EXPERIENTIA

Vol. VI - Fasc. 1 Pag. 1-40 15. I. 1950

The Chromatic Aberration of the Human Eye and its Physiological Correction

By H. HARTRIDGE[1], London

(1) *The chromatic aberration of the human eye*

A problem which has been a subject for discussion for many years concerns the chromatic aberration of the human eye. When a beam of white light passes into a glass lens, the violet rays are the first to come to a focus owing to the greater refrangibility of rays of short wavelength in comparison with those of long wavelength. Then follow in the order of the spectrum the foci of the blue, the bluegreen, the green, the yellowgreen, the yellow, the orange, and the red rays. In consequence of this arrangement, if a screen be placed at the focus of the yellow rays, all the other rays will form blurs which will be of different sizes according to the positions of their focussing points.

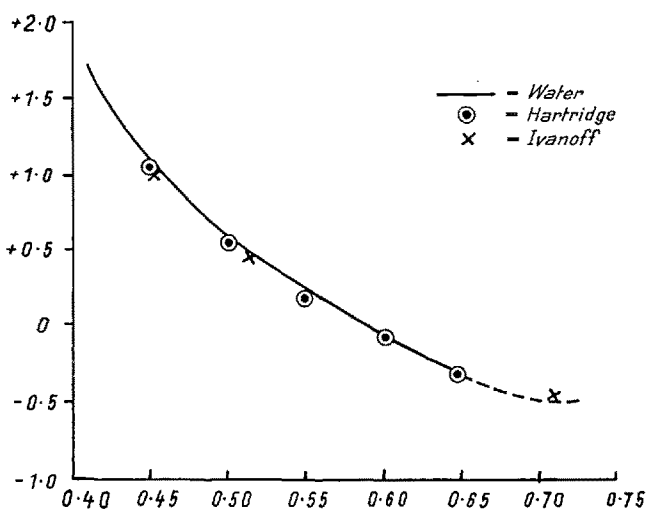


Fig. 1. – The chromatic aberration of the human eye. Aberration measured in dioptres, vertical. Wavelengths, horizontal.

The image formed on the screen will be found under these circumstances to consist of a bright yellow dot which is surrounded by a blueviolet halo, the former being produced not only by the yellow rays but also by the orange and the yellowgreen rays; while the latter is produced by the red rays together with the bluegreen, the blue and the violet rays. This arrangement of the colours can be seen easily if the image produced by a simple convex glass lens is examined by means of a low-power microscope.

Since the lens system of the eye is not found to include any colour correcting elements, such as those found in camera lenses, or in the object glasses of telescopes or microscopes, it must from images similar to those produced by an uncorrected glass lens, and instead of a single focus there must be a number of different foci, the relative positions of which will depend on the refrangibility of rays of different wavelength. The focal lengths of the eye for the different coloured rays have been determined recently by IVANOFF[1] and by the writer[2], and there is good agreement between them, so that it may be stated with confidence that not only does the eye suffer from chromatic aberration but also that its amount is accurately known, see Fig. 1. In fact it is nearly equal to that of a light flint glass lens and thus is somewhat greater than that produced by one made of crown glass.

Experiments performed by the writer in 1918 showed that of the different coloured rays, the yellow and the yellowgreen rays are those which are usually sharply focused on the retina when white light is being used to illuminate the objects under examination. Under these circumstances, as explained above, the image of a point of white light will consist of a bright yellow dot which is surrounded by a blueviolet halo. If on the contrary the object looked at be a black dot on a bright background then the retinal image will consist of a blueviolet dot surrounded by a yellow halo. In the case of a grating test object consisting of parallel black and white bars of approximately equal widths, the retinal image will consist of blueviolet and yellow bars, the former taking the place of the black bars and the latter taking the place of the white ones. Now when an observer who has emmetropic vision looks at these test objects he does not observe the colours produced by chromatic aberration. Thus a bright point of white light looks white to him, he does

[1] Director, Vision Research Unit, Institute of Ophtalmology (University of London), Medical Research Council, London.

[1] A. IVANOFF, Rev. d'Opt. *26*, 145 (1947).

[2] H. HARTRIDGE, Phil. Trans. B *232*, 592 (1947).

not observe any yellowness, nor does he notice the blueviolet halo which surrounds the yellow central spot. With a black dot on a bright background, or with a grating test object consisting of black and white bars it is the same, that is to say the colours due to chromatic aberration are not detected. So complete is the process of colour elimination that certain workers have supposed that the eye must be corrected in some way for this aberration, thus bringing rays of all wavelengths to the same focus. There are however two other puzzling features to be mentioned. Attempts by HELMHOLTZ to improve the acuity of the eye by placing in front of it a combination of crown and flint glass lenses, which would have the effect of achromatizing the lens system of the eye, failed to produce any noticeable benefit, and the replacement of white light by monochromatic light by LUCKIESH, by ROAF, by SHLAER and his coworkers, and by the writer has also been found to effect an improvement in acuity which is so small that it may well be within the experimental error of the method used. All these facts lend support to the idea that the lens system of the eye is corrected in some way for chromatic aberration. But against this idea can be arrayed the following facts: (*a*) When by means of a colour filter of cobalt blue glass, or in some other way, the rays illuminating a test object are limited to those of long and those of short wavelength, all those of medium wavelength being eliminated, it is found either that the long wavelength rays, the red and orange rays, are sharply focused and that the short wavelength rays form out of focus blurs; or that the short wavelength rays, the blue and violet rays, are sharply in focus, and that the red and orange rays form out of focus blurs; or that neither long nor short wavelength rays are in focus, and both form blurs. (*b*) When half or more than half of the pupil is covered over by means of an opaque screen, then the images of point or line test objects are found to consist of spectra which are produced by the refraction of the light rays as they traverse the uncovered parts of the pupil. (*c*) The good agreement between the measurements of refraction made by different observers, mentioned above. Would there be such good agreement between these values, and would the cobalt blue glass experiment, and the partial occlusion of the pupil, demonstrate so clearly that chromatic aberration is present unless it really is there? But if it is present, how can we explain the absence of its effects from normal vision, and how does it come about that the correction of the aberration by means of a Helmholtz lens, or its elimination by means of monochromatic light, produce such negligible improvements in acuity? On the one hand the evidence that the aberration is present is conclusive; on the other hand the evidence that it is absent appears to be equally conclusive. That is the paradox which must now be considered.

