Test for colour vision – and Save Sight.

"Colour tests have been with us for a long time- but new techniques promise a revolution in the early detection of serious disease."

About 1 in 12 males has an inherited defect of colour vision. While this is important to a boy who wants to be a police marksman or a seaman, in most situations the defect does not matter very much and no-one tests routinely for such problems. Older people, with severe blinding eye diseases, also suffer from colour vision defects.—but again we tend never to test for them. But there are very good theoretical reasons why colour vision tests should be of use in detecting and managing treatment in cases of acquired eye disease, and as a result of developments in computer game technology, it is now possible to carry out these tests simply, effectively, quickly and economically.

The basis of seeing in colour is of course the three different types of cones in our eyes. But when the cone pigments were first analysed, there was a great surprise. The three pigments are not equally spaced over the spectrum. The "blue" pigment (short wave length) is a very good blue, but the two remaining pigments both absorb best in the green part of the spectrum: there is no long wavelength pigment which allows us to see red. Although the quality of redness seems so unique, the sensation is due to layer after layer of neural processing, that begins in the retina and continues into the higher visual brain centres. It is not surprising that anything that interferes with the computation should affect colour vision. Furthermore, 90% or more of the fibres of the optic nerve are concerned with colour processing. They belong to the 'midget' or parvocellular system. If we only test vision with black and white achromatic stimuli (as with an optotype) most of the time we are only testing the large ganglion cells of the 'magnocellular' system and exclude most of the visual neurones! The black and white system is of great importance: it is essential for perceiving motion, for detecting delicate shading, for stereopsis and depth perception, and can handle all detail up to a surprisingly high level, equivalent to a visual acuity of > 0.5. The "private pathways" by which the foveal cones send coded colour information can also handle ultra-fine detail – but are not often needed. In fact, the main tasks the magnocellular system cannot handle are to help thread a needle and detect when fruit is ripe. But the *quality* of our visual sensation is entirely dependent on the colour-coded midget system. The greatest obstacle to using colour tests in the clinic is the lack of good, rapid, sensitive tests for the blue yellow colours, because with these we could exclude the majority of congenital diseases and concentrate on acquired blinding conditions. This is because blue vision suffers earliest in almost all retinal disease, possibly because the blue cones are very fragile, but also because they are very few in number. A single blue cone has a large receptive field. If it, or its ganglion cell, dies there is a significant "hole" for blue in the visual field, and proper tests of colour vision can demonstrate this easily. However until recently all tests of blue vision were relatively non-quantitative, and slow. The situation has been revolutionised by using computers to present coloured images to the patient, using the techniques developed for use in computer games.

Colour- the basics

The test in practice looks deceptively simple: a coloured letter (optotype) is flashed on the screen. The letter is visible only because it has a different hue to the background on which it appears. The difference in hue can be made more or less obvious, by moving the hue along a predetermined line in colour space. One of these lines is the red –green axis that is important for congenital colour deficiency and the line is a protanopic colour confusion line. To persons who lack "red" cones, all colours on this line are confusible. Another colour confusion line, at right angles to the first, is a blue-yellow or tritan colour confusion line. Persons who lack "blue" cones confuse all colours on this line. The two lines form the minor and major diameters of an ellipse, known as the MacAdam ellipse, after the colour scientist who first made the measurements. All the colours inside a MacAdam ellipse seem the same to the observer. In eye disease, one of the axes increases in length. In congenital loss of colour vision, the minor (red green) axis increases in length, and in acquired retinal disease it is common to find a large and disproportionate elongation of the major blue-yellow axis. It is simple for a computer to make the background and optotype a known distance apart along a colour confusion line. If the distance is great, the colour difference is obvious. If it is reduced, the colours become less visible. The minimum displacement at which the letter can be recognised gives the colour threshold. Modern psychophysical methods allow thresholds to be determined very quickly- one can find the protan and tritan thresholds in about the time it takes to measure visual acuity.

