The Blue Arcs of the Retina

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ABSTRACT Around a dim light viewed in a dark room can be seen faint blue-gray arcs which occupy that part of the visual field corresponding to the retina where the arcuate nerve fiber bundle passes from macular ganglion cell bodies to the optic nerve. These blue arcs of the retina are an entoptic phenomenon in which action potentials of the arcuate nerve fiber bundle presumably excite adjacent neurons. The experiments here described show that the light stimulus initially evoking the blue arcs excites cones and not rods as has been generally believed until now. Another commonly held idea is that the blue arcs are produced by bioluminescence or fluorescence associated with the action potentials in the arcuate nerve fiber bundle. The experiments described here disprove this hypothesis.

If a practised observer monocularly views a weak light in a dark room he sees, in that part of his visual field corresponding to the region of his retina extending between the fovea and the optic nerve, a faint bluish gray double arc (the "blue arcs"). The pattern of this entoptic perception agrees exactly with the pattern of the arcuate nerve fiber bundle extending between the fovea and the optic disc. A variety of evidence leads to the inference that the weak light excites the retina in the usual way, and that the resulting activity of some arcuate nerve fibers produces secondary excitation of that part of the retina immediately surrounding these fibers. The evidence upon which this inference has been based, and a good historical review of the experimental work on this effect, have been summarized by Judd (1929) and by Newhall (1937).

It is quite generally believed that to observe blue arcs the initial excitation must be of rods and of rods alone. Amberson (1924) suggested this idea because he found that if the initiating stimulus was confined to the center of the rod-free fovea, blue arcs were not visible. Amberson found that the arcs only became visible provided that the initiating stimulus excited a region of the retina which contained rods. Subsequent mapping of the visual field in which the initiating stimulus may fall and continue to produce the blue arcs has not always confirmed this blue arc blind area in the center of the fovea (Dolecek and de Launay, 1945). Experimental differences of this kind are always difficult to evaluate when (as is here the case) it is impossible to be certain under what conditions exact foveal fixation took place.

A second reason for believing that the initiating stimulus for the blue arcs must excite rods and not cones is that the only action spectrum for threshold visibility of the arcs (Judd, 1929) seems to be more nearly scotopic than photopic.

Rods and cones differ in a number of other ways besides their spatial distribution in the retina and their respective spectral sensitivities. Thus it seemed important as a first step in a better understanding of the phenomenon to provide a firmer empirical basis for this "rods only" hypothesis. Far from doing this, the experiments described below indicated arc visibility when (and only when) cones, not rods, were excited by the initiating stimulus.

A second, more difficult (and less successful) objective of this paper is to attempt an evaluation of the means whereby the physiological activity of nerves might cause "secondary" excitation of the retina. At present only two possibilities are seriously considered: (*a*) Druault (1914) suggested that the nerve impulses were associated with a weak bioluminescence which (because of the extreme sensitivity of the retinal rods) was detected by those rods immediately beneath the active arcuate nerve fibers. (*b*) H. Gertz (1905) suggested that the flow of current associated with the nerve impulses along some active fibers of the arcuate nerve bundle caused resting adjacent nerve fibers belonging to ganglion cells distributed along their path to become active. In support of this latter idea it is sometimes pointed out that ganglion cell axons are unmedullated as long as they remain in the retina. Moreover, recent electron photomicrographs (Cohen, 1961) show that these unmyelinated fibers are packed extremely close together in this region, frequently without intervening glial cell connections.

METHOD

In general, the minimum intensity of stimulus light required to evoke a blue arc was measured. To an experienced observer the arc is easily seen if he fixates the stimulus light directly (or looks just slightly to its temporal side). The stimulation of the retina slightly nasal to the center of the fovea gives rise to a similar entoptic perception, except that while the arcs are invisible the area between the upper and lower arcs becomes a visible bluish gray. This is the blue "spike." These relations between nasal retinal excitation and blue spike, and temporal retinal excitation and blue "arc," agree precisely with the well established distribution of arcuate nerve fibers from nasal and temporal macula, respectively.

The observer viewed the primary stimulus light through a 1.3 mm artificial pupil, and the intensity of this light was gradually increased by rotation of a calibrated neutral wedge until the arcs were clearly visible. The dimension of this light stimulus was a vertical rectangle 132 min by 54 min for the dark adaptation and directional sensitivity measurements, and a 30 min diameter circle for the spectral sensitivity measurements. The room was otherwise dark.

THE PHOTORECEPTORS EXCITED BY THE INITIATING STIMULUS

1. *Dark Adaptation*

If the photoreceptors which must be excited by the stimulus light in order to see the blue arcs are only rods, the change in blue arc sensitivity following bleaching of the rhodopsin in these rods should closely follow the dark adaptation curve for rod vision.

