J. Physiol. (1961), **156**, *pp.* 179–192
With 4 text-figures
Printed in Great Britain

INCREMENT THRESHOLDS IN A SUBJECT DEFICIENT IN CONE VISION

BY M. G. F. FUORTES,* R. D. GUNKEL AND W. A. H. RUSHTON†
From the Ophthalmology Branch, National Institutes of Health, Bethesda, Maryland, U.S.A.

(*Received* 28 *November* 1960)

It is well recognized, particularly from the work of Hecht (1937), that measurements of visual function at different levels of illumination generally fall into two categories, a low intensity scotopic portion and a high intensity photopic portion. Thus in the time course of dark-adaptation, and in the curves where log. increment threshold, log. acuity or flicker fusion frequency is plotted against log. field intensity, the results are found to fall upon two distinct branches. The low-intensity branch has the spectral sensitivity of rhodopsin, and it is not seen in measurements made upon the fovea centralis; it is therefore considered to be due to rod function. The other branch has a different spectral sensitivity, it is found upon the rod-free fovea and hence it is considered to be due to cone function.

Now though there are considerable difficulties in distinguishing the functions of the various types of cone, it is easy to separate cone curves from rod curves over the whole intensity range of the cones. For not only may measurements be made upon the fovea where rods are absent, but even elsewhere it turns out that cones have an increment sensitivity and fusion frequency so much higher than rods that the plotted curves generally show the cone branch substantially in its entirety.

It is otherwise with rod curves, for not only is there no part of the human retina which contains rods without cones, but the very features which make cone measurements outstanding in a mixed population make rod measurements unavailable. In fact, we can only measure rods at intensities below the cone threshold. This does not mean that rods are necessarily inactive at higher intensities, but their activity cannot be measured by ordinary threshold procedures, and we are left wondering what course the rod branch may take after it disappears behind the cone branch.

This question could be answered if we were able to investigate a subject who had no cones but whose rods and rod pathways to the brain were

* Instituto Neurologico, Via Celoria 11, Milano.
† Present address: Physiological Laboratory, University of Cambridge.

normal. The subject to be described in this paper and in the sequel comes near enough to that condition to be of interest.

Photanopia. A rare congenital condition resembling pure rod vision has been recognized for about a century and has played an important part in establishing the duplicity theory of vision (see the historical review in Walls & Heath, 1954). The subjects have little or no colour vision and are hence called 'achromats' or 'monochromats', more specifically rod-monochromats, to distinguish them from an entirely different type of achromat with good photopic vision (cone monochromats). But all the subjects fully investigated have shown features inconsistent with what would be expected of an eye with normal rod vision and nothing else. They have generally exhibited some features resembling a limited cone vision (Lewis & Mandelbaum, 1943; Hecht, Schlaer, Smith, Haig & Peskin, 1948). The degree and the manner in which cones enter varies considerably in reported cases and the cone spectral sensitivity may be maximal either at 440 mμ (Blackwell & Blackwell, 1959), 510 mμ (Sloan, 1954, 1958; Alpern, Falls & Lee, 1960) or in our case at 550 mμ. There is no reason to believe that all these represent the same genetic condition, and indeed the mode of inheritance of the 'blue cone' type appears to be different from that of the 'rhodopsin cone' type (H. F. Falls, personal communication). It seems convenient to have a name shorter than 'incomplete typical achromatopsia' to describe these various cases of very deficient photopic vision, and we shall use 'photanopia' to include them all.

Our object here is to study rod vision in one photanope for the information this may give concerning normal rod vision, and so we shall not be concerned except incidentally with the complex and fascinating questions as to the nature of the cone malfunction. This matter has been discussed extensively by Walls & Heath (1954) and investigated by Alpern *et al.* (1960) with a very impressive range of techniques.

Description of the photanope D.M. She was 17 years of age with no brothers and one younger sister who was said to exhibit the same condition in milder form. There was no known comparable visual defect in parents or relations. D.M. had had the condition all her life and some years back exhibited a marked nystagmus which has now largely vanished (as it usually does in this condition with advancing years).

She saw well (i.e. normally) in dim light, but with increasing illumination she saw worse, and upon questioning it appeared that contrast was lost and the whole field assumed a bright undifferentiated appearance. A retinal illumination of 1000 td would produce this effect, and produce it instantly, and upon return to 1 td the sense of contrast returned within a second or so. It therefore was not linked with the bleaching and regeneration of rhodopsin which takes 4–5 min to recover half way (Rushton, 1961).

Although she did not like bright illumination, it gave her no pain, and she could submit to 30 sec fixation upon 1,000,000 td of retinal illumination without complaint and without the subsequent 2 hr of disability described by Walls & Heath (1954).