Take the test

When one investigates more deeply, there are tricks that are employed to improve the clinical value of such a test. There has to be close control over the brightness and colour properties of the system, or else, it might be possible for a patient to use any artefactual brightness to make the distinction. Since we all have different amounts of yellow pigment in our lenses (and in the macula), such variations are bound to occur, but they can be masked by having an ever-changing pattern of "luminance noise" on the screen, to mask any residual brightness clue the coloured letters might give . The pattern must flash briefly, for then; the patient cannot make eye movements to scan the letter. If the optotype is chosen correctly and is equivalent to the top line on an ordinary test type, small defects- perhaps the loss of single blue cones - will make it impossible to recognise the letter of the optotype, even if the colour is clearly visible. When the optotype is made very large, of course more than just the fovea is tested, and this is important, because diseases like diabetic retinopathy need not begin in the foveal retina. In other forms of the same test, the optotype is discarded, and the patient is presented with a fixation point, while stimuli are confined to the retinal periphery. The arcs of the retina where glaucomatous field losses occur can thus be selectively probed. Normally when an image is presented to the retinal periphery it quickly fades with time (the Troxler effect). With the flashed images produced by a computer this does not happen.

To take such a test is simplicity itself. The operator chooses the type of disease to be investigated, and the computer does the rest. It prompts the

operator for patient's details, and shows the letters to the patient. Only approximate refractive correction is required. The patient names the letters (or an illiterate E or a symbol can be used), and the operator notes whether the response is correct. The computer homes in on the threshold, and when preset criteria are satisfied, it terminates the test. It displays the threshold, and relates it to normal age-corrected values. The print–out gives a coloured indication: blue if the threshold is normal, green, yellow, orange or red for increasing losses.

What results can be expected? In the 3 major blinding conditions can be made very early. In Glaucoma blue scotomas develop before black-and-white ones. The colour contrast test is much less 'noisy' and easier for the patient than automated perimetry, because instead of determining thresholds at many locations, one can concentrate on just a few. The results are as good as those obtained by imaging scanning laser ophthalmoscopes that measure changes to the optic disc. In Diabetes, colour vision begins to be lost before there is any visible change in the fundus. By the time that even a few microaneurysms are visible, the thresholds are significantly elevated. To conduct such a rapid screening test is far preferable to dilating the pupils, waiting, and conducting a detailed search with an ophthalmoscope. The test is more sensitive than any other, including fundus photography with sophisticated digital systems. Furthermore, progression can be monitored; by the time a background retinopathy has deteriorated to a sight-threatening condition, the tritan threshold will be much elevated, and protan thresholds will also rise. At this stage, of course, specialist intervention is required; but up to this point, a lay person can administer and interpret the test results.
The same is true for age-related maculopathy. Even in very sophisticated societies, it is difficult to detect or monitor the progress of the early stage of this condition. However, blue losses begin when only a few drusen or a slight irregularity of pigmentation can be seen. By the time the patient is in danger of an acute degenerative change, a flashed optotype of a size that more than covers the fovea is indistinguishable no matter how intense the colour. Now that new treatments are proposed for ARM, the problem of early detection and selection is becoming important. Nor is this a complete list of the uses of colour-vision testing. A variety of conditions, from drug intoxications, to inherited degenerations and optic neuropathies also produce extraordinary losses of colour vision. For example, Stargardt's disease alone of the retinal degnerative conditions frequently spares blue cones. Dysthyroid eye disease is best monitored by losses of blue-yellow discrimination, and the success of surgical or medical intervention can be seen within 24 hours. Optic neuropathies classically affect all colour systems equally, so if there is a disproportionate loss of blue, then retinal damage has also occurred. In summary, there are inexhaustible reasons for colour vision testing, and because of the cheapness and familiarity of the PC it is possible to screen everyone who requires a new prescription. Optometrists have reason to be grateful for the computer-games industry!

