz), and it is approximately proportional

to the logarithm of the product of stimulus intensity and area. This relationship is called the Ferry-Porter Law [Ferry, 1892; Porter, 1902] which is to say that CFF rises linearly with log luminance over middle range of intensities. This relationship has been found to hold true, but only for middle ranges of intensities. However, for low and mid ranges of intensities, the periphery can detect higher frequencies than the fovea.

The changes of CFF during the course of dark adaptation have long been confusing and conflicting in accordance with the past literature. For example, Lythgoe and Tansley [1929], Krakov [1938], White and Baker [1976], concluded that CFF increases during light adaptation and decreases during dark adaptation. However, Allen [1900], Peckham et al. [1952] showed that CFF decreases during light adaptation and increases during dark adaptation. Shikano [1951] stated that CFF follows an alternating course during dark adaptation. Akos and Akos [1966] later showed that CFF involves a drift up to 10Hz (higher and lower) over time which is characteristic of the subject. These drifts were reportedly due to variations in the methods of the previous studies [White and Baker, 1976]. There are mainly two methods used: (1) CFF is measured with a stimulus of constant intensity as a function of time in the dark, and (2) the intensity as a function for a stimulus flickering at a constant frequency is measured as a function of time in the dark.

White and Baker's methodology involved the measurement of changes occurring in foveal CFF as a function of time in the dark. They implemented a technique in which both flicker frequency and luminance were varied, but always near the absolute threshold.

From this they concluded: (1) that CFF measured during dark adaptation of the foveal area is a function of the subject's retinal sensitivity (state of adaptation) and not necessarily time in the dark, and (2) that the Ferry-Porter Law holds for foveal stimulation when the stimulus is near absolute threshold.

Experimental Design

The object of this research is to clarify the course of CFF during dark adaptation and to demonstrate rod-cone interaction at the retinal level. While some of the methodology was similar to that of White and Baker, the apparatus was entirely different and many additional conditions were assessed. These additional conditions provide for proof of rod suppression of the cone contribution to threshold. A general rationale will be presented here followed by a detailed explanation of the various conditions and methodological designs.

The controversial results of CFF during light and dark adaptation need further clarification. Some of the previous studies can be questioned because of their technique. Recent studies have reported that prolonged observation of a stimulus flickering at a constant frequency may promote specific forms of adaptation for temporal frequency [Alpern et al., 1961; Smith, 1970].

DeLange [1958] maintained that for the frequency bands 0 → 70 Hz the visual brightness system shows a low-pass filter action for temporally modulated or intermittent light. However, linear processes cannot fully account for the steep slopes of the DeLange curves above 10 Hz.

D. H. Kelly [1970] provides an excellent theoretical model (and review) of flicker and transient responses involved in photopic (cone level of brightness) flicker. He describes a model of two retinal processes; the first being a linear diffusion process at the receptor level, the second is a non-linear inhibiting system or "neural feedback at the synapses of the plexiform layers" of the retina which has control over the sensitivity and time constants of the model.

METHOD

Six observers, having normal or corrected vision, served as subjects (four males and two females). After three trials for each of the six observers, analysis and comparison showed very low variability. It was then decided that two observers would be quite sufficient for the remainder of the experiments.

Apparatus

The glow modulator tube has fast rise and decay times. It produces a square wave and was chosen for the test flash in this research mainly because prior work with CFF during stages of adaptation involved the glow modulator tube. This glow modulator tube produced a 2° variable flicker test stimulus. Furthermore, a 50-50 duty cycle was always implemented whereby the flickering stimulus was off as often as it was on, producing a normal square wave. The apparatus was designed to include a Maxwellian view test flash and adapting source. The Maxwellian view [see J. C. Maxwell, 1860] utilizes a source which is focused by lenses to image on the eye's entrance pupil. This schema was used in this research to provide maximum intensity (or efficiency) of luminance, to insure that the proper field of view enters the subject's pupil, and to minimize distortions and non-uniformity [Westheimer, 1966]. A glow modulator

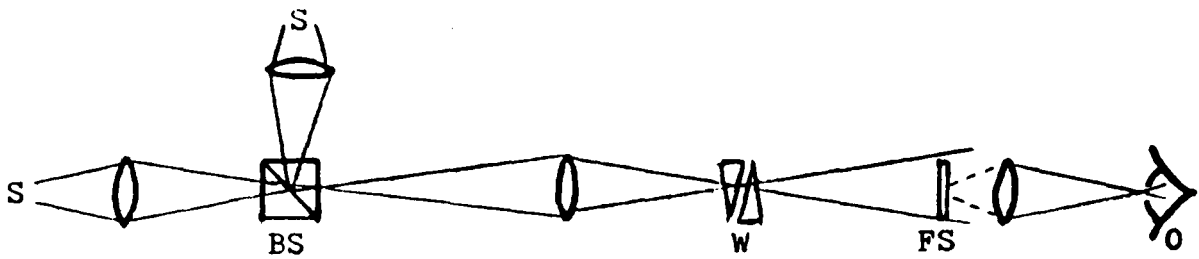
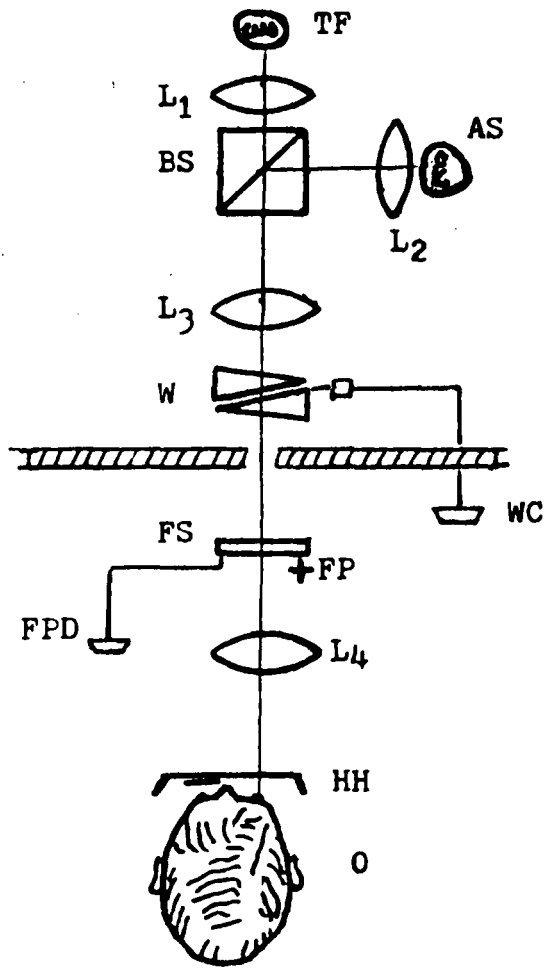
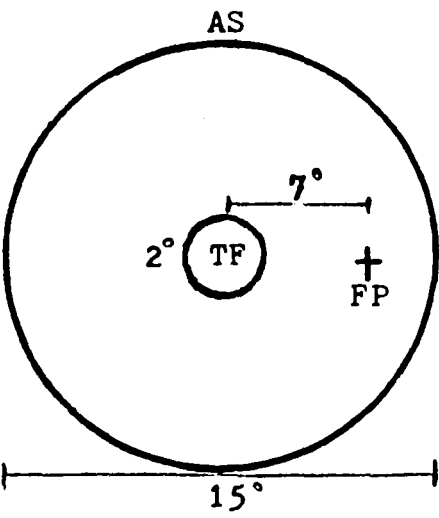
tube was used as the test flash and a Maxwellian view Tungsten-filament lamp was used as the adapting field. [See Fig. 1.]

The apparatus further consisted of a Marietta adaptometer, L.D.C. chart recorder, and a Lafayette flicker fusion unit. The retinal illuminance of the adapting source was 5.9 log trolands for 45 seconds, which converts to 7.6 Log td seconds, which bleaches 99% of the rhodopsin [See Rushton and Powell, 1972]. The intensity was determined with an SEI exposure meter and converted to Log td retinal illuminance [Westheimer, 1966; Rushton, 1956a, b]. The adapting field subtended 15° , the test flash subtended 1° (and 2°), when in use, the fixation cross hairs were 7° from the test flash. At 7° , the retina has approximately an equal number of rods and cones [Østerberg, 1935] and provides clear photopic (cone) and scotopic (rod) branches in the dark adaptation curve.

Procedure

The design of this research involves essentially four types of experimental conditions, each containing dark adaptation procedures. The dark adaptation proceeded as follows: The Os head was held by an American Optical chin and head rest. Threshold measurements were made using von Bekesy's [1947] tracking method in which the O increased a neutral density wedge until the test flash was no longer visible, then reduced the density of the wedge until the test flash was just perceptible, again increased the wedge density until the test flash

Fig. 1. Diagrammatic representation of apparatus. The observer (O) sits behind a head holder (HH) and chin brace that can be adjusted to fit the subject. The final lens (L₄) is also adjustable in order to position the beam of light within the O's pupil. The observer fixates on the cross hairs making up the fixation point (FP) and observes the field stop (FS). The FS is at the focal distance from the last lens so as to present a clear image on the retina. The O can attenuate the wedge (W) by a wedge control (WC) and also attenuate the FP by the fixation point dimmer (FPD). The test flash (TF) and adapting source (AS) are combined into the same beam by a beam splitter (BS).



was no longer perceptible, etc. In this manner, the subject continuously tracked his (or her) threshold for the test eye and his judgments were constantly recorded by the chart recorder [Prestrude, 1976]. [See Fig. 2.]

