Colour vision deficiency in the medical profession

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SUMMARY
Colour is often used as a sign in medicine, yet there have been few studies into the effects of a colour vision deficiency (CVD) on doctors' medical skills. Using a literature search, the results indicate the prevalence of CVD in the medical profession and its effects on medical skills. For the congenital form among male doctors in the United Kingdom, the prevalence is shown to be probably about the same as for the population at large; i.e. 8%. However, the data is insufficient for any estimate to be made of the small number of female doctors and for the acquired forms of CVD. The effect on skills is also shown. Because of certain features of their work, general practitioners may have special problems. Thus, it is concluded that medical students and doctors should be screened for the deficiency and advised about it, and that there should be more study of the effects of CVD on decision-making in general practice and some specialties.

Keywords: congenital and acquired colour vision deficiency; prevalence; screening.

Introduction

COLOUR is often used as a sign in the practice of medicine. It gives information about surface and below surface phenomena. Many descriptive and diagnostic terms in common use indicate its value when used in this way: jaundice, cyanosis, erythema, melaena, and rubella are examples. It is also used in histology, biochemistry, and coding for many new technologies. Yet there have been few enquiries into the effects of a colour vision deficiency (CVD) on doctors' medical skills.

Awareness of the implications of CVD is often limited among those who suffer from it, and even more so among those who do not. But doctors have given accounts in published articles about the difficulties it causes in medicine, and a few research articles have been published on this subject. Conclusions drawn from personal accounts inevitably have a subjective element, but recent studies have provided better evidence on the types of difficulty encountered and the effects on performance.

Congenital CVD has a prevalence in the general population of 8% for men and 0.4% for women; therefore, its prevalence in the medical profession is likely to be high if there is no widespread self-selection out of the profession as a result of it. More people will have suffered from the acquired forms, but the prevalence is not known.

Prevocational screening for the deficiency and further testing for severity are practised for a number of occupations where certain standards of colour vision are required, but, as far as is known, medical students are screened at only one university in the United Kingdom (UK)[1,2] and only at a few in the rest of the world — screening for CVD is practised by all medical schools in Taiwan (personal communication: Hwei-Zu Wang, Kaohsiung Medical College, 1995).

This article gives background information on CVD, and then reviews the literature on the prevalence of the deficiencies in the medical profession and the effect on medical skills. A study of general practitioners (GPs), which is not widely known, gives evidence of problems particular to the profession.[3] The implications are discussed.

Method

Studies to be reviewed were identified from three sources. First, MEDLINE and BIDS ISI, from 1996 to August 1997; secondly, from reference lists of the articles so identified; thirdly, from personal recommendations. Studies, with two exceptions, were in English.

Studies for prevalence data were rejected if they used inadequate screening methods or did not give the male to female ratio.

Results

Background information

Many doctors may be unfamiliar with some of the modern concepts of colour vision; however, excellent reviews are to be found.[4-7] Salient features are that normal vision is trichromatic, meaning that all spectral hues can be matched by additive mixtures of three primary hues taken from the red, green, and blue parts of the spectrum. The three primary hues are detected by three types of cone cells containing pigments with photosensitives that overlap but peak in the green (long-wavelength), yellow-green (middle-wavelength), and violet (short-wavelength) parts of the spectrum. By comparing the rates of absorption of photons, the visual system is able to discriminate colours. It is varieties of hue, saturation (the similarity of a colour to white), and brightness that lead to an estimate that the human eye is able to distinguish, at equal luminance, 17 000 perceptible differences in colour.[8] Context, for example, adjoining colours (colour contrast and colour constancy), expectation,[9] and memory[10] are among factors that influence the colour actually perceived.

Widespread interest in CVD followed John Dalton's[11] description (1798) of his own deutan (middle-wave) deficiency,[12] but, for the preceding centuries, the deficiency has been described as 'an immensely well-kept secret'.[13]

The deficiency, its nature, terminology, inheritance, and prevalence

Congenital CVD is the result of an abnormality or absence of one or other of the three forms of photopigment in the retinal cones. The three types of deficiency are protan ('red' or long-wave), deutan ('green' or middle-wave), and tritan ('blue' or short-wave). (These words derive from the Greek *proto* meaning first, *deuteros* meaning second, and *tritos* meaning third.) The terms 'red', 'green', and 'blue' are widely used but lead to a misunderstanding because the failure to discriminate colour is of a much wider range in each case. Classifying by severity of the 'red-green' forms is much more useful for predicting the effect on performance. When tested in this way, approximately equal numbers grade as mild, moderate, or severe.

The acquired deficiencies are caused by ocular and intracranial

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Submitted: 14 July 1998; final acceptance: 7 December 1998.