To study this dark adaptation process, the observer was first exposed for 45 sec to a circular field (white light) $2\frac{1}{2}°$ in diameter which was centered on the region of the retina where the stimulus for the primary light appeared. This light adaptation field was sufficiently large to cover the entire region of the retina to which the light stimulus was exposed, and sufficiently small so that it did not include any of the retina with which the observer sees the blue arcs themselves (except for entoptic scatter). The intensity of this light adaptation was 2.04×10^5 trolands, which suffices to bleach 60% of rhodopsin in the rods in the region of focal illumination when exposed for 45 sec.

Fig. 1 shows the change in minimum intensity of the stimulus required in order to see the blue arcs at various time intervals in the dark following this 60% rhodopsin bleach. The orange test stimulus was provided by a Wratten No. 22 filter (dominant wavelength 599 mμ). It is evident that after 5 or 6 min in the dark there is no further increase of sensitivity to the blue arcs.

The test stimulus in Fig. 1 was orange, which can be expected to excite rods only relatively weakly. It was not used in these experiments for this reason but because the arcs are much easier to see with long wave stimulation than with lights of short wavelengths. However, dark adaptation curves like the one illustrated in Fig. 1 have also been obtained using stimulus lights of other colors including blue (λ = 453 mμ; Wratten No. 47B), yellow (λ = 576 mμ; Wratten No. 73), white and green (λ = 538 mμ; Wratten No. 74). A typical result in the latter case is illustrated in Fig. 2. While the shape of the recovery of sensitivity for the blue arcs is quite similar to that measured with orange light, the variability of the measurements particularly during the later intervals in the dark is considerably greater for a green light than for orange. This result is in agreement with those of previous investigators, almost all of whom report difficulty in obtaining good measurements with shorter wavelength stimuli (cf. Newhall, 1937).

If the photoreceptors excited by the (light) stimulus were rods and only rods, a reasonable expectation would be that maximum sensitivity for the blue arcs would be achieved only after 30 min in the dark following a 60% rhodopsin bleach, and that in the intervening interval the threshold would fall

over some 7.0 $\log_{10}$ units (Rushton, 1961). This result was never obtained regardless of the color of the stimulus light employed. Nor was it possible to demonstrate a rod-cone break which might be anticipated if the photoreceptors for the stimulus were both rods and cones. The most reasonable description of all the data is a simple exponential:

$$\log \phi/\phi_0 = 3e^{-t/125}. \tag{1}$$

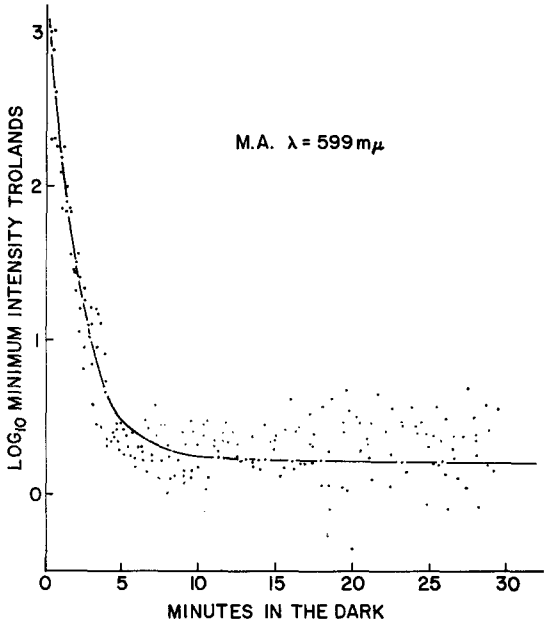

FIGURE 1. Ordinate is the minimum intensity of an orange test stimulus required to observe blue arcs. Abscissa is the time in the dark after 60% of rhodopsin has been bleached by white light at $t = 0$. The bleaching and test lights were projected upon the retinal region associated with excitation of blue arcs but excluded the retinal region in which blue arcs are observed. The results are from five successive experiments. The smooth curves in Figs. 1 and 2 both describe regeneration of chlorolabe (or erythrolabe).

In this equation t is the time interval in the dark in seconds after bleaching, ϕ_o is the minimum intensity required to visualize the blue arcs after 30 min in the dark, and ϕ is that intensity at any other moment t. The smooth curves in Figs. 1 and 2 are drawn according to equation 1. Rushton (1963) has proved that equation 1 describes (*a*) the foveal dark adaptation curve of the protanope and (*b*) the resynthesis of chlorolabe in the protanope's fovea where:

$$\log \phi/\phi_0 = 3(1 - \rho), \tag{2}$$

is the relation between the dark adaptation threshold and ρ the fraction of chlorolabe present at any given moment. Since chlorolabe and erythrolabe have exactly the same bleaching and regeneration constants (Rushton, 1965), the curves in Figs. 1 and 2 are the most reasonable estimate of the recovery of cone pigments following a bright bleaching exposure.

2. *Directional Sensitivity*

These dark adaptation results are precisely what one would expect if the receptors for the stimulus which evokes blue arcs were cones, and not rods.