Visual acuity was 20/80 as measured by the Snellen chart (no serious error of refraction). Colour vision was nearly absent, but good red or blue lights were always correctly named. Blue-green and yellow were both called grey, and she failed in all ordinary colour-vision tests.

It is important but not easy to know whether the fixation point falls upon the fovea or not. When an ophthalmoscopic arrangement focused a bright 2° field upon the retina and D.M. was asked to fixate the centre of it, the illuminated retinal region was seen to have a point of light (foveal reflex) situated near the centre. This point of light is the real image formed by the ingoing beam after reflexion in the foveal cup. The point is situated about 30 μ in front of the centre of the cup. Thus if the fovea is defined as the region of the cup the illuminated retinal image was seen to be situated near the centre of the fovea, and hence in these conditions D.M. fixated with her fovea. However, this is not to say that she did so in other conditions of illumination.

D.M. was a very favourable subject for this work because her rods appeared quite normal and such cones as she possessed had familiar properties and hence could easily be understood. Moreover she was most co-operative, with quick understanding, sharp observation and sustained attention. Nevertheless, in spite of long hours and repeated sessions much of the work is less complete and well confirmed than could be wished, but the period at our disposal was limited.

In this paper we investigate the increment threshold, and in the sequel the course of dark-adaptation in relation to the regeneration of rhodopsin.

METHODS

The optical arrangement of Fig. 1 was designed to superpose a small flashing light upon a background field and to present these to the subject by Maxwellian view, in such a manner that the points of entry of flash and field through the pupil could be adjusted to centre or periphery. The light source was a zirconium arc (not shown) whose crater was focused by an achromatic lens upon the stop S_1 of 3 mm diameter. The light could be attenuated by neutral filters F_c in the common path. Light entering through S_1 was divided by the beam-splitter B_1, and each beam could be modified by neutral wedges W_1, W_2 and neutral and narrow-band interference filters F_1, F_2. The beams were recombined at the beam-splitter B_2 and fell upon L the Maxwellian lens, which imaged S_1 upon the pupil at $\frac{1}{4}$ of the size. Beam 1 usually filled the lens and provided the background field; beam 2 was restricted by stop S_2 and subtended a 1° patch in the centre of the lens (see inset, Fig. 1). The rotating sector R continually interrupted the light so that the 1° patch was on for half a second and off for half a second. A small fixation light D was placed at the edge of the lens L when parafoveal increment thresholds were measured. The brightness was adjustable and the distance from the 1° patch was 7°.

It was sometimes required to send the flashing light through the centre of the dilated pupil (8 mm diam.) and the background field through the periphery, or vice versa. The separation of the two focal points of entry upon the pupil as indicated by the inset of Fig. 2 was adjusted by rotating B_2 in the horizontal plane. The shift across the pupil was achieved by a horizontal movement of L across the beam. Mechanical stops were fixed so that the lens could instantly be pushed from one position to the other to change between situations A and B (inset, Fig. 2). When the light is passing through the pupil, it is not easy to see the point of entry; that only becomes obvious when the light is caught upon the iris muscle.

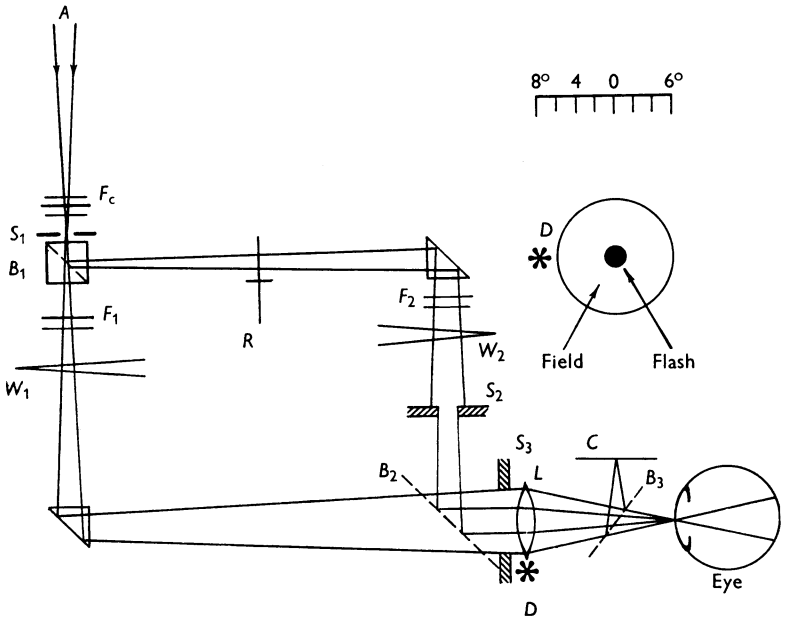

Fig. 1. Apparatus for measuring increment thresholds. Inset: Visual field. A, light from zirconium arc focused upon stop; B_1, B_2, B_3, beam-splitters; C, white card; D, fixation light; F_1, F_2, interference filters; F_c, neutral filter; R, sector disk, $\frac{1}{2}$ sec on, $\frac{1}{2}$ sec off; S_1, S_2, S_3, stops; W_1, W_2, wedges.

