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HUMAN COLOUR VISION: ITS BASIS AND CLINICAL SIGNIFICANCE

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Abstract

To understand abnormalities in colour vision a full knowledge of the properties of light, the eye and the brain, all of which are responsible for colour vision is necessary.

This paper presents a review of the physiology of colour vision and gives a detailed explanation of colour vision abnormalities.

Key words: *Trichromatic colour vision, dichromatic colour vision, monochromatic colour vision, deutan, protan, tritan.*

INTRODUCTION

In order to understand any abnormality in colour vision it is necessary to have a full knowledge of the properties of light, the eye and the brain all of which are responsible for colour perception.

This paper explains normal colour vision and gives a detailed explanation of colour vision abnormalities.

Light is the physical stimulus that initiates the visual process. It is a form of electromagnetic energy which is radiated (or emitted) from a source such as the sun or an electric light. The source releases particles of matter (photons) as an electromagnetic vibration. These particles vibrate at various different frequencies and the reciprocal of the frequencies occur is refered to as the 'wavelength'. The wavelengths of light that are visible to the human eye range from 380nm to 750nm (1 nanometer, nm = 1 millionth of a millimeter or 10^{-9} meter) and form the visual spectrum. Wavelengths shorter than 380nm (ie particles that vibrate too quickly to stimulate cone receptors and thus be seen by the eye) include ultraviolet rays and X-rays. Those wavelengths longer than 750 nm include infra-red rays and radiowaves. (These particles vibrate too slowly to stimulate cones and be seen by the human eye).

In the normal eye light within the visual spectrum excites rod and cone photoreceptors. These in turn excite colour coding cells in the retinal ganglion cell layer, the lateral geniculate nucleus (LGN) and the occipital cortex allowing the person to experience colour vision.

It is commonly believed that the colour of an object depends on the pigment and texture of its surface. This is untrue. The colour of an object depends on the following:

(1) the light source (eg. the sun or an electric light). As slightly different wavelength light is emitted from different sources the exact colour of an object will vary under different light sources.
(2) the dominant wavelength of light emitted from the source that bounces off the object. When light emitted from any source hits an

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object some wavelengths are absorbed by the object and others are reflected from (bounce off) it into the eye. One dominant wavelength is always reflected from an object.

Objects which absorb most of the light which strike them appear to be black. Those which reflect all the light appear to be white. Grey objects partially absorb the same proportion of light at each wavelength. Coloured objects selectively absorb various wavelengths and transmit others hence colour depends on the dominant wavelengths of light transmitted or reflected from the object. For example, a green chair absorbs all wavelengths of light except green light. Green wavelengths are reflected from the chair into the eye. The mixture of pigments which gives green is called a SUBTRACTIVE colour mixture. When white light hits the chair both the very short and the very long wavelengths are absorbed. By absorbing these wavelengths the pigment on the chair is virtually subtracting the short and long wavelengths from the white light. Thus the only wavelengths left to be reflected are the mid wavelengths so the chair appears green.

The other type of colour mixture is known as ADDITIVE colour mixture. Physical superimposition of different wavelengths of light on the same points on the retina is known as additive colour mixing. This affect can be achieved by projecting blue, red and green (ie the primary colours) from three projectors onto a white screen. By varying the brightness of each colour (using neutral density filters) all the different hues that can be perceived by the human eye may be produced.

The light source and the dominant wavelength of light which bounces off the object are the only objective factors affecting its colour. The rest of the colour interpretation depends on physical factors (the eye, the brain etc) and the individuals past perceptual experience.

The colour that the human brain perceives an object to be depends on the following[1]:

(1) the eye itself. The media must be normal allowing a clear passageway for wavelengths of light to reach the retina.

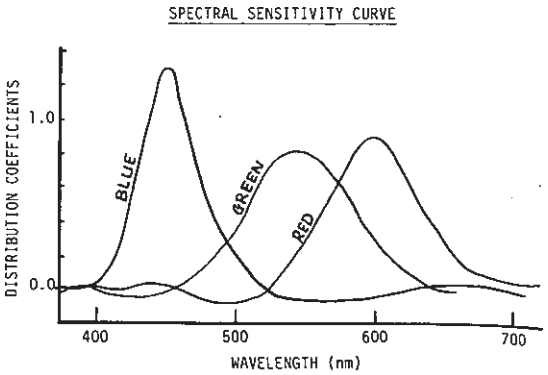

Figure 1: Spectral sensitivity curve.