Table 1. Classification and prevalence (in Caucasians) of congenital deficiencies.[7]

Type	Name	Prevalence (%) Male	Female	Hue discrimination
Dichromat (two cone photopigments only)	Protanopes ('red' deficiency)	1	0.01	Severely impaired
	Deuteranopes ('green' deficiency)	1	0.01	
Trichromat (three cone photopigments, one abnormal)	Protanomalous ('red' deficiency)	1	0.03	Continuous range from severe to mild[15]
	Deuteranomalous ('green' deficiency)	5	0.53	

The suffix, anomalous, denotes the possession of an abnormal photopigment. Protan and deutan are terms denoting both dichromats and trichromats. Tritan ('blue') prevalence = dichromats: 1 in 10 000; trichromats: unknown. Monochromats are extremely rare.

pathologies, and also by many drugs.[15] Diabetic retinopathy, hypertension, glaucoma, macular degeneration, and yellowing of the lens[16] owing to ageing are common causes. Tritan deficiencies are the most common. The prevalence of acquired deficiencies are not known but are probably greater than the congenital form, particularly in older people.[17]

Inheritance for the 'red-green' forms is X-chromosome linked, recessive, and are the result of deleted or tandem genes.[18] The tritan form is autosomal dominant.

Testing and screening

There are screening, classifying, and vocational tests for CVD.[7] The Ishihara is the most commonly used screening test but does not measure severity. Computer graphic tests are under development[19] and not widely used.

The school health service is responsible for screening children aged between seven and 12 years. The Department of Education and Science published information in 1971[20] stating that some form of test was administered in all but five health authorities in England and Wales, but the Department of Health, now responsible, does not now provide data on this subject. A working party report of the British Paediatric Association[21] recommends a grading test such as the City University Plates for those identified by the Ishihara test, but there is now a trend away from routine universal screening towards selective checks (personal communication: JC Read, Department of Health, 1996).

The deficiency as a disability

The identification of a bush that has holly berries on it, the observation by a pilot of the patterns of coloured lights at an airport, and learning about a person's health by their complexion are all tasks in which a person with CVD may fail. The skills involved have been traditionally classified into the discrimination, naming, and matching of colours. A more useful classification for many common activities derives from the study of the advantages of normal trichromat vision in the life of humans and animals.[22,23] These advantages are in object detection, object or pattern recognition, and in determining the significance of the colour of an object. The three tasks mentioned above each illustrate one of these.

Reds, oranges, yellows, browns, greens, purples, and violets are the colours that those with the most common 'red-green' deficiencies can fail to discriminate. Protans see reds as dark and tend to have more difficulties in practice than deutans. Failures are greater with dark and light colours. Dichromats fail to distinguish even some bright colours. Dalton,[11] for example, wrote that stockings spotted with blood or with dirt would scarcely be distinguished. Circumstances can also cause failures; for example, the absence of cues, poor illumination,[24] working at speed,[25] and viewing objects that subtend a narrow angle at the eye.[26]

There is an impressive body of evidence to show that some of those with CVD perform less well than others in some occupations.[27] Occupations have been classified according to the standards of colour vision required, and these have been applied, notably in the transport and electrical industries and the armed forces, but not always effectively.[28-30] For many occupations, little consensus exists about the standards to apply.[27]

Difficulties in everyday life have been reported; for example, dress, home decorating, cooking, and sport. In one study, 75% of 102 people with a deficiency reported some types of difficulty, and there was a correlation ($P<0.01$) between the percentage reporting difficulties and the severity of their deficiency.[29]

There are, however, some advantages to having a CVD. Colour can rival other visual perceptions, and this explains advantages in detecting camouflage and textures.[31] An advantage also seems to be given by the greater care that a person with CVD takes with certain tasks,[3,32] and because deutans see dark/light differences at twilight better than others.[33]

Awareness

People with a colour deficiency often lack awareness of its extent and effect.[34] A few react by denial.[35] In the study by Cole and Steward,[29] 5% of dichromats and 25% of anomalous trichomats were not aware of their deficiency. Students at art school can be unaware of it even when severe.[36] In one study, lack of awareness of the acquired form was shown by all 34 diabetics[37] with retinopathy; and, of eight people who made more than two-step errors on home testing of urine, none were aware of any difficulty. The tritan deficiency that occurs with ageing tends to go unnoticed owing to adaptive processes occurring slowly over time.[38]

The lack of awareness must in part be because of the effective use of cues, often unconsciously, to guess the colour of the object observed. For example, brightness can be a cue to red, the texture of a lawn to green, and the shape of a banana to yellow. In addition, it has been observed that individuals with a congenital deficiency learn from childhood to keep quiet about it[39] and so are deprived of the opportunity of fully understanding its implications. To understand these implications takes a sustained effort,[40] and few attempt this.

The difficulty of gaining awareness that colours are not being perceived can be shown by asking individuals with normal colour vision about their colour vision at night. Many do not realise that in dim light (scotopic conditions) they see only in black, grey, and white, except when viewing direct sources of light.[6] Experiments have shown how strong the tendency is of even those with normal colour vision to rely on assumptions about the colour of an object.[9] They can match, for example, the image of a brown lemon as yellow; and the poorer the colour information given, the stronger this tendency is. This helps to explain how those with a CVD can make assured but incorrect judgements of colour.