For further information visit the website http:/www/ ChromaTest.com
Or e-mail c.h.electronics @virgin.net

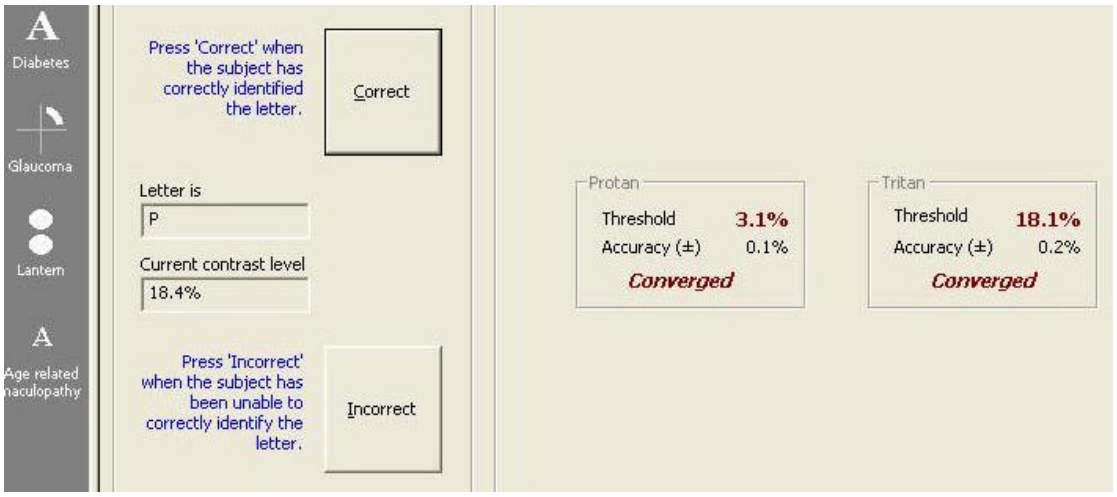


Figure 1.
The operator's panel of the colour test. The "buttons" on the vertical bar to the left start the tests for various pathologies. The large panels are clicked by the mouse, to indicate if the patient has correctly identifies a letter, (the correct letter is also indicated). The progress of threshold finding is indicated on the rest of the screen.

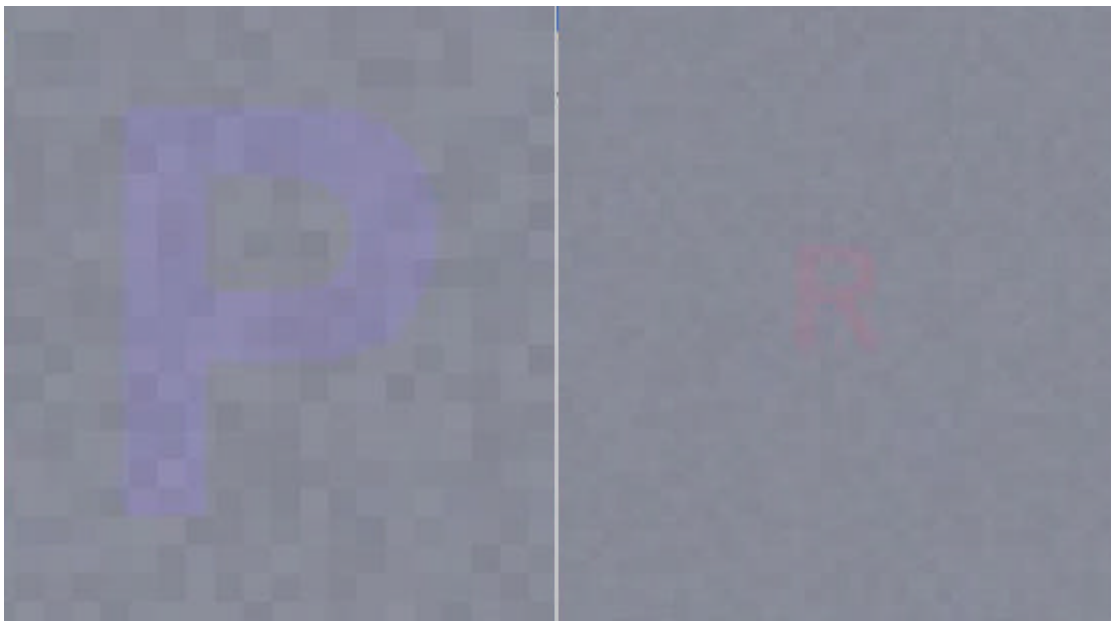


Figure 2.
The patient's view of two of the stimuli, in a test for ARM. These stills show the luminance noise, (squares) the size of which matches the optotype size.

