RECENT ADVANCES IN THE PHYSIOLOGY OF VISION—PART III*

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Section I: Human Colour Perception

Three papers appeared at the end of last year which are of great importance to the theory of human colour-vision. In the first paper the author, Dr. Motokawa (1949), describes his method, which has both interesting and novel features. It consists in exposing the eye to light and immediately afterwards investigating the sensitivity of the eye to an electrical stimulus of known current strength and duration. He used lights which either were white or were monochromatic of chosen wavelengths from the visible spectrum. The electrical apparatus consisted of batteries, switches, and resistances so arranged that the duration of the stimulus was always one-hundredth second. The current was applied to the eye by means of two electrodes, one above the eyebrow and the other on the temple. A subject with normal sight gave electrical sensitivity curves after exposure of the eye to red, to green, or to blue lights, the shapes of which depended on which light was used.

Only two of these, that for blue and that for green, were found in a red-blind subject; only two, that for red and that for blue, were found in a green-blind subject; whereas all three were found in an anomalous trichromat. In other words, the results were in satisfactory agreement with current views on the type of vision present in these colour-defective subjects.

In the second paper the method is extended to the investigation of different regions of the retina of a subject who has normal colour-vision. The fovea was found to be trichromatic in accordance with the findings obtained by means of colour-mixing experiments, thus supporting Young's theory, with, however, this strange difference, that whereas the anomalous trichromat after exposure to white light was found to exhibit a three-humped electrical sensitivity curve—one hump for the red component, one for the green component, and one for the blue component (see Fig. 1)—the subject with normal colour-vision exhibited a smooth curve without humps (see Fig. 2), thus clearly showing that his retina comprises some additional factor or factors not present in the anomalous trichromat, which fill in the gaps between the three main humps. Let us then consider what the modifying factor or factors present in the normal-sighted are likely to be.

Wright and his co-workers showed that, although the normal-sighted and the proto- and deuto-varieties of anomalous trichromat are classified as trichromats on the basis of Young's theory, they are found to differ greatly in colour perception. Thus the ability to distinguish grada-

tions of colour is very good in the normal-sighted from one end to the other end of the visible spectrum (see Fig. 3). On the contrary, the ability to recognize these same gradations in the spectrum is greatly reduced in the two types of subject with anomalous vision (see Fig. 4). It will be noticed that in the yellow and the blue-green the difference between the normal-sighted and the anomalous subject is quite clearly marked but that it is even more marked in the red, in the green, and in the blue. In fact, many of

FIG. 1.—Three-humped electrical sensitivity curve of a deuteranomalous subject for white light which has been separated diagrammatically into its three R, G, and B components. Arbitrary electrical units, vertical; time in seconds, horizontal.

FIG. 2.—Smooth electrical sensitivity curve of a normal-sighted subject for white light. Arbitrary electrical units, vertical; time in seconds, horizontal.

FIG. 3.—Hue-discrimination curve of a normal-sighted subject. Wavelengths in μ , horizontal; acuity of discrimination in m. μ , vertical.

FIG. 4.—Hue-discrimination curve of an anomalous trichromat. Wavelengths in μ , horizontal; acuity of discrimination in m. μ , vertical.

these anomalous subjects have hardly any hue discrimination at all at these parts of the visible spectrum.

No satisfactory explanation of these important differences has ever been given on the basis of the three-colour theory. Now, this is one of many points where superiority over the three-colour theory is shown by the polychromatic theory. According to this theory persons who have normal colour-vision possess not only the three fundamental types but have several additional types of receptor, which have the function of enhancing hue perception. It is these which give the improved hue discrimination in the red, the green, and the blue that is so valuable a feature of normal

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colour-vision. Much evidence supports the idea that four additional types of receptor are required in order to effect this improvement—one with a maximum response in the extreme red, another in the yellow, a third in the blue-green, and a fourth in the blue.

On these grounds it would be expected that Motokawa's method would demonstrate not a major difference between the sensitivity curves of the normal-sighted and the anomalous, since the foveas of both possess the three important types of receptor postulated by Young's theory, but rather some minor difference caused by the sensitivities of the four additional types of receptor which according to the polychromatic theory are present in the retinas of normal-sighted subjects. The filling-in of the gaps between the electrical sensitivity curves of the orange, the green, and the blue-violet receptors is exactly the kind of change that would be expected from the presence of these four additional types of receptor, just as these same receptors fill in the gaps which are observed in hue discrimination when only the orange, the green, and the blue-violet types of receptor are in operation.

It probably will have been noticed that an apparent inconsistency exists in this reasoning: in the case of electrical sensitivity it has been claimed that the additional receptors fill in the gaps between the three major components, whereas in the case of hue it has been claimed that the additional receptors fill in the gaps, not in between the major components, but at their very crests. Thus, in the case of electrical sensitivity the gaps appear in the yellow and the blue-green, whereas in the case of hue they occur in the orange, in the green, and in the blue.

The explanation of this apparent discrepancy lies in the fact that not one receptor alone, but two at least are required in order to provide discrimination of hue, because a change of wavelength must bring about not a change in the degree of stimulation of one type of receptor only, since this could be imitated by a change of light intensity, but a differential change in at least two types of receptor. The most sensitive arrangement is met with where with a change of wavelength one type of receptor receives a greater strength of stimulation and another type of receptor receives a less strength. Thus, good hue discrimination in the orange would be provided by the presence of two types of receptor, the response curves of which slope in contrary directions in this region of the spectrum. Satisfactory situations for these would be in the yellow and in the extreme red. Similarly, for improved discrimination in the green one type could be in the yellow and the other type in the blue-green; and for improvement in the blue one type could be in the blue-green and the other type in the blue or the blue-violet. Thus, the same additional types of receptor are required to smooth out the gaps in hue discrimination as are required in order to fill in the gaps in electrical sensitivity.

The presence of these additional types of receptor is supported by the evidence which Motokawa obtained when he investigated retinal areas at 15, at 25, and at 35 degrees with the axis of fixation, for he found irrefutable evidence for a yellow type of receptor which played a part of increasing importance as the periphery of the retina was approached. He also found that the orange and the green receptors present in the fovea altered in wavelength, the one towards the red, the other towards the blue-green. The arrangement found was in conformity, therefore, with the tenets of the polychromatic theory and was also strongly reminiscent of Hering's theory with its four receptors—red, yellow, blue-green, and blue. Thus, there is at the fovea an arrangement substantially in conformity with Young's theory.

On the other hand, away from the fovea the arrangement resembles that postulated by its rival. It will be recollected that the polychromatic theory comprises both these older theories in requiring not only the orange, the green, and the blue-violet receptors of the three-colour theory but also the red (or crimson), the yellow, the blue-green, and the blue demanded by the four-colour theory, so that seven types of receptor are required in all. Of these types, six have been identified so far by means of this new technique.

It should be noted that in the case of Hering's theory the red (or crimson) receptor is regarded as possessing one response curve in the red and a second response curve in the violet. In the case of the polychromatic theory the same assumption is made, but it does not form an essential feature of the theory.