(2) *The elimination of the coloured fringes*

It was found by the writer that several factors are involved in the elimination of the colours of the fringes which are produced by chromatic aberration: (*a*) the yellow fringe is diluted by a considerable amount of white light; (*b*) the blueviolet fringe is of very low luminosity; (*c*) at small pupil diameters the colours of the chromatic fringes are partially neutralized by those produced by diffraction; (*d*) at large pupil diameters the sizes of the fringes are reduced by the STILES-CRAWFORD retinal direction effect. Thus the major problem concerns the elimination of the coloured fringes when a pupil of medium diameter, for example 2 to 4 mm, is in use. It was noticed by the writer that yellow and white test objects, when placed on a black background, become indistinguishable when the angle which they subtend at the eye is sufficiently reduced. It was also noticed that blueviolet and black test objects, when placed on a white background, also become indistinguishable with a suitable reduction of visual angle. When the intensity of illumination of these test objects was varied it was found that a low intensity tends to favour the resemblance between yellow and white objects and between blueviolet and black ones, and vice versa that bright illumination tends to increase the differences between them. Many years later these experiments were repeated, and extended, in order to ascertain if these colour changes play any essential part in the reduction, or the elimination, of the fringes and the halos which are produced by chromatic aberration. When yellow and white, with reduction of visual angle, resemble one another, one of three things may be happening: (*a*) white may become yellow; (*b*) yellow may become white; (*c*) both yellow and white may alter to an intermediate yellowish white. Of these possibilities it was found that (*b*) is the correct one, because, whereas white undergoes no apparent alteration, yellow is replaced by white, or to be more exact by very pale grey. There are three similar possibilities in the case of blue, but in this case also it is found to be blue, which changes, and black which does not. Thus yellow is replaced by pale grey, blue is replaced by dark grey. It was found further that these colour losses are much more likely to take place when yellow and blue are in close proximity than when they are widely separated. So potent is the effect of close proximity that a grating test object consisting entirely of alternate yellow and blue stripes appears to be replaced by one consisting solely of white and black ones, when the visual angle which the test object subtends at the eye is sufficiently reduced. Similar tests were now performed with a yellow dot surrounded by a blueviolet halo, and with a blueviolet dot surrounded by a yellow halo. In every case it was found that the colours disappeared when the visual angle was sufficiently reduced. Here then is a physiological process by means of which colours are eliminated, and

in particular the yellow and the blueviolet which are the colours of the fringes normally produced by chromatic aberration. Not only is this process capable of eliminating the usual colours of the fringes but also the colours of actual yellow and blue test objects at which the observer is looking, provided that the latter resemble the fringes in being in close proximity and in being approximately the same sizes as the fringes.

Additional tests were now performed in order to ascertain whether the sizes of the fringes produced by chromatic aberration are correct in order that their complete elimination can occur, and it was found that such is the case, because the eliminating process is normally found to be capable of dealing not only with the aberrations of a normal human eye, but of an externally placed glass lens as well. It is for this reason that spectacle lenses, and simple hand magnifying lenses, do not usually require achromatization.

With these observations in mind it is not difficult to understand why the effects of chromatic aberration do not show themselves, when the eye is being used for everyday vision. Take for example the case of a bright point on a black background. When as is normally the case yellow or yellowgreen rays are in the sharpest focus on the retina, the retinal image of the bright point consists, as we have said above, of a yellow dot surrounded by a blueviolet halo. Now the correcting process comes into play and changes the yellow to pale grey or white and alters the blueviolet to dark grey or black, thus presenting to consciousness an impression which is completely colour-free and which resembles closely the object originally looked at. If a small black dot on a white background be considered similarly the image on the retina will be a blueviolet dot with a surrounding yellow halo. But the correcting process alters blueviolet to dark grey or black, and alters yellow to pale grey or white, and this merges with the white background, thus largely replacing the original appearance of the object. In the case of a grating test object, consisting of alternate black and white bars, owing to chromatic aberration these are changed to blueviolet and yellow bars, but the correcting process changes them back again to black and white. Lastly let us take the case of the sharp line at the junction between a large white field and a large black one; both calculation and experiment show that the line of junction is occupied by two narrow parallel fringes, one yellow and one blueviolet; the former merging with the white field and the latter merging with the black one. But after correction has occurred the yellow is replaced by pale grey or white which becomes part of the white field, and the blueviolet is replaced by dark grey or black which becomes part of the black field, so that the observer appears to see what he was originally looking at.

We now see why it is that the correction of chromatic aberration by means of a Helmholtz lens, or by the use of homogeneous monochromatic light, produces so little improvement in visual acuity. It is because this aberration has been corrected already in the visual mechanism itself. Thus the paradox of the lens system being uncorrected in the optical sense, but of behaving as if it were corrected in the physiological sense, is now solved, but it clearly leaves a further problem to be investigated, namely to account in a satisfactory manner for the colour changes which have just been described.