The beam-splitter B_3 (Fig. 1) was found very helpful, for by placing the white card C to coincide with the image of the pupil, the two points of entry upon the pupil also appeared as bright points upon the card. Then, looking through the beam-splitter, these points could easily be seen, and so could the whole reflected eye, if a weak light was shone upon the subject's face. In this way the two focal points could be seen clearly in relation to the pupil as in the insets A, B (Fig. 2) and the entry points properly adjusted. B_3 was left in position to check from time to time the possibility of small head movements which could impair the setting.

The subject bit upon a dental impression, her head was steadied by a forehead rest, and she fixated either the central flashing 1° field or else the red fixation light D, 7° away. The pupil was dilated with cyclopentolate HCl 1%.

Calibration of interference filters. The interference filters were all about 10 mμ band-pass at half maximum. They were treated as though they transmitted monochromatic light at peak wave-length. We needed to know the relative energies of the light incident upon the cornea. An E.M.I. red-sensitive photomultiplier cell was covered except for a 2 mm hole

drilled in a metal cap above the photocathode. The cell was substituted for the subject's eye so that the $\frac{3}{4}$ mm focal point of light entered the 2 mm hole and the light fell upon the cathode. An opaque screen was interposed at F_1 and the wedge W_2 (Fig. 1) was adjusted so that the output of the photocell was always the same whatever the interference filter F_2. The spectral sensitivity of the photocell was known and thus the relative quantal flux could be directly calculated. The calibration was made in beam 2 (Fig. 1) and all the spectral sensitivity measurements were made in this beam; thus the calibration gives directly the relative quantum flux at the cornea.

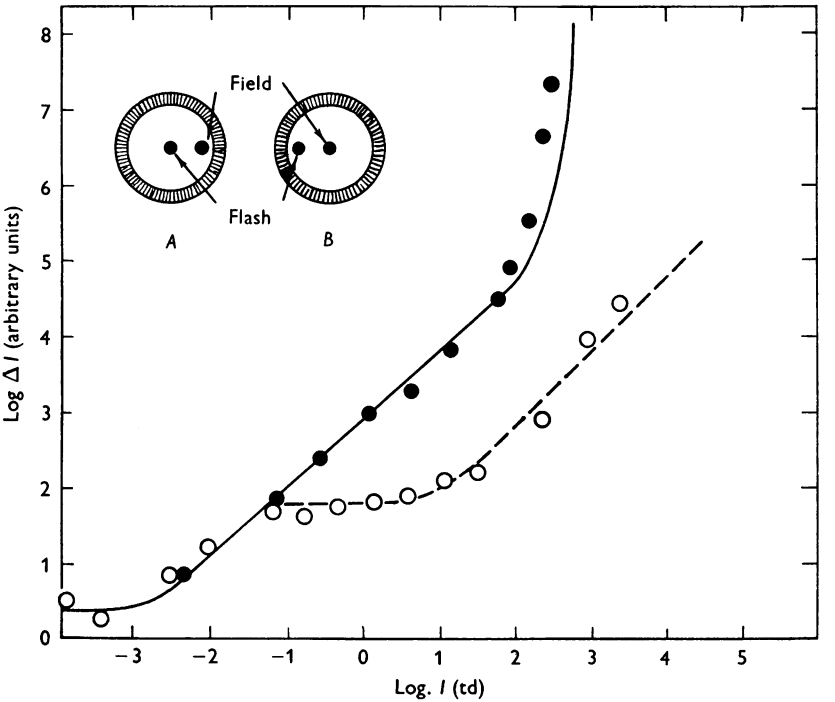

Fig. 2. Increment threshold 7° parafoveal. Ordinate, log. increment flash intensity (arbitrary units); abscissa, log. background field. ○, normal subject; ●, photanope; continuous curve, normal rod increment threshold curve (from Aguilar & Stiles, 1954). Inset: Points of entry of flash and field lights through dilated pupils in two cases *A* and *B*.