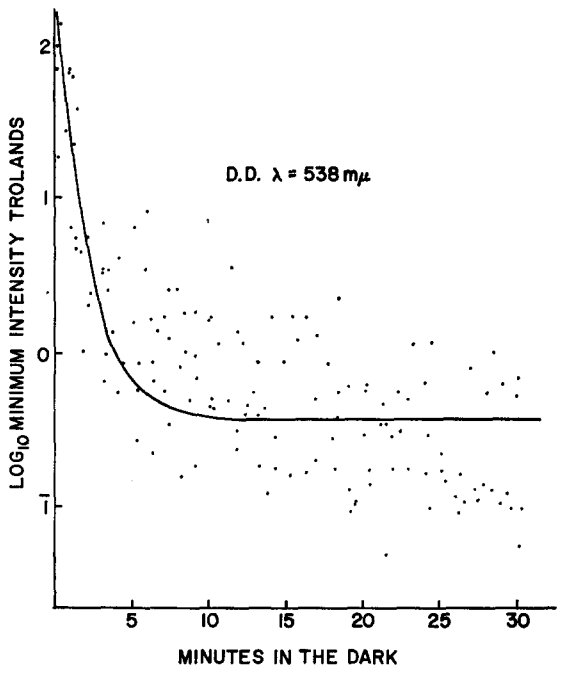

FIGURE 2. Experiment similar to that illustrated in Fig. 1 with a green rather than an orange test stimulus. The increased variability of the measurements is due to the difference in dominant wavelength of the stimulus, not to individual differences in the observers.

If this is the case one expects further that the intensity threshold for exciting blue arcs would vary systematically depending upon the region of the pupillary entry. Flamant and Stiles (1948) showed in man, as did Donner and Rushton (1959) for the frog, that while the cones are quite sensitive to the direction of incident light the rods are not.

The dental bite which held the observer's head fixed in the experiments just described was itself mounted to a very rigid frame which could move the head in any given meridian with respect to the center of the observer's pupil. This allowed the (1.3 mm) aperture stop of the apparatus to be centered on different parts of the pupil (dilated with 3 drops of 1½% Mydriacyl). Measurements were begun with the light entering through one edge of the pupil.

Three measurements were made in succession. The biting board was horizontally displaced 0.5 mm and the measurements repeated. This same procedure was followed until the horizontal meridian of the pupil was completely traversed. In making these measurements it was found that adaptation was best controlled by turning the room lights on between each successive measurement. This served to keep the eye moderately light-adapted and to give results with the smallest variability.

The data in Fig. 3 are typical mean results of three repetitions of this ex-

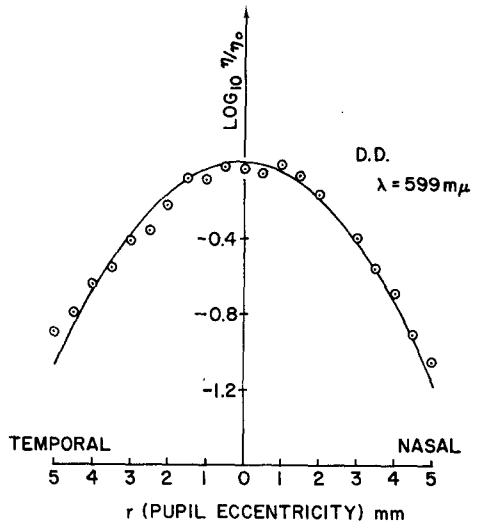

FIGURE 3. Intensity of an orange vertical rectangle required for threshold blue arc visibility when the stimulus light is directed through different parts of the widely dilated pupil. The ordinate is the logarithm of the ratio of the intensity at any eccentric point to that at the center of the pupil. The points are the means of nine measurements completed in three separate experimental sessions. The smooth curve describes Stiles' (1937) measurements of the directional sensitivity of his foveal cones by a brightness matching method.

periment. The smooth curve has the equation:

$$\log_{10} \eta/\eta_0 = -\alpha r^2 \tag{3}$$

in which η_o is the sensitivity to blue arcs when the light goes through the center of the pupil, and η its sensitivity when the light goes through any eccentric pupillary point r (in millimeters), and α is a constant (0.045 in this particular case). Stiles (1937) proved that equation (3) satisfactorily described the directional sensitivity of his own foveal cones, and Fig. 3 shows it is also a reasonable description of the directional sensitivity of the photoreceptors which are excited by the light giving rise to the blue arcs. The inference is that these latter are cones, not rods.

3. *Spectral Sensitivity*

Both the dark adaptation and directional sensitivity measurements lead to this same conclusion. But a convincing argument requires substantiation with

action spectrum measurements which correspond to the known form of one or more of the cone mechanisms, or to the photopic spectral sensitivity curve.