Prevalence of CVD in the medical profession and allied occupations

Table 2 gives the results of all known studies that include prevalence figures for doctors, dentists, and medical and dental students when reliable methods of testing for CVD have been used.

Two large studies,[1,2] not included in the table, give prevalence for all students, medical and non-medical, attending Queen's University of Belfast for two periods of five years (1949–1954 and 1954–1959). CVD testing was part of the health assessment for all entrants. The prevalence of CVD was 6.9% for 1966 men and 5.5% for 2405 men. The Ishihara test was used but with 'no special illumination'. If tungsten lighting was used, it could account, by missing some of the deutans, for the low figures.

All prevalence figures came close to, or a little above, the average 8% for men with inherited CVD in the general population, except for ophthalmologists[45] who had a prevalence of 6.7%, which suggests some self-selecting out for this specialty and possibly for the students of Belfast University.

Doctors with CVD

Personal accounts

Five doctors with CVD have published accounts of their experiences in medical practice. Haenel,[52] a physician, deliberately induced snowblindness by skiing in the Alps so that he could experience the effects of CVD, which are mainly those of a tritan deficiency.[53] He was most struck by the change in normal people's complexion. The other four doctors had congenital CVD: Ahlensteil,[54] a physician; Logan,[40] a physician; Spalding,[55] a GP; and Currier,[56] a neurologist. They reported a wide range of difficulties and many were common to all. Blushing, pallor, faint rashes, cyanosis, erythema, blood in body products, ophthalmoscopy, otoscopy, and microscopy could all cause difficulties in observation. Logan[40] and Spalding[55] recommended the screening of medical students. Logan commented that difficulties could be overcome by awareness, self-training, and effort.

Reports relating to individual doctors with CVD

Little,[57] an ophthalmologist, described the difficulties of a doctor studying under him, who failed to make normal progress. After first observing 'a black person's eyes', he found no difficulty with conditions of the 'eye ground' even in white people, but red appeared to him as bluish; for example, in kerato-conjuctivitis and the retinal reflex.

Other reports include Jeffries,[58] who referred to a physician with faulty colour vision 'who had trouble with the colour of throats, ulcers, gangrene, and some sores'. Wilson[59] had encountered five colour-blind physicians; one of whom confessed to him that 'red in the lips, cheeks, nose … inflammation, and the like look blue'. And Voke[28] quotes from a physician, surgeon, anaesthetist, endoscopist, ophthalmologist, and a nurse who reported difficulties that included identifying organs, the presence of pus, blood, cyanosis, jaundice, and facial discolouration.

A study of general practitioners with CVD

In a study of 40 (38 men and two women) doctors with congenital CVD, of whom 37 were GPs, who had responded to letters in medical journals, Spalding[3] used a questionnaire and a battery of colour vision tests to determine their range of difficulties in medical practice, and to relate these to the type and severity of their deficiency. A difficulty was defined as a problem, whether or not it was overcome.

The doctors reported a wide range of difficulties. The most common were:

- the widespread body colour changes of pallor, cyanosis, jaundice, and cherry red (25 doctors);
- rashes and erythema of skin (25 doctors);
- charts, slides, prints, and codes (24 doctors);
- test-strips for blood and urine (22 doctors);
- ophthalmoscopy (18 doctors);
- blood or bile in urine, faeces, sputum, or vomit (18 doctors); and
- otoscopy (14 doctors).

To overcome their difficulties, 17 doctors reported close observation, seven asked for the help of others, and four reported paying more attention to the history of the patient. Difficulties in microscopy, chemistry, clinical examination, and teaching methods as medical students were reported by 23 of the doctors. Seventeen doctors reported no or very few difficulties: this percentage corresponds fairly closely with that of Cole and Steward[29] for subjects with difficulties in everyday life. It is notable that five of those who reported no or very few difficulties had severe deficiencies, and this suggests that these doctors were unaware of their observational failures.

Doctors with mild deficiencies reported fewer difficulties compared with the combined results of those with moderate or severe deficiencies ($P<0.03$; $n = 40$). No significant difference was found between protans and deutans in the numbers of difficulties experienced.

The following comment, made by a GP (moderate deutan) of his experience as a medical student, is one of many:

> *My loss of confidence might have been more sympathetically handled had they [my teachers] been on the lookout for this problem.*

Studies of specialists and medical students with CVD

Tocantins *et al*[41] identified, with Ishihara plates, nine junior medical students with CVD in a class of 70, and studied their skills in chemistry and microscopy. These nine made many incorrect matches compared with normal controls, who made a few, when tested for matching phenolphthalein solutions, identifying Gram-negative and acid-fast bacteria, and eosinophils.

Olson[42] studied the help given by the use of coloured filters for microscopy to 26 students with CVD out of 400 studying histology. He reported that those with CVD had varying degrees of difficulty with the course material, and the majority of these found they were helped by a magenta filter.