There is still much ground to be covered—namely, to investigate the retina at high and at low intensities of illumination and also for test fields of different sizes. When these have been completed—and so far as can be seen they should offer no difficulty—we shall be well on the road which leads to a more complete understanding of human colour-vision.

Section II: Perception of Yellow by the Human Eye

According to the three-colour theory of Thomas Young the sensation of yellow is perceived when the red and the green receptors are stimulated in a certain ratio. When the ratio is altered in favour of the red, the yellow sensation is replaced by an orange one, and when the ratio is altered in the reverse direction the sensation is one of greenish yellow or of yellowish green. If it be true that a sensation of yellow is perceived when the red and the green receptors are stimulated in a certain ratio, then an identical yellow should also be perceived when the red receptors are stimulated by red light and at the same time the green receptors are stimulated by green light, the intensities of the lights being such that the ratio of stimulation is the same as before. But experiment shows that such is not the case, because to persons with normal sight the pure yellow is found to be more saturated than the red-green mixture.

An attempt is made by supporters of the three-colour theory to meet this difficulty by supposing that the response of each of the receptors not only occupies its own particular part of the spectrum but certain neighbouring parts as well. Two important points should be noted: first, experimental evidence is against the acceptance of this supposition; and, secondly, this supposition leads to completely wrong conclusions with regard to the colour-vision of the protanomalous subject. Thus, the three-colour theory is faced by serious difficulties so far as the perception of yellow is concerned. But these are not by any means the only ones. If it be true, as three-colour theorists suppose, that yellow stimulates red receptors and green receptors just as a red-green mixture does, we should expect (1) that a pure yellow and a mixed yellow which have been adjusted to match one another would have visual fields of the same size when measured by means of a perimeter; (2) that the two yellows would produce identical results when the eye, after being adapted to them in turn, examines the different spectral colours; (3) that the two yellows would continue to match when alterations are made in the part of the retina receiving stimulation, in the size of the visual field, and in light intensity; and (4) that in other ways the two yellows would produce indistinguishable results. But experiment shows that none of these expectations is realized in practice.

According to the polychromatic theory there are four different types of receptor in operation in the red-orange-yellow-green part of the spectrum. Of these, the orange

and the green receptors play a major part and the red and the yellow a minor one at the foveal centre. Pure yellow light stimulates the yellow receptors strongly, the orange and the green receptors less strongly, and the red receptors feebly. On the contrary, a mixed yellow, consisting of red rays and green rays, stimulates the red and the green receptors strongly, the orange receptors less strongly, and the yellow receptors feebly. Owing to these differences in stimulation produced by the pure and by the mixed yellow, any alteration in the relative sensitivities of these receptors will disturb a match which has been made between these two yellows. It would be expected that the disturbances would be of three kinds: (a) in hue, (b) in intensity, and (c) in saturation. Experiment shows that all three disturbances are present. It is these disturbances which cause the perimetric field of a mixed yellow to be much smaller than that of a pure one, and which cause mismatching when alterations occur either in the intensity of the light which is illuminating the test object or in the angle which the latter subtends at the observer's eye.

Section III: Two Unusual Cases of Colour Deficiency

Pickford (1948) has recently reported a case of colour-blindness in the left eye which followed injury to the left side of the face in a road accident. Three years after the accident the patient's condition was as follows.

Right side of face normal. Left side of face partially paralysed. Normal hearing in right ear, stone-deaf in left. Both eyes myopic and with poor acuity even after correction. Colour-vision of right eye normal, and all Ishihara's plates correctly identified. None could be identified with the left eye. The perimetric fields were normal for all colours using the right eye. The left eye had a normal green field but very small fields for red, yellow, and blue.

The spectrum appeared normal to the right eye, except that the violet was called pale blue. To the left eye the blue-green, the green, and the yellow-green appeared one uniform green and the other spectral colours were called grey or dark grey.

In several respects Pickford's case outlined above resembles one recently examined by me.

A middle-aged subject, who previously had good colour-vision, suddenly experienced a distortion of vision and then a scotoma in the macula of his left eye. This scotoma seemed to form a partial obstruction so that everything seen through it appeared to be a dark bluish green. At ordinary light intensities the right eye had good acuity, the left eye having only 6/36. But with very intense illumination this improved to 6/9. Colour-vision tests, using Ishihara's plates, Edridge-Green's lantern, Holmgren's wools, and colour-filters, showed the right eye to be normal but the left completely colour-blind. Thus bright red and bright blue were found to match one another, since both looked bluish-green. They were found to resemble in hue a spectral colour of $0.52\text{--}0.53\ \mu$ wavelength, as seen by the normal eye.

At first it was suspected that the scotoma in the left eye was due to an extravasation of blood, which then became altered to a greenish substance resembling haematoidin. That this could not be the explanation was proved by further tests: (a) no absorption bands were detectable; and (b) very intense red or orange light, which should have penetrated the pigment and thus have been seen in its true colour, failed to do so and appeared bluish green. The condition present in the eye seemed to be one of a special type of cone monochromatism. Against this diagnosis are two facts: cone monochromats do not usually suffer from scotomata and they have normal acuity at ordinary intensities of illumination.

The resemblances between this case and Pickford's are remarkable. In both patients the left eye was affected by "green vision" and was otherwise completely colour-blind, and both patients had normal colour-vision in the affected eye before the onset of the condition.

Section IV: Colour-blindness and the Theories of Vision

As soon as it appeared likely that human colour-vision is polychromatic in character two questions presented themselves for consideration: (1) will it be easier, or more difficult, to account for the many varieties of colour-blindness on the new theory than it was on the older three-colour theory?; and (2) will any alterations be required in the methods used in testing for the presence of colour defectiveness? The second question is obviously one of considerable practical importance, but so also is the first, though to a somewhat less extent.

It has always proved a matter of great difficulty to give an adequate account of the various classes of trichromat on the basis of the three-colour theory. According to Judd (1947) there are four classes: (1) the normal subject; (2) the protanomalous subject who requires more red in a red-green mixture which matches a given spectral yellow; (3) the deuteranomalous subject who requires less red in that mixture; and (4) the tritanomalous subject who requires more blue in a blue-green mixture which matches a given spectral blue-green. With regard to the first three types, they differ from one another not only in the results of spectral mixture but also in hue and in saturation discrimination. As already pointed out in Section I, three-colour theorists find themselves faced by great difficulties when they attempt to account for these differences. It was thought at one time that they were due to abnormality in the response of one or more of the three types of colour receptor—red, green, or blue. It is now known that the differences cannot be accounted for in this manner. Take hue discrimination, for example; not only are protanomalous subjects (type 2 above) inferior to the normal in this respect in the red part of the spectrum, but they are inferior in the green and the blue parts as well. These latter differences cannot be accounted for on the basis of Young's theory on the supposition that the red receptors have reduced sensitivities. Neither does this account for the requirement of more red in a red-green mixture which is matching a given spectral yellow, because the red deficiency should affect both the mixed and the pure yellow to precisely the same extent; so that if they matched to the normal-sighted subject they should also match to the protanomalous subject. But in fact they do not match.