In the dark adaptation experiments (Fig. 1) with various dominant wavelengths of stimulus light, it was found that all the thresholds were more or less the same after 30 min in the dark. This suggested that the spectral sensitivity of the effect was a photopic curve rather than the scotopiclike curve described by Judd (1929). This impression was confirmed by measurement of the minimum intensity of initiating light required for a blue arc threshold at 10 mμ steps across the visible spectrum. The apparatus has been described elsewhere (Alpern et al., 1960). For the present purpose we did not use the 1° surround and this beam was occluded. The test field consisted of a 30 min circular narrow band (4 to 6 mμ band widths) monochromatic field. The measurements were made in the dark, but the room lights were turned on between measurements while data were recorded and adjustments in the wavelength drum and neutral wedge were completed. The elapsed light adaptation time was about 1 min. The measurements themselves could be made in a period about half that long. We always increased intensity until arcs were barely visible. These conditions differ somewhat from those of Judd, who kept the room lights on during the measurements, varied the spatial positions of the fixation point, and decreased intensity to the point of invisibility of the arcs. While these different conditions are probably responsible for part of the difference in the results in the two cases, we have little doubt as to the validity of our measurements nor of the inference to be drawn from them.

Fig. 4 gives the results of such measurements. The rectangles show the means $\pm$ SEM of 30 observations for each wavelength obtained in 10 separate experimental sessions. The circles in the same graph illustrate the foveal luminosity measurement at a level of 2 trolands measured by the cascade heterochromatic brightness matching method on the same observer. The solid line shows the C.I.E. standard observer's photopic luminosity curve.

The agreement between the measured foveal luminosity function and the C.I.E. curve serves as a check on the calibration of the apparatus. The agreement is satisfactory except for wavelengths smaller than 450 mμ. It is well documented (Wald, 1945; Hsia and Graham, 1957; and others) that sensitivity of the standard observer is too low in this region.

The comparison of the circles and rectangles in Fig. 4 shows reasonable agreement through the yellow-green and red parts of the spectrum, and somewhat less satisfactory agreement in the blue-green and blue. The discrepancies are never very large, however. Discrepancies of this same order are readily obtained between two foveal luminosity curves on the same observer measured at different luminance levels (Wright, 1946; Alpern, unpublished observations), and it seems quite likely that the discrepancies illustrated in Fig. 4

are attributable to the difference in luminance levels of the test stimulus when the blue arcs were measured compared to that used to obtain the luminosity curve (2 trolands).

However these differences are to be accounted for, these spectral sensitivity measurements verify the conclusion already drawn from the previous experiments. The photoreceptors excited initially by the light stimulus when we perceived the blue arcs in our experiments were cones and cones alone.

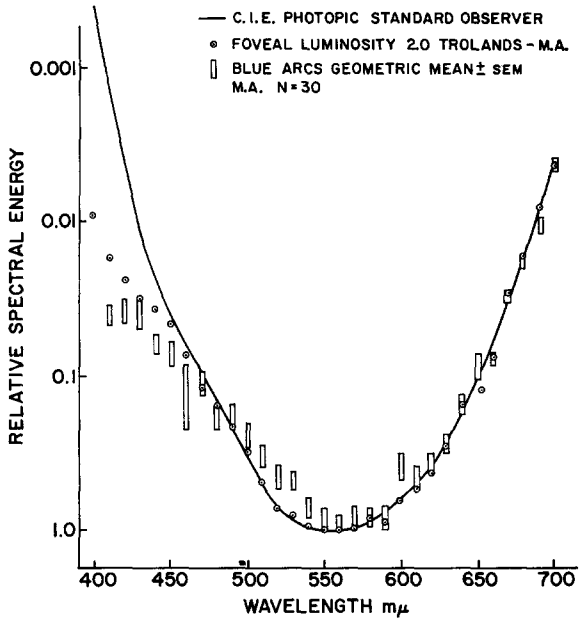

FIGURE 4. Relative energy of different wave bands in the spectrum required for threshold visibility of the blue arcs. The rectangles are the geometrical means ±1 SEM of thirty repetitions, the circles show foveal luminosity measurements by cascade method at 2.0 trolands of retinal illuminance. The smooth curve is the C.I.E. photopic curve.

Of course, it remains to some extent an open question whether or not under some peculiar set of circumstances excitation of the rods may possibly give rise to blue arcs. The limited conditions of the experiments described above have by no means exhausted all possible stimulus arrangements under which blue arcs can be seen, and it is still possible that under some of those not studied here, the arcs could be produced by initial rod excitation. The above experiments: (*a*) show that the arcs are produced under conditions of initial excitation of cones, and only of cones, and (*b*) outline the kind of documentation that is required in any experimental demonstration that initial rod excitation may also produce blue arcs.

THE HYPOTHESIS OF RADIATION OF VISIBLE LIGHT ASSOCIATED WITH NERVE ACTIVITY IN THE ARCUATE NERVE FIBER BUNDLE

In the introduction it was pointed out that only two hypotheses are still seriously considered to explain how nerve action potentials in the arcuate nerve fiber bundle can give rise to secondary excitation of surrounding quiescent nerve cells in the retina. In this section, one of these (Druault's notion that the action potentials are associated with the radiation of visible light which excites photoreceptors beneath the pathway of the arcuate nerve bundle) is examined.