(2) all retinal receptor cells and neural cells must be intact and functioning normally.
(3) neural pathways from the eye to the brain must be functioning normally.
(4) the individual must have past perceptual experience on which to base their interpretation of the stimulus.

Although the eye must be functioning normally in order to perceive colour, the eye itself does NOT recognise colour. Colour interpretation occurs in the brain. However, the eye must recognise the different wavelengths of light that enter it. Just as various wavelengths of sound stimulate the human ear differently enabling us to hear different noises, various wavelengths of light entering the eye stimulate cone photoreceptors differently. This ultimately enables us to see different colours.

When white light (ie a combination of all wavelengths of light from 380nm to 750nm) passes through a medium that is denser than air it is refracted or bent by that medium. As short wavelength (quickly vibrating) particles are bent more than slowly vibrating long wavelength particles white light passing through a prism is dispersed into the colours of the spectrum. This phenomenon was first reported by Newton[2].

The six colours (red, orange, yellow, green, blue and violet) that a prism breaks white light up into are known as SPECTRAL colours. All other colours such as pinks and browns are known as NON SPECTRAL colours.

ANATOMY

Cone receptor cells

When entering the eye light passes through the media and the outer retinal layers to the layer of rods and cones (ie the photoreceptors). Light is absorbed by the photoreceptors and this is the first stage in the perception of colour.

There are estimated to be 7 million cones in the human retina.[3] Light is absorbed by the pigment within each cone. Although little is known about cone pigments, using microspectrophotometry, researchers[4,5] have established that there are three varieties of photopigment. Each individual cone responds to all wavelengths of visual spectrum light however the response differs as each cone contains one of the three different photopigments. Thus the individual cone is maximally stimulated by (or maximally absorbs) either short wavelength 450nm blue light (ie 'blue' cones), mid wavelength 540nm green light ('green' cones) or long wavelength 590nm red light ('red' cones). Therefore a cone is named after the wavelength of light which the cone pigment it contains maximally absorbs.

The sensitivity of an individual cone to a particular wavelength of light is known as spectral sensitivity. The spectral sensitivity of the individual cone pigments to the various wavelengths of light is clearly shown on the spectral sensitivity or luminosity function curve (see Figure 1).[6] This curve represents the absorption function of the three cone pigments.

The difference in spectral sensitivities of the individual cone pigments is essential for normal colour perception. Without it all wavelengths of light would be absorbed equally by cones thus different wavelengths reflected from objects would produce only differences in brightness and the world would look like the picture on a black and white television set.

RETINAL GANGLION CELLS

As the cone receptors are not colour specific there must be neural mechanisms that can contrast the relative amounts of absorption of the three cone pigments in order to colour match. When light maximally stimulates red cones for example it causes these cones to send an excitatory input to the retinal ganglion cells. The same wavelength of light causes an inhibitory response to be sent from the green and blue cones to the retinal ganglion cells. In the retina the mechanism for colour coding consists of two groups of ganglion cells:

(1) spectrally opponent cells[7]
(2) spatially opponent cells[8]

Red and green are mutually exclusive colours. In daily life we see red-yellows, red-blues, greeny yellows and greeny blues however we never see red-greens or blue-yellows. This is because red and green are mutually exclusive colours. Blue and yellow are the only other pair of mutually exclusive colours. In the literature these colours are described as 'opponent' colours.[7,8]

(I) Spectrally opponent ganglion cells

After staring at a red spot on a white background for a number of minutes then looking away, the after image of the red spot is a green spot. This occurs because red and green are SPECTRALLY OPPONENT colours.

There are two sets of spectrally opponent colours in the human visual system. They are red — green and blue — yellow. Each individual spectrally opponent ganglion cells is thought to have both an excitatory and inhibitory field thus each individual ganglion cell is excited by stimulation of one type of cone photoreceptor and inhibited by another.

In 1975 De Valois and De Valois[7] reported that there were 4 types of spectrally opponent retinal ganglion cells. These cells are:

(1) red (long wavelength) excitatory — green (mid wavelength) inhibitory (R+ and G−)
(2) green excitatory — red inhibitory (G+ and R−)
(3) blue excitatory — yellow inhibitory (B+ and Y−)
(4) yellow excitatory — blue inhibitory (Y+ and B−)

Red — green cells would for example be excited by input from red cones that were stimulated by long wavelength light and inhibited by input from green cones that were stimulated by mid wavelength green light (see Figure 2).