Supporters of the polychromatic theory find themselves faced by no such difficulties. The protanomalous subject, according to this theory, is a true trichromat, possessing three types of receptor alone—namely, orange, green, and blue-violet. The subject with normal sight, on the contrary, is not a trichromat but a polychromat, having the three types of receptor which are found in the protanomalous subject and four additional types as well—namely, red, yellow, blue-green, and blue. These enrich colour-vision by making the hues of nearly all the spectral colours more saturated. It is they which improve the hue discrimination of the normal-sighted subject in the red, the green, and the violet over that possessed by the protanomalous subject. Moreover, the reason why the latter requires more red in a red-green mixture which has been adjusted to match a certain spectral yellow is because the protanomalous do not possess the red receptors which are present in the retina of the normal-sighted. Consequently, to them the red lacks luminosity, and in consequence more of it must be used. In the deuteranomalous the situation is reversed, because they have red receptors which are too active. Thus, to them the red part of the spectrum is too luminous, and so less red is required in the red-green mixture which matches a given yellow. In this manner all

the differences between the various classes of trichromats can be adequately accounted for. But it is known that normal subjects differ one from another, so that one observer will not accept the colour matches made by another. Moreover, the matches made by a subject on one day may not be accepted by him on the next. These individual and day-to-day differences are exceedingly difficult to account for on the three-colour theory, whereas to supporters of the polychromatic theory they present no difficulties, since they are due to small changes in the activities of receptors of different types.

With regard to the dichromats there are three clearly defined classes: the protanopes, the deuteranopes, and the tritanopes. All these types can be accounted for fairly satisfactorily on the three-colour theory except that there seem to be two essentially different types of tritanope and at least two types of deuteranope. These fit into the framework of the polychromatic theory much more easily than they do into that of the trichromatic theory.

With regard to the classification of colour-defective subjects, it is fortunate that no essential modification need be made. Those who see at least three different colours in the spectrum are all "trichromats." They do not find colour-matching difficult, and are of no danger to the community because of their colour-vision. Those who see only two colours in the spectrum are all "dichromats." They match colours badly and are always potential sources of danger. Lastly, there are the "monochromats," who are completely colour-blind.

Colour-vision tests which are designed in order to differentiate these three types, such as Ishihara's test cards and the lantern tests, may still be used with complete confidence, even if we now regard human colour-vision as polychromatic, because they separate the dangerous from the non-dangerous. In consequence, these practical tests need undergo no modification. Where, on the contrary, human colour-vision is being examined in order to identify as completely as possible any abnormality which may be present, the situation is entirely different. Much more precise methods of investigation are needed, and usually more than one type of measurement is required. Thus, the exact shapes of the luminosity curve, of the hue-discrimination curve, of the saturation-discrimination curve, and of spectral mixture data should all be known before a complete diagnosis is attempted.

Section V : Vitamin A and Some of its Coloured Derivatives

It is now an established fact that the important pigments visual purple and retinene are both closely related chemically to vitamin A. It is therefore of special interest to record the fact that Morton and his co-workers (1948) have prepared from vitamin A and from retinene, which may itself be derived from vitamin A by processes of oxidation, a number of pigments, having narrow absorption bands, which occupy a number of different positions in the spectrum. One set of pigments has been prepared using concentrated sulphuric acid and another set employing concentrated phosphoric acid (see Table). The interesting

the visible spectrum, and the fact that these pigments bleach readily under the action of light just as visual purple does. It is not suggested that these actually are the pigments present in the human retina for purposes of colour-vision, still less that the natural pigments depend on the action of strong acids for their development. What is so suggestive is that so many pigments of the required type can be prepared from vitamin A, which on other grounds is known to be present in the retina. It seems likely that facts of importance from the point of view of human colour-vision will be derived from a further study of these synthetic vitamin-A derivatives.

Section VI : Coloured Lights for Signals

It has long been known that certain colours are good for signalling owing to the fact that they can be recognized at a distance with ease. Thus, red and blue-green are found to be satisfactory, whereas yellow, blue, and purple are not. König (1897) thought that these results were due to the centre of the fovea being blue-blind or tritanopic, and this idea has persisted to the present day. The blue-blindness has been accounted for in various ways: (a) that it is due to the absorption of the blue and the violet rays by the foveal or macular pigment; (b) that it is due to the absence of blue receptors at the fovea; and (c) that it is due to the absence of rods, which are supposed to function not only as colourless receptors for scotopic vision but also as blue receptors for photopic vision.

After some experiments I arrived at two conclusions: (a) that the condition is not one of blue-blindness; and (b) that the condition is not limited to the foveal centre. With regard to the first point, blue-blindness would cause white to appear yellow, would reduce the luminosity at the short wavelength end of the spectrum, and would leave the hue and the luminosity of the green, the yellow, the orange, and the red parts of the spectrum unaffected, as anyone can prove for himself by looking through a piece of canary-yellow glass. But in fact we find that white undergoes no alteration, that green is replaced by pale blue-green, that yellow is replaced by pale grey, and that orange and red become indistinguishable from crimson. With regard to the second point, the change in colour-vision is found to occur anywhere in the retina where detail perception is good enough for the experiment to be performed successfully. This makes the pigment hypothesis untenable, because if in order to account for the change in colour-vision we suppose that there is yellow pigment everywhere throughout the retina, we then find ourselves faced with the difficulty of accounting for the existence of normal colour-vision, particularly in the blue and violet parts of the spectrum. Similar reasoning applies to the hypothesis that the phenomenon is due to absence of blue receptors. For if, in order to account for the so-called blue-blindness, we suppose that blue receptors are absent, then they must be absent everywhere throughout the retina, because every part is found to exhibit the phenomenon. But if blue receptors are absent everywhere then no part of the retina can have normal blue-vision—that is, that every part suffers permanently from blue-blindness—a conclusion which is clearly contrary to the facts.

In order to account for the existence of normal colour-vision for near objects, and of reduced colour-vision for distant ones, I advanced a neurological hypothesis which was based on the three-colour theory. This hypothesis has recently been criticized by Middleton and Holmes (1949) on the grounds that the "switching" mechanism in the connexions between retina and brain is a complicated one, and that independent evidence for such a mechanism

Wavelengths of Maximum Absorption in μ

Vitamin A in conc. H_2SO_4 ...	465	530	590	620*
Vitamin A in conc. H_3PO_4 ...	440-480	520	600	
Retinene, in conc. H_2SO_4 ...	440-460	520	560	664*
Retinene, in conc. H_3PO_4 ...	470	500	550	590
Rhodopsin		500		
Iodopsin			560	
Dominators		500	560	
Modulators	450-465	500	520-530	580-610

* Shown better with the antimony trichloride reagent.

points to be considered here are the narrowness of the absorption bands, their position in various parts of

is entirely lacking. With this criticism I entirely agree, and it was because of the unsatisfactory nature of this hypothesis that I abandoned it in favour of an alternative one based on the polychromatic theory (Hartridge, 1947). It is interesting to find that Middleton and Holmes have arrived at a similar conclusion—namely, that a satisfactory hypothesis can be framed on the basis of the polychromatic theory, but not on the three-colour theory.