Voke[28] studied endoscopists attending a conference. Colour was considered a major factor by all endoscopists. Using Ishihara plates, five were found to have a CVD and three admitted that it was a handicap. Testing skill, using a model stomach and circular coloured targets, showed some errors compared with two controls with normal vision.

Koningsberger *et al*[44] found 15 endoscopists with CVD out of 139 attending a meeting. Skill was assessed by use of a video excerpt of endoscopies of a variety of conditions. No effect of CVD was shown by these studies, but the F2 test was used, which is only for screening, therefore, severity was not established, which reduces the value of this study.

Poole *et al*[46] studied the skills of 132 histopathologists (15 with CVD) and 138 medical laboratory scientific officers (13 with CVD) by testing them with 20 projected transparencies of histopathology slides with various staining techniques. The colour vision tests used were Ishihara, City University, and Farnsworth–Munsell 100-line. Both groups with CVD were sig-

Table 2. Summary of studies giving evidence of prevalence of congenital CVD in the medical and dental professions.

Authors, date and country of study	Occupation of subjects	Method of selection	Colour vision test	Total number of male subjects	Percentage (number) with CCVD
Tocantins et al[41] 1933 (USA)	Junior medical students	Not known	Ishihara	70	12.8 (9)
Olson[42] 1971 (UK)	Histology students Medical: 240 Dental: 100 Science: 60	All students	Ishihara	320	8.7 (26) (1 not R/G)
Rigby et al[43] 1991 (UK)	Histopathologists: 28 Cytologists: 2	Volunteers	Farnsworth-Munsell 100-line	23	10.3 (3)
Koninsberger et al[44] 1994 (Holland)	Gastroenterologists	All male consultant and resident pathologists and physicians of internal medicine with at least one year's experience in gastroenterology and colonoscopy visiting a conference	Modified F2	139	11.0 (15)
Arden et al[45] 1995 (UK)	Ophthalmologists	96.1% of all UK ophthalmologists	Computer graphic	810	6.7 (54) (1 tritan)
Poole et al[46] 1997 (UK)	Histopathologists and medical laboratory scientific officers	Volunteers from 45 hospitals who had spent more than one year in histopathology	1. Ishihara 2. City University 3. Farnsworth-Munsell 100-hue	132	13.0 (15)
McMaugh[47] 1977 (Australia)	Dentists, dental students, and ceramic technicians	Students: 86 Dentists: 20 Technicians: 88 (Sample of 88 tested for CVD)	Ishihara	88	7.9 (7)
Barna et al[48] 1981 (USA)	Dentists	Not given	American optical company charts	50	14.0 (7)
Moser *et al*[49] 1983 (USA)	Dentists	Participants in a health assessment programme	1. Dvorine 2. Ishihara	630	10.0 (66)
Davison *et al*[50] 1990 (USA)	Dental students/faculty and staff of university	Not given	SPP-C-Part one	635	7.8 (18)
Wasson *et al*[51] 1992 (USA)	Dental students, dentists, dental hygienists, dental assistants, and university staff	'Recruited'	SPP-C-Part one	75	9.3 (7)

Total number of female participants = 599. Number of females with CVD = 1.

nificantly poorer at identifying slides than normal sighted people. When subjects with CVD were categorized as severe, moderate, or mild, there was a significant trend towards those with severe deficiency making more mistakes (*P*<0.001).

Doctors' awareness of their CVD

In their study of 30 histopathologists, Rigby *et al*[43] reported that two of the three found to have a CVD were unaware of this. Spalding[3] reported that, of 40 doctors known to have CVD, in 24 it was severe but 19 of these did not know this. Three of the 40 doctors specified that it was mild, three did not know of the deficiency until after qualification, and one of these, a moderate deutan, did not know until six years after qualification, and later he reported many difficulties as a physician.

Colorimetric tests for urine and blood

There have been many studies[60-70] showing that diabetics with retinopathy or congenital CVD make errors in reading certain colorimetric tests. For diabetics with retinopathy, the tests were Diastix, Clinistix, Clinitest, Haemoglucotest (Bayer Diagnostics), and BM Glycemie (Boehringer Mannheim). For diabetics with congenital CVD, the tests were Glucostix, Diastix, and Clinitest (Bayer Diagnostics). Greater than one-step errors were reported for both groups. One study used colorimetric methods[61] to show that the chromaticity of the charts used in these tests was in the range that would be expected to cause errors for quantitative testing for glucose but not for detection of its presence. Only one study[71] out of 11 did not show more errors by diabetics with retinopathy than those without it.

There has been one study[72] of laboratory technicians with congenital CVD using a variety of colorimetric tests, but Spalding,[3] in his study of 40 doctors with inherited CVD, found that 22 reported difficulties with these tests. Only one reported the use of a reflectance meter.