RESULTS

Parafovea

In Fig. 2 are plotted some increment thresholds with the adaptation field in log. trolands as abscissa and the superposed threshold flash as ordinate also upon a logarithmic scale. The white circles show the result upon a *normal* subject with our apparatus obtained with yellow light (580 mμ) for the flash, fixation 7° temporal to the test-flash and with both lights sent through the centre of the pupil. The curve is familiar from the work of Stiles (1939, 1949, 1953). It consists of two branches each of the

same characteristic shape and each corresponding to one visual mechanism. In shape each branch is a horizontal line which runs indefinitely from the left (the absolute threshold for the mechanism) and turns rather sharply to become a line of slope nearly 45° (the Weber–Fechner relation). The lower branch to the left corresponds to rod vision. At a field strength of -1 log. td the cone threshold is reached, and a second branch of the curve is obtained corresponding to one or more of the cone mechanisms, as Stiles has demonstrated very fully.

Now if the photanope has normal rods and no cones in the retina at a point 7° temporal to the fixation spot, we should expect the above experiment to give a normal lower branch to the curve but no upper branch. The black circles (Fig. 2) show that this expectation is exactly fulfilled, and the Weber–Fechner line continues to rise for 3 log. units above the point where it is normally hidden by the greater sensitivity of the cones. At a background field luminance of about 2 log. td, however, the rod curve begins to change, and in a sense contrary to that expected from the entry of a new mechanism. For instead of thresholds appearing below the Weber–Fechner line, at about 2 log. td they rise above it, and at 3 log. td of background illumination the brightest white flash we delivered remained undetected.

Now though in normal subjects at moderate illumination rod function is usually masked by cone activity, Aguilar & Stiles (1954) have succeeded in demonstrating it by an ingenious technique. The activity of cones was depressed by using as background field a red light introduced through the centre of the pupil, and the test flash excited the rods relatively well by being green and by entering through the periphery of the pupil. With this technique the threshold of the cones was so far raised that the flash only began to excite them at a background field of 3 log. td or more. It was therefore possible to plot the increment threshold curve for rods in normal subjects up to a background field of 3 log. td. Their results are shown as the unbroken curve in Fig. 2 and it is seen that our results upon the photanope agree with theirs upon the normal subject so well that the two curves may nearly be superimposed.

Test upon the fixation point

When the background field was 3 log. td of retinal illumination our rod monochromat could not see the brightest flashes when she fixated 7° away from the 1° flashing light. But when she looked directly at the flashes she could see them. Thus she clearly possesses at the fixation point a visual mechanism that does not become ineffective at a retinal illumination which incapacitates normal rods. Figure 3 shows an experiment, plotted as Fig. 2, which throws some light upon this mechanism.

At each background intensity the photanope made two increment threshold determinations (each repeated) one fixating upon the test flash and one upon the fixation light (*D*, Fig. 1) 7° temporal. Both field and flash entered the dilated pupil coincident and near the centre in both fixation positions. The flashing beam passed through a 580 mμ interference filter, the field through a 520 mμ filter together with a 0·68 neutral, which gave it the same scotopic brightness as the 580 mμ filter alone.

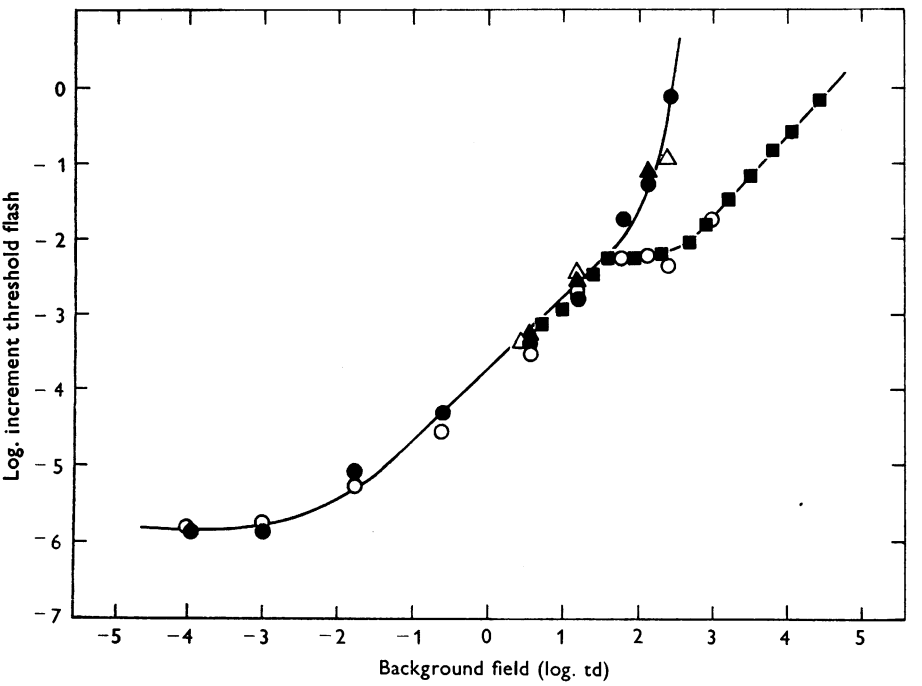

Fig. 3. Increment thresholds of photanope with flash and background of wave-lengths indicated below.