Taking up the practical question concerning good and bad light for signals, Holmes (1949) has arrived at the following conclusions: red is the ideal colour for signals, giving high probability of recognition. Yellow and white are distinct colours, but the white should be of high colour temperature—that is, it should be a bluish and not yellowish white, and the yellow should be near 0.590μ —that is, a little on the orange side of sodium yellow. Green and blue are distinct colours at high intensities but not at low ones. Purple is not a suitable colour for signals.

With these conclusions I agree except for the use of both yellow and white signal lights in the same code. If illumination is good there is not much risk of a mistake occurring; but if illumination is poor, or if the signal is seen through mist or fog, then there is, in my opinion, grave danger of confusion taking place between yellow and white.

Section VII: Subjective Alterations of Brightness and Colour

When stars “twinkle” alterations are observed in both brightness and colour. Similar phenomena are sometimes noticed in the case of distant terrestrial light sources. It is usual to account for these alterations in appearance on purely physical grounds. The air through which the light rays pass is stated to be at different temperatures; some parts are warm and have a lower refractive index than other parts which are cooler. As the warm air rises, and the cool air sinks, the light rays which pass through them are subject to refraction in an irregular and unpredictable manner, just as they would be if a plate of glass having irregular surfaces were moved in front of the eyes. These changes in refraction are supposed to produce the alterations of brightness and colour.

On performing the experiment with the glass plate just referred to, I found that different phenomena are noticed if a distant point source of white light be looked at—namely, (a) lateral movements first in one direction and then in another, and (b) the production of short spectra, the lengths of which vary with the amount of the lateral displacement. The long axis of these spectra always passes through the real position of the point source and the short wavelength colours (for example, the blue and violet) are always near to that position, and the long wavelength colours (for example, the red and orange) are always displaced away from it. These findings would be expected if the light rays on their way into the eye were refracted and dispersed by the prism action which is produced by the irregular thickness of the glass plate, and similar findings would be expected to follow from refraction and dispersion produced by zones which consist of air at different temperatures. On examining the images of stars and of distant terrestrial point sources, movements are not obviously present nor are spectra to be seen. These facts seem to cast doubt on this physical explanation. I found that a point source quite close to the observer can be observed to twinkle under suitable conditions, and it has recently been found by Weale and me that, using a micro-stimulation apparatus, a point source may be seen to undergo changes of colour and brightness when the distance of the source from the eye is less than two metres, which

would seem to exclude the probability of rising currents of warm air as being the cause.

Since physical explanations do not appear to account for the observed facts, physiological ones must be examined instead. Present evidence suggests that each of the retinal receptors possesses specific colour properties of its own. Thus, one of these may be a red receptor and another may be a blue one. These receptors of different types (Hartridge, 1947) are distributed in an irregular manner, with a consequence that clusters occur, there being in one part of the retina a cluster consisting predominantly of one type of receptor, and in a part near-by a cluster consisting principally of another type. Also here and there small scotomata are to be found, the exact causes of which are not known at present. Now, the retinal image, even of the smallest source, does not consist of a bright point of light surrounded by darkness, but of a disk of light of finite size which is produced by diffraction and by the aberrations present in the lens system of the eye. When this disk of light falls on a heterogeneous collection of receptors of different kinds a white or nearly white response results. But when it falls on a cluster of red receptors there is a red response, and when it falls on a cluster of green receptors there is a green response. Moreover, when it falls largely or entirely on a scotoma the response is feeble, and may indeed be absent altogether. In this manner it is possible to account satisfactorily for the changes which are observed in the appearances of local light sources. Distant light sources may be dealt with in a similar manner. But the possibility that in their case some physical phenomenon also plays a part is not ruled out.

In the case of both local and distant sources the colours observed are pink and a greenish blue, and I have never been able to identify other colours such as yellow, blue, or purple. This is in agreement with the view that when the angle subtended by the objects which are being looked at is small, there is dichromatic colour-vision, with red or crimson as one primary colour and blue-green as the other.

Section VIII: Eye Movements and Methods of Recording Them

A number of different methods have been used for studying the movements of the eyes: some of these have been mechanical, some optical, and some electrical. Mechanical methods are obsolete and are not being used at the present time. Optical methods are of several different kinds. Some workers make use of the corneal reflection—for example, Lord and Wright (1948). Others have applied a small galvanometer mirror to the sclera, and have reflected light on to a moving photographic plate—for example, Adler and Fliegelman (1934). Barlow attaches a tiny globule of mercury to the sclera and records its movements photographically. All these methods suffer from two major defects: (a) they record head movements as well as eye movements; and (b) they record lateral shifts of the eyeball within its socket as well as eyeball rotations.

It is sometimes not realized how very free is the lateral mobility of the eyeball within the orbit. It is usual to make the statement that these structures form together a ball-and-socket joint. So they do, but the socket is very different from that provided for the head of the femur by the acetabular cavity of the hip. In the case of the eye, the socket is composed of connective tissue and smooth muscle, which is backed by a mass of semi-liquid fat. In this manner the eyeball is permitted very free rotation in all directions, but it is also provided with very free lateral movement at the same time. This as a rule seriously curtails

the precision of optical methods which are intended to record eyeball rotations only. So far as head movements are concerned a method of eliminating their effects has been devised and used by Hartridge and Thomson (1948) by employing a special optical principle.

The electrical method seems to have advantages which are not present in other methods. When suitable electrodes of small size are attached to the skin near the eye, differences of electrical potential may be recorded by means of a valve amplifier of high gain and a cathode-ray tube. On rotating the eye axis alterations in the potential are found to occur. Meyers (1929) thought, incorrectly, that the potential was due to the action currents produced by the external eye muscles. On the contrary, Mowrer, Ruch, and Miller (1936) showed that the potential originates within the eyeball itself, and that as the latter rotates the corresponding changes of potential occur in neighbouring eye structures. This method has been used by Carmichael and Dearborn (1948) for studying the movements of the eyes during reading.

The relative advantages of this electrical method and the optical methods may be compared as follows: the optical method requires a dark-room, and part of the apparatus must be situated within the subject's field of vision. The electrical method does not suffer from these disadvantages, but there are several points on which further information is required: (a) how constant are the potentials when a subject is performing fixation? (b) are the potentials entirely unaffected by head movements? and (c) do changes in the illumination of the visual field affect the values of the potentials?

Section IX: Refractive Errors of the Eye and their Causes

Many theories have been advanced in order to account for the origin of the refractive errors of the eye. Some of these theories have been examined recently (Hartridge, 1949).

Theory 1.—That they are largely due to variations in eyeball length. The eyeball is stated to be short at birth; so that accommodation has to be used in order to focus parallel rays on the retina; thus less accommodation is left for near vision and consequently the condition present is one of hypermetropia. As age increases the eyeball slowly elongates, thus reducing the long sight until in the adult the emmetropic state is reached. If, however, the elongation is not checked as it ought to be, and thus proceeds too far, there is myopia.

Theory 2.—That since the anterior corneal surface produces about two-thirds of the total refraction of the eye, this structure is largely responsible for the presence of refractive errors, both spherical and astigmatic. This theory led to the use of keratometers in order to determine the corneal curvature.