(3) *The three-colour theory and the inflections in the luminosity curve*

At first it seemed possible that an explanation of these colour changes would be obtained on the basis of the three-colour theory in the following way: suppose that two alterations take place in the nervous pathways between the retina and the brain when the

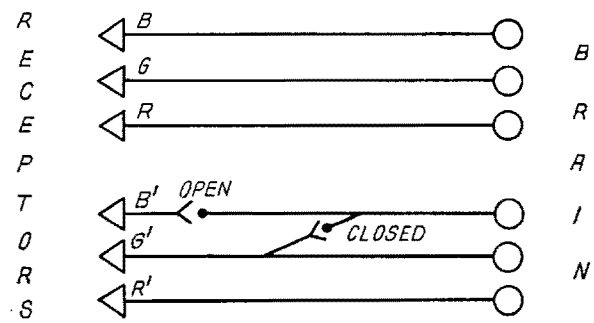


Fig. 2. – Arrangement for normal colour vision.
B Nerve path for blue perception
G Nerve path for green perception
R Nerve path for red perception

Arrangement for reduced colour vision.
B′ Nerve path for blue perception severed
G′ Nerve path for green perception also joined to brain centre for blue
R′ Nerve path for red perception

visual angle subtended by a test object is reduced; (*a*) a severing of the path which connects the blue receptors with the brain centre for the perception of blue and (*b*) the joining of the path which connects the green receptors with the brain centre for the perception of green, also with the brain centre for the perception of blue. See Fig. 2. Now the essential difference between blueviolet and dark grey is that the former reflects much more blue and violet light than does the latter. If then the blue receptors are put out of action either by severing their nervous connection, or in some other way, the essential difference between blueviolet and grey will disappear and the former will have the same appearance as the latter. In this manner the change undergone by blueviolet may be accounted for satisfactorily. Now taking the case of yellow the essential difference between this colour, and pale grey is that the latter reflects blue and violet rays strongly whereas the former does not do so, but does reflect

strongly the green, the yellow, the orange and the red rays. Thus on the basis of YOUNG'S theory yellow stimulates both the green and the red receptors and therefore produces activity in both the corresponding brain centres. But owing to the use of the cross link referred to above the nerve impulses also travel from the green receptors to the brain centre for the perception of blue, so that all three centres are set into activity just as they would be either by white or by pale grey. In this manner the resemblance between yellow and either white or pale grey, which is produced by a reduction of visual angle, is satisfactorily accounted for. We see then that two additional hypotheses enable an explanation to be offered on the basis of the three colour theory for the colour changes which the writer found to occur, namely, (*a*) the cutting of the nerve path for the perception of blue, and (*b*) the use of a cross link between the green receptors and the brain centre for the perception of blue. But there are other changes besides those of colour, and these cause supporters of this theory much more difficulty.

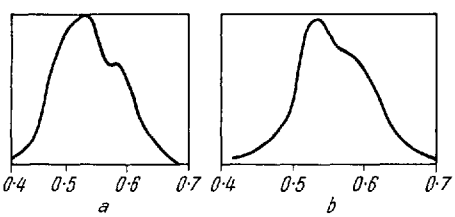


Fig. 3.
a WRIGHT'S notch. *b* SLOAN'S notch.
Sensitivity in arbitrary units, vertical. Wavelengths, horizontal.

WRIGHT[1] found that a reduction of visual angle causes an inflection to develop in the luminosity curve in the yellow region of the spectrum (see Fig. 3*a*). This the writer has called "WRIGHT'S notch". This resembles closely the inflection in the luminosity curve in the yellow which SLOAN[2] found to be produced by a reduction of light intensity, which the writer has called "SLOAN'S notch" (See Fig. 3*b*).

It is well known that difficulties arise when comparing the shape of one luminosity curve with that of another because it is usually found that alternative explanations of the differences in shape are possible. In consequence one has to be guided by the circumstances of the case. Thus in the case under consideration the alternative views are (*a*) that proposed by FORBES and (*b*) that proposed by the writer. According to FORBES the inflection in the yellow part of the spectrum is due to an increase in the relative luminosity in the orange-red of the spectrum at about 0·62 μ, brought about by the red receptor process which in his opinion maintains its sensitivity better, as light intensity is reduced, than either the green or the blue receptors do. According to the writer, on the other hand, the inflection is due to a relative decrease in sensitivity of yellow receptors which operate more strongly than the red and the green receptors when light intensity is high, but which operate less strongly than the red and the green receptors when light intensity is reduced. Now there is one important fact to be kept in mind, namely that white maintains its whiteness as light intensity is reduced, and therefore the inflection found by SLOAN at the long wavelength end of the spectrum must have a counterpart at the short wavelength end. Another point is that these inflections would be expected to be accompanied by changes in hue, that is if it be true, as most physiologists suppose, that hue and luminosity are mediated by the same receptors. On the basis of FORBES' suggestion, namely that the inflection in the long wavelength end of the spectrum is due to a relative increase in sensitivity in the red, it would be expected that there would be a corresponding relative increase in sensitivity in the bluegreen because otherwise white cannot remain unchanged but must alter to pink. It would also be expected that both red and bluegreen would show some colour changes. But none of these anticipations is found as the result of experiment. Now, on the basis of the writer's suggestion, namely that SLOAN'S inflection in the luminosity curve is due to reduced sensitivity in the yellow, it would be expected that there would be a corresponding reduction in the blue in order that white may remain unchanged. It would also be anticipated that there would be changes in colour in both yellow and blue. Now all these anticipations are in agreement with experiment because a notch has been found in the blue corresponding to the one in the yellow on reducing the light intensity, and the colour changes of yellow to pale grey, and of blueviolet to dark grey, have already been noted above.

As already stated with reduction of the visual angle which the test object subtends at the eye an inflection was found by WRIGHT in the luminosity curve in the long wavelength end of the spectrum. This, as in the case of SLOAN'S inflection, might be due either to enhanced sensitivity in the red or to reduced sensitivity in the yellow. If the former were true there should be a corresponding increase in the bluegreen, but if the latter were true there should be a corresponding decrease in the blue. Moreover, there should be changes of colour both of the yellow and of the blue. THOMSON has found recently the notch in the blue, and the writer has found both these changes of colour. Moreover, these have been confirmed by WILLMER and WRIGHT[1], who found neutral points both in the yellow, at 0·578 μ, and in the blue, at 0·41 μ, with fields subtending 15 minutes of arc at the observer's eye.

There is one way in which the notches in the yellow and in the blue could be accounted for satisfactory,

[1] W. D. WRIGHT, The Refractionist *31*, 53 (1942).
[2] L. L. SLOAN, Psych. Mon. *38*, 1 (1928).