The idea that retinal rods and cones are excited by the bioluminescence or fluorescence associated with action potentials in ganglion cell axons is not very satisfying on theoretical grounds. On the other hand, none of the other possibilities is of very great theoretical appeal either, and so it would be a mistake to dismiss Druault's idea without direct experimental evidence that it is untenable. The following experiments provide such evidence.

A. *The Nature of the Photoreceptors*

If action potentials in the retinal ganglion cell axons give off visible light, which of the photoreceptors in the retina does this radiation excite? There are only four possible answers to this question, namely π_5 (red) cones, π_4 (green) cones, π_1 (blue) cones, or π_o rods. It is quite unlikely that excitation of either of the first two of these could account for the blue color of the blue arcs. Newhall (1937) made colorimetric matches between the blue arcs and an adjacent field. He found, using a wide variety of different colors for the initiating stimulus, that the color of the arc always clustered in the blue corner of the chromaticity diagram. These experiments lead to the idea that if visible light radiation does cause the blue arcs, it is because it is perceived either by π_1 cones or by rods.

In order to decide between these two possibilities one has merely to take advantage of the well documented fact that threshold of rods and of cones for a given light stimulus is a function of the intensity of the background against which the stimulus is perceived. Stiles (1939) has shown that when a stimulus excites one of the cone mechanisms or the rod mechanism, the elevation of the threshold of that stimulus depends only upon the extent to which the background excites the same mechanism.

METHOD

An image of a tungsten filament was focused on a 1.3 mm artificial pupil providing a Maxwellian view optical system for a background field. The color of this field could

be varied with appropriate filters. Two such were used in different experiments, a red plastic filter (dominant wavelength 625 mμ) and a Wratten gelatin blue No. 98 (dominant wavelength 482 mμ). The scotopic transmittances of these two filters were almost exactly equated in the tungsten light used. The intensity of this background field was varied by Wratten neutral density (No. 96) gelatin filters. The stimulus for the blue arc was provided by the same light source, its beam being split off from the background field beam by a beam-splitting cube. Light in this stimulus beam passed through neutral (No. 96 Wratten) filters, a lens providing a Maxwellian view, and a red Wratten No. 26 (621 mμ) gelatin filter. This converging light pencil was reunited with the light providing the background by a second beam-splitting cube. Between the lens and this cube a field stop was placed to restrict the stimulus light to a vertical rectangle about 10 min wide and about 2° high. A neutral wedge behind this stop permitted variation in the intensity of the red rectangle. A piece of cardboard blocked out one edge of the lens providing the Maxwellian view of the background, and the stimulus for the initial blue arc excitation appeared against this opaque background. The variable color and intensity background appeared just to the right of this stimulus so that it occupied precisely the region of the field in which the blue arc was visible (but none of the region where the initiating stimulating light was seen) when the right eye was used for observation.

The technique used to measure the action spectrum of the red and blue backgrounds for π_1 excitation has been described in detail elsewhere (Alpern and Rushton, 1965). In brief, it involved measurement of a threshold vs. intensity (t.v.i.) curve for a 0.5° blue (Wratten 47B gelatin, dominant wavelength 453 mμ) test flash seen by foveal fixation against a yellow (590 mμ) background field provided by light passing through an interference filter. Stiles (1939) has shown that the π_1 threshold lies about 0.75 $\lg_{10}$ unit above the absolute threshold under conditions similar to these. This was verified for the present case also. At the intensity of the yellow background which sufficed to insure that the test flash excited π_1 an auxiliary background was added to this field. This auxiliary background was the same field as that provided in the blue arc experiments, since it was in this field that we wished to equate the red and blue filters in so far as their ability to excite π_1 was concerned. To do this one merely measured the t.v.i. curves when first the red and then the blue filters were placed in this auxiliary background field. The amount of displacement of the blue t.v.i. curve which was necessary to superimpose it upon the red one represented the neutral density filter which had to be combined with the blue filter in order to give it equal effectivity with the red filter in exciting π_1. This value proved to be 1.35 density units.

RESULTS

Fig. 5 shows the results of the 30 measurements of the intensity required by the red vertical rectangle in order that the blue arc should be visible against red and blue backgrounds of different intensities. In Fig. 5B the abscissa scale shows the background intensity in scotopic trolands. In Fig. 5A the same data are replotted but this time on an abscissa scale equated so that the red and blue backgrounds are equally effective in exciting the π_1 (blue) mechanism for this observer.

The results in Fig. 5 show clearly that if the arcs are produced by secondary excitation of photoreceptors following radiation of visible light associated with the action potential, then these photoreceptors must be rods and not π_1 (blue) cones.