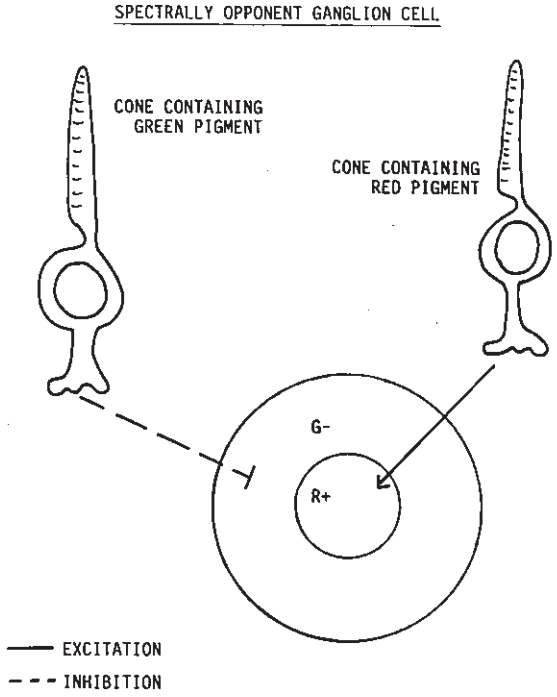

Figure 2: Spectrally opponent retinal ganglion cell.

(II) Spatially opponent retinal ganglion cells
These ganglion cells are also thought to have an excitatory and an inhibitory field. However, they respond to increases and decreases in surrounding light.

There are two types of spatially opponent retinal ganglion cells. They are:

(1) black excitatory — white inhibitory (Bl+ and Wh−)
(2) white excitatory — black inhibitory (Wh+ and Bl−)

These cells enable us to perceive objects clearly in the absence of colour stimulus; for example when watching black and white television, objects are perceived individually.

As the receptive field organization of the retinal ganglion cells is similar to that of the LGN neural coding of colours occurs in much the same way in the LGN.[6] Little is known about the mechanisms involved with colour vision in the visual cortex, however it is suspected on the basis of work done on the Rhesus monkey cortex[9] that neural coding occurs in much the same manner in the visual cortex as it does in the retinal ganglion cells.

NORMAL COLOUR VISION

Colour perception in individuals who use all three cone photopigments to colour match is called normal TRICHROMATIC colour vision (i.e. Young-Helmholtz trichromatic theory). Such individuals mix the three primary coloured lights in various proportions to perceive all the colours in the spectrum.

ABNORMAL COLOUR VISION

There are two major subgroups of colour vision abnormalities (excluding colour blindness). These are:

(1) anomalous trichromatic colour vision (deuteranomaly, protanomoly and tritanomaly).
(2) dichromatic colour vision (deuteranopia, protanopia and tritanopia).

(The prefix deut- means green, prot- means red and trit- means blue. The suffix -anomaly means there is an anomaly of pigment whereas -anopia refers to an absence of pigment).

(I) Anomalous trichromatic colour vision
These patients have cones containing the three photopigments, however they use abnormal proportions of these to colour match. This is a congenital condition which is associated with normal visual acuity and no other ocular problems.[10] Usually the patients are unaware of any colour vision defect.

These patients are labelled 'colour blind' and this term is extremely confusing. The patients do see colours, they just perceive them slightly differently. For example, if an orange block and a yellow block were placed on the table in front of an anomalous trichromat (protanope — see below) and the patient was asked to pick up the orange block, he would most probably select the yellow block stating that it was orange.

Most patients with anomalous trichromatic colour vision have red-green colour abnormalities. This abnormality occurs in 8% of the male

population.[10] Congenital blue-yellow colour abnormalities are extremely rare.[10]

Red-green 'colour blindness' can be further subdivided into:

(a) deuteranomalous 'colour blindness' (green deficiency)
(b) protanomalous 'colour blindness' (red deficiency)

Patients with blue-yellow defects are said to have a tritanomalous defect.