Condition I involved the traditional tracking of the threshold stimulus at 2 Hz, but initially and after every 2 minutes, a CFF measurement was made ascending and descending at or near threshold attenuation. Thus, without disturbing the threshold tracking or the level of adaptation, CFF measurements were made throughout the recovery of sensitivity at 2 minute intervals. Results of these data are shown on Fig. 3.

Condition II follows precisely that of condition I, except after a full 30 minutes of dark adaptation, the experimenter then presents the subject with the specific frequencies that were recorded at 2 minute intervals during recovery. For each of these frequencies, the subject is asked to decrease the wedge density (increase the brightness) of the flickering test flash until it is just perceived as flickering. An example of the results is illustrated in Fig. 4. The purpose of this procedure is to show the difference in flicker detection due to state of adaptation. Why should the intensity required to detect flicker not be the same--since the frequency is the same? After 30 minutes of recovery, the sensitivity of the eye is near a maximum--shouldn't it require less intensity to detect the same frequency than when 50 percent or so of the visual pigment is in a

Figure 2. Dark adaptation curve obtained from our lab. Each dot represents the midpoint from the tracking procedure. Measurements were made over a 3.5 log unit range of luminance.

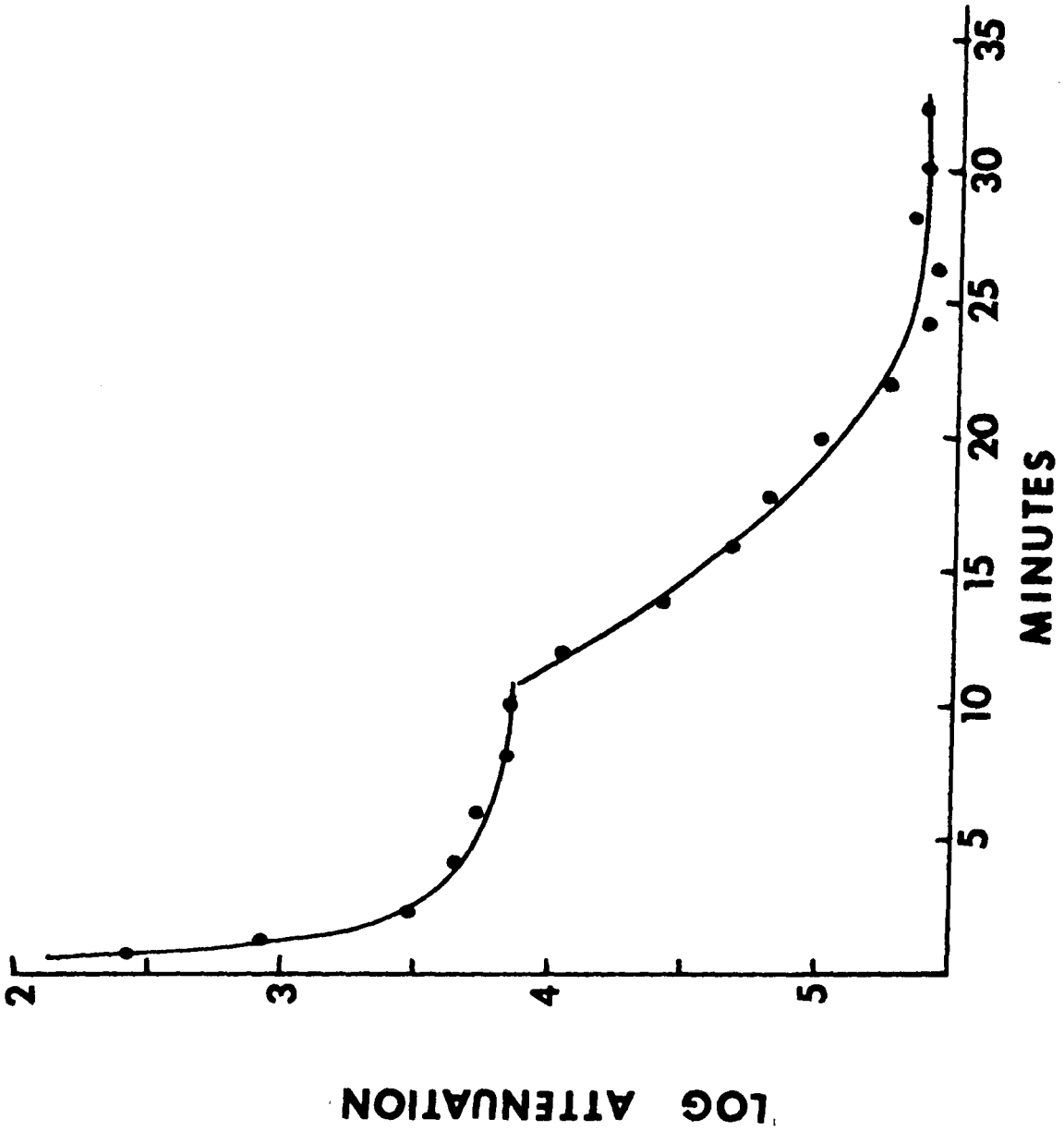


Figure 3. Representation of CFF plotted against time in dark. Note the marked decrease in CFF as time in dark increases. The slight "kink" at 14 minutes might represent 92% regeneration of rhodopsin.