Rectal bleeding and haematemesis

A proctologist[73] reported one case of a patient with CVD who had failed to recognize rectal bleeding, which evoked subsequent correspondence in the *American Journal of Ophthalmology*.[74,75] Surgeons[76] also reported three other cases where treatment had been delayed for the same reason. No mention was made of the possibility that doctors might have the same difficulty with the stools of their patients, but one deutan GP reported, 'I once diagnosed a haematemesis as bile. The patient was lucky to survive.'[3] Eighteen of the 40 doctors (37 GPs) in this study reported difficulties with detecting blood or bile in urine, faeces, sputum, or vomit.

Discussion

The evidence presented here suggests that the prevalence of congenital CVD in the UK medical profession is about the same as for the population at large. It follows that, in the year 1994, there were likely to have been approximately 5800 doctors in general practice in the UK with this deficiency,[77-80] and more would have had an acquired deficiency. The evidence[3,34] also suggests that many of these doctors will not know of its severity in their own case and a few will not know that they have any deficiency at all.

Published studies described here have shown that, in some common medical procedures, individuals with CVD, particularly if it is moderate or severe, perform less well than those with normal colour vision. This has been shown for patients using colorimetric tests for glucose in urine and in blood,[61-71] both with congenital deficiencies and those owing to diabetic retinopathy, and it clearly is likely also to be true for doctors with these conditions. Medical students working in laboratories[41] have been shown to perform less well with certain colorimetric tests and in microscopy. Recently, rather strong evidence has shown that histopathologists[46] perform less well in examining slides using a wide range of staining techniques.

In the direct observations for physical signs in patients, there have been a number of accounts by doctors with CVDs, both in their present practice and retrospectively as students.[3,28,40,41,52-57] However, there have been no published studies providing objective evidence of the effect on performance for this type of observation, apart from two of endoscopy, where the results are not conclusive.[28,44] It cannot be assumed that performance is not affected. The observations that are reported to cause difficulties, for example, pallor, erythema, cyanosis, and body products, comprise the range of colours that are known to cause failures of discrimination for those with CVD.

However, doctors have many leads in the diagnosis and management of patients that do not involve colour. For outpatients, the history of the patient has been shown to be the most valuable.[81] It is likely to be this, together with the greater care that is taken by those with CVD and the uncommon occurrence of serious error, that account for the fact that doctors with CVD are generally considered by themselves and others to perform as well as other doctors with normal colour vision.[3] However, diagnosis can be a complex process and is often, in part, intuitive, so that it can be difficult to single out the part played by one type of observation. But from what is known of colour vision and from doctors' comments, some features of the consultation can be predicted as likely, on occasion, to cause errors by those with CVD. In some cases there is the so-called pivotal observation,[82,83] a single sign that it is essential to observe for the correct course of action to be taken. There is the unexpected and the unfamiliar case presentation that can make special demands on the doctor as observer — there is the scanning of an area for the detection of small features, for example, bacilli or a rash;[22,23] and there are conditions of work, for example, at speed,[24] alone, in poor illumination[25] when visiting, and the patient who cannot give a history.[3] Most of these features are particularly likely to affect the GP, the underlying reason being the difficulty in controlling the conditions of observation.

There is much evidence that doctors with normal colour vision often disagree about physical signs,[87,88] but what is at issue in the present case is disagreements that may be, at least in part, resolvable or avoidable. For example, in the detection of blood in body products, a sign that is missed can have serious consequences; a doctor with CVD might be helped by viewing with a coloured filter,[86] if carefully advised on the method, and then using an adapted peroxide test to confirm its presence. In addition, a reflectance meter could be used for some colorimetric tests.

Clearly, where error is involved, risk has to be balanced against hazard.[87] In a valuable paper, Cole[27] points out that, in relation to non-medical occupations, in analysis of the causes of accidents it is difficult to identify risk factors because they have so many causes and are relatively infrequent. He further comments that, if we insisted on incontrovertible accident data before taking action to protect society from unnecessary risk, there would be very little action taken.

The evidence for action in the medical profession, such as the screening of all medical students for CVD, derives from many sources that need different types of assessment. The finding that doctors commonly do not know the severity of their deficiency is, for example, very significant,[3] and what can hardly be doubted is that doctors who are aware of their limitations are more likely to make corrections.

Conclusion

The evidence points to the need for screening for CVD in medical students and doctors. This would allow testing for severity, counselling, and an informed choice of career. Doctors with known CVD could seek testing for severity, and, because of the wide range of specialties, the question of non-acceptance of applicants to medical school need hardly arise. Screening has implications for the work of occupational health physicians and teachers and examiners in medicine.

There is a case for a more detailed examination of the effects of CVD on decision-making in general practice, but also in a number of specialties; for example, ophthalmology, ENT, paediatrics, gastroenterology, and pathology.