Theory 3.—That chance variations occur independently in focal length and in eyeball length, and in consequence different combinations are found in different subjects. If both are long, both are average in length, or both are short, there is emmetropia or near emmetropia. If, on the other hand, the focal length is longer than the eyeball length there is hypermetropia; whereas the opposite causes myopia.

Theory 4.—That growth proceeds in both eyeball and lens system simultaneously. In consequence emmetropia is more frequently met with than would be expected on the previous theories. Measurement has shown that this is in fact the case.

Theory 5.—That heredity plays an important part in determining the adult condition of the eye. This theory is supported by the work of Sorsby (1933) on the refraction of Jewish and non-Jewish children. He concluded that myopia is commoner among adult Jews because emmetropia is commoner among Jewish children. Vice versa, emmetropia is commoner among adult Gentiles because hypermetropia is found to be commoner when they are children. Thus the

hypermetropic child becomes an emmetropic adult, but the emmetropic child becomes a myopic adult. According to this theory Gentiles inherit emmetropia, but Jews inherit myopia.

If theory 5 is true it raises the question regarding the way heredity acts. We know that tallness and shortness are inherited characteristics. Does the same thing apply to the errors of refraction? Height is controlled by a specific hormone of the anterior pituitary. Does a special hormone, secreted by this gland, control the growth of the different parts of the eye? Can refraction be influenced by the local application of such hormones as thyroxine, adrenaline, "pituin," corticosterone, or by extracts of the anterior part of the pituitary gland? Now that the x-ray methods used by Goldmann and Hagen (1942) in Switzerland and by Deller, Sorsby, and O'Connor (1947) in this country have provided means of measuring both the eyeball size and the focal length of the lens system in the living human eye, answers to these questions should not be difficult to obtain.

Section X: Inequalities in Sizes of Retinal Images and their Correction

To normal-sighted subjects the perception of size is one of considerable accuracy. Moreover, the size of an object when its image falls on the fovea is substantially the same as it is when its image falls on a part of the periphery of the retina instead. But to some subjects this is not so, and sizes seem to alter with retinal situation. Again, to a subject who has normal sight a vertical wall looks vertical, and does not appear to be toppling forward or to be falling over backward. But there are subjects who see either one or other of these illusions. There are found to be eight simple types of abnormality: (a) top closer than bottom; (b) vice versa; (c) left side closer than right side; (d) vice versa; (e) top left corner closer than bottom right corner; (f) vice versa; (g) top right corner closer than bottom left corner; and (h) vice versa. How may the condition present be diagnosed? One simple way is to place in front of the subject a square with its two diagonals. This is then tilted until by trial and error a position is found at which the square appears to be vertical with its sides and corners equidistant from the observer.

To what are these abnormalities due? If the retinal images of the two eyes were compared it would be found that the images of near objects are laterally displaced relative to the images of distant objects. The displacement is to the right on the right retina and to the left on the left one. If, then, the same displacements were produced, by suitable means, in part of the image of a distant object, this part would appear to move closer to the observer. The displacements could be produced either by the lateral deviation of some of the light rays while they are passing through the eye media or by the lateral deviation of part of the retina relative to the remainder. In some patients it is not difficult to diagnose the precise cause of the condition which is responsible for the effects, but in other patients it is much more difficult.

One very obvious source of error is that the retinal image of one eye differs in dimensions from that of the other. This may either be present in the natural state of the two eyes or may occur as the result of correcting some error of refraction. Thus, suppose there to be unequal refraction in the two eyes—say one eye normal and the other either hypermetropic or myopic; if the latter defect be corrected by means of a spectacle lens this may alter the size of the image on the retina at the same time. Suppose the image of the right eye to be smaller than that of the left, then the left-hand half of the image is displaced to the right,

and so objects on this side appear nearer to the subject. At the same time the right-hand half of the image is displaced to the left, and so objects on this side appear further away to the observer.

How do these anomalies agree with visual theory? They support Hering's theory of stereoscopic vision extremely well. According to this theory the perception of the position of objects in space is due to the precise estimation of the lateral displacements which some parts of the retinal image have undergone in relation to other parts. In fact, it would not be too much to say that these abnormalities of the size-and-position sense provide the strongest possible evidence of the correctness of Hering's theory.

How can these abnormalities be treated? Most of them can be treated by the employment of special "size" correcting spectacle lenses, which are so designed that they displace the defective part of the retinal image and so cause it to correspond precisely with the part of the retina on which it should fall. Thus, if the error present is due to inequality in the sizes of the two retinal images, then the spectacle lenses which are prescribed make them equal once more. This subject of inequality of the retinal images, "aniseikonia," is one of great theoretical and practical value; an interesting account has recently been published by Lord Charnwood (1948).

Section XI: Blood Supply of the Fovea

It has been known for a long time that the fovea centralis is devoid of the superficial blood vessels which are branches of the central artery and vein of the retina. These vessels send out capillary loops which ramify richly over the macula, but which do not extend over the fovea itself, so that an avascular area is left. The term "avascular" is not perhaps a well-chosen one, because the choroidal blood vessels which supply the retina on its outer side are particularly well developed at the fovea. These vessels are not encountered by light on its way through the eyeball to the retinal receptors and so cause no obstruction to vision. That the superficial blood vessels obstruct light is shown by the Purkinje experiment, in which a bright point of light is focused on to the sclera by means of a convex lens. Another very satisfactory method that I have used is to focus the light on one of the closed eyelids, the focusing point being moved about from one spot to another. So long as the point of light is in movement the branches of the blood vessels are usually visible. But they disappear rapidly if the point remains stationary for a few moments. This disappearance indicates a fact of great interest—namely, that the normal retina has the property of very quickly adapting itself to any partial obstruction of the light rays which are incident on it.

When the eye is directed towards a uniformly lit surface, particularly when this is illuminated by light in which blue or violet rays predominate, streaks are seen to travel in various directions over the visual field in proximity to the fixation point. These streaks have recently been called "flying saucers" by Dr. Willmer. In fact, they look more like little fish with black bodies and bright tails. That they are caused by the shadows cast by the red blood cells as they travel through the superficial blood capillaries is shown by the following facts: (a) they are not seen in that part of the visual field which corresponds to the "avascular" area mentioned above; (b) they move rapidly with each systolic contraction of the heart and come nearly to rest during each diastole; and (c) the widths of the "fish" as seen in the visual field correspond approximately to the diameters of the red blood cells. Now, the corpuscles are circular biconcave disks; how, then, are the "fish"

produced? The bodies of the "fish" are presumably due to the shadows cast on the receptors by the red blood cells as they are driven along the retinal capillaries. The bodies are somewhat elongated owing to persistence of vision. The bright tails are probably due to illumination falling on retina which has just recently adapted itself to the reduced illumination produced by the shadows of the red blood cells.

The fact that blood vessels are not found at the fovea does not eliminate the possibility that haemoglobin is present in solution in the tissues of the retina, just as it is in both cardiac and skeletal muscle. This is, however, very unlikely in view of the colourless appearance of the retina when after removal from a recently excised eyeball it is spread on a piece of milk-white opal glass.