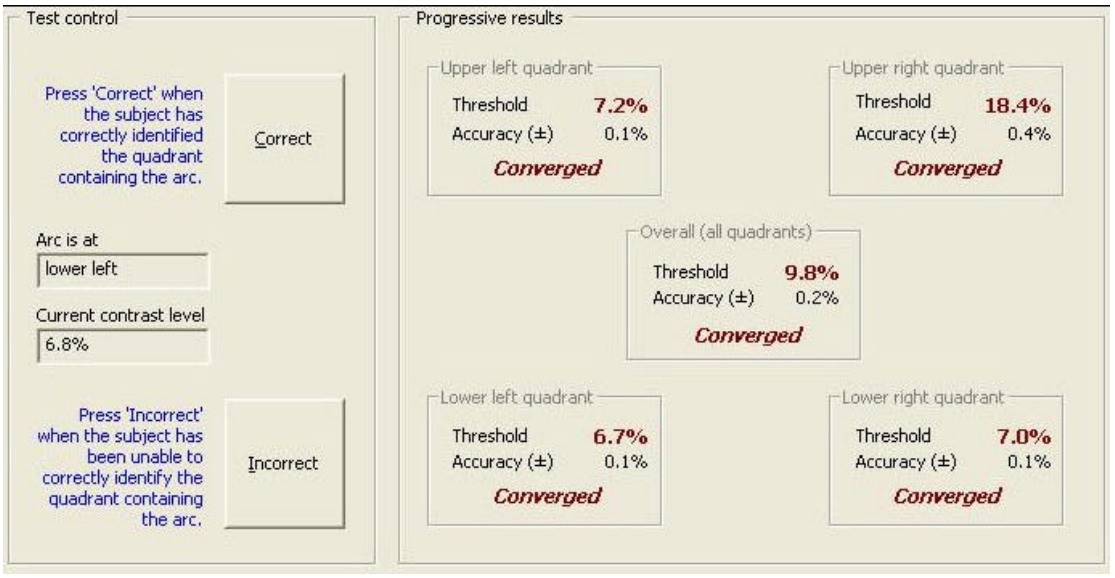


Figure 3.
The operator's panel for the glaucoma test exposes arcs in one quadrant. The patient sits nearer the monitor, so the blue image falls on the Bjerrum region. Thresholds for each arc, and the average, are collected together.