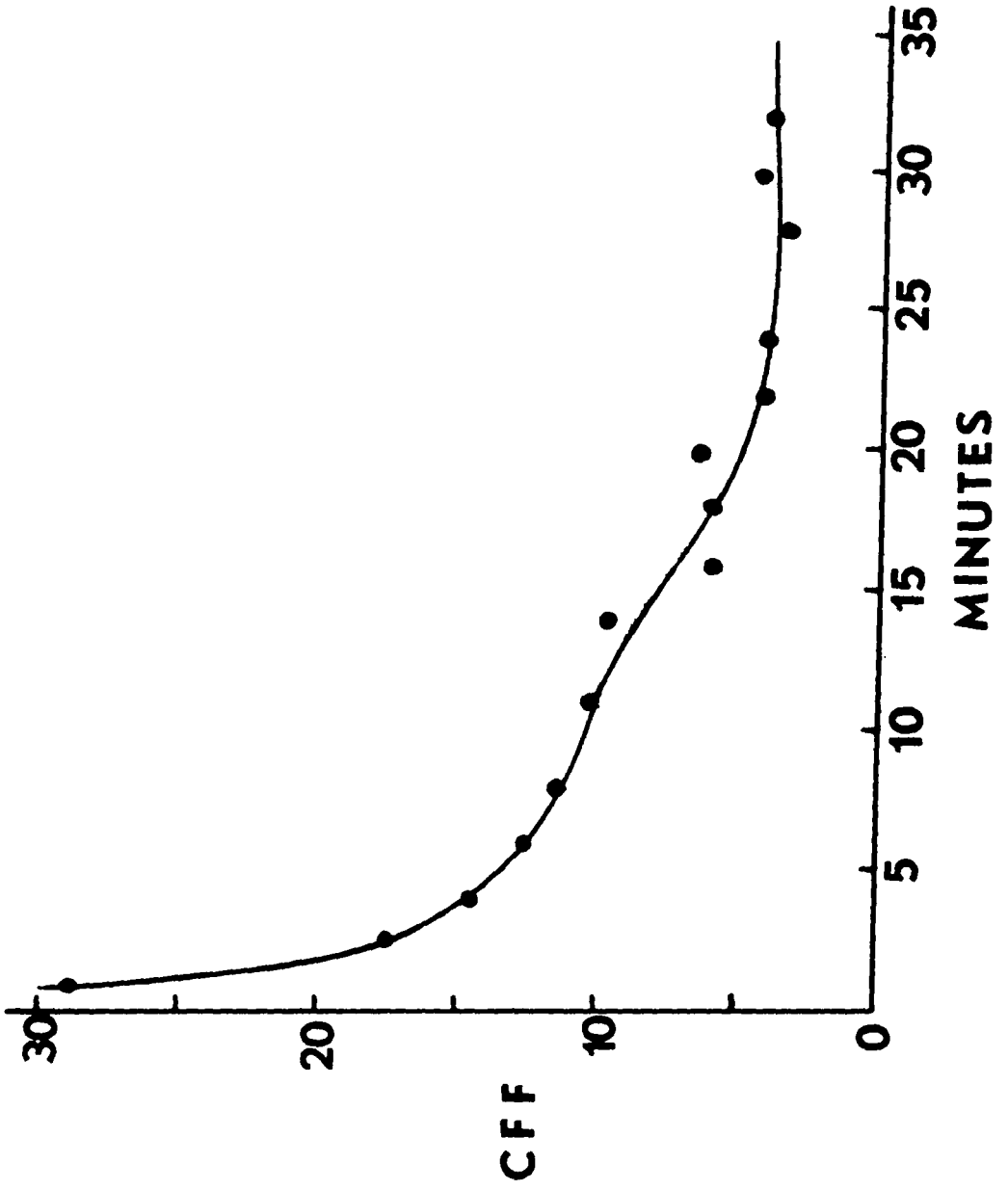
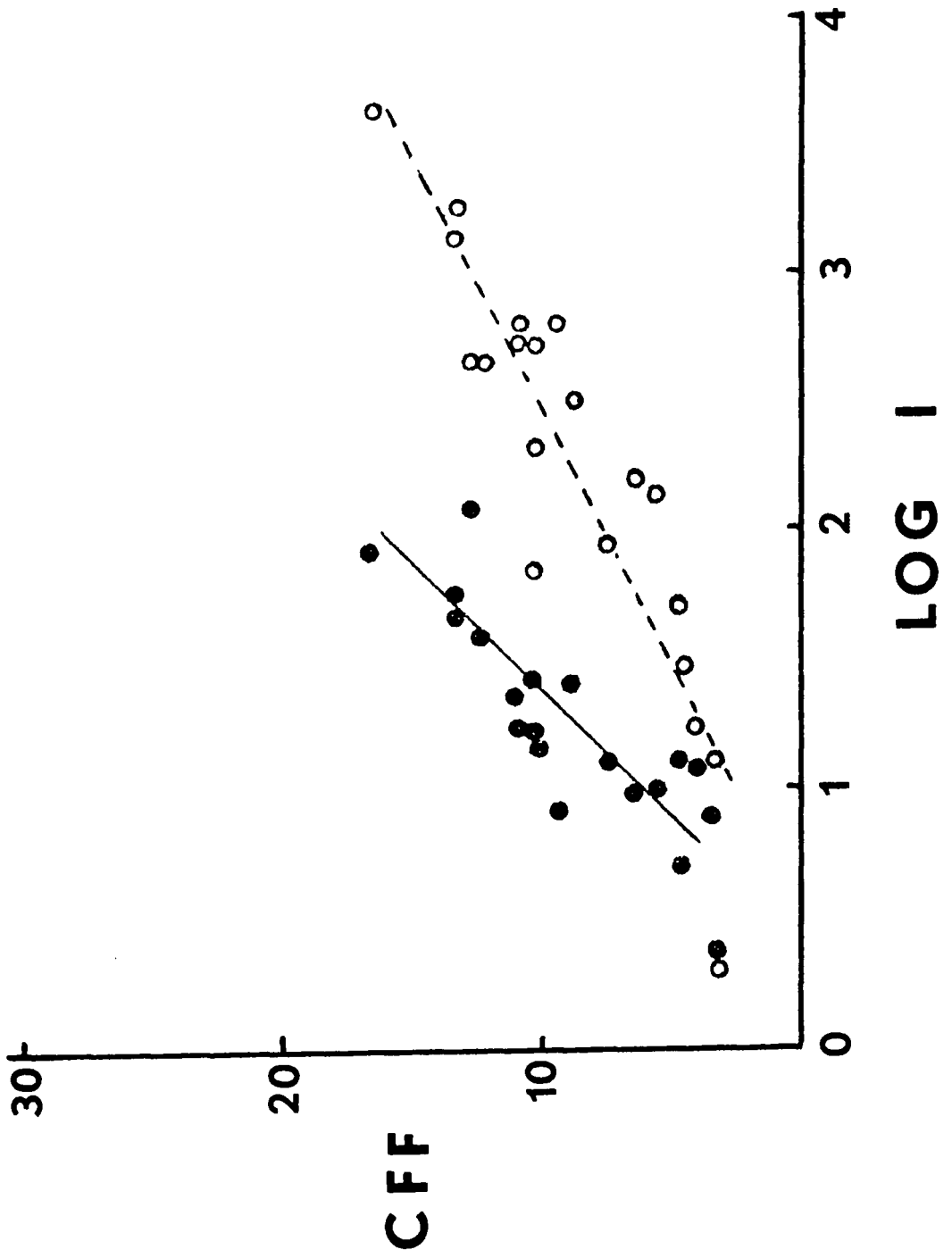


Figure 4. Plot of CFF against log intensity. The filled circles represent measurements made during dark adaptation; and the open circles are measurements made after 30 minutes of dark adaptation.



bleached state? The data show that actually the more sensitive condition (near full recovery) requires up to 1.5 log units more intensity to detect the same flickering stimulus or frequency.

Condition III is similar to Condition II, except specific frequencies (8, 10, 12, 14, 16, 18, 20, 24, 28, 30) were chosen before dark adaptation. Normal tracking at 2 Hz occurred until 5 minutes of adaptation (the level "cone plateau"), then the specific frequencies were tested for flicker detection in the manner of the procedure described in Condition II. These measurements were taken from 5 to 10 minutes. Then normal 2 Hz tracking was resumed; when after 30 minutes of adaptation, the very same chosen frequencies were tested again for flicker detection (after full recovery). [Since this study has been completed, Stabell and Stabell (1977) have also used this method of studying rod and cone contributions to threshold.]

In the same condition, we also tested an experienced rod monochromat. A rod monochromat was used in the experiment because this person presumably has no functional cones. This would allow us to observe the contribution of the isolated rod signal. Thus, we have a natural condition in which the subject should show no rod-cone interaction. The monochromat was diagnosed as such from measurements of pseudo isochromatic plates and anomaloscopic data. (The anomaloscope is a portable Nagel type built by the author and designed by T. P. Piantianida.)

Condition IV involved the subject tracking at flicker detection the same frequency throughout the session--either 10, 12, or 20 Hz, whereas the usual dark adaptation procedure (condition I) is run at 2 Hz. Precautions were taken so that the subjects sensitivity was not influenced or altered by observing flicker throughout the trial. Foveal and parafoveal (7°) trials were performed at each flicker frequency.

Summary of Experimental Conditions

The conditions of the experiment are summarized as follows:

Condition

- I. CFF determinations during dark adaptation at 2 minute intervals.
- II. CFF determinations during dark adaptation and CFF determinations after recovery (30 minutes).
- III. Flicker determinations after 5 minutes of dark adaptation and after 30 minutes of dark adaptation for normals and for a rod monochromat.
- IV. Flicker threshold throughout dark adaptation for foveal and parafoveal areas.

RESULTS

The following data demonstrate two clear cut results: (1) CFF decreases during dark adaptation, rather--during the process of regeneration of the bleached pigment. (2) Cone contribution to flicker thresholds can be inhibited by rod activity during these threshold measurements. Fig. 3 (Condition I) demonstrates that CFF decreases as photopigment regeneration occurs. When the eye has completely dark adapted, all of the rhodopsin has essentially regenerated completely. However, this is not to say that rhodopsin regeneration follows hand in hand with dark adaptation, for when in the course of dark adaptation the rod branch first appears below the level of the cones--rhodopsin has regenerated about 92 percent [Rushton, 1969]. If the rod branch of the traditional dark adaptation curve (See Fig. 2) is related to the regeneration of rhodopsin, it must relate to this final 8 percent. In Conditions I, II, and III, flicker detection measurements were made during various states of adaptation. As a result, the following data support the conclusion that CFF decreases as a result of changing state of adaptation, which effects a change in sensitivity thereby causing CFF to decrease during dark adaptation.

Fig. 4 represents Condition II, which compares the same flicker detection frequencies during and after dark adaptation.

Presented are the averaged data for six subjects, three trials each. The largest difference between the two measurements occurs at the maximum difference in state of retinal adaptation. In this figure, the lower intensity measurements were made at a closer interval during dark adaptation than the higher intensity measurements--since the higher CFF values resulted from early measurements during the dark adaptation process. This represents the change in CFF as a function of change in time.

Figures 5 and 6 illustrate condition III in which flicker detection measurements were made during dark adaptation from 5-10 minutes and again after 30 minutes. These are the averaged data of three trials for each of two O's. Figure 6 was derived by subtracting the Log I from early measurements (5-10 min.) from the Log I required for later measurements (30+ min.). Note that in Fig. 6 the change in Log I is always a positive value--meaning that after full adaptation more intensity is required to detect the flickering component of the stimuli than when some of the photopigment is still in the bleached state. At about 5 min. into dark adaptation, approximately 44% of the pigment is still in the bleached state.* This was calculated from Rushton's [1961a] formula

$$(1 - p) = (1 - p_0) 10^{-t/14}$$

where $(1-p)$ is the fraction of rhodopsin in the bleached state after time t (min.) in the dark. $(1-p_0)$ is the initial fraction bleached.

* I am grateful to J.F. Kehoe for computational advice.