Section XII: Yellow Pigment of the Macula

It is well known that there is a yellow spot, or macula lutea, in post-mortem retinas near the posterior pole of the eyeball, and that at the centre of this yellow spot there is a depression called the fovea centralis where visual acuity for fine detail reaches its greatest perfection. The questions which arise concern the chemical nature of this pigment; its origin, the retinal structures in which it is situated, and, lastly, its effects on vision.

In two recent papers opposite views have been expressed concerning the existence of this pigment in the normal living fovea, because according to Professor Nordenson (1949) its presence is doubtful, whereas according to Dartnall and Thomson (1949) not only is it present but, further, it plays an essential part in retinal metabolism by assisting the fovea to get enough oxygen in spite of the absence of the retinal blood vessels in this important part of the retina.

Let us then review the evidence.

According to Gullstrand (1905) the yellow pigment is not present in the retina during life, but appears as a post-mortem effect. Wald (1945) believes that it is a carotenoid, probably xanthophyll, similar to that found in the green leaves of plants. According to Baly (1935) chlorophyll exists in two forms, A and B, and the former is changed to the latter by light. Whereupon carotene takes up oxygen from chlorophyll B, thus reconverting the A type, and itself becomes oxidized to xanthophyll. Now the blood contains carotene, which might undergo oxidation after death and therefore might stain the retina. Alternatively, the retina may itself contain sufficient carotene to produce staining; so that actually Gullstrand's views are not incompatible with those of Wald.

With regard to the structures in which the pigment is found, Polyak (1941) states that these are retinal layers Nos. 4 to 10—that is, the external molecular layer, the external nuclear layer, the internal molecular layer, the internal nuclear layer, the ganglion-cell layer, the layer of nerve fibres, and the internal limiting membrane. If this be so we should expect the pigment to be most dense in a ring round the fovea centralis, because it is here that these layers are thickest, but to be absent, or nearly absent, from the fovea centralis, because these layers are almost entirely absent in this part of the retina. The probable effects of this pigmented ring with its faintly pigmented centre must now be briefly referred to. In the first place no pigment may be present at all in a normal eye. Thus Wolff (1948) writes: "The yellow colour of the macula is best seen in post-mortem eyes in which the retina has already become clouded. It is not seen in perfectly fresh eyes, nor ordinarily with the ophthalmoscope." I have seen a yellow pigment

in some pathological human eyes, after removal from the body, but have never seen it in the freshly removed eye of any animal. It is stated that it may be seen by ophthalmoscopic examination using red-free light and in darkly pigmented fundi if daylight is used for illumination, also if the retina be detached immediately after enucleation and then be spread over a glass slide held in front of a white background.

In fact, the ophthalmoscopic appearances might very well be due not to the presence of macular pigment but to the absence of blood pigment, since the central area of the macula is devoid of superficial blood vessels. Thus, the evidence for macular pigmentation during life is conflicting.

When König (1897) investigated the special colour-vision which is found at the centre of the retina he identified it as tritanopia and claimed that the condition is due to macular pigment. Abney (1913) found that the proportion of the rays of different colour which are required in order to match the various spectral colours varied in different observers. He ascribed these variations to differences in the depth of the macular pigment. In fact, the centre of the retina is not tritanopic and its special colour-vision cannot be due to this or to any other pigment. Also, Wright (1946) found that, when the effects of spectral matching are expressed in such a manner that the effects of pigment would be eliminated, the personal variations are still found to occur. "It would seem certain that the differences in the curves represent genuine differences in the colour-matching characteristics of the observers, and, since the effect of macular pigmentation has been eliminated, this implies differences in the receptor processes themselves." These are but examples of some of the facts which have been erroneously ascribed to the foveal pigment. If this pigment were indeed present during life we would expect that colour-vision would be altered, not at the centre of the fovea, because the pigment is almost completely, if not completely, absent there, but in a ring with the fovea as centre, because this is the distribution of the pigment described by Polyak. But no such visual effects have ever been described so far as I am aware. Considering the facts as a whole there seems to be little to support the view that macular pigment is present in the normal human fovea in the living state.

Let us suppose, however, that this pigment is present in order to consider what probable effects it would have on vision, and let us assume that Wald is correct in stating that the macular pigment is xanthophyll. Now, this substance, in addition to producing some general absorption from 0.38μ to 0.52μ , has three clearly marked narrow absorption bands which have crests at 0.424μ , 0.447μ , and 0.478μ . Thus the absorption is limited to the short wavelength part of the spectrum. If this pigment is present in the macular ring it will reduce the intensity of the blue and violet parts of the spectrum so that there will be some degree of tritanopia or blue-blindness. I have pointed out elsewhere (Hartridge, 1947) that there are two essentially different forms of this condition—congenital and acquired. To subjects of the congenital type the short wavelength part of the spectrum looks red or crimson. To the acquired type this part of the spectrum may appear to be entirely absent, but its normal appearance is restored on sufficiently increasing its intensity. If pigment is present in the macula it is this type of tritanopia which must be produced to some extent, so that more blue light will be required in order to replace the colourless vision, due to the rods, by the colour-vision, due to the cones, and more blue will be required in a blue-green mixture which has been adjusted to match a given spectral blue-green. Will white objects

look yellow? They probably will not do so if the macular pigment maintains a constant concentration, because of the very marked power of adaptation possessed by the retina. It is for this reason that candle-light, which is very deficient in blue and violet rays in comparison with daylight, ceases to look yellow after a few minutes' use. If the retina can adapt for a temporary change in blue and violet brightness, such as this, it is much more likely to do so for a condition which is constantly present. It must be emphasized once more that even if pigment is present in the normal macular ring it will not affect the colour-vision of the fovea, because the evidence is that there cannot be more than a trace of this pigment at this part of the retina.

Section XIII : Perception of Brightness or Luminosity

When advancing their theories of colour-vision both Young and Hering considered how the perception of brightness may take place. According to the three-colour theory, specialized receptors for luminosity are unnecessary because the receptors for colour mediate luminosity at the same time. According to the four-colour theory, on the contrary, there are specialized receptors for luminosity—that is, for black, for white, and for the various shades of grey. What are modern views on this subject?

If Hering's views are correct it should be possible to have brightness without colour, and colour without brightness; the colour-deficient should have spectral luminosity curves of normal shape, they should not require more light at the photopic threshold, and they should not demand an increase of illumination in order to achieve a given measure of acuity. Moreover, so far as normal colour-vision is concerned any alterations which it undergoes—for example, at different parts of the retina—should not be accompanied by changes in the shape of the luminosity curve. All these results must accompany the presence of an independent mechanism for the perception of luminosity. Young's theory, on the other hand, would require the exact opposite of Hering's theory. It would not be possible to have colour without brightness, or brightness without colour, except when, under conditions of low illumination, the twilight (scotopic) visual mechanism which is devoid of colour sense is in use. Each type of colour-blindness should have a luminosity curve of a typical shape, and these subjects should require more light in order to achieve colour-vision than normal sighted subjects do; moreover, they ought to require more light in order to reach a given standard of visual acuity.