	Wave-length (mμ)	
	Flash	Background
○ fovea	580	510
● parafovea	580	510
△ fovea	510	580
▲ parafovea	510	580
■ fovea	White	White

The results of Fig. 3 were taken in the order from left to right. The first point had zero background field, so this measures the absolute threshold, but the subject had not remained in the dark long enough for it to represent the fully dark-adapted threshold. It is seen that for all backgrounds from zero to + 2 log. td the threshold is the same when fixation was central

(white circles) or peripheral (black circles), and the results correspond well enough to those of Fig. 2. But at backgrounds higher than 2 log. td there is a marked divergence in the curves. With peripheral fixation the threshold rapidly rises above the Weber–Fechner line and exhibits the features of normal rod saturation seen in Fig. 2. With central fixation, on the contrary, the threshold falls below the line and evidently indicates the appearance of a new visual mechanism. In order to investigate this mechanism more extensively the colour filters were removed from flash and field and a two-branched curve was obtained as indicated by the squares. The removal of filters, of course, causes a shift in both branches of the curve depending upon their spectral sensitivity. The two-branched curve with the squares has been plotted in Fig. 3, shifted (without rotation) so that the kink coincides with the kink of the white circles.

The spectral sensitivity of the new mechanism is not that of rhodopsin, as may be seen from the effect of interchanging the (580 mμ) and the (520 mμ + 0·68 neutral) filters which had been in the test flash and field respectively. Both for the normal eye and for our photanope these two filters had about the same scotopic brightness, so their interchange could have no effect upon pure rod function. At a field intensity of 0·5 log. td the interchanged thresholds (triangles for circles) are seen to be unaltered, confirming that this part of the curve records the activity of rhodopsin receptors. The next point to the right shows the same thing. At 2·2 log. td the black (parafoveal) circles and triangle still nearly coincide, signifying that this branch still records a rhodopsin receptor. But at 2·4 log. td the white (foveal) triangle lies above the white circle by about 1·4 log. units. Thus the new mechanism is much more sensitive to yellow (as compared with green) than is rhodopsin. This suggests that the second visual mechanism at the fixation point is due to cones. If so, they should exhibit the Stiles–Crawford retinal direction effect.

Stiles–Crawford effect

As is well known, Stiles & Crawford (1933) showed that light entering through the centre of the pupil is much more effective in stimulating cones than is light which enters through the periphery of the dilated pupil and falls upon those same cones. But this retinal direction effect applies to cones only, and hardly or not at all to rods (Stiles, 1939; Flamant & Stiles, 1948). Thus if the flash and background beams enter the pupil in different places, as in Fig. 2 (inset), it should not make any difference to the *rod* threshold whether light entered the pupil as at A or at B. But if cones are excited in a situation where the increment threshold lies upon the Weber–Fechner 45° line, a change from A to B should increase the log. increment threshold by twice the change expected

from moving the entry point of either flash or field alone, i.e. we might expect a total change of about 1 log. unit.

In the experiment of Fig. 3 a change of pupil entry from A to B caused an increase in threshold of 0·1 log. unit in the lower region of the curve, but it was otherwise with the second visual mechanism. With conditions corresponding to the last circle to the right (Fig. 3) the shift from A to B caused 0·5 log. unit of threshold change.

Alpern *et al.* (1960) have measured the Stiles–Crawford effect in some photanopes more thoroughly than we did. Using a flicker method they plotted the efficacy of the light as a function of its point of entry through the pupil and found a marked asymmetry, indicating tilting of the foveal cones. The magnitude of the Stiles–Crawford effect therefore appeared greater or less than normal, depending upon the particular point of entry round the edge of the pupil, and our low value may well have been due to the one peripheral point we used chancing to be at a low-valued region.