Figure 5. Results of condition III. Filled circles are flicker detections recorded from 5-10 minutes of dark adaptation. Open circles are flicker detections measured after 30 minutes (full recovery).

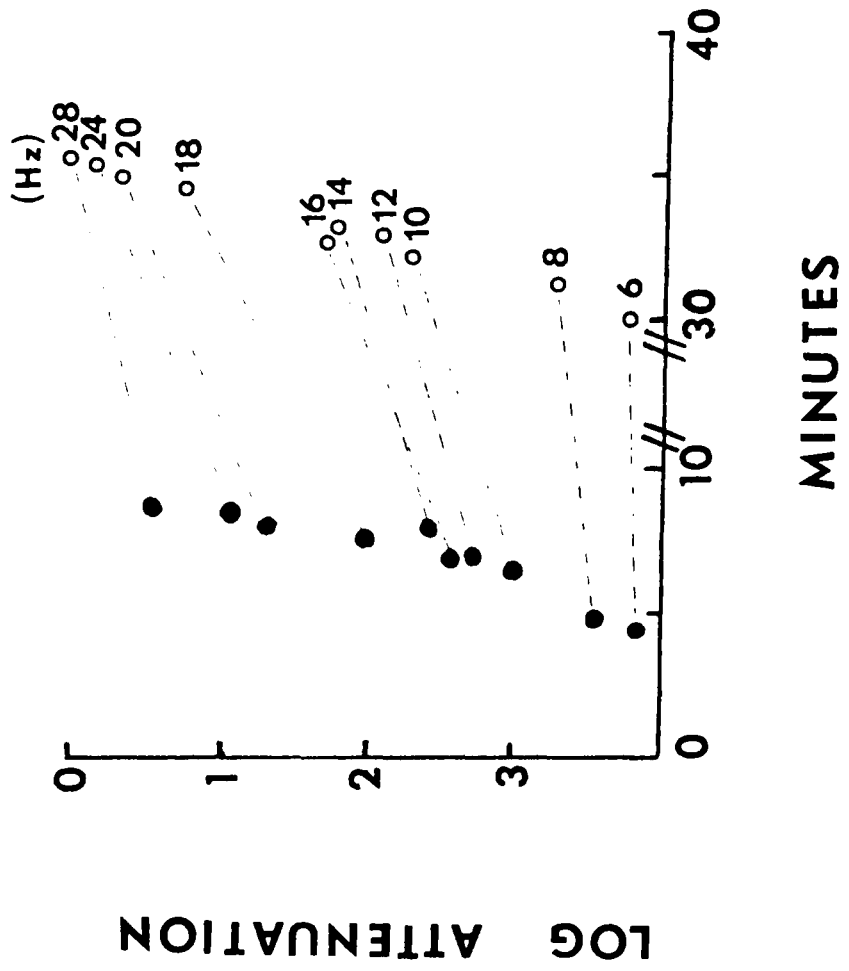


Figure 6. Results of condition III. The ordinate is the difference in Log I between measurements made from 5-10 minutes and those made after 30 minutes of dark adaptation. Vertical lines represent ± 1 standard error of the mean.

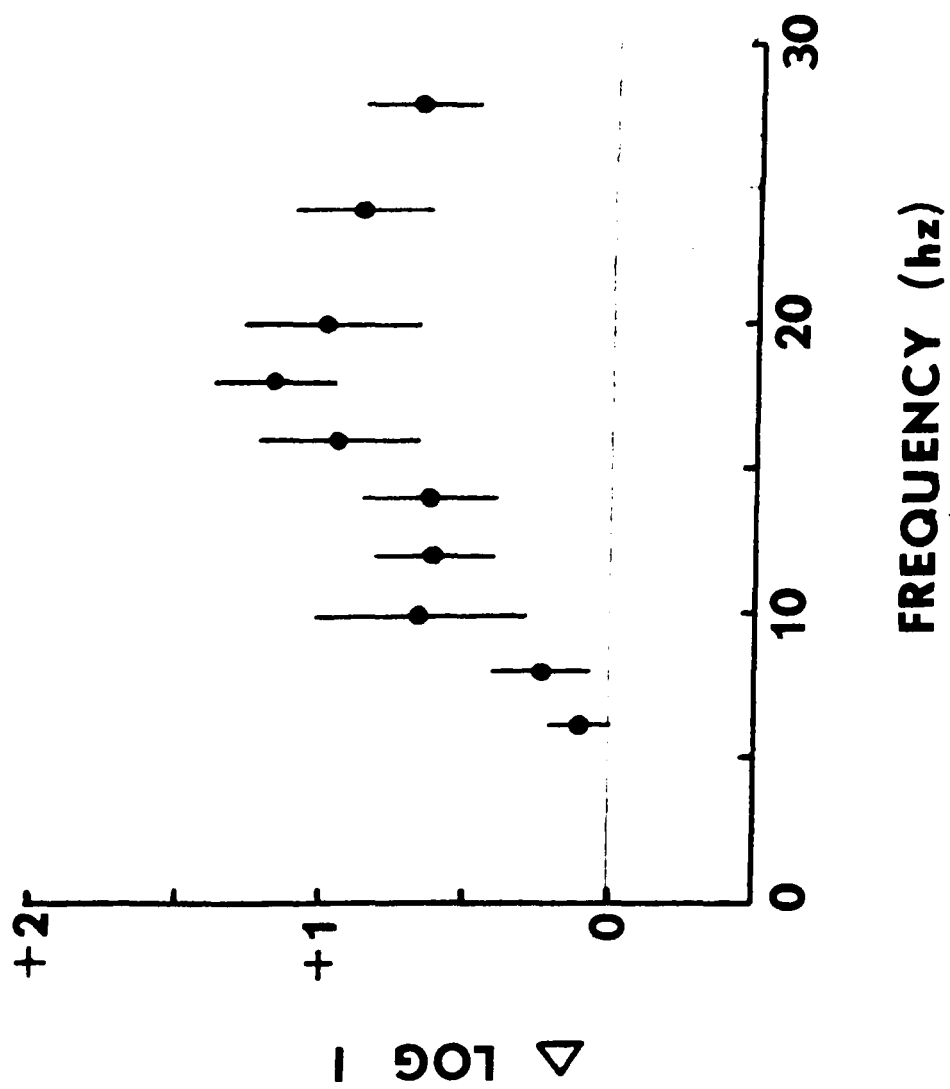


Figure 7. Results of condition III were recorded from a rod monochromat. The ordinate is the difference in Log I between measurements made from 5-10 minutes and those recorded after 30 minutes. Vertical lines represent ± 1 standard error of the mean.

