METHODS OF TESTING FOR COLOUR VISION AND THEORETICAL DEDUCTIONS FROM OBSERVATIONS ON COLOUR VISION*

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Colour Discrimination

As the result of the work of many observers it has been established that in the spectrum there are two main maxima of colour discrimination, corresponding to the sensations of yellow and of blue-green. There may be some doubt as to the existence of one or more additional minor maxima (Corbett, 1936; Wright and Pitt, 1934). The method by which these conclusions have been established is by exposing two identical regions of the spectrum alongside each other and altering the wave-length of one until the two do not appear to match even after adjustment of the intensities has removed any difference in brightness.

At the two main maxima of discrimination the two lights can be recognized to be of the same colour in spite of differences in brightness, while at other wave-lengths a difference in brightness will cause a difference in colour even if the wave-lengths are the same (Roaf, 1927, 1932a; Corbett, 1936). These two spectral regions have been studied to find out whether the sharpness of discrimination is related to any special recognition of these colours when there is no fixed wave-length with which to compare the colour.

Yellow Sensation

The subjects were asked to set a spectroscope so that the field of view appeared to them to be yellow. They were asked to alter the wave-length until the light seemed to be slightly tinged with orange, then to alter back until the light appeared to be slightly tinged with green. The wave-length difference between these two extremes measures the "range" of the "yellow" part of the spectrum, and the mid-point between the two wave-lengths can be called "mid-yellow"—that is, the centre between the two points where yellow just begins to be tinged with the next colour in the spectrum should be the purest "yellow" in the spectrum.

As the result of the examination of 100 individuals it was found that eighty-seven normal persons showed an average "range" of $67 \pm 29\text{A}^\circ$ and the "mid-yellow" was at $5853 \pm 38\text{A}^\circ$, while thirteen of the subjects had defective colour vision and possessed an average "range" of $314 \pm 168\text{A}^\circ$ with a "mid-yellow" at $5547 \pm 237\text{A}^\circ$ (Hailwood and Roaf, 1937-8). The narrower the "range" of wave-lengths over which the colour is seen the greater is the power of discrimination. These figures confirm the conclusion, arrived at as the result of measurements of discrimination, that those with defective colour vision do not show a maximum of discrimination in the "yellow" region of the spectrum.

Blue-green Sensation

Comparable observations were made in which the subjects were asked to mark out the "blue-green" region by making the field first distinctly bluish and then dis-

tinctly greenish. The average "range" of the "blue-green" was $61.7 \pm 34.4\text{A}^\circ$, and the "mid-blue-green" was situated at $4926 \pm 51\text{A}^\circ$ for 100 cases. Of these cases nine had defective colour vision as shown by the Ishihara test. These nine persons gave an average "range" of $81 \pm 54\text{A}^\circ$ with a "mid-blue-green" of $4965 \pm 67\text{A}^\circ$. These figures confirm the conclusion that those with defective colour vision have no difficulty in distinguishing colours produced by this region of the spectrum (unpublished experiments by Mrs. J. C. Hay).

We cannot say that there are not individuals who have defective colour vision for this region of the spectrum, as ten cases showed a range of more than 100A° (one actually 207A°), but only one of these was classified as having defective colour vision. The reason may be that the Ishihara test is designed to detect the usual type of red-green confusion, hence it might not detect a failure to discriminate in the "blue-green" region of the spectrum. The mere width of the "range" does suggest a lower degree of discrimination by these subjects in this region of the spectrum, provided they have realized that the first clearly recognized difference in colour should be noted. The recorded figures are the medians from five separate readings for each limit of "range."

Tests for Defective Colour Vision

The importance of colour recognition for railway, marine, and aeroplane signalling has led to the use of many methods of testing. Of these the fundamental method is some form of lantern, as the recognition of coloured lights is necessary in all the above services (Edridge-Green, 1920). Owing to the low visibility of "blue" light and the deficiency in "blue" rays of many sources of illumination not much attention has been paid to the distinction between blue and green. ("Signal green" is really a "blue-green" allowing all the spectrum except the "red" to pass through.) The distinction is between red, yellow, and green. (White is not distinguished from yellow because many sources of light—for example, a paraffin lamp—are really yellow.)

Distinction between different degrees of defective colour vision may be made by using diaphragms of different areas, by altering the intensities of the light, and by changing the duration of the exposure of the light (Guttman, 1907, 1908). The duration of the exposure requires further study, and a preliminary examination of this factor has been made by Corbett and Roaf (1937). The new lantern devised for testing colour vision in the Navy makes use of the variations in area and intensity but does not change the duration of exposure.

By means of the spectroscope we can measure the extent of difference in wave-length necessary to produce a recognizable difference in colour in the "yellow" region. "Ranges" greater than 100A° suggest defective colour vision, but all subjects with these "ranges" do not make mistakes with the lantern or other tests.