We conclude that the receptors responsible for the main curve of Fig. 3 are normal rods, since they have the spectral sensitivity of rhodopsin, the absence of Stiles–Crawford effect and they coincide with Aguilar & Stiles's (1954) curve for normal rods, becoming saturated at the normal value of 3 log. td. The receptors of the high-intensity branch, on the other hand, appear to be cones, for they are more yellow sensitive than is rhodopsin, and they exhibit the Stiles–Crawford effect. But they differ from normal cones in that they are only detectable on or near the fovea, their threshold is very high, and their acuity is poor.

Spectral sensitivity

From the results of Fig. 3 it was hardly to be doubted that the spectral sensitivity measured at absolute threshold would be that of rhodopsin, whether at 7° parafoveal or by direct fixation. We therefore contented ourselves with the rather irregular results of Fig. 4 (black circles and triangles) which roughly correspond to the rhodopsin sensitivity shown by the continuous curve (that plots the log. absorption of rhodopsin against wave number).

Of more interest is the spectral sensitivity of the upper branch of the curve in Fig. 3, namely the mechanism left when a bright background field has abolished the response of rods to a superimposed flash.

The intensity of the white background was raised until no increment flash was detectable at 7° parafoveal even with a strong white flash. Now the subject fixated directly and was able to see the flash even when greatly reduced. By interposing a filter transmitting either 492, 527, or 590 mμ the threshold energies for these lights could be found. The white circles of Fig. 4 show the average quantum energies obtained plotted as log.

sensitivity. The interrupted curve is the sensitivity of a visual pigment which fits Dartnall's nomogram (1952) and has its maximum at 550 mμ. This shift in spectral sensitivity corresponds closely to the change seen in Fig. 3 when the green and yellow filters in flash and background were reversed.

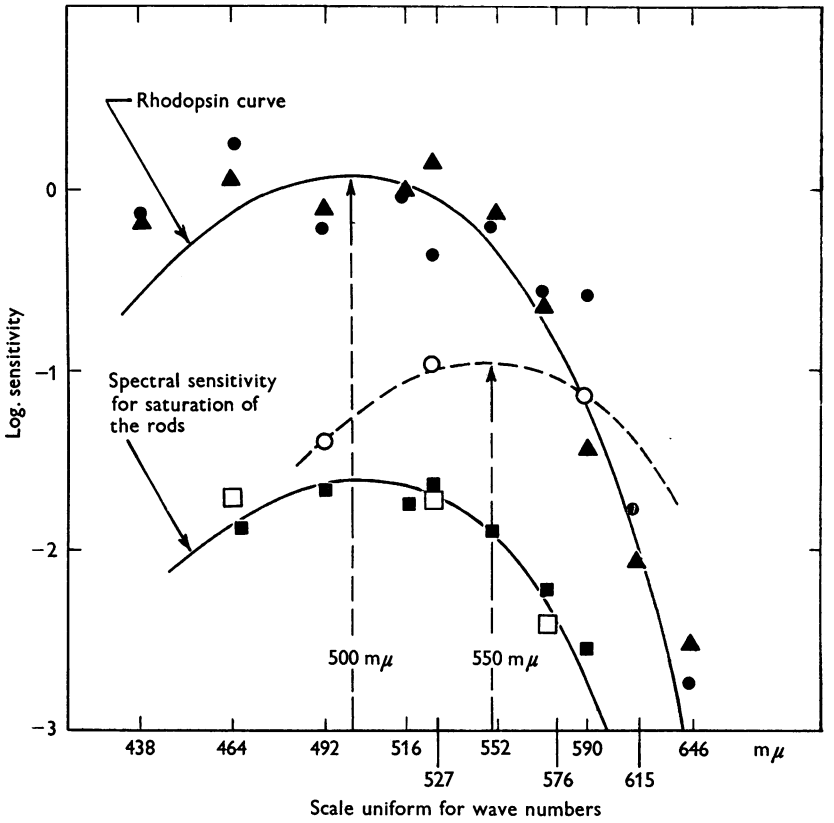

Fig. 4. Spectral sensitivity for various visual processes. Ordinate, log. sensitivity (arbitrary units); abscissa, wave-lengths plotted upon a scale uniform for wave frequency. Continuous curves, log. absorption of rhodopsin; interrupted curve, the rhodopsin curve displaced to give maximum at 550 mμ. ▲ parafoveal threshold; ● 'foveal' threshold; ○ foveal increment threshold upon a background which saturates rods.

Now in Fig. 3 it will be noticed that the level at which the rods lose their capacity to signal a superposed flash is just about the level where cones enter the picture, so those who hold that cones can inhibit rods may incline to the view that it is the entry of the cones which causes what we (following Aguilar & Stiles, 1954) have called 'rod saturation'. According to this view, inhibition of the rods will be caused by background lights of

various wave-lengths when the cones have been stimulated to a fixed extent. The spectral sensitivity of a background which just inhibits the rods should therefore correspond more or less to the white circles of Fig. 4, which represent the sensitivity of the cones actually present.