The facts are as follows: each type of colour-blindness is found to have its own specific luminosity curve, by means of which it can be identified. Thus the protanope, who suffers from red-green blindness, also has reduced luminosity in the red and orange parts of the spectrum. On these grounds it is stated that his defect is due to absence of, or failure on the part of, the red receptors. But, further, these subjects were found by Hecht, Hsia, and Shlaer (1947) to require 119% more white light than that wanted by subjects with normal colour-vision in order to achieve a given degree of visual acuity. Thus there is consistent evidence not only that these subjects suffer from a colour deficiency but that they have a brightness deficiency as well.

Here, then, are strong grounds for the view that the red receptors mediate not only the sensation of colour but also the sensation of luminosity in the particular part of the spectrum by which they are stimulated.

This view is supported by observations on human colour-vision, because when, owing to lack of illumination or to the employment of a small visual field, full colour-vision is replaced by reduced colour-vision not only are losses of

colour to be found in the yellow and in the blue parts of the spectrum, so that these parts become a neutral grey, but at the same time there are losses of luminosity, producing the notches in the luminosity curve which have been described by Sloan (1928), by Wright (1943), by Thomson (1949), and by others.

As the result of his work on the colour-vision of animals, employing the micro-electrode technique, Granit (1947) found evidence for colour receptors (modulators) and for luminosity receptors (dominators). At first sight these discoveries support the opposite view to the one advanced above. It must be recollected, however, that Granit has himself proposed, as the result of further work on dominators, that they are not a specific type of receptor but are derived from the combined action of many, and possibly a great many, modulators. If this be true the luminosity response of the dominator must surely be obtained from the luminosity responses of some or all of the modulators of which it is composed. One further point: if it be true that the dominators are luminosity receptors, as Granit has suggested, this does not make it less likely, but more likely, that the colour-perceiving modulators are also luminosity receptors—that is, so far as their own particular colour is concerned.

Returning now to the subject of human colour-vision, we do not know for certain whether there are dominators in the retina or not. For purposes of discussion let us suppose that these are present and that they have the function of mediating luminosity. Then there will be two mechanisms for this function—the dominators which operate when they are stimulated by light rays of all wavelengths, and the colour receptors which are stimulated selectively by their own particular wavelengths. We can ascertain the respective parts which these two mechanisms play by examining what happens in colour deficiency. Thus in protanopia the luminosity of the spectrum in the red is reduced to a very low value, if not to zero.

It seems evident, therefore, either that the contribution made by dominators is small, or that, for some reason at present unknown, not only the red receptors are inactive but the dominators as well so far as the red part of the spectrum is concerned.

In cone monochromatism there is defective luminosity perception at both ends of the spectrum, so that also in this case dominators seem to be making only a small contribution. In other types of colour deficiency the evidence is not so clear as in the two cases just cited, but, so far as can be judged at present, the evidence is in favour of the view that luminosity is mediated in normal human vision less by specialized dominators than by the colour receptors themselves.

At very high and very low intensities of illumination things may be widely different. The evidence here is that there is colourless vision when the illumination has been sufficiently increased. Could this not be due to dominators which may play only a small part at ordinary light intensities but an increasingly important part as intensity is increased? A similar question arises with regard to low intensities at which there is also colourless-vision; can this be due to dominators or are the red receptors alone responsible for this?

This is a question which will have to be decided as the result of future research, but there is another aspect to which reference must be made. Pickford has investigated colour-blind subjects recently in order to ascertain the degree of correlation between loss of colour sense and loss of luminosity perception. He points out, correctly, that

according to the classical three-colour theory there should be the closest possible correlation between them on the basis that the same receptors mediate both colour-vision and luminosity-vision. He finds, however, that experiment demonstrates no such close association between these two attributes, and he therefore accepts Hering's theory which has separate mechanisms for colour and brightness perception, or, better, Houston's modification of this theory. I have pointed out elsewhere, however, that difficulties confront this theory just as they do the three-colour theory, and because of these inconsistencies I have advanced the polychromatic theory. How does this theory deal with the important point discovered by Pickford? The nerve fibres on their way from retina to brain are supposed to encounter at some part of their course a bifurcation where the nerve fibres divide or separate into two divisions. One division conveys the impulses to the brain centre for the perception of luminosity, and the other division conveys the impulses to the brain centre for the recognition of colour. Now if all the nerve fibres were cut below the bifurcation there would be complete loss both of brightness and of colour sense. If they were all cut after the bifurcation, but in one division only, there would be either complete loss of luminosity but retention of colour sense or complete loss of colour sense and retention of luminosity perception. Similarly, a partial loss, concerning, for example, the red receptors only, would cause complete loss of both perception of red and of luminosity in the red part of the spectrum if the interruption occurred below the bifurcation; but if it occurred above this structure there would be either loss of luminosity in the red and retention of red perception, or vice versa, according to which of the two divisions of the nerve paths had been affected by the lesion. In this manner Pickford's conclusion may be accounted for satisfactorily on an orthodox physiological basis.

Section XIV: Some Effects of Polarized Light on Vision

When a uniformly lit white surface is examined through a Nicol prism or a piece of polaroid, the cross described by Haidinger can usually be seen; this consists of a yellow bar intersected at right-angles by a blue one. On rotating the Nicol prism or polaroid this cross is also seen to rotate. If the polarizing apparatus remains still the cross slowly fades and may disappear completely owing to colour adaptation on the part of the retinal receptors. On the other hand, rotation of the polarizing apparatus, by interfering with this process of adaptation, causes the cross to persist. Some persons can see the cross distinctly without the use of a Nicol prism or polaroid when they look at an object from which partly polarized light is coming, such as the sky, or reflecting surfaces such as water, glass, and furniture. These facts, which have been known for many years, are usually ascribed to the lamellar structure of certain parts of the eye—for example, the nerve-fibre layer of the retina.

Recently some suggestions of very great interest have been made by Von Frisch (1947, 1949) concerning this property of the eye in the case of the honey bee. Briefly the facts are as follows:

When a bee, while out foraging, discovers a new and rich source of nectar, it conveys the information to the other occupants of the hive by an excited dance. Wagging its abdomen from side to side, it ascends the face of a comb and by the direction it takes it indicates to other foragers both the direction and the distance away of the new source of nectar. Suppose that during the return flight to the hive the forager had the sun on its right hand, so that the sunlight made an angle of 90 degrees with the direction of travel, then on entering

the hive the bee will turn to the right and will proceed along the comb face in such a direction that gravity now has in the same direction as the sun's rays had previously. Suppose, on the other hand, the sun was directly behind the forager during its return flight, then it would proceed vertically upwards on entering the hive. The direction taken by the forager when inside the hive is carefully noted by the other bees, and they direct their flight accordingly.

The distance away of the source of nectar is indicated by the number of waggles of its abdomen which the forager makes while it travels over the comb face. If the distance of the source of nectar be 100 metres, the forager on returning to the hive is found to perform 10 waggle runs in 15 seconds. If it be 1,500 metres then 5.5 waggle runs are performed in 15 seconds. Shorter distances are indicated by more frequent waggle runs and greater distances by less frequent ones.