References

1. Johnston W, Cheeseman EA, Merrett JD. Observations on routine medical examinations of university entrants in Northern Ireland. *Br J Prev Soc Med* 1957; **11:** 152-161.
2. Johnston W, Merrett JD. Further observations on routine medical examinations of university entrants in Northern Ireland. *Br J Prev Soc Med* 1962; **16:** 76-83.
3. Spalding JAB. Doctors with inherited colour vision deficiency: their difficulties in clinical work. In: Cavonius CR (ed). *Colour vision deficiencies XII. Proceedings of the International Research Group for Colour Vision Deficiency 1995.* Dordrecht, Boston, London: Kluwer Academic Publishers, 1997.
4. Mollon JD. Colour vision. *Ann Rev Psychol* 1982; **33:** 41-85.
5. Wandell BA. *The foundations of vision.* Sunderland: Sinauer Associates, 1995.
6. Hubel DH. *Eye, brain and vision.* New York: Scientific American Library 1995.
7. Birch J. *The diagnosis of defective colour vision.* Oxford: Oxford University Press, 1993.
8. MacAdam DL. Visual sensitivities to colour differences in daylight. *J Opt Soc Am* 1942; **32:** 247-274.
9. Bruner JS, Postman L, Rodrighes J. Expectations and the perception of colour. *Am J Psych* 1951; **64:** 216-227.
10. Duncker K. The influence of past experience on perceptual properties. *Am J Psychol* 1939; **52:** 255-265.
11. Dalton J. Extraordinary facts relating to the vision of colours: with observations. *Memoirs of the Literary and Philosophical Society of Manchester* 1798; **5:** 28-45.
12. Mollon D, Kanwaljit S, Hunt D, Hunt DM. Dalton's colour blindness: an essay in molecular biography. In: Dickinson C, Murray I, Carden D (eds). *John Dalton's colour vision legacy. Selected proceedings of the International Conference 1995.* Bristol: Taylor and Francis, 1995.
13. Bradley N. Colour blindness: notes on its development and clinical significance. *Int J Psychoanal* 1970; **51:** 59-70.
14. Birch J. Classification of anomalous trichromatism with the Nagel anomaloscope. In: Drum B (ed). *Colour vision deficiencies XI. Proceedings of the International Research Group on Colour Vision Deficiency 1991.* Dordrecht, Boston, London: Kluwer Academic Publishers, 1993.
15. Ruddock KH. *Acquired deficiencies of human colour vision. Baillière's Clinical Neurology.* London: Baillière Tindall, 1993.
16. Lakowski R. Is deterioration of colour discrimination with age due to lens or retinal changes? *Die Farbe* 1962; **11:** 69-86.
17. Francois J, Verriest G. Nouvelle observations de déficiences acquises de la discrimination chromatique. *Annales D'Oculistique* 1968; **201:** 1097-1114.
18. Nathans J, Piantanida T, Eddy RL, Shows TB. Molecular genetics of inherited variations in human colour vision. *Science* 1986; **232:** 203-232.
19. Arden GB, Gundez K, Perry S. Colour vision testing with a computer graphics system. *Clin Vis Sci* 1998; **2:** 203-220.
20. Department of Education and Science. *Health of schoolchildren 1971-72. Report of the Chief Medical Officer.* London: HMSO, 1974a.
21. British Paediatric Association. *Report of the working party on health needs of school age children.* London: British Paediatric Association, 1995.
22. Jacobs GH. *Comparative colour vision.* New York: Academic Press, 1981.
23. Mollon JD. 'Tho' she kneel'd in that place where they grew.' *J Exp Biol* 1989; **146:** 21-38.
24. Kudo H, Obaro F, Ohta Y. Panel D15 test in colour vision deficiencies at reduced illumination levels. In: Drum B (ed). *Colour vision deficiencies XIII. Proceedings of the International Research Group on Colour Vision Deficiencies 1995.* Dordrecht, Boston, London: Kluwer Academic Publishers 1997.
25. Cole BL, MacDonald WA. Defective colour vision can impede information acquisition from redundantly colour-coded video displays. *Ophthalm Phsyiol Opt* 1988; **8:** 198-210.
26. Smith VC, Pokorny J. Large field trichromacy in protanopes and duteranopes. *J Opt Soc Am* 1977; **67:** 213-220.
27. Cole BL. Does defective colour vision really matter? In: Drum B (ed). *Colour vision deficiencies XI. Proceedings of the International Research Group on Colour Vision Deficiencies 1991.* Dordrecht, Boston, London: Kluwer Academic Publishers 1993.