[1] E. N. WILLMER and W. D. WRIGHT, Nature *156*, 119 (1945).

namely by supposing that in addition to colour receptors of other types there are some which have the specific property of responding to yellow, and others which have the corresponding property of responding to blue. When the visual angle is large all types respond but when it is sufficiently reduced the yellow and the blue ones become inactive, thus causing a reduction of light intensity which shows itself by notches in the luminosity curve in the yellow and in the blue, and at the same time causing a loss of colour in both these regions of the spectrum. There is, however, another possibility which must now be examined, namely that both notches may be caused by the absorption of light by pigments, the yellow by hæmoglobin for example and the blue by xanthophyll or by some other yellow pigment. These possibilities will be examined in the next section.

(4) *Macular pigmentation*

Hæmoglobin, which occurs normally in the bloodstream in two forms namely oxidized and reduced, exhibits two sharply defined absorption bands between the D and E Fraunhofer lines when in the oxidized state, and a single broad ill defined band when in the reduced condition. The oxyhæmoglobin bands have maximæ of absorption at 0·577 μ for the alpha band, and at 0·540 μ for the beta band. In addition to these two bands there is the beginning of a gamma band in the bluegreen and this extends into the ultraviolet. The reduced hæmoglobin band has a maximum of absorption at 0·55 μ, and there is a second band in the ultraviolet. If light on its way through the eye to the retinal receptors encountered a layer which contained hæmoglobin then this pigment will absorb some of the incident light and the absorption would be greatest in that part of the spectrum which correspond in wavelength to the maxima of the absorption bands of the hæmoglobin. If then part of the retina contained hæmoglobin, and part did not, the luminosity curve of the one part might be expected to differ from that of the other part at those wavelengths at which the absorption bands of hæmoglobin occur. Is it possible that the notch in the luminosity curve in the yellow part of the spectrum is due to absorption on the part of hæmoglobin? It seems unlikely that such is the case for four reasons: (*a*) the whole of the central area of the fovea of man is devoid of superficial blood vessels, that is of branches of the central artery and vein of the retina so that this central area appears to depend for its nutrition solely on the choroidal blood plexus which lies behind the pigment cell layer, and in consequence is not traversed by light which is passing through the retina on its way to the receptors. (*b*) If a test object of large size produced an image on the retina which lay partly on retina with superficial blood vessels and partly on retina without such vessels, whereas a smaller test object produced an image which lay entirely on retina without superficial vessels, it would seem likely that the luminosity curve of the larger field would show notches whereas the smaller one would not. In fact, as we have seen above the exact opposite is the case. (*c*) It was shown by the writer that the colour changes, and it was found by WRIGHT that the notch in the yellow, are not limited to the central part of the fovea only. According to the writer both the colour and the luminosity changes can occur anywhere, in the retina depending solely on such factors as light intensity and size of field. When these together exceed a certain critical value there is full colour vision and a smooth luminosity curve (the precise shape of which depends on the relative contributions of the receptors present in the part of the retina which is receiving stimulation). When, on the contrary the critical value is not reached, there is reduced colour vision in which both yellow and blue appear to be replaced by neutral tints, and notches are found in the luminosity curve both in the yellow and in the blue. (*d*) Neither oxyhæmoglobin, reduced hæmoglobin, nor a mixture of the two would produce a notch in the yellow of the same shape as those found in the luminosity curve.

A similar enquiry must now be made with regard to the notch in the blue part of the spectrum, and in this case it is the yellow foveal pigment which comes up for special consideration. Reports concerning this pigment are conflicting. According to GULLSTRAND the pigment is not present in the retina during life but only develops as the result of a *post mortem* reaction. Its presence is not observed in the normal human retina when it is examined by means of the ophthalmoscope using ordinary white light for purposes of illumination. But it is claimed to be visible when red free-light is used. According to POLYAK the pigment is seen in histological sections in the fourth and succeeding layers of the retina. In this case it would be almost completely absent from the foveal centre, but would rapidly increase in amount as the retina thickens on passing from the foveal centre towards the outer rim of the fovea. WALD[1] extracted a yellow pigment from a number of human foveas, but unfortunately he does not give any details concerning them. Thus we do not know whether they were obtained from eyes within a few minutes of their removal from the orbit, nor whether the retinas were normal and were free from pathological change. As the result of examining the extracted pigment he identified it as xanthophyll. But there seems to be some doubt as to the exact light transmission by this pigment at different wavelengths. In spite of these uncertainties as to the nature of the foveal pigment it seems to be possible to find a definite answer to the question "Can the notch in the luminosity curve, which develops when visual angle is made

[1] G. WALD, J. Gen. Physiol. *31*, 377 (1948).

small or when light intensity is reduced, be due to foveal pigment?". It seems most unlikely, if not impossible, for this to be the case, for if POLYAK is correct as to the distribution of this pigment, then since it is absent from the foveal centre a small test object would be less likely to exhibit a notch in the blue than a larger one. But as we have seen the exact opposite is the case.

Both in the case of yellow and in the case of blue it seems most unlikely that the notches which are found in the luminosity curve would be caused by pigments, through which the light has to pass on its way to the receptors. But even if the notches were produced in that manner the latter would not cause the alterations in colour which both yellow and blue are found to undergo. We must therefore conclude, in the absence of some other acceptable explanation, that both the notches and the colour changes are due to an alteration in receptor activity, that is that yellow and blue receptors undergo modification whereas, for example, red ones and green ones do not.

(5) *Alterations in receptor activity*

The mechanism of the colour changes proposed at the end of the previous section is at variance with the tenets of the three-colour theory, and is based on the supposition that the inflections in the luminosity curve found in the yellow and in the blue are depressions of activity, that is are notches or troughs. In order to make certain on this point, the alternative view should be reconsidered, namely that they are enhancements of activity as suggested by FORBES in the case of SLOAN's inflection. In order to account for the yellow inflection we must suppose that the red receptors maintain their sensitivity with reduction of visual angle or of light intensity better than do the green or the blue ones. In order to account for the inflection in the blue we must suppose similarly that the blue receptors maintain their sensitivity better than do the red ones or the green ones. But it will be remembered, in order to account for the replacement of blue by dark grey or black, that it was postulated, on the basis of the three-colour theory, that the nervous pathway is severed which connects the receptors for blue rays and the brain centre by which blue is perceived. Now on the contrary in order to account for the inflection in the luminosity curve in the blue it is necessary to suppose that the blue receptors maintain their sensitivity better than the green ones or the red ones. How could they do this if their nerve path had been cut? Either the blue receptors maintain their sensitivity, or they do not, and they cannot do both at the same time. Thus supporters of the three-colour theory find themselves faced by a dilemma because they are able to account either for the colour change in the blue, or for the luminosity inflection in the blue, but not for both at the same time.