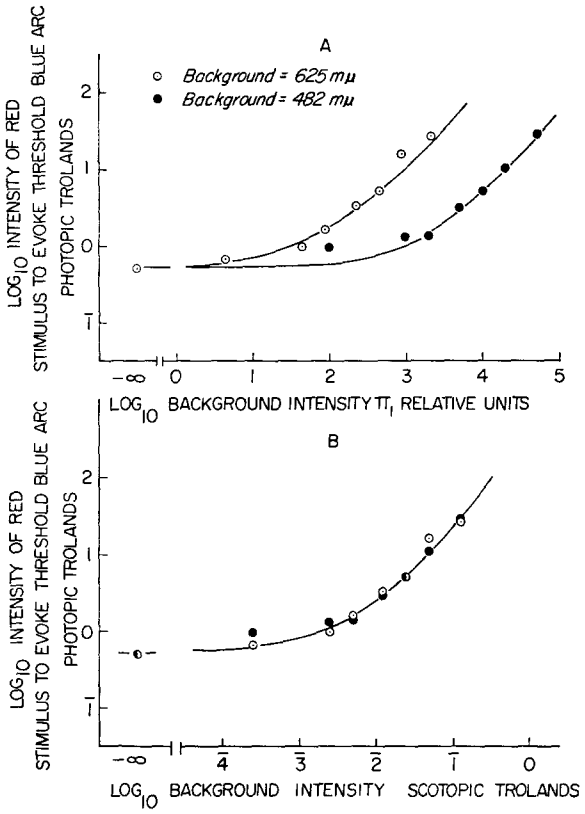

FIGURE 5. The intensity of a red vertical rectangle required for threshold blue arc visibility for different colored backgrounds of various intensities. The data in both figures are the same, but the abscissa scale in A is arranged so that the red and blue backgrounds have equal effectivity in the excitation of π_1 (the blue) mechanism; in B it is arranged so that the red and blue backgrounds have equal effectivity in the excitation of rods.

B. *Evidence against Secondarily Emitted Radiation*

1. BLUE ARC INTENSITY AT THRESHOLD DURING DARK ADAPTATION

If the action potential is associated with radiation of visible light, and this causes the blue arc by photic excitation of underlying rods, it is to be expected that the threshold intensity for excitation of these rods will vary exponentially over a range of 10^7 to 1 with full recovery achieved only after 30 min in the

dark following a 50% bleach of their rhodopsin (Rushton, 1961). This prediction has been tested in the following way: A lens providing a 20° Maxwellian view of a bright tungsten light was partly covered at one edge by a strip of black electrical tape. An observer who adapted to the bright light provided by this optical system when fixating the middle of the strip of tape succeeded in exposing to the adaptation light all that part of the visual field where the blue

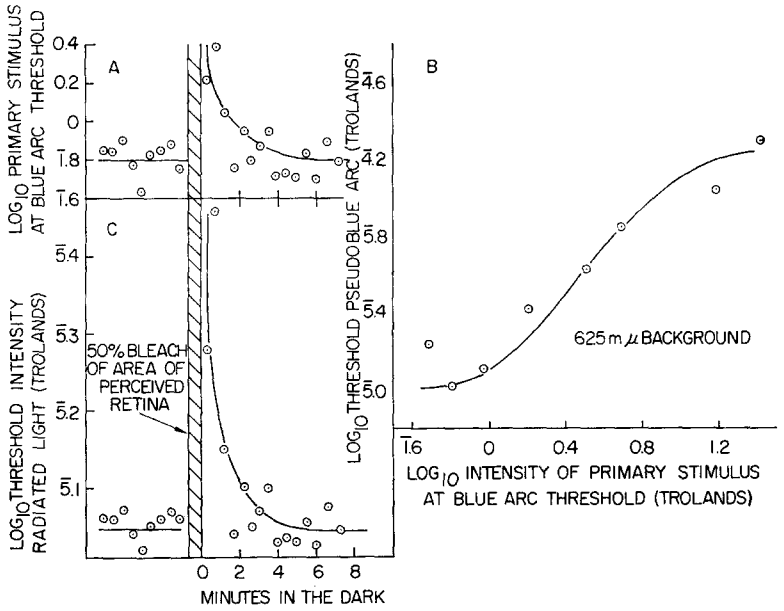

FIGURE 6A. Ordinate is the minimum intensity of a red test light required to observe blue arcs. Abscissa is the time in the dark after 50% of the rhodopsin has been bleached by white light at $t = 0$. The bleaching light was applied only to that retinal region with which the arcs are observed and excluded the retinal region where the test stimulus appeared (except for entoptic scatter). 6B. The relation between the threshold intensity of a pseudo blue arc and intensity of the red vertical rectangle required for arc visibility for different intensities of a red background. The smooth curve shows the trends of the data. 6C. The data in A have been transformed to obtain the intensity of the inferred secondary radiation at blue arc threshold, using the smooth curve in B.

arc (and spike) appeared, and none of that part of the field where the initiating stimulus appeared. Fig. 6A illustrates the variation in the intensity of the primary (initiating) stimulus at the blue arc threshold following 50% bleach of the rhodopsin in the rods presumably excited by the secondary radiation.