DEUTERANOMALOUS COLOUR VISION

These patients are labelled as 'green blind'. Despite the fact that they have a red-green deficiency, they CAN DISTINGUISH pure greens and pure reds. On colour vision testing these patients perform as though they are using more green to colour match. This can be explained in terms of a shift in the spectral sensitivity curve towards longer wavelengths (i.e. to the right).

As a result deuteranomalous patients have most trouble distinguishing colours in the yellow, orange and blue sections of the colour circle, that is the colours that are a mixture of red and green.

PROTANOMALOUS COLOUR VISION

Society calls these patients 'red blind'. Once again they have a red-green defect but have no problem distinguishing pure reds and pure greens. On colour vision testing, these patients perform as though they are using more red to colour match. This can be explained in terms of a shift in spectral sensitivity towards the shorter wavelengths. In performing the same test with colour blocks, if asked to select the YELLOW block the protanomalous subject would pick the ORANGE block. Protanomalous subjects have most problems distinguishing between yellows, oranges and blue colours. In dim lights these subjects may have problems distinguishing long wavelength reds.

TRITANOMALOUS COLOUR VISION

This category of colour vision defect is extremely rare.[10] They are called 'blue blind' patients yet they have no trouble with either pure blue or pure yellow. Their problems occur with greens and reds.

Aetiology of Anomalous Trichromatic Vision

The hypothesis which explains the aetiology of anomalous trichromatic colour vision suggests that these patients have a slight abnormality in the cone pigments causing them to maximally absorb slightly shorter (protanomaly) or slightly longer (deuteranomaly and tritanomaly) wavelength light.[10]

Dichromatic Colour Vision

This condition may be congenital or acquired. When it is congenital the patients usually have normal vision and no other ocular problems. Once again these patients are said to be colour blind but in fact they are not. Patients with dichromatic colour vision abnormalities can be subdivided into three groups:

(a) deuteranopia — green 'blind'
(b) protanopia — red 'blind'
(c) tritanopia — blue 'blind'

DEUTERANOPIA AND PROTANOPIA

The nomenclature of these abnormalities is more appropriate because the patients do have problems with red-green discrimination and they cannot distinguish green from reds yet show no confusion between yellow and blues. All colours appear as various saturations of blue and yellow or as grey or black. The deuteranopic patient frequently describes the green object as black and the protanopic patient may describe a red object as black. In either group red and green may be confused with yellow when tested on the anomaloscope.

TRITANOPIA

These patients cannot distinguish blues from yellows yet they show no confusion between reds and greens. Sometimes they may confuse blue with grey when tested on the anomaloscope.

Aetiology of Dichromatic Colour Vision

There are two hypotheses as to the cause of dichromatic colour vision. The first, and by far the most popular is that the patients have a

normal population of cones all of which contain only two of the three cone pigment[10,11] (i.e. either the red, green or blue cone pigment is missing). As a result the patient colour matches using only two rather than the three cone pigments. This hypothesis fails to explain acquired dichromatic colour vision resulting from disease or injury to the higher neural centres not affecting the cones. Some authors suggest acquired dichromatic colour vision is thought to be due to post receptoral loss of colour discrimination in one of the two pairs of opponent neural colour coding cells either in the ganglion cell layer, the LGN or the visual cortex.[12] Protanopic and deuteranopic individuals would have loss of red-green opponent neural colour coding cells and tritanopic individuals would have loss of blue- yellow opponent colour coding cells.

COLOUR BLINDNESS

Monochromatic Vision

Individuals with monochromatic vision are the only group of so called 'colour blind' patients who ARE actually colour blind. They see the world in black, white and shades of grey.

There are several subgroups within the monochromatic group. They include;

(1) Cone monochromatism:
- simple cone monochromats
- blue cone monochromats

(2) Rod monochromatism:
- partial rod monochromats
- total rod monochromats

CONE MONOCHROMATISM

(a) Simple cone monochromatism: Patients have good foveal vision, no nystagmus and no photophobia. Their only visual abnormality is their total colour blindness[10].

Simple cone monochromatism is thought to occur because of a loss of both red-green and blue-yellow opponent ganglion cells.

(b) Blue cone monochromatism: This is an extremely rare condition in which there is decreased visual acuity.[10] The decreased visual acuity occurs because foveal cones do not respond to short wavelength light (i.e. blue light. It is suggested by the author that this situation arises because the macular pigment, Xyanthyll, absorbs all the short wavelength light before it reaches the layer of foveal cones). Patients with blue cone monochromatism have nystagmus and a central scotoma. This condition is thought to occur due to a congenital absence of both green and red cone pigments.