28. Voke J. *Colour vision testing in specific industries and professions.* London: Keller, 1980.
29. Cole BL, Steward JM. Some (but a few) colour defectives have no difficulty with color. In: Dickinson C, Murray I, Carden D (eds). *John Dalton's colour vision legacy. Selected proceedings of the International Conference 1991.* Bristol: Taylor and Francis, 1997.
30. Vingrys AJ, Cole BC. Are standards of colour vision in the transport industry justified? *Ophthal Physiol Opt* 1988; **8:** 1257-1274.
31. Morgan MJ, Adam A, Mollon JD. Dichromats detect colour-camouflaged objects that are not detected by trichomats. *Proc R Soc London B* 1992; **248:** 291-295.
32. Cole BL, Maddocks JD. Defective colour vision is a risk factor in driving. In: Cavonius CR (ed). *Colour vision deficiencies XII. Proceedings of the International Research Group on Colour Vision Deficiencies 1995.* Dordrecht, Boston, London: Kluwer Academic Publishers 1997.
33. Adam A. A further query on colourblindness and natural selection. *Soc Biol* 1969; **16:** 197-202.
34. Kalmus K. *Diagnosis and genetics of defective colour vision.* Oxford: Pergamon Press, 1965.
35. Pickford RW, Cobb SR. Personality and colour vision disorder. In: Streiff EB, Verriest G (eds). *Modern problems in ophthalmology* 1970. Basel: Karger, 1974.
36. Pickford RW. Colour defective art students in four art schools. *Br J Physiol Opt* 1972; **24:** 102-114.
37. Bresnick GH, Crawford J, Groo A. Urine testing inaccuracies among diabetic patients with colour vision deficiencies. In: Verriest G (ed). *Colour vision deficiencies VII. Proceedings of the International Research Group on Colour Vision Deficiencies 1983.* Dordrecht, Boston, London: Kluwer Academic Publishers 1984.
38. Werner JS. *Visual problems of the retina during ageing: compensation mechanisms and colour constancy across the life span. Progress in retinal and eye research.* London: Elsevier Science, 1996.
39. Bacon L. Colour vision – an educational handicap. *Medical Officer* 1971; **16:** 199-209.
40. Logan JS. The disability in so-called red-green blindness. An account based on many years of self-observation. *Ulster Med J* 1977; **46:** 41-45.
41. Tocantins LM, Jones JW. Defective colour vision and its handicaps in medicine. *Am J Science* 1993; **293:** 243-249.
42. Olson IA. The use of colour filters by students with congenital colour defects in the learning of histology. *Medical and Biological Illustrations* 1997; **21:** 52-53.
43. Rigby HS, Warren BF, Diamond J, *et al*. Colour perception in pathologists: the Farnsworth-Mansell 100-hue test. *J Clin Path* 1991; **44:** 745-748.
44. Koningsberger JC, van Norden D, van Niel JCG, Dekker W. Does colour vision deficiency in the endoscopist influence the accuracy of endoscopic diagnosis? An anonymous study with Dutch gastrointestinal endoscopists. *Endoscopy* 1994; **26:** 549-553.
45. Arden GD, Hall MJ. Does occupational exposure to argon laser radiation decrease colour contrast sensitivity in UK ophthalmologists? *Eye* 1995; **9:** 686-696.
46. Poole CJM, Hill DJ, Christie JL, Birch J. Deficient colour vision and interpretation of histopathology slides: cross sectional study. *BMJ* 1997; **315:** 1279-1281.
47. McMaugh DR. A comparison analysis of the colour matching ability of dentists, dental students and ceramic technicians. *Aus Dent J* 1977; **22:** 165-167.
48. Barna GJ, Taylor JW, King GE, Pelleu GB. The influence of selected light intensities on colour perception within a colour range of natural teeth. *J Prosth Dent* 1981; **46(4):** 450-453.
49. Moser J, Wayne WT, Nageway CA, Ayer WA. Colour vision in dentistry: a survey. *J Am Dent Assoc* 1985; **110:** 509-510.
50. Davison SP, Mysilinski NR. Shade selection by colour vision defective dental personnel. *J Prosth Dent* 1990; **63:** 97-101.
51. Wasson W, Schuman N. Colour vision and dentistry. *Quintessence International* 1992; **5:** 249-352.