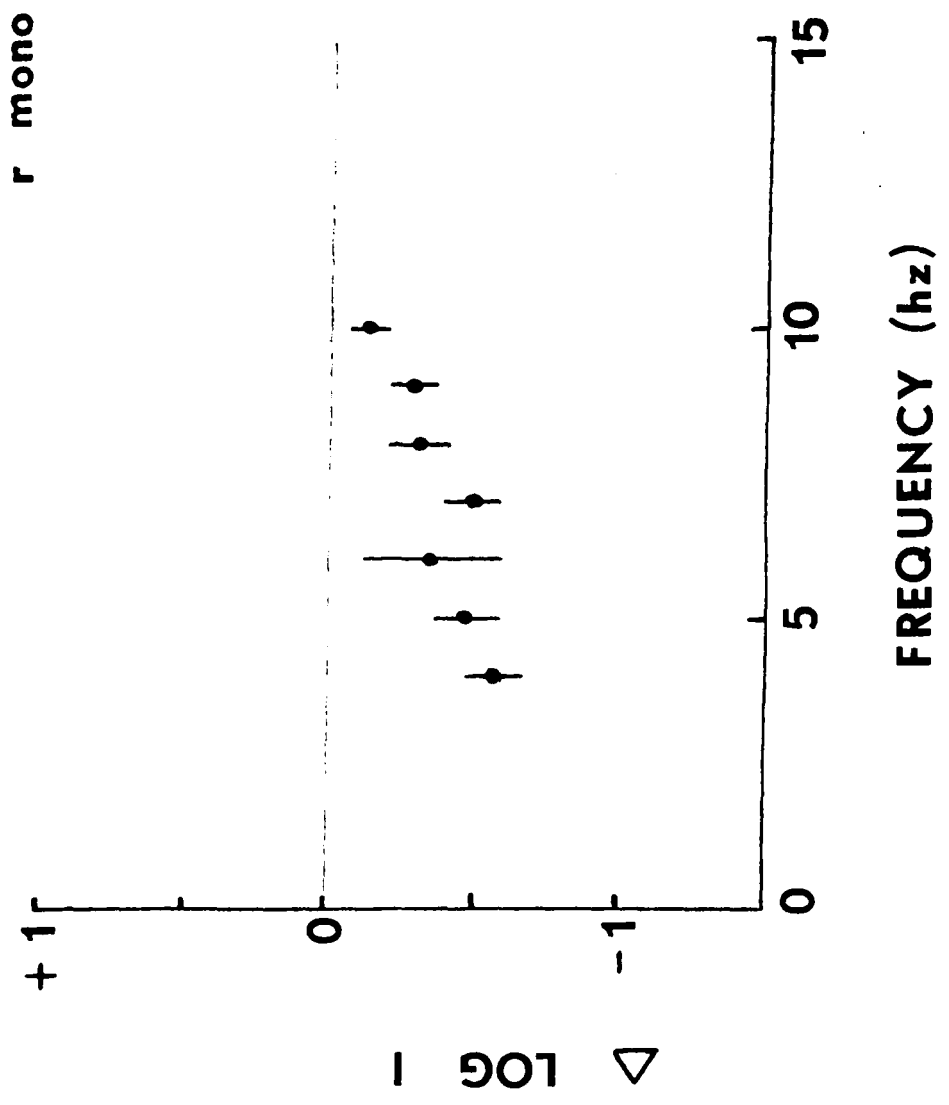
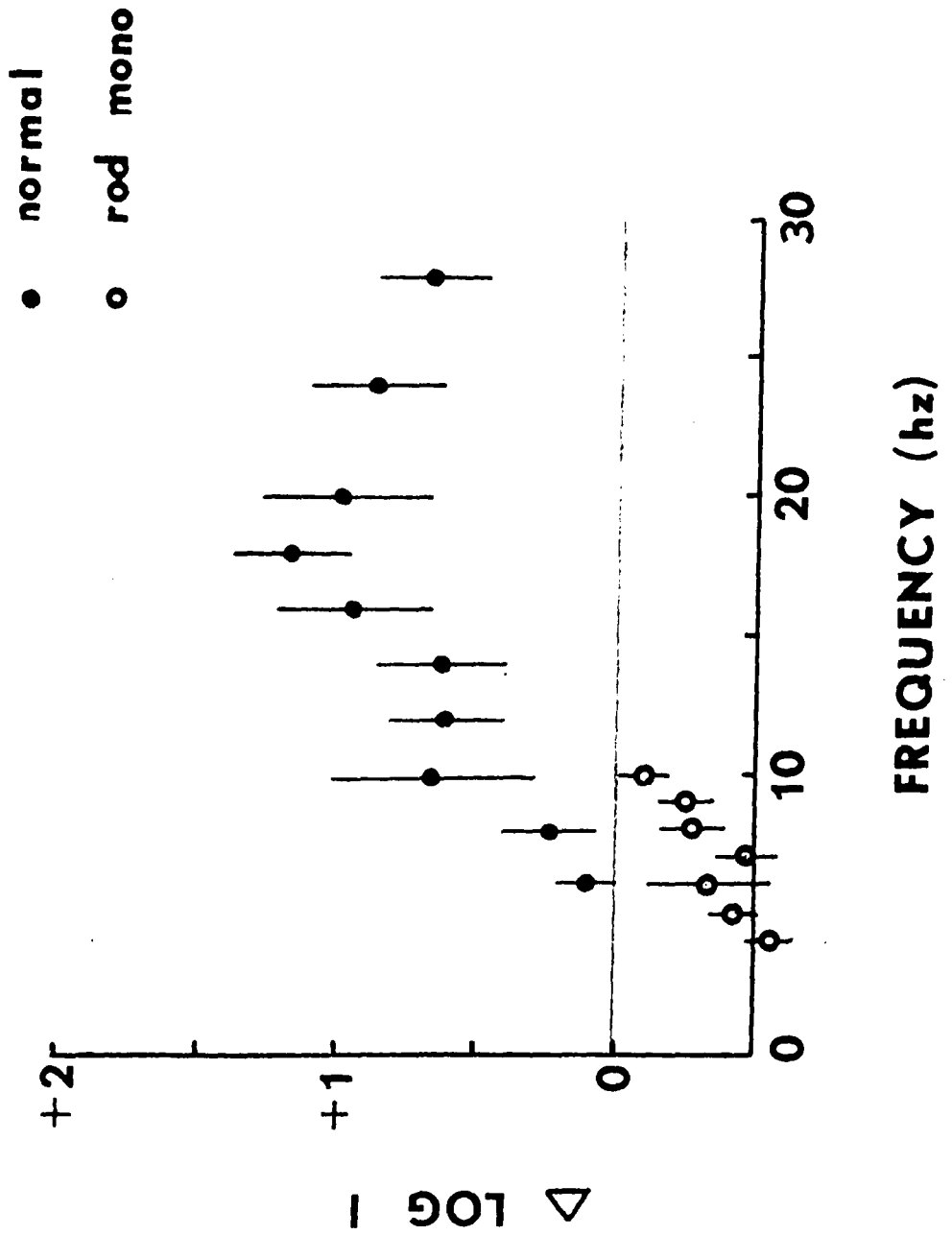


Figure 8. Compares results recorded from a rod monochromat (open circles) to those recorded from normal observers (filled circles).



Furthermore, these large differences in intensity required to detect the same frequency suggest that the cone signal is inhibited or suppressed due to rod intervention. The only real physical difference in flicker detection between 5-10 min. and after 30 min. is due to the amount of photopigment in the bleached state. Which is to say that the difference is due to only the cones being able to contribute to the threshold vs. both cones and rods contributing to the threshold. This definitely implies a rod-cone interaction which causes a decrease in the flicker sensitivity of the system.

Fig. 7 shows the data collected from a rod monochromat (Condition III) over two trials. Rod monochromacy was diagnosed after tests from an anomoscope, pseudo-isochromatic plates, and acuity measurements. Monochromacy itself was a factor limiting the test frequencies to 10 Hz and below. However, observe the significant discrepancy in the obtained frequencies from the monochromat compared to those obtained from normal subjects (Fig. 7 vs. Fig. 6). Theoretically, the only physical difference between the monochromat and the normal is that the monochromat is devoid of functional cones. This should mean that the difference between Fig. 6 and 7 would be attributed to the effect of cones interacting with the rods in the normal eye. The comparison of normal vs. rod monochromat is reported in Fig. 8. Since there is no positive change in sensitivity in the monochromat's data, but actually a negative change--this obviously suggests rod-cone interaction in the normal eye.

Condition IV is represented by Fig. 9 and Fig. 10. Both of these figures are data averaged from two subjects with two trials each. Fig. 9 represents data from the subject observing just perceptible flicker from a test stimulus flickering at 10 Hz throughout the dark adaptation procedure. Foveal and 7° parafoveal conditions are compared in both figures. Fig. 10 presents data of similar conditions using 12 Hz as the continuous frequency of the test flash. Fig. 11 presents data for 20 Hz. Fig. 12 represents one trial for one subject using a 10 Hz flickering stimulus throughout dark adaptation. During this run, specific control procedures were utilized as follows. The stimulus was withdrawn at 25 min. into dark adaptation including the fixation cross hairs. At 30 min., when the conditions were resumed, note that the attenuation required to perceive flicker was the same as for 25 min. for both foveal and parafoveal conditions. This control was implemented so as to illustrate that our procedure shows virtually no real drift or change in sensitivity due to observing flicker itself. Furthermore, for each run (foveal and 7° pf) at 33 min. the subject was asked to switch to the other fixation and track for a few minutes. For example, if the subject was tracking the 10 Hz stimulus at 7° fixation, he would change at 33 min. and observe the 10 Hz stimulus at foveal fixation and vice versa. This was undertaken to provide measurements of internal consistency. Note how close in log attenuation the measurements are. Note also, the consistency between Figs. 9 and 12. This provides

evidence that there is no real shift or change in flicker threshold due to flicker itself.

Figure 9. Intensity required to detect 10 Hz flicker at foveal fixation and at 7° parafoveal. Open circles represent measurements from 7° ; filled circles were recorded at foveal fixation.