(6) *The evidence for independent yellow receptors*

It was shown in the previous section that the alterations in colour vision, which occur at small visual angles and at reduced light intensities, can only be accounted for it there are independent yellow receptors. In this section other evidence will be summarized for the existence of this type of receptor. If this type of receptor did not exist then a monochromatic yellow light would stimulate red receptors and green receptors only, and so would resemble closely the stimulation produced by a mixture of red rays and green rays which have been mutually adjusted to match the yellow. Vice versa, if in a number of respects a yellow and a redgreen mixture differ from one another then these differences provide evidence for independent yellow receptors. When, employing a perimeter, ABNEY measured the sizes of the fields for rays of different colour, he found that a pure yellow had one of the largest fields, whereas a redgreen mixture had one of the smallest.

When STILES and CRAWFORD[1] determined the retinal direction effect for rays of different colour, they found that the magnitude of the effect for yellow was quite different to those either of red or of green.

When the various colours of the spectrum are matched by means of three selected spectral colours, for example, red of 0·65 μ, green of 0·53 μ, and blue of 0·46 μ, it is found that pure spectral yellow cannot be matched by persons with normal colour vision by a mixture of red and green. This is because the pure yellow stimulates the yellow receptors strongly, and the red and the green receptors less strongly, whereas the opposite is the case with the redgreen mixture. There is an alternative explanation of these facts which is based on the three-colour theory, namely that the receptors have broad responses with very considerable overlap. As the writer has pointed out elsewhere there is no confirmatory evidence of this hypothesis and moreover it leads to the erroneous conclusion that the partially colour-blind protanomalous trichromats have more selective receptors which do not overlap nearly as much as those present in the normal-sighted subject.

The writer, employing an optical device called a microstimulation apparatus for producing tiny rays of monochromatic light, found two pieces of evidence in favour of the existence of independent yellow receptors: (*a*) There was a definite fixation point for this colour intermediate in retinal position between that for orange and that for green. (*b*) There was a specific sensation produced by rays of about 0·58 μ,

[1] W. S. STILES and B. H. CRAWFORD, Proc. Roy. Soc. B. *112*, 422 (1933).

which differed from those produced by orange rays or by green rays.

EDRIDGE-GREEN and MARSHALL, employing monochromatic sodium light for producing adaptation of the eye, then looked at the spectrum of a source of white light. They noticed that red is now crimson, that orange is pale pink, that yellow is grey or even purplish grey, that yellowgreen is pale bluegreen, that green is bluegreen and that blue and violet are dark grey or even black. EDRIDGE-GREEN and MARSHALL concluded that this is evidence for an independent yellow receptor, which is selectively reduced in sensitivity when the retina has been adapted by means of sodium light. BURCH repeated these experiments and was unable to confirm this conclusion. He did, however, decide, as the result of adaptation to other monochromatic radiations, that there are four types of receptor, red, green, blue and violet, a conclusion to which HOLMGREEN had arrived previously as a result of stimulating the human retina by means of narrow beams of yellow and of blue light. The writer repeated EDRIDGE-GREEN and MARSHALL'S experiments (*a*) using filtered sodium lamp-light so that only the radiations of ·5890 and ·5896 μ were present, also using (*b*) a monochromatic yellow light, and (*c*) a redgreen mixture which matched this yellow light as closely as possible in hue, brightness and saturation, both of the latter (*b* and *c*) being obtained by using specially prepared colour filters. Employing these three yellow lights for producing adaptation of the eye, the writer confirmed in every respect the descriptions of their experiments given by EDRIDGE-GREEN and MARSHALL, and he arrived at the same conclusion as they had done, namely that here there is definite evidence for independent yellow receptors.

There are other circumstances in which yellow and a redgreen mixture do not resemble one another physiologically, thus lending further support to the view that there are specific receptors for yellow as well as those for red, for green and for blue.

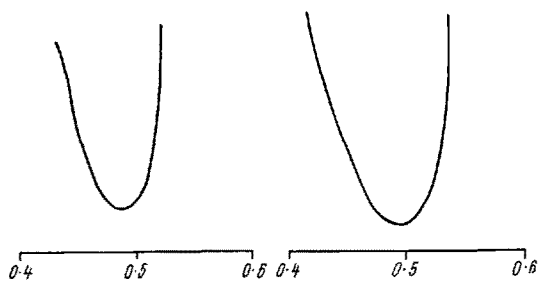
(7) *Alterations of colour vision with light intensity*

It is not proposed to go thoroughly into all the phases of colour vision and to show that in every case a polychromatic theory is necessary, but rather to concentrate attention on the chromatic aberration of the eye and to describe, so far as is known at present, the exact nature of the physiological mechanism by which it is corrected. If, at a medium intensity of illumination, we look at coloured test objects we appreciate all the colours of those objects, that is if we have normal colour vision. If now, the intensity be greatly reduced colours are found to fade and disappear, and everything appears black, white or grey, that is there is colourless vision. If on the contrary the intensity be greatly increased, in this case also it is found that colours disappear and once more there is colourless vision. In between these two extremes of light intensity, both of which are accompanied by colourless vision, there is colour vision but this is not homogeneous since it is found to vary in quality according to the intensity of illumination. Thus at high intensities, below that required to cause colourless vision, there is dichromatic vision resembling that of the protanope or the deuteranope, that is the long wavelength end of the spectrum looks yellow, and the short wavelenght end looks blue. On the other hand at low intensities, above those required to cause colourless vision, there is again dichromatic vision, but this time it resembles that of the tritanope, and the long wavelength end of the spectrum looks a purplish-red and the middle looks a bluishgreen. When this type of vision is in use there is no trace of yellow or of blue. Now here is the important point. Not only is this form of dichromatic vision found when tests objects are of normal size and light intensity low, but also when light intensity is normal and the test objects are small in size. Experiment shows clearly that the factor which determines whether there shall be this type of vision or not is the product of the intensity and the area of the retinal image. If the product is large there is normal colour vision, but if it is small then there is abnormal vision. At ordinary light intensities the fringes produced by chromatic aberration are of such a size on the retina that there is either dichromatic or colourless vision, and consequently the yellow and the blue colours of the fringes are not observed. Thus chromatic aberration is effectively dealt with by these physiological alterations in colour vision.