If the sun be hidden behind clouds the bees are still found to detect its true position. Von Frisch suggests that this is done by determining the directions in which the light is polarized. I have endeavoured to do this by using a Nicol prism held before the eye, but without success, because the Haidinger cross, which is visible on looking at the sky, is invisible when clouds are looked at instead, and the situation is not improved by the use of the Nicol prism. If, then, bees can deduce the sun's position from the direction of polarized light given by clouds they must have at their disposal better means of detecting polarization than that provided by a Nicol prism. In human eyes it is the nerve-fibre layer which is supposed to be responsible for Haidinger's cross. But in bees' eyes the light rays do not pass through the nerve fibres on their way to the receptors as they do in human eyes. If, then, bees see Haidinger's cross it must be produced in a different manner.

Other structures besides the nerve fibres produce polarized-light effects in the human eye—for example, the cornea and the crystalline lens. But these do not normally affect vision.

A more detailed account of Von Frisch's interesting experiments has recently been published by Thorpe (1949).

Section XV: Stimulation of the Retina by Electricity and Magnetism

It has been known for almost two centuries that the passage of an electric current through the eyeball gives rise to a visual sensation or phosphene. Unfortunately, much of the earlier work was performed at a time when the exact measurement of voltages, currents, and resistances was impossible.

A direct current of constant amperage acts as a stimulus to the eye. Thus, when the cathode is over the closed lids, and the anode somewhere on the body, a yellow colour is perceived; but when it flows steadily in the reverse direction the colour seen is bluish violet. The closure and interruption of a constant current also stimulate the eye, causing a bright flash of light. The flash is brighter on closing the current when the anode is on the eyelids and the cathode is on the body, whereas it is brighter on interrupting the current when the electrodes are in the reverse position.

When the eye is stimulated by light and by an electric current at the same time some interesting effects are found to occur. With the anode on the open eyelids, and a constant current flowing, the shorter wavelengths are more readily perceived, and vice versa for the longer wavelengths. On reversing the direction of flow of the current the opposite effects are produced. There are three spectral regions for which there is no change of sensitivity. These are (a) in the red, near 0.70μ ; (b) in the yellow, near 0.57μ ; and (c) in the blue-violet, near 0.40μ .

Not only do electric currents modify the thresholds of light as described above but the reverse effects are also found—namely, that exposure of the eyes to light modifies the threshold to electrical stimulation. Thus Barlow, Kohn, and Walsh (1946) found that light-adaptation raised and dark-adaptation lowered the threshold to electric stimulation produced by the discharge of charged condensers.

When an after-image has been produced by the incidence of light on the eye it is found that the flow of an electric current modifies it in the following ways. When the anode is in contact with the closed eyelids the current tends to accentuate a negative after-image but to diminish a positive one; when the cathode is on the lids the after-images are affected in the reverse manner.

It seems probable that the electric current stimulates the retina rather than some other part of the visual apparatus.

Barlow, Kohn, and Walsh (1946) also investigated the effects of magnetic fields on vision. In this case, also, it is the retina itself which is stimulated. Thus, placing a powerful electro-magnet, through which an alternating current is passing, near the occipital region of the skull close to the visual centres was found to produce no noticeable result.

It will be recollected that Motokawa (1949) has recently used a light stimulus closely followed by an electrical stimulus in order to investigate the properties of human colour-vision. Some further notes about his method, and some of the conclusions to which he has come, will be found above in Section I.

REFERENCES

- Abney, W. de W. (1913). *Researches in Colour Vision*. Longmans Green and Co., London.
- Adler, F. H., and Fliegelman, M. (1934). *Arch. Ophthalmol.*, N.Y., n.s. **12**, 475.
- Baly, E. C. C. (1935). *Proc. roy. Soc. B.*, **117**, 218.
- Barlow, H. B., Kohn, H. I., and Walsh, E. G. (1946). *Amer. J. Physiol.*, **148**, 372.
- Carmichael, L., and Dearborn, W. F. (1948). *Reading and Visual Fatigue*. Cambridge, Mass.
- Charnwood, Lord (1948). *The Optician*, April 23.
- Dartnall, H. J. A., and Thomson, L. C. (1949). *Nature*, **164**, 876.
- Deller, J. F. P., O'Connor, A. D., and Sorsby, A. (1947). *Proc. roy. Soc. B.*, **134**, 456.
- Goldmann, H., and Hagen, R. (1942). *Ophthalmologica, Basel*, **104**, 15.
- Granit, R. (1947). *Sensory Mechanisms of the Retina*, p. 252. Oxford Univ. Press, London.
- Gullstrand, A. (1905). *v. Graefes Arch. Ophthalmol.*, **62**, 1.
- Hartridge, H. (1947). *Phil. Trans. B.*, **232**, 522.
- (1949). *Transactions of Optical Convention of Worshipful Company of Spectacle Makers' Convention*, November, 1948, p. 91.
- and Thomson, L. C. (1948). *Brit. J. Ophthalmol.*, **32**, 581.
- Hecht, S., Hsia, Y., and Schlaer, S. (1947). *International Conference on Colour Vision*, Cambridge.
- Holmes, J. G. (1949). *Docum. ophthalmol.*, **3**, 240.
- Judd, D. B. (1947). *International Conference on Colour Vision*, Cambridge.
- König, A. (1897). *S.B. Akad. Wiss., Berlin*, p. 559.
- Lord, M. P., and Wright, W. D. (1948). *Nature*, **162**, 25 and **163**, 803.
- Meyers, I. L. (1929). *Arch. Neurol. Psychiat.*, Chicago, **21**, 901.
- Middleton, W. E. K., and Holmes, M. C. (1949). *J. opt. Soc. Amer.*, **39**, 582.
- Morton, R. A., Ball, S., Collins, F. D., and Stubbs, A. L. (1948). *Nature*, **161**, 424.
- Motokawa, K. (1949). *J. Neurophysiol.*, **12**, 291, 465.
- Mowrer, O. H., Ruch, T. C., and Miller, N. E. (1936). *Amer. J. Physiol.*, **114**, 423.
- Nordenson, J. W. (1949). *v. Graefes Arch. Ophthalmol.*, **149**, 540.
- Pickford, R. W. (1948). *Brit. J. Psychol.*, **39**, 73.
- Polyak, S. L. (1941). *The Retina*. Univ. Chicago Press, Chicago.
- Sloan, L. L. (1928). *Psychol. Monogr.*, **38**, No. 1.
- Sorsby, A. (1933). *Annual Report L.C.C.*, **4**, Pt. 3, p. 55.
- Thorpe, W. H. (1949). *Nature*, **164**, 11.
- Von Frisch, K. (1947). *Bull. anim. Behav.*, **1**, No. 5, 1.
- (1949). *Experientia, Basel*, **5**, 142.
- Wald, G. (1945). *Science*, **101**, 653.
- Wolff, E. (1948). *The Anatomy of the Eye and Orbit*, 3rd ed., p. 103. Lewis, London.
- Wright, W. D. (1943). *Nature*, **151**, 726.
- (1946). *Researches on Normal and Defective Colour Vision*, pp. 149, 150. Kimpton, London.