In the experiment to find the spectral sensitivity of the saturating background, the stop S_2 (Fig. 1) and the rotating shutter R were transferred to the other beam, so the subject by moving the wedge W_2 as usual could vary the background field. The test flash was white and was made very bright so that its exclusion would correspond to fairly complete saturation or inhibition of the rods. The black squares (Fig. 4) represent an attempt to investigate many points in the spectrum, the white squares give the results of another experiment where just three wave-lengths were chosen and re-checked several times.

The points are seen to lie close to the (continuous) curve of rhodopsin sensitivity displaced vertically downward, but they cannot be fitted by vertical displacement of the (interrupted) curve of cone sensitivity. Thus the abolition of rod increment function is due to quanta absorbed by rods and not by cones.

DISCUSSION

The photanope whom we studied appeared to have normal rod function, and her rod increment threshold curve on the parafovea coincided with that of Aguilar & Stiles (1954) in their special conditions which excluded increment thresholds of the cones. In particular, rod saturation was reached at the same value of about 1000 td.

When their paper appeared there seemed three kinds of explanation for the abolition of increment perception against this moderate background.

(*a*) Rhodopsin might be all bleached away at this illumination. This was rejected by Aguilar & Stiles in consideration of the small incidence of quanta per second per rod, and more recently the direct rhodopsin measurements of Campbell & Rushton (1955) have confirmed their conclusion.

(*b*) Rods might be inhibited by cones, so that though rods are still active in responding to light they do not send a signal down the optic nerve fibres. This is a plausible concept, for the electrophysiological records from ganglion cells of animals usually show both rods and cones converging upon the same optic nerve fibre, the rod signal preponderating in dim vision, the cone signal in bright (Granit, 1947). It is reasonable to think that the rods have sole use of the common communication line below the cone threshold, but that some mechanism is needed to turn off the rods at high illumination, otherwise all the channels might be jammed full of high frequency rod discharges and be insensitive to the near-threshold cone response which in fact is the signal transmitted.

The results of this paper, however, are very difficult to reconcile with inhibition of rods by cones, for our photanope, though apparently quite devoid of active cones in the parafoveal region, nevertheless, exhibited the 'inhibition' of rods there precisely as does the normal subject, and at the same background of 1000 td. The evidence is even stronger; for the spectral sensitivity curve Fig. 4 (squares) shows that it is rods not cones upon the background field that catch the quanta which abolish increment threshold.

(*c*) There are two ways in which rods might become 'saturated'. (i) The signal generated by each rod in response to light will increase with illumination but only to some fixed upper limit, and when this is reached an increment flash can do nothing further. Upon this view, saturation is a property of the light transducer mechanism of the rod. (ii) Rods may in fact be inhibited not by cones but by other rods. In electrophysiological studies of ganglion discharges from the cat's retina Donner & Willmer (1950) confined themselves to ganglions whose spectral sensitivity was throughout that of rhodopsin. The somewhat bewildering diversity of on/off patterns of discharge which they recorded from various receptors at different intensities makes their conclusion readily acceptable that some interaction between rod and rod may be exhibited in the time course of discharge. A clear case of spatial interaction which varied with the level of dark-adaptation was studied by Barlow, FitzHugh & Kuffler (1957*a*, *b*), in the cat. In their situation both rods and cones generally interacted but there was no evidence of inhibition of rods by cones; rather the rod–cone complex of the centre of a ganglion's receptive field was inhibited at high illumination by the similar rod–cone complex of the periphery.

The electrophysiology of vision is embarrassing in urging us to explain, by nerve discharges of the utmost complexity and variety, visual phenomena which had appeared both uniform and simple. Here we go no deeper than to point out the alternative mechanisms of 'rod saturation'. However, in view of the results of the following paper the first alternative of (*c*) seems the more likely.

CONCLUSIONS

1. In our photanope the rods and rod vision appeared normal, judged from all the experiments we have made as described in this and the following paper.

2. At a background illumination of 1000 trolands the rod increment threshold was abolished in the photanope as it is in the normal subject, and the abolition is not caused by cones. This in fact is the level of illumination at which our subject lost the sense of visual contrast in objects around her and became virtually blind.

3. Cone vision was restricted to the neighbourhood of the fovea. The spectral sensitivity was roughly normal (maximum 550 mμ) and there was a Stiles–Crawford effect. But the cone organization was not normal, since threshold was extremely high, colour vision practically absent and acuity very poor.