52. Best F, Haenel H. Rotgrün blindheit nach schneeblendung. Kin Monatsbl Augenheilkd. *Beilagen* 1880; **45:** 88-105.
53. Hansen E. The disturbance of colour vision after sunbathing. In: Verriest G (ed). *Colour vision deficiencies V. Proceedings of the International Research Group on Colour Vision Deficiencies 1979.* Bristol: Adam Hilger, 1980.
54. Ahlenstiel H. *Red-green blindness as a personal experience*. London: Kodak Research Library, 1951.
55. Spalding JAB. The doctor with an inherited defect of colour vision: the effect on clinical skills. *Br J Gen Pract* 1993; **43:** 32-33.
56. Currier JD. A two and a half colour rainbow. *Arch Neurol* 1994; **51:** 1090-1092.
57. Little WS. Experience of a red-blind physician with one ophthalmoscope. Practical advantage of colour-blindness with a case. *Arch Ophthalm* 1881; **10:** 20-22.
58. Jeffries BJ. *Colour blindness – its dangers and detection.* Cambridge, USA: Riverside Press, 1983.
59. Wilson G. *Research on colour blindness with a supplement*. Edinburgh: Southerland and Knox, 1855.
60. Smith VC, Burns SA, Pokorny J. Colorimetric evaluation of urine-sugar tests by diabetic patients. In: Verriest G (ed). *Colour vision deficiency VI. Proceedings of the International Research Group on Colour Vision Deficiencies 1981*. Dordrecht, Boston, London: Kluwer Academic Publishers 1982.
61. Thompson DG, Howarth F, Taylor H, *et al*. Defective colour vision in diabetes: a hazard to management. *BMJ* 1979; **1:** 859-860.
62. Muntoni S, Serra A, Mascia C, Songini M. Dyschromatopsia in diabetes mellitus and its relationship to metabolic control. *Diabetic Care* 1982; **5:** 375-378.
63. Lombaril P, Cathelineau G, Gervais P, Thibult N. Abnormal colour vision and reliable self-monitoring of blood glucose. *Diabetic Care* 1984; **7:** 318-320.
64. Aspinal PA, Hill AR, Cameron D. An evaluation of the Ames Clinitest. In: Verriest G (ed). *Colour vision deficiency V. Proceedings of the International Research Group on Colour Vision Deficiencies 1979.* Bristol: Adam Hilger, 1980.
65. Green FD, Chafur IM, Allan D, *et al.* Colour vision in diabetics. *Br J Ophthalm* 1985; **69:** 533-536.
66. Zisman F, Adams AJ. Influence of colour deficiency on home blood glucose monitoring. In: Verriest G (ed). *Colour vision deficiency VIII. Proceedings of the International Research Group on Colour Vision Deficiencies 1985.* Dordrecht, Boston, London: Kluwer Academic Publishers 1987.
67. Allwood MC, Tyler R. Colour vision and blood glucose self-monitoring in diabetics. *Practical Diabetes* 1988; **5:** 110-112.
68. Leid J, Leid V, Messabie P. The influence of diabetic dischromatopsia on the interpretation of self-monitoring coloured reagent strips. In: Verriest G (ed). *Colour vision deficiency VIII. Proceedings of the International Research Group on Colour Vision Deficiencies 1985.* Dordrecht, Boston, London: Kluwer Academic Publishers 1987.
69. Fletcher R. Colour perception warning for self-testing diabetics. In: Moreland JD, Serr A (eds). *Colour vision deficiency X. Proceedings of the International Research Group on Colour Vision Deficiencies 1989.* Dordrecht, Boston, London: Kluwer Academic Publishers 1991.
70. Mantyjarvi M. Screening of diabetics who read incorrectly colour-dependent glucose test strips. In: Drum B (ed). *Colour vision deficiency XI. Proceedings of the International Research Group on Colour Vision Deficiencies 1991*. Dordrecht, Boston, London: Kluwer Academic Publishers 1993.
71. Graham K, Kesson CM, Kennedy HB, Ireland JT. The relevance of colour vision and diabetic retinopathy to self-monitoring of blood glucose. *BMJ* 1980; **281:** 971-973.
72. Fetter MC. Colorimetric tests read by colour-blind people. *Am J Technol* 1963; **29:** 349-355.
73. Pearl SS, Elizabeth NJ. Colour blindness and bowel bleeding. [Letter.] *JAMA* 1978; **12:** 1132.
74. Pokorny J, Smith VC. Colour blindness and bowel bleeding. (Reply.) [Letter.] *Am J Ophthalm* 1978; **85(5):** 723.
75. Porter J. Letter. *Am J Ophthalm* 1978; **86(4):** 179.
76. Stiff GJ, Harray PN, Foster ME. Dischromatopsia (number 97) and rectal bleeding. *BMJ* 1996; **313:** 594.
77. Department of Health. *Health and personal statistics for England*. London: HMSO, 1996.
78. National Health Service in Scotland. *Manpower distribution.* London: Information and Statistics Division, 1995.
79. Welsh Office. Health Statistics Analysis Unit, 1996.
80. Northern Ireland Department of Health and Social Services. *Annual Statistic Report.* Centre for Information Analysis, 1996.
81. Hampton JR, Harrison MJG, Mitchell JRA, *et al.* Relative contribution of history-taking, physical examination, and laboratory investigation to diagnosis and management of medical outpatients. *BMJ* 1975; **2:** 486-489.
82. Bradley GW. *Disease diagnosis and decision*. Chichester: John Wiley and Sons, 1986.
83. Eddy DM, Clanton CH. The art of diagnosis. *N Engl J Med* 1982; **21:** 1263-1267.
84. Koran LM. The reliability of clinical methods, data and judgement. *N Engl J Med* 1975; **293:** 642-646, 695-701.
85. Feinstein AR. A bibliography of publication of observer variability. *J Chron Dis* 1985; **38(8):** 619-632.
86. Fletcher R. Experiences with assisting Daltonics. In: Verriest G (ed). *Colour vision deficiency VI. Proceedings of the International Research Group on Colour Vision Deficiencies 1981.* The Hague: Dr W Junk, 1982.
87. Calman KC, Royston HD. Risk, language and dialects. *BMJ* 1997; **315:** 939-942.

Acknowledgements
I would like to thank Professor Geoffrey Arden for scientific advice and for help with the manuscript, Dr John Campbell of UMDS for suggesting this article, Fraz Asif Mir for help with the literature search, and the Special Trustees at Guy's Hospital for his research studentship funding.

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