The Nagel anomaloscope based on Rayleigh's observations (1881) enables us to test the match between a fixed "red" + a fixed "green" with a fixed "yellow." In the examination of 100 subjects all those (thirteen in number) who made abnormal readings could be classified as possessing defective colour vision, and all those who made the match within the normal range had normal colour vision. It is difficult to reconcile these results with the statement made that those with dichromatic vision will agree with the normal match but will also agree with an abnormal match. Perhaps there is some systematic defect in the anomaloscope method, otherwise some of the

* Paper read in the Section of Physiology and Biochemistry at the Annual Meeting of the British Medical Association, Plymouth, 1938.

subjects should have made settings in the normal region. Perhaps these subjects with defective colour vision were not dichromatic cases, but in that event defective colour vision does not correspond to dichromatism. On the other hand Pitt (1935) gives matching curves with two colours by subjects with dichromatic vision. There seems to be some need for an explanation of the fact that persons with defective colour vision are not always satisfied with the match made by a normal person.

Pseudo-iso-chromatic Plates

The introduction of coloured plates on which a normal person could see a design which was not visible to the individual with defective colour vision was due to Stilling. Ishihara's plates are probably the best to use. Dr. Collins found that in an examination of 379 persons no normal persons made a mistake with No. 2, 3, 7, 8, or 9, and that no individual with defective colour vision could see the number read by the normal person in No. 6 or 11. The degree to which the other plates were reliable in distinguishing normal from abnormal subjects is given (Collins, 1937).

Ishihara's plates are formed of small disks which vary in area and in purity of colour, so that when they are made into a design this can be recognized only by the colour. Any recognition of the design by differences in intensity is rendered impossible because the same colour may be present in different degrees of purity—that is, with more or less white in it—so that the disks appear paler or darker. In addition, the variation in area of the disks tends to confuse anyone who does not recognize the difference in wave-length composition of the reflected light.

The whole test is based on the outstanding psychological value of red for the normal person while the individual with defective colour vision cannot distinguish red from green. As those with defective colour vision can distinguish blue from green, blue is introduced to make designs which are not noticed by the normal person because of the outstanding effect of red on him.

The reason why a person with defective colour vision sees a different design from that seen by a normal subject is that he classifies the colours differently, as shown in the following table:

	Colours classed together	but distinguished from
Normal person	Yellow-red or vermilion and blue-red or carmine	Yellow-green and blue-green
Person with defective colour vision	Vermilion and yellow-green	Carmine and blue-green

The only exceptions are plates 12 and 13, which are said to distinguish "red-blindness" from "green-blindness." Two red figures are seen on each of these plates by the normal person. If red as such is not distinguished, neither number will be seen, as can be proved by looking at them through a blue-green light filter. One figure in each case is vermilion and lighter in shade than the other, which is a dark carmine or mauve. The blue in this latter makes it look to the normal person more like the blue-grey of the background spots. The vermilion number is said to be seen by the "green-blind," while the mauve number is said to be seen by the "red-blind."

This difference may be due to a decrease in sensitivity to the "red" end of the spectrum in scoterythrous (protanope) cases.

The vermilion disks reflect mainly "red" light, the mauve disks reflect "red" and "blue" light; hence a

decrease in sensitivity to "red" will cause a greater relative decrease in brightness of the vermilion disks. The number formed by the vermilion disks may thus fade into the background. The mauve disks reflecting "blue" light may not be so much decreased in brightness because of the "blue" light reflected, hence the number formed by the mauve disks becomes visible. The number seen by the "green-blind" actually reflects less "blue" than that seen by the "red-blind."

Up to the present no evidence has been brought forward to explain why different numbers are seen in plates 12 and 13 by different individuals with defective colour vision.

Binocular Colour Fusion

Each eye can give rise to an independent judgment of intensity, colour, and shape. When both eyes are used combination of the results may occur, in a similar manner to that in the spinal cord. With low intensities summation is possible, as shown by Lythgoe and Phillips (1937-8). At higher intensities occlusion may be present, hence a landscape may appear darker with both eyes than with one eye. If a neutral glass is placed in front of one eye, that eye being obstructed by an opaque screen, it is found that when the opaque screen is removed the landscape appears darker, whereas it ought to appear brighter because the impulses from the unobstructed eye are reinforced by further impulses from the eye which has just been exposed by removal of the screen. This effect is observed over a certain range of illumination. If the neutral glass is too opaque or too transparent the difference in darkness is not observed. This experiment suggests some sort of compensation between the impulses coming from the two eyes, which, of course, is analogous to occlusion.

By presenting different colours to each eye it is possible to obtain an addition of the sensations very similar to the result of simultaneous stimulation of one eye by the two colours. Thus if the intensities of the two colours are properly adjusted a red screen in front of one eye and a green screen in front of the other produce a yellow sensation (McDougall, 1901). This shows that the sensation of yellow is due to fusion behind the optic chiasma and that Hering's hypothesis cannot be true of retinal processes, although it may apply to cerebral interpretation of nerve impulses.