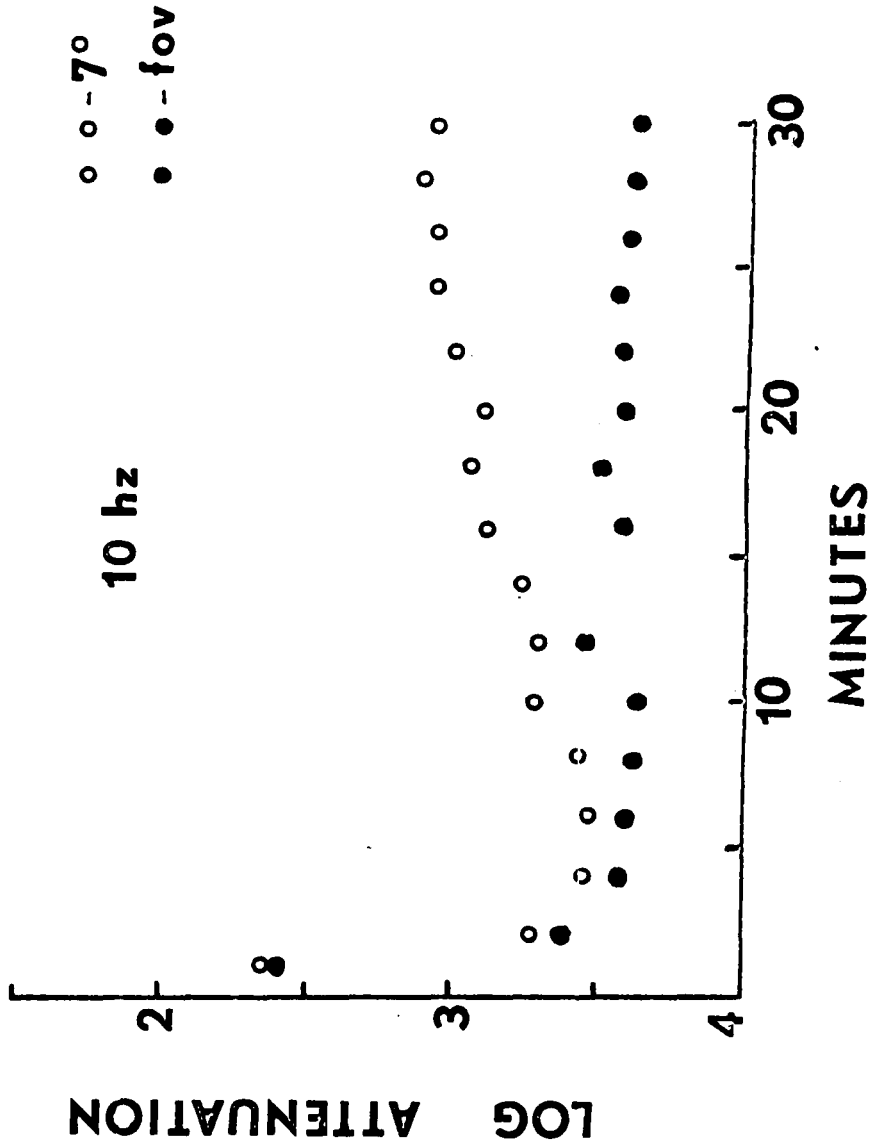


Figure 10. Intensity required to detect 12 Hz recorded at foveal fixation (filled circles) and at 7° parafoveal fixation (open circles).

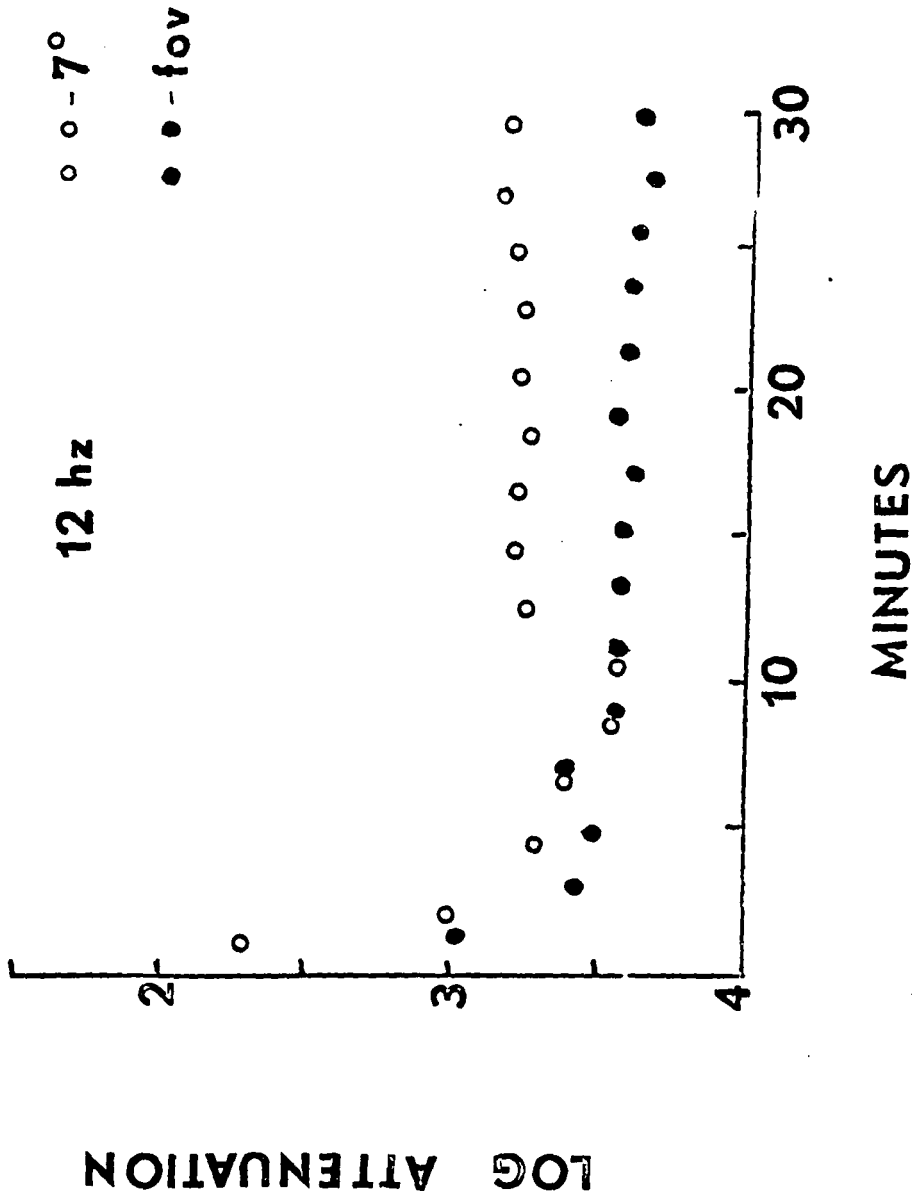


Figure 11. Intensity required to detect 20 Hz recorded at foveal fixation (filled circles) and at 7° parafoveal fixation (open circles).