ROD MONOCHROMATISM

(a) Partial rod monochromatism: This is a rare condition in which the patient presents with extreme photophobia, nystagmus (which improves with age), very decreased visual acuity for distance, slightly reduced visual acuity for near (N8, N12) and total colour blindness.[10]

Partial rod monochromatism is thought to be due to either a congenital decrease in the number of functioning cones in the retina.[13,14] Some authors suggest that there may be a number of rods in the foveal area.[15,16,17] The author suggests that foveal cones may actually contain rhodopsin (the rod pigment), in which case it would be interesting to speculate on the performance of receptors containing rhodopsin connected to ganglion cells in a one to one relationship.

(b) Total rod monochromatism: This is an extremely rare condition. Signs and symptoms are similar to but more severe than those for partial rod monochromatism. Patients with total rod monochromatism are believed to have no functioning cones in their retina.

DISCUSSION

An understanding of the basis of colour vision is vital if clinical colour vision tests are going to be applied correctly. Also, correct interpretation of the results of such tests cannot be made without a complete understanding of the basics.

It is also important that the clinician is able to explain any abnormality in colour vision to the patient or parent. For example the parent should understand why their child who has been called red-green colour blind can actually see reds and greens. Also parents should be aware of some employment opportunities which may be limited due to defective colour vision.

CONCLUSIONS

There are many fallacies commonly associated with colour vision abnormalities. The term 'colour blindness' itself is extremely misleading as only a very small percentage of the population (approximately 0.009%[10]) have no perception of colour. Up to 8% or 9% of the population are described as being 'colour blind' yet they do see all the colours of the visual spectrum. These patients would be better described as having an alteration in their perception of colour.

References

1. Boynton RM. Colour Vision. In: JA Kling & LA Riggs (eds), Woodworth & Schlosberg's experimental psychology. 3rd ed. New York: Holt, Rinehart and Winston, 1971: 315-368.
2. Haber RM & Hershenson. Perception of Colour in Psychology of Visual Perception 2nd ed. Holt, Rinehart and Winston, 1980, 5: 92-112.
3. Davson H. Retinal Structure and organisation. In Physiology of the Eye. 4th ed. London, Churchill Livingstone, 1980; 167-177.
4. Marks WB, Dobelle WH & MacNichol E.F. Visual pigments of single primate cones. L Science 1964; 143: 1181-1964.
5. Wald G. The receptors of human colour vision. Science 1964; 145: 1007.
6. Wright WD. Research on normal and defective colour vision. 1946. London, Kimpton.
7. De Valois RL & De Valois KK. Neural coding of colour. In: Carterette EC and Friedman H, (eds), Handbook of Perception. New York: Academic Press, 1975; Vol. 5; 117-116.
8. De Valois RL. Processing intensity and wavelength information. Invest Ophthalmol 1972; 11: 417-427.
9. Zeki SM. Colour coding in rhesus monkey prestriate cortex. Brain research 1973; 53: 422-427.
10. Krill AE. Congenital colour vision defects. In Hereditary Retinal and Choroidal Diseases, Vol 2, Clinical Characteristics. London: Harper and Rowe, 1972: 353-387.
11. Bowmaker JK. How is colour perceived: the visual pigments of human cones. Trans Ophthalmol Soc UK 1983; 103: 373-379.
12. Hurvich LM. Colour deficiencies: dichromats. 1981. In Colour Vision, Massachusetts: Sinauer Associates Inc, pp241-258.
13. Blackwell, HR & Blackwell OM. Rod and Cone Mechanisms in Typical and Atypical congenital achromatopsia. Vision Res 1961; 1: 62-107.
14. Walls GL & Heath GG. Typical total colour blindness reinterpreted. Acta Ophthalmol 1954; 32: 253-297.
15. Falls HF, Wolter JR & Alpern M. Typical total monochromacy. Arch Ophthalmol 1965; 74: 610.
16. Larsen H. Demonstration mikroskopischer. Praparate von einem monochromatischen auge. Klin. Mbl. Augenheilk 1921; 67: 301.
17. Harrison R, Hoefnagel D & Hayward JN. Congenital total colour blindness. A clinicopathological report. Arch. Ophthalmol 1960; 64: 685.