The data in Fig. 6A could be used to find the variation in the intensity of the secondary radiation at the blue arc threshold during dark adaptation provided a relation between the intensity of the initiating stimulus and the intensity of the hypothesized secondary radiation could be established. It is possible to work out this relation empirically by measuring the threshold

intensity of a real light of the same shape, field position, and color as the blue arc (a "pseudo blue arc") for different intensities of background and comparing the result to the intensity of the initiating light at the blue arc threshold against these very same background intensities. Fig. 6B shows such a comparison for the case of a red background whose intensities were varied over the entire range in which the arcs were at all visible. The results with the pseudo blue arc are the means of ten repetitions in a single experimental session and show considerable scatter. However, it is possible to describe the relation between the intensity of primary light and the pseudo blue arc threshold as a relatively smooth curve (drawn by eye) to show the trends of the data. This has been done in Fig. 6B. This smooth curve can then be used to infer a dark adaptation curve from data like those illustrated in Fig. 6A in which now the ordinate is the $\log_{10}$ of the threshold intensity of the inferred secondarily radiated light. From the smooth curve in Fig. 6B each datum in Fig. 6A can be used to infer a threshold intensity of secondary radiation, and this is the ordinate in Fig. 6C.

The result in Fig. 6C shows that the intensity of threshold following 50% rhodopsin bleach of the blue arc perception area does not take 30 min to fall over 10 million-fold. On the contrary it requires only about 6 min for full recovery, and the threshold falls only about fivefold.

It is almost certain that even this small transient increase in intensity of the blue arc threshold illustrated in Fig. 6A is not at all due to the bleaching of rods in that part of the retina which perceives the blue arcs, but to the effects of the adaptation light on the cones in that part of the retina excited by the primary stimulus light. While, in theory, the eye's first order geometric image of the adaptation light excluded these cones from bleaching by the adaptation light, in practice because of the inability of the observer to control exactly steady fixation during the 45 sec of bright bleaching and because of entoptic scatter, a certain amount of bleaching of these cones does in fact occur. Fig. 7 illustrates that this effect accounts for most if not all of the elevated blue arc threshold illustrated in Fig. 6A. This figure shows a repetition of the experiment illustrated in Fig. 6A, except that not only the intensity of the primary light to evoke a blue arc (filled circles) but also the intensity of the primary light for visibility threshold (open circles) was measured. In Fig. 7 the bleaching exposure was increased so that 90% of the rhodopsin was bleached, producing a much more pronounced threshold change. The results show that independent of the state of light or dark adaptation of that part of the retina which perceives the arcs, the intensity of the primary stimulus for arc visibility is always approximately a constant multiple of the absolute threshold of that stimulus. Thus the effects illustrated in Fig. 6 are apparently merely a result of the bleach of the cones in that part of the retina which is exposed to the primary light, and not at all a result of the bleaching of the rods in the region

where the arcs are seen. The interpretation seems clear and unequivocal. The idea that blue arcs are caused by photoreceptor excitation by secondarily emitted light associated with action potentials in the arcuate nerve fiber bundle is untenable.

2. BLUE ARC VISIBILITY WHEN THE PERCEIVING RODS ARE INEXCITABLE

It is possible to come to this same conclusion in quite another way. Is there any interval of time following a bleach (of the retinal region used to perceive the arcs) when the blue arcs are clearly visible but a physical stimulus of the same brightness is not? If the answer to this question is "no," then the visible

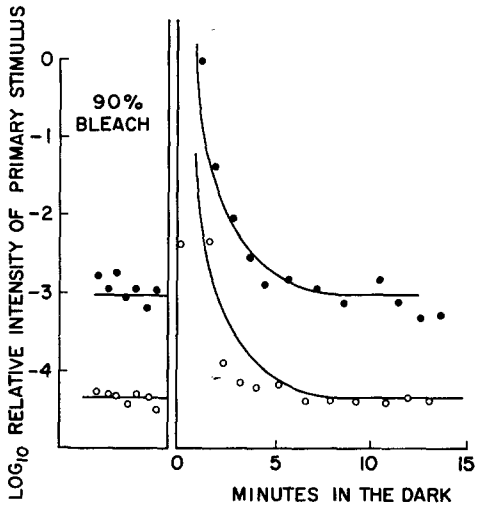

FIGURE 7. Filled circles illustrate an experiment just like that of Fig. 6A except that the bleaching light is much stronger. The open circles show the intensity of the red light at visibility threshold. Single experimental session.

radiation idea may or may not be valid; but if the answer is "yes" then that theory must be wrong. Bleaching rhodopsin so that the relevant rods were insensitive to light of a luminance sufficient to match the arcs in brightness could not permit visibility of the arcs either, if they were perceived by excitation of these same rods by "secondarily" emitted light.