Let us now turn our attention to the larger issue and consider how these changes in colour vision are themselves brought about. To begin with let us restate the facts; at very low light intensities there is colourless vision, probably mediated by the rod receptors. At low intensities there is crimson-bluegreen dichromatic vision. At medium intensities there is full colour vision. A further increase in intensity causes this to be replaced by yellowblue dichromatic vision. Lastly at extremely high intensities there is colourless vision once more. How can these facts be accounted for? One very simple explanation would be to suppose that there are four types of receptor: purplish red, yellow, bluegreen and blue; at high light intensities and at large visual angles the second and fourth types are functioning alone; at low light intensities and at small visual angles the first and third types are functioning alone; at ordinary light intensities and visual angles all four types are functioning. This slightly modified form of HERING'S theory will be considered in the next section, and it will be shown that this theory fails to comply with certain important aspects of human colour vision. An alternative hypothesis will therefore be considered in the last section of this paper.

(8) *The four-colour theory*

The evidence produced in the previous section of this paper for red (crimson), yellow, bluegreen and blue receptors, leads to a consideration of HERING's four-colour theory. Unfortunately this theory fails to comply with certain facts of colour vision, the most important of which is hue discrimination. As is to be expected, the appreciation of hue depends on the differential stimulation of not less than two different types of receptor. Thus suppose a human subject to have in his retina two types of receptor only, one type having its maximum sensitivity at one wavelength and the other type having its maximum sensitivity at another wavelength, then the spectrum will be divisible into three parts: a long wavelength part "A" to which one type of receptor alone is sensitive; a short wavelength part "C" to which the other type of receptor alone is sensitive; and in between these a third part "B" comprising all the medium wavelengths to which both types of receptor are sensitive. Now in



a Fig. 4. *b*

a Hue discrimination of a protanope
b Hue discrimination of a deuteranope
Discrimination in arbitrary units, vertical. Wavelengths, horizontal.

parts A and C no hue discrimination is possible because only one type of receptor is stimulated, and therefore a change of wavelength produces an alteration in the degree of stimulation of one type of receptor only, that is, a variation of brightness is found to occur. In part B on the other hand hue discrimination is provided for and it will become most acute at that part of the spectrum at which a small change of wavelength produces the maximum alteration in the stimulation of the two types of receptor. If the response curves of both these two types are smooth ones it is likely that the maximum acuity will be found at some wavelength in between those at which the two response curves reach their maximum values, and hue discrimination will be found to be represented by a simple U-shaped curve. In confirmation of this reasoning the hue discrimination of the protanope and of the deuteranope may be pointed to, and the fact that in both these classes of dichromat the hue discrimination curve has a simple U-shape. See Fig. 4.

Now a human subject who has three types of receptor, one operating at long, one at medium, and one at short wavelengths, should have two separate U-shaped hue discrimination curves, one due to the long and the medium wavelength receptors acting in conjunction with one another and the other due simularly to the medium and short wavelength receptors. Moreover, such is found by experiment to be the case, as shown by an examination of the hue discrimination of the protanomalous trichromat who has three types of receptor. See Fig. 5.

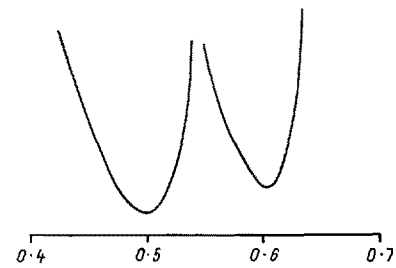


Fig. 5. – Hue discrimination of a protanomalous subject. Discrimination in arbitrary units, vertical. Wavelengths, horizontal.

On the same basis a human subject who has four types of receptor, one with its maximum response in the red, one with its maximum in the yellow, one with its maximum in the bluegreen and one with its maximum in the blue or blueviolet, and this is the arrangement suggested by HERING, should have hue discrimination conforming to three U's; one due to red and yellow receptors, one due to yellow and bluegreen

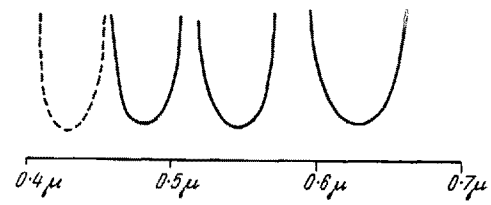


Fig. 6. – Hue discrimination based on HERING's theory. Discrimination in arbitrary units, vertical. Wavelengths, horizontal.

receptors and one due to bluegreen and blue (or blueviolet) receptors. But if the red type of receptor also operates at short wavelengths as well as at long ones there will be a fourth U-curve due to the interaction of the blue (or blueviolet) receptor with the repetition of the red receptor in the extreme violet. See Fig. 6. Now when these theoretical curves derived from the

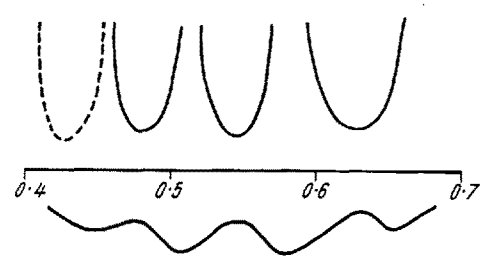


Fig. 7. – Upper curve: Hue discrimination based on HERING's theory. Lower curve: Hue discrimination found by experiment. Discrimination in arbitrary units, vertical. Wavelengths, horizontal.