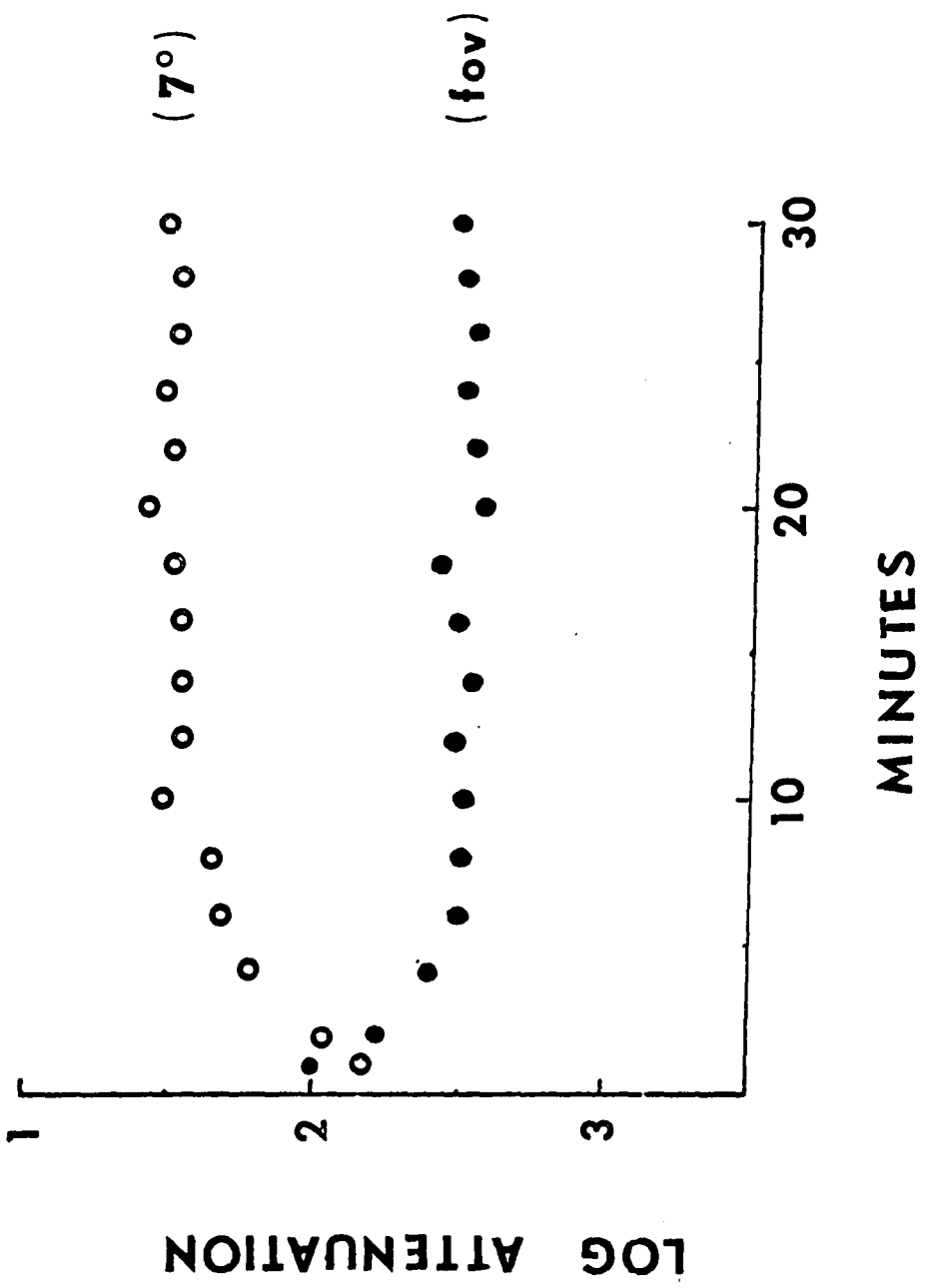
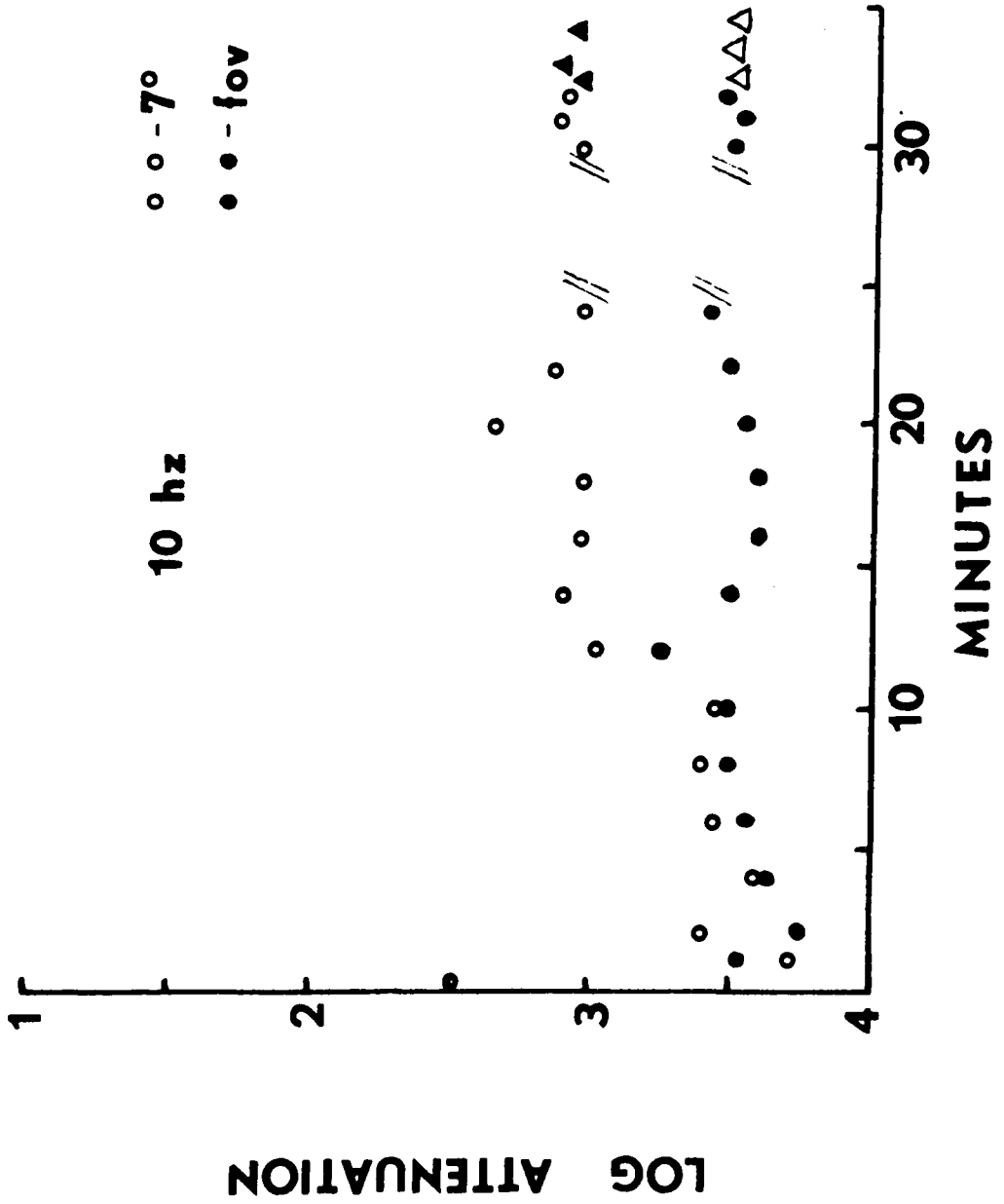


Figure 12. Shows the difference between 10 Hz recorded at foveal fixation (filled circles) and at 7° parafoveal fixation (open circles). All visual stimuli were extinguished at 25 minutes for 5 minutes, then tracking resumed. Open triangles represent foveal fixation measurements after tracking at 7° parafoveal for 30 minutes. Filled triangles were recorded at 7° parafoveal after tracking at foveal fixation for 30 minutes (minus the 5 minutes of darkness).



DISCUSSION

The duplicity theory of man's retina has long been accepted and it is generally assumed that rods and cones act in a relatively independent fashion. The two systems also seem to vary independently in their sensitivity. This is exemplified by the familiar dark adaptation curve whereby cones recover full sensitivity soon after bleaching, while rods are still insensitive. An example of interaction is discussed later in the floating of the cone plateau during dark adaptation.

This question of rod-cone independence during the recovery of sensitivity after bleaching was well discussed in a paper by Hayhoe, MacLeod and Bruch [1976]. They discussed the reasons for experimentally testing the alternatives of independence or interaction. They suggest that rod-cone independence would imply that the rod and cone signals are separated into different channels as they are transmitted through the afferent stages where the sensitivity loss occurs, which is to say that the sensitivity loss originates solely within the retina, even before the rod and cone signals are pooled at the ganglion cell. However, if there is interaction, then the hypothesis is plausible that the ganglion cell possesses a sensitivity regulating mechanism; and there are interacting channels as the signal passes through the retina. Leaving aside the possible involvement of central processes in the recovery of sensitivity, there is a need

for more evidence on the interaction of rod and cone signals in the retina itself. The appearance of both cone and rod contributions to the interocular light adaptation effect [Lansford and Baker, 1969; Paris and Prestrude, 1975; Prestrude, 1974; Prestrude, 1976] suggest that cones and rods interact during pre-adaptation and dark adaptation.

Anatomical interconnections of rods and cones are abundant, but it is not at all clear as to the functional significance of these connections [Rodieck, 1974]. The question will definitely need psychophysical evidence in order to decide the functions. The electron microscopic studies of Dowling [1967] and Dowling and Boycott [1965] indicated that there are ample provisions for interactions among the rods and cones--as did the histological evidence of Gouras [1965] which indicates that both rods and cones converge on the same ganglion cell. In Rushton's [1965] Ferrier lecture to the Royal Society, he presented a model of the input-output relations of the "Automatic Gain Control" (A.G.C.) box in equilibrium conditions. He described the system as follows: Every quantum caught by a rod generates a signal, and the ganglion cell is excited by a quantity proportional to the sum total of these signals within certain space-time limits. But the proportional factor is not constant--it is regulated by adaptation or summation pools. These receive the whole flux of signals and regulate their size so as to comprise an

automatic gain control. Such a mechanism keeps the output at a fairly constant level while preserving the contrast of neighboring regions.

Stiles [1939] and Flamant and Stiles [1948] demonstrated rod-cone independence of rod increment thresholds. Rushton [1968], under certain conditions, verified rod-cone independence psychophysically in parafoveal areas. Dodt and Jessen [1961] found rod-cone independence during bleaching recovery in the frog ERG. In contrast, it has been noted that some parafoveal dark adaptation curves tend to gradually increase after 7 minutes [Wooten, 1975; Hayhoe et.al., 1976; and in our own laboratory]. If so, this could be evidence for rod-cone competition. MacLeod [1972] reported an experiment in which a colored stimulus was observed as flickering at rod (dim) and cone (bright) levels of intensities, but appeared steady over a range of mesopic (middle) intensities. He concluded that the steady mesopic appearance was due to a cancellation between rod and cone signals being equal in amplitude to the cone signal, but delayed by a 180° difference in phase.

Our results concerning changes of CFF during dark adaptation are in agreement with those reported by White and Baker [1976]. It appears that earlier findings on this subject may be influenced to an unknown degree by the aforementioned methods which were used. However, the results from this research demonstrate without a doubt that critical flicker frequency decreases with state of adaptation

after a strong bleach. We have demonstrated this in all 28 trials of conditions II, III, and IV.

Figures 9, 10, and 11 show that flicker perception requires more intensity in the periphery (7°) than in the fovea for 10 and 12 Hz. This is perplexing--since the periphery contains rods (and cones) which should be more sensitive requiring less intensity for flicker detection. However, this is not the case, and we believe that there is a rod-cone interaction in the periphery which suppresses temporal sensitivity. However, recently Baker and Bargoot [1977] have demonstrated that the absolute threshold for light detection was suppressed when the flicker rate of the stimulus is slightly increased--i.e., from 1 Hz to 4 Hz. Our results imply that peripheral CFF is lower (in frequency--meaning less sensitive to flicker) than foveal CFF. Brown [1965] states that whether CFF increases or decreases from the fovea toward the periphery is a clouded issue, mostly due to varying pupil size.