A small 7½ w tungsten bulb was placed behind a 47B Wratten gelatin filter (dominant wavelength 453 mμ) in a light-tight box. The front end of the box about 1 ft from the bulb was milk glass, and its front end was covered by electrical tape cut to expose a region of glass conforming to the shape of the inside part of the blue arcs. The left end of this box contained a small red slit of light whose intensity could be varied to initiate a blue arc. The current to the 7½ w lamp was varied by connection with a variable resistance transformer. This varied the luminance of the field to be matched with the blue arcs, without appreciable change in its color temperature (over the small range varied), because of the relatively narrow band transmittance of the colored filter. The observer's pupil was fully dilated by 3 drops of Cyclogyl

0.5%. He could under these conditions make a reasonably satisfactory brightness match between the "milk glass pseudo blue spike" and the entoptic blue arcs, viewed at about 2 ft distance. The photometry of the milk glass then provided an estimate of the retinal illuminance necessary to match the blue arcs once the size of the dilated and fixed pupil had been measured. Ten adjustments of this sort, each made immediately after the room lights had been extinguished, yielded a mean value of 0.74 troland. This value is in agreement with the value of 0.75 troland obtained by Newhall as the upper limit of the brightness of a "typical" (suprathreshold) arc in similar (though better controlled) experiments.

Measurements were made of the time intervals following a bleach of the region of the retina where both the arc and spike appeared until (*a*) the milk glass pseudo blue spike and (*b*) the entoptic blue arc first appeared. These time intervals were measured with two stopwatches. The same light adaptation field with which the data in Figs. 6 and 7 were obtained was also used here. This experiment has been done a number of different times, and the result is always the same. A typical result of 15 successive measurements (mean ± SEM) gives the time interval of 139 ± 10 sec following a 17% rhodopsin bleach in which the milk glass pseudo blue spike was invisible, while the time interval between the bleach and the appearance of the first blue arc was 48 ± 5 sec. Thus following a 17% bleach there was a time interval of about a minute and a half in which the blue arcs were quite visible but a physical light of the same brightness was not.

The conclusion of this experiment is inescapable. The blue arc phenomenon cannot be explained as the perception of visible radiation secondarily emitted because of the physiological activity of the nerve fibers of the arcuate bundle.

DISCUSSION

It has been shown experimentally (Arvanitaki, 1942; Katz and Schmitt, 1940) in several very artificial situations that an action potential in one nerve can affect the excitability of a neighboring cell, even though there are no specialized anatomical structures connecting them. It is not known whether such effects are of any physiological importance but, as far as we are able to determine, the phenomenon of the blue arcs is the one likely possibility of such an effect known to occur regularly in the normal organism. It is in this light that the examination of the mechanisms involved in giving rise to the blue arcs is of interest.

The present experiments have provided convincing evidence against two commonly held ideas about blue arcs. The first of these is the idea that the initiating (primary) light must excite rods in order to obtain the blue arcs. The second is that blue arcs are caused by excitation of retinal rods (and/or cones) by light quanta originating in association with the action potentials in

the arcuate nerve bundle. It has not been too difficult to prove that neither of these ideas is correct, but it seems very difficult indeed to know by what means action potentials in the arcuate bundle cause a secondary excitation of quiescent neighboring nerve cells in the retina.

What are the remaining possible explanations for this effect? We can think of only three: all these have theoretical objections to them, but in the absence of convincing experimental evidence against them, they cannot be dismissed from consideration. Since the accumulation of such evidence appears to be the most probable way of excluding one or more of these hypotheses, it seems inappropriate to present here a detailed theoretical discussion of these remaining possibilities in the light of existing evidence. We have instead elected merely to enumerate them:

(*a*) The action potential in the arcuate nerve bundle excites the adjacent retinal cells in exactly the same way that an electric pulse to the eye excites a phosphene. The evidence of Brindley (1955) and of Crapper and Noell (1963) suggests that whatever this mechanism is, it remains a different one from that given in (*b*) below.

(*b*) The action potential in one or a small group of fibers in the arcuate nerve bundle excites adjacent quiescent nerve fibers or quiescent ganglion cells (or prevents propagation in spontaneously firing cells) by flow of electric current (Gertz, 1905). The electron micrographs of the nerve fiber layer (Cohen, 1961) clearly show that these unmedullated axons may be contiguous, often without intervening glial connections.

(*c*) The accumulation (or depletion) of a relatively high concentration of extracellular K^+ (or some other substance or substances) in a small interstitial space which only slowly diffuses away leads to an ephaptic excitation of otherwise inactive nerve cells by active ones (J. B. Ranck, Jr., personal communication). In the squid axon Frankenhaeuser and Hodgkin (1956) found that the accumulation of extracellular K^+ had rather marked effects on nerve impulses, and neurons of the cat hippocampus apparently behave in a similar way (Kandel and Spencer, 1961). Maturana (1960) advanced the same idea to explain an ephaptic effect produced in a frog optic nerve, a preparation in which the ultrastructure is in many ways analogous to that of the human retinal arcuate bundle.

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