	42	455	48	50	52	54	585	605	55
Spectral mixture (normal subject)	P	450	–	498	–	543	600	600	P
Hue discrimination (normal subject)	–	458	P	P	–	535	P	616	P
Spectral mixture (in protanomaly)	–	460	–	–	–	538	–	590	–
Hue discrimination (in protanomaly)	–	P	–	–	–	552	–	P	–
Subjective colour method	420	450	480	510	–	540	575	610	640
Fixation point method	420	460	–	500	–	540	580	620	660
Retinal direction effect	–	P	P	P	P	P	P	P	–
Conditioning method	–	–	P	–	–	–	585	–	–
Luminosity curve (reduced foveal vision)	–	–	–	–	–	–	(585) (565)	–	–
Periphery of normal subject	P	–	471	495	–	–	(576) (585)	–	P
Fovea at high intensities	P	–	476	507	–	–	574	–	P
Fovea at low intensities	P	–	P	500	–	–	570	–	P
Fovea at small visual angles	P	–	P	500	–	–	578	–	P
Granit using the microelectrode	420	460	480	500	530	540	580	600	–
Granit using electropolarization	–	450	–	–	–	520	–	610	–

four-colour theory are compared with those obtained by experiment it will be seen how very unlike they are. See Fig. 7.

We are forced to the conclusion that supporters of the four-colour theory can give no adequate account of the hue discrimination of normal sighted subjects.

A similar statement must be made concerning at least three other aspects of human colour vision: (*a*) the retinal direction effects of STILES and CRAWFORD; (*b*) the number of separate fixation points at the foveal centre, and (*c*) the number of subjective colours produced by stimulation of the fovea by means of monochromatic sources of small size. Now it seems likely that no one of these taken alone would be regarded as having sufficient weight to necessitate the abandonment of HERING'S theory; taken together, however, they constitute a serious setback to that theory which cannot be overlooked.

(9) *The polychromatic theory*

We have seen in the previous sections that four types of receptor alone would provide all that is necessary in order to comply with certain aspects of human vision, but that other aspects are not so favourably dealt with, one of these being hue discrimination. This is found by experiment to be good in the yellow and the bluegreen, and to be poor in the green and at both ends of the spectrum. In order to conform with these facts it is necessary that there should be receptors of three main-types: orange, green, and blueviolet. Where the response curves of the first pair, the orange and green, overlap, there should be good hue discrimination, and this provides the optimum in the yellow. Where the response curves of the second pair, the green and blueviolet, overlap, there should again be good hue discrimination and this provides the optimum in the bluegreen. Now three types of receptor, such as these, would agree with spectral mixture data and with current descriptions of cases of colour blindness.

The plan which is therefore suggested is that there are seven types of receptor in the human retina: (1) red or crimson, (2) orange, (3) yellow, (4) green, (5) bluegreen, (6) blue, and (7) blueviolet. The yellow and the blue receptors function at high intensities of illumination and at large visual angles. The red (or crimson) and the bluegreen receptors function at low light intensities and at small visual angles. Receptors of all types function at medium light intensities and normal visual angles, but the major role is played by the orange, the green and the blueviolet receptors. For convenience these combinations of receptors have been called by the writer the yellow-blue (Y–B) unit, the red-bluegreen-red (R–BG–R) unit. The "R" is repeated because red or crimson receptors must have two separate response curves, one in the red part and one in the violet part of the spectrum. Lastly there are the orange, the green and the blueviolet receptors which comprise what has been called the tricolour unit.

The positions in the spectrum of the apices of the response curves of certain of these receptors has been identified with some degree of certainty and are given in the Table.

Various hypotheses have already been advanced in order to account for these seven types of receptor. GRANIT[1] has suggested that those of the Y–B and of the R–BG–R units are derived from the receptors of the tricolour unit by processes of inhibition and summation. WRIGHT[2] has supposed that during evolution a single type of receptor first specialized into yellow and blue types, and that then many of the yellow specialized into orange and green types. If this process was repeated a third time, so that some of the blue ones have specialized into bluegreen and blue violet it would go far to comply with the polychromatic theory.

The author has been struck by the following curious point: at low intensities the long wavelength receptor

[1] R. GRANIT, J. Neurophysiol. *11*, 239 (1948).
[2] W. O. WRIGHT, verbal communication (1948).

is red (or crimson); at medium intensities it is orange; at high intensities it is yellow. Thus there is a change in spectral position with change of light intensity. Can it be that the same spectral change takes place with the short wavelength receptor, which is bluegreen at low light intensities and is blue at high ones? If so it would comply with some very interesting experimental results of THOMSON's[1]. He has found, what he has aptly called, "the reverse Purkinje effect", namely a movement of the apex of the photopic luminosity curve towards shorter wavelengths as light intensity is increased.

Whatever may be the outcome of these speculations one thing is certain: we know that the century old problem of the correction of the chromatic aberration of the eye is now solved. All the details of the correcting process are not yet known because they involve facts which lie at the very bases of human colour vision.

Zusammenfassung

1. Eine Untersuchung des Linsensystems des menschlichen Auges zeigt, daß es eine chromatische Abweichung haben muß. Dies wird mit folgendem bestätigt: a) beim Sehen durch ein Kobaltglas, b) mit einer teilweisen Verlegung der Pupille und c) durch die Messungen der chromatischen Aberration, wie sie übereinstimmend von verschiedenen Forschern erhalten wurden. Die durch die chromatische Abweichung verursachten farbigen Ränder sind beim Normalsehen nicht vorhanden; auch wird die Sehschärfe weder durch den Gebrauch einer Helmholtzschen Korrektivlinse noch im monochromatischen Licht verbessert. Es war zu fragen, wie sich diese anscheinend widersprechenden Befunde auf eine befriedigende Weise erklären lassen.