Our results show a definite decrease of parafoveal flicker sensitivity which may be attributed to the regeneration of rod photopigment--since the effect is unnoticeable until rod sensitivity begins to function (about 10-15 min. after a bleach). However, under certain conditions we have found evidence for rod-cone interaction--at a point in time before the rods appreciably contribute to the threshold. The rod free fovea shows no lasting change in sensitivity from 5 to 30 min. as the parafovea does. Note that in Figs. 9, 10,

11 the curves for foveal and 7° are about equal from 5 to 15 min. but after that they distinctly separate. Furthermore, some foveal data points rise above that of the parafoveal points at 5 to 15 min. This could suggest interaction due to a floating effect, similar to that reported in traditional dark adaptation curves at the same time period [Wooten, 1976; Hayhoe et al., 1976; and in our own lab.].

The rod-cone interaction leading to suppression of flicker thresholds is quite robust. Figs. 4, 5, 6, and 7 demonstrate this rod-cone interaction most significantly. The data support the contention that the interaction is due to state of retinal adaptation. Figs. 5 and 6 show the effect due to different levels of retinal adaptation. At 5 min. about 44% of the photopigment is still in a bleached state. At 10 min. about 15% is still bleached, whereas at 30 min. about 99% has regenerated (only 1% in a bleached state). This difference of remaining bleached pigment (or non-regenerated pigment) represents the change in sensitivity in which rod-cone interaction can be observed.

Many have tried to discover whether bleaching raises the threshold by desensitizing the rods, or by decreasing the effect of their signal. What was not clear was why or how bleaching raises the threshold. Recently, it has been discovered that bleaching does not affect the capacity of the rods to convert quanta caught into signals. Rather, through "adaptation pools," bleaching reduces the effect of those incoming signals [Rushton, 1969]. These adaptation

pools are a special retinal organization with the following properties: they collect signals from those rods which catch quanta and by feedback modify the conducting paths from all rods to ganglia in their region so that the unit signal is reduced in size. Thus, it can be said that in all conditions (up to rod saturation) each quantum caught generates a signal. In light of this, it would not be unreasonable to propose that the cone signal for flicker thresholds acts in a similar (but reversed) manner. That is, as dark adaptation proceeds and regeneration of rhodopsin occurs, the effect of the rod signal becomes greater. This large rod signal can inhibit the effect of the cone signal (or vice versa--cones may act on rod signals) as shown by the photopic or mesopic flicker threshold measurements made when nearly all of the rhodopsin has regenerated (Conditions II and III). By virtue of temporal priority and relatively low levels of light, this rod-cone suppression of the sensitivity could occur at or possibly before the adaptation pool.

The cone pigments regenerate about 5 times as fast as the rod pigment does [Wald, 1967]. Therefore, the cones have a much greater affinity for the molecular structure necessary for regeneration (11-cis retinol). "Thus, during dark adaptation, rods fare badly in their competition with the nimble cones." Rushton [1968] also showed that rods regenerate even more slowly when cones in their neighborhood are regenerating. However, it may be possible that once the cones are regenerated, and the rods now have "the line" for regeneration--they may not give it up so easily. (This should not be confused with the

signals from "dark" receptors which may be inhibited at the summation pools.) The foregoing exerts a suppression on the rod signal arising from moderate light intensities--if regeneration is directly related to the signal. However, it may be that when the rods have "the line" (after 15 min. of adaptation) they may not give it up so easily--exerting a suppression of the total signal.

Recently, Rodieck and Rushton [1976] performed a clever experiment in which an electrode recorded responses of a ganglion cell supplied by rods and cones. They were able to observe cone cancellation of the rod signals as well as rod cancellation of the cone signals. They concluded that it could only be the increasing sensitivity of the rods that quietened the cone signals, and that when the rods became sensitive to the stimulus, the cones began to be silenced.

In light of this and our results, we conclude that the rod-cone interaction and cone-rod interaction results from the cones and rods both being able to contribute to the threshold (or both being able to send a signal to the ganglion cell). So that when both are active, the suppression of the total signal is possible and does occur. As for the "How" of suppression, this study supports MacLeod's idea of additive cancellation. In which rod signals are 180° out of phase to the cone signals but equal in amplitude and this causes an additive cancellation or inhibition of the total signal probably at the ganglion cell. Rodieck and Rushton [1976] also suggest this cancellation hypothesis.

At any rate, it is apparent that some sort of interference is occurring when the rods and cones are able to both contribute to a threshold. In this way, it may be attributed to the state of adaptation. The site of this rod-cone interaction appears to be within the retina. The possibilities include (in the order that the signal is traversed) (1) photoreceptors, (2) horizontal cells, (3) bipolar cells, (4) amacrine cells, (5) ganglion cells. It is extremely unlikely if not impossible, that the interaction would occur at the photoreceptors themselves, since there is no immediate interconnections or filtering mechanism at this site. It is also unlikely that the horizontal cells carry out this interaction. Many horizontal cells lack axons, and recent electron microscopy show that they only make synaptic contact with rods [Rodieck, 1973]. The bipolar cells, however, make contact with rods and cones, and it has been shown that some bipolars receive visual information from both rods and cones. Furthermore, Dowling [1967] states that the main site of visual adaptation is probably in the bipolar cell layer of the retina. He cites studies that have demonstrated reciprocal synapses between the bipolar terminals and amacrine processes, and he suggests that such a synaptic arrangement could account for visual adaptation by a mechanism of inhibitory feedback on the bipolar cell. This is also congruent with Rushton's point of view.

Amacrine cells have no identifiable axon and rarely have influence on ganglion cells--if they do communicate with ganglion cells, it is only in specialized parts of the retina. Furthermore,

the horizontal-to-bipolar-to-ganglion cell chain constitutes the principal neuron network that feeds into the ganglion cell [Chan and Naka, 1976]. Amacrine cells are thought to function as connections to and from other bipolar cells. Amacrine cell functions remain the most obscure of the 4 cell types.

Ganglion cells are known to convey many types of information. They do this entirely via their pattern of spike discharge. Many have suggested that they perform certain "brain" functions such as recoding signals. Rushton [1969], Brindley [1955], Prestrude [1971], and others cite the ganglion cell as the most probable location for the summation pool and automatic gain control. The main function of the ganglion cell is to fire impulses up the optic nerve.

This research supports past evidence which contends that this type of interaction occurs at the ganglion cell layer of the retina. However, it may actually be the bipolar cell which acts on the ganglion cell to inhibit or suppress the total signal. However, we are not yet convinced that the results are caused purely by rod suppression of the cone signal or vice versa. We would rather state that the interaction is a result of rods and cones contributing together to a total signal but rods as well as cones can be the inhibiting factor. Rushton [1968] and others have demonstrated that this type of sensitivity loss can be regulated by the ganglion cell. Remember, it is the ganglion cell that has the "last say" on sensitivity before the signal is traversed up the optic nerve.

SUMMARY

It can be concluded the CFF decreases during dark adaptation due to the changing level of adaptation. This decrease in CFF during dark adaptation is also linked to the recovery of rod sensitivity.

Furthermore, we have clearly demonstrated an interaction of the rod signal and the cone signal effecting a general suppression or inhibition of the total signal contributing to the threshold. Now that the interaction itself is the obvious, the mechanism of this interaction should be uncovered. Is it the rod system suppressing or interfering with the cone system or is it the cones interfering with the rod signal? We conclude that when both rod and cone signals are both contributing to a stimulus response, additive cancellation or suppression of the total signal occurs under certain conditions.

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ROD-CONE INTERACTION THROUGH FLICKER DETECTION AND
THE COURSE OF CFF DURING DARK ADAPTATION

by

Thomas A. Bruch

(ABSTRACT)

Critical flicker fusion (CFF) functions are reported during the course of dark adaptation under various states of cone and rod adaptation. Also, measurements were made of the stimulus intensity necessary to just perceive flicker for a wide range of frequencies. The resulting data clearly demonstrate a rod-cone interaction causing a suppression of the threshold.

Our results reproduce the familiar rod and cone branches of the CFF function, and confirm the assertion that CFF is dependent upon state of retinal adaptation. Intensities necessary for flicker detection were recorded at various frequencies during the cone plateau. However, the very same frequencies required as much as 1.5 Log units more intensity when recorded after complete rod recovery. We were able to detect the cone-rod interference only at the time when the rods begin to significantly contribute to the threshold. Our data suggest a channel whereby the rods have an accessible mechanism of directly influencing the cone signal as well as the total contribution to a threshold. The probable retinal area of this interaction is

also discussed. Results are reported from a rod monochromat and six color normals. This study discusses other stimulus properties which could also reveal the rod-cone suppression.