By such types of experiment it is hoped to be able to distinguish between the effect of different stimuli on common receptors in the eye and the effect of impulses from different receptors on a common interpreting mechanism (Roaf, 1928, 1932b). A practical application of binocular colour fusion might be to train individuals with defective colour vision to recognize colours by the use of a tinted colour filter in front of one eye.

Conclusion

In this paper an attempt has been made to point out the difference between normal and defective colour vision, to describe the means of detecting defects in colour vision, and the possibility of compensating for defects in colour vision by binocular fusion of colours. The usual type of defective colour vision is a failure to distinguish red, yellow, and green—that is, red-green confusion. There is no failure to distinguish blue from green. We can say that the normal person and the hypochromat distinguish blue from not-blue. The normal person distinguishes red from not-red. That which is neither red nor blue is called green. The hypochromat does not distinguish red from not-red, and anything which

is not blue he tends to call yellow, as that is the brightest part in the not-blue region of the spectrum.

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SOME CARDIOLOGICAL FALLACIES

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Even to-day unfounded beliefs regarding cardiovascular disease appear to be so widespread in the profession that we think it worth while to discuss some of the commonest of them in a short paper.

Fatty Heart

For a number of reasons, into which it would take too much time to enter, all obese people tend to become short of breath on exertion. On this account alone a diagnosis of "fatty heart" is often made. In post-mortem examinations of the obese—and not only of the obese—it is not unusual to find fat stored in and around the heart, but during life its presence can so rarely be proved to give rise to cardiac insufficiency that it is never wise to make the diagnosis. When definite symptoms of cardiac insufficiency are present in an obese patient another cause for them should be sought. The commonest by far is hyperpiesis, but there are, of course, others.

Physical Strain

The belief that a healthy heart can be injured by physical strain still unfortunately persists, in spite of the immense amount of work that has been done on the subject during the last thirty years. The truth of the matter has never been better put than by Lewis (1937) when he says: "It is reasonable to conclude that the burdens imposed by physiological acts upon the normal heart, however heavy these burdens may be, never injure the heart and fibres, never produce injurious dilatation, and never exhaust the heart's reserve." That the heart tends to be a little larger and rounder than the average in persons who habitually do hard physical work is not denied, but it is a normal and healthy reaction of the

muscle to exercise and no more to be accounted pathological than the hypertrophy of, say, their biceps.

There is a corollary to this: Every now and then one sees a patient in consultation whose heart, according to his medical attendant, has recently dilated enormously within the space of a few minutes or, at most, a few hours, usually, though not always, as a result of effort, and has returned to its normal size, either with rest or through his treatment, just as rapidly. Such acute dilatation of the heart, in our experience, never occurs. In this connexion it is interesting to observe that in a prolonged attack of "paroxysmal tachycardia," in which the heart beats continuously at a rate of over 200 a minute, appreciable enlargement—when it develops—is a matter of several days.

Screening the Heart

Even in a patient neither emphysematous nor obese it is impossible to estimate the transverse diameter of a heart with any accuracy by percussion—one has only to screen a normal chest in the lateral or oblique position to be persuaded of this. And, supposing it were, no one has ever suggested that the depth of a heart—a very important matter in cases of mitral stenosis, congenital morbus cordis, etc.—can be estimated by this means. Yet not only can both of these points be elucidated by screening, but a reasonably accurate estimation of the size of each individual chamber of the heart—surely a consummation earnestly to be desired—is as a rule easily made by this means.

Displacement of the apex beat to the left, where this cannot be attributed to pulmonary causes, though very suggestive of an enlarged heart, is not absolutely diagnostic. Scoliosis, even of so slight a degree as to be only recognizable by radiography, is a fruitful cause of such displacement. Again, a high diaphragm—the constant accompaniment of obesity—by causing the lie of the heart to become more transverse, tends to produce it. Through ignorance of these facts on the part of medical men, multitudes of healthy people are accounted victims of heart disease who, were their hearts screened by anyone conversant with the technique, could be safely assured as to the integrity of that organ.

Enlargement and Dilatation

The heart is generally believed to increase in size with age, as a normal process. There is no evidence for this. It may even diminish in size. When it is found to be enlarged in old age another cause should be looked for—for example, hyperpiesis, bundle-branch block, etc.

It is almost an article of faith in the profession that in all toxæmic states of any severity there is necessarily some dilatation of the heart through flabbiness of its musculature. It is forgotten that in cases of very advanced phthisis where there has been the severest degree of toxæmia for months or years the heart in life usually appears smaller than normal under the screen, and at necropsy is found to be wasted, perhaps, but not dilated. Again, in a recent radiological study of 119 hearts in pneumonia by Davies, Hodgson, and Whitby (1935), definite enlargement was witnessed in no more than three.

It is, we believe, practically only when auricular fibrillation, auricular flutter, or heart-block develops in the course of toxæmia that the heart dilates. Circulatory failure, when it occurs in toxæmia, is in our opinion almost always due to loss of contractility of the muscular coat of the peripheral arterioles and not to myocardial weakness.