2. Wird der Sehwinkel reduziert, dann wird aus Gelb, Blaßgrau und aus Blau Dunkelgrau. Dieses Abblassen der Farben findet leichter statt, wenn Gelb und Blau einander nahestehen; es wird durch physiologische Vorgänge verursacht. Diese sind es, die die Randfarben ausschalten und damit die chromatische Abweichung des Linsensystems des Auges auskorrigieren. Die Helmholtzsche Linse und das monochromatische Licht verbessern das Sehen deswegen nicht, weil das bereits durch diese physiologischen Vorgänge geschehen ist.

3. Als vorläufige Erklärung nach der Dreifarbentheorie wurde für das Abblassen von Gelb und Blau folgendes angeführt: Die Verbindung der Blaurezeptoren mit den entsprechenden Hirnzentren wird aufgehoben. Dabei sind die Grünrezeptoren sowohl mit ihrem eigenen Zentrum für die Grünwahrnehmung wie auch mit dem Zentrum für die Blauwahrnehmung verbunden. Dies läßt das Gelb dem Weiß ähneln, da beide nun alle drei Hirnzentren reizen. Zugleich wird aus Blau Dunkelgrau oder Schwarz, da die Blaurezeptoren außer Funktion gesetzt sind. So sind diese Farbveränderungen befriedigend erklärt. Es gibt aber außerdem noch umgrenzte Helligkeitsveränderungen im gelben und blauen Teil des Spektrums, welche sich auf Grund einer Dreifarbentheorie nicht deuten lassen.

4. Die Einschnitte in der Helligkeitskurve im Gelb- und Blaubereich könnten auf Absorption durch Farbstoffe in der Macula beruhen. Das Hämoglobin würde den Einschnitt im Gelben hervorrufen, während der gelbe Farbstoff der Macula den im Blauen vorhandenen verursachen könnte. Die Untersuchung der Lichtabsorption und der Verteilung dieser Farbstoffe in der Netzhaut zeigt aber, daß die erwähnten Substanzen nicht mit den Einschnitten in der Helligkeitskurve zusammenhängen.

5. Die Ansicht, daß die Einschnitte im Gelben und Blauen nicht durch eine Herabsetzung der Tätigkeit der Gelb- und Blaurezeptoren, sondern durch eine gesteigerte Aktion der Rezeptoren für Rot und Violett verursacht werden, wird durch die experimentellen Befunde nicht gestützt.

6. Die Farbeinbuße im Gelben und die Helligkeitsherabsetzung durch Reduzierung der Lichtstärke oder des Sehwinkels wird am besten mit dem Ausfall besonderer Gelbrezeptoren erklärt. Diese sind in Funktion, wenn der Sehwinkel groß und die Lichtstärke hoch ist. Sie stellen aber ihre Tätigkeit ein, wenn diese Bedingungen nicht erfüllt sind. Die Unabhängigkeit der Gelbrezeptoren wird durch zwei Gruppen von Befunden bestätigt: a) in verschiedener Hinsicht gleicht das reine Gelb nicht der ihm ähnlichsten Mischung aus rotem und grünem Licht. Solche Unterschiede sind bezüglich der Größe des Sehfeldes, der retinalen Richtungseffekte, der Sättigung, der Lage der Fixationspunkte auf der Netzhaut und der Wirkung der selektiven Adaptation festzustellen. b) Die Übereinstimmung zwischen reinem Gelb und der Rot/Grün-Mischung wird durch verschiedene Faktoren gestört: vermehrte bzw. verminderte Lichtstärke, herauf- bzw. herabgesetzte Weite des Sehwinkels, Adaptation an ein helleres bzw. schwächeres Licht.

7. Mit der Lichtstärke wird auch das Farbensehen verändert. Bei sehr geringen Lichtstärken wird nur ein farbloser Eindruck in der Fovea hervorgerufen. Bei niedrigen Lichtstärken gibt es nur ein dichromatisches Sehen von Karmesinrot und Blaugrün. Bei mittleren Lichtstärken werden die Farben normal wahrgenommen. Bei hohen Lichtstärken gibt es blau-gelb dichromatisches Sehen und bei sehr hohen Lichtstärken ist der Eindruck wieder farblos. Die Größe des gereizten Netzhautbezirkes ist ebenfalls ein wichtiger Faktor. So gibt es bei kleinen Sehwinkeln karmesinrot-blaugrün dichromatisches Sehen, während Gelb und Blau nicht wahrgenommen werden. Auf diese Weise werden die durch chromatische Abweichung verursachten gelben und blauen Ränder ausgeschaltet.

8. Nach der HERINGschen Theorie gibt es vier Farbempfindungen: Karmesin (rot), Gelb, Blaugrün (grün) und Blau. Diese würden die im vorausgehenden Teil beschriebenen Arten des Farbsehens erklären. Leider stimmt aber diese Theorie schlecht mit den an normalsichtigen Versuchspersonen erhobenen Befunden über Unterscheidungsfähigkeit für Farbtöne überein. Diejenigen Bereiche des Spektrums, in denen nach dieser Theorie das Unterscheidungsvermögen besonders gut sein sollte, erweisen sich im Experiment als schlecht und umgekehrt. Die Theorie hält auch in anderer Beziehung nicht stand.

9. Um die Befunde über Farbtonunterscheidung und um andere experimentelle Tatsachen zu deuten, müssen mehr als drei oder vier Arten von Farbrezeptoren angenommen werden. Nach den vorliegenden Daten dürften besondere Rezeptoren für sieben Farben angenommen werden: 1 Karmesin, 2 Orange, 3 Gelb, 4 Grün, 5 Blaugrün, 6 Blau und 7 Blauviolett. 3 und 6 funktionieren auch bei hohen Lichtstärken und großen Sehwinkeln. 1 und 5 arbeiten bei sehr niedrigen Lichtstärken und kleinen Sehwinkeln. Alle Rezeptoren sind bei mittleren Lichtstärken und Sehwinkeln in Tätigkeit. Die Lage der Reaktionskurven gewisser Rezeptoren im Spektrum ist mit einiger Sicherheit ermittelt worden. Es wird diskutiert, wie diese sieben Rezeptortypen aus ursprünglich weniger Arten abgeleitet werden könnten.

[1] L. C. THOMSON, J. Physiol. *106*, 59 (1947).