SUMMARY

1. These investigations were all made upon one 'incomplete rod-monochromat' (with some comparison upon normal subjects).

2. Increment thresholds measured upon the fovea showed a normal rod branch and also a cone branch with spectral sensitivity maximal at about 550 mμ but with threshold some 2 log. units above that of normal cones.

3. At 7° away from the fovea only rods were active, and the increment threshold relation coincided with the normal rod relation as measured in full extent by Aguilar & Stiles (1954).

4. At a background of about 1000 trolands normal rods become saturated and so do the monochromat's rods, and this is the level where she lost all luminous contrast from her surroundings.

5. The spectral sensitivity of the background which just saturates is that of rhodopsin. Thus the abolition of rod function in monochromat and normal subject by a bright background is not due to inhibition of rods by cones. For it occurs in normal fashion where there are no cones, and rhodopsin is the pigment which catches the quanta.

REFERENCES

AGUILAR, M. & STILES, W. S. (1954). Saturation of the rod mechanism of the retina at high levels of stimulation. *Optica acta*, **1**, 59–65.

ALPERN, M., FALLS, H. F. & LEE, G. B. (1960). *Amer. J. Ophthal.* (In the press.)

BARLOW, H. B., FITZHUGH, R. & KUFFLER, S. W. (1957*a*). Dark-adaptation, absolute threshold and Purkinje shift in single units of the cat's retina. *J. Physiol.* **137**, 327–337.

BARLOW, H. B., FITZHUGH, R. & KUFFLER, S. W. (1957*b*). Change or organization in the receptive fields of the cat's retina during dark adaptation. *J. Physiol.* **137**, 338–354.

BLACKWELL, H. & BLACKWELL, O. M. (1959). Identification of rod and cone receptor mechanisms in atypical congenital achromatopsia. *Arch. Soc. Amer. oftal. optom.* **2**, 73–94.

CAMPBELL, F. W. & RUSHTON, W. A. H. (1955). Measurement of the scotopic pigment in the living human eye. *J. Physiol.* **130**, 131–147.

DARTNALL, H. J. A. (1952). Visual pigment 467, a photosensitive pigment present in tench retinae. *J. Physiol.* **116**, 257–289.

DONNER, K. O. & RUSHTON, W. A. H. (1959). Rod-cone interaction in the frog's retina analysed by the Stiles–Crawford effect and by dark adaptation. *J. Physiol.* **149**, 303–317.

DONNER, K. O. & WILLMER, E. N. (1950). An analysis of the response from single visual-purple dependent elements in the retina of the cat. *J. Physiol.* **111**, 160–173.

FLAMANT, F. & STILES, W. S. (1948). The directional and spectral sensitivities of the retinal rods to adapting fields of different wave lengths. *J. Physiol.* **107**, 187–202.

GRANIT, R. (1947). *Sensory Mechanisms of the Retina.* Oxford University Press.

HECHT, S. (1937). Rods, cones and the chemical basis of vision. *Physiol. Rev.* **17**, 239–290.

HECHT, S., SHLAER, S., SMITH, E. L., HAIG, C. & PESKIN, J. C. (1948). The visual functions of the complete colour blind. *J. gen. Physiol.* **31**, 459–472.

LEWIS, S. D. & MANDELBAUM, J. (1943). Achromatopsia: report of three cases. *Arch. Ophthal., N.Y.*, **30**, 225–231.

RUSHTON, W. A. H. (1961). Dark-adaptation and the regeneration of rhodopsin. *J. Physiol.* **156**, 166–178.

SLOAN, L. L. (1954). Congenital achromatopsia: A report of 19 cases. *J. opt. Soc. Amer.* **44**, 117–128.

SLOAN, L. L. (1958). The photopic retinal receptors of the typical achromat. *Amer. J. Ophthal.* **46**, 81–86.

STILES, W. S. (1939). The directional sensitivity of the retina and the spectral sensitivities of the rods and cones. *Proc. Roy. Soc.* B, **127**, 64–105.

STILES, W. S. (1949). Increment thresholds and the mechanisms of colour vision. *Docum. ophthal.* **3**, 138–163.

STILES, W. S. (1953). Further studies of visual mechanisms by the two-colour threshold technique. In *Coloquio sobre problemas opticas de la vision*, pp. 65–103. Union internationale de physique pure et appliquée. Madrid.

STILES, W. S. & CRAWFORD, B. H. (1933). The luminous efficiency of rays entering the eye pupil at different points. *Proc. Roy. Soc.* B, **112**, 428–450.

WALLS, G. L. & HEATH, G. G. (1954). Typical total color blindness reinterpreted. *Acta Ophthal.* **32**, 253–297.