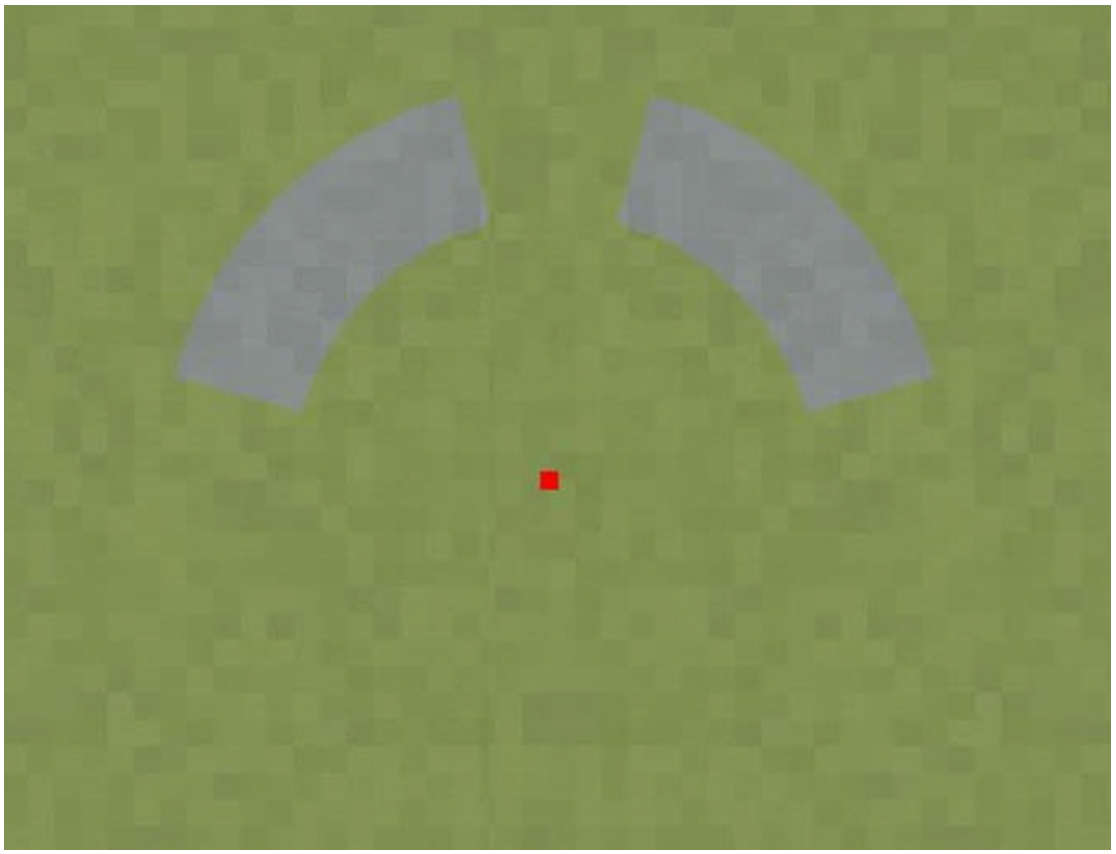


Figure 4.
Patients view of glaucoma test. The montage shows 2 off the 4 blue arcs, but in practice only one appears at any one time. The patient fixates the red spot.

Subject of **Mr A. N. Other**

Birth date 1/01/1950

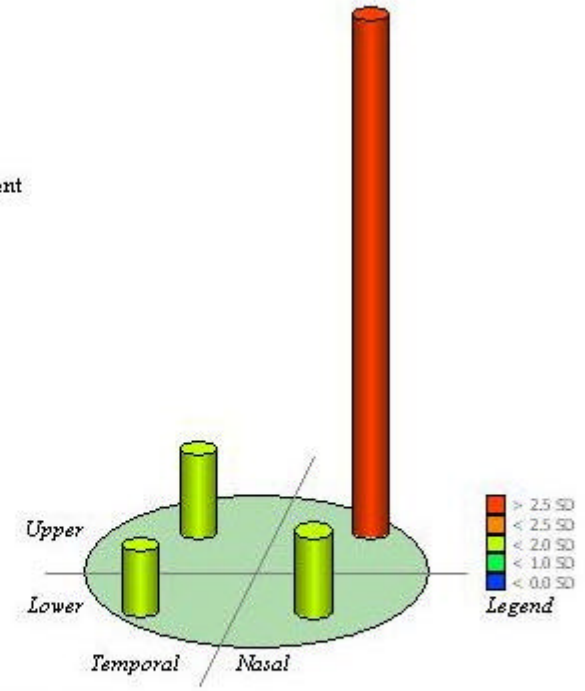
Tested 3/01/2003

at Electrophysiology Department
Moorfields Eye Hospital
162 City Road
London
EC1V 2PD

Eye Left

Tritan Results

	Temporal	Nasal
Upper	1.5 SD	9.0 SD
Lower	1.1 SD	1.4 SD



Thresholds are

Overall:	9.8% ± 0.2% (3.3 SD from normal mean)
Upper Nasal:	18.4% ± 0.4% (9.0 SD from normal mean)
Upper Temporal:	7.2% ± 0.1% (1.5 SD from normal mean)
Lower Temporal:	6.7% ± 0.1% (1.1 SD from normal mean)
Lower Nasal:	7.0% ± 0.1% (1.4 SD from normal mean)

Figure 5.
The printed report from the glaucoma test. Note the elevated threshold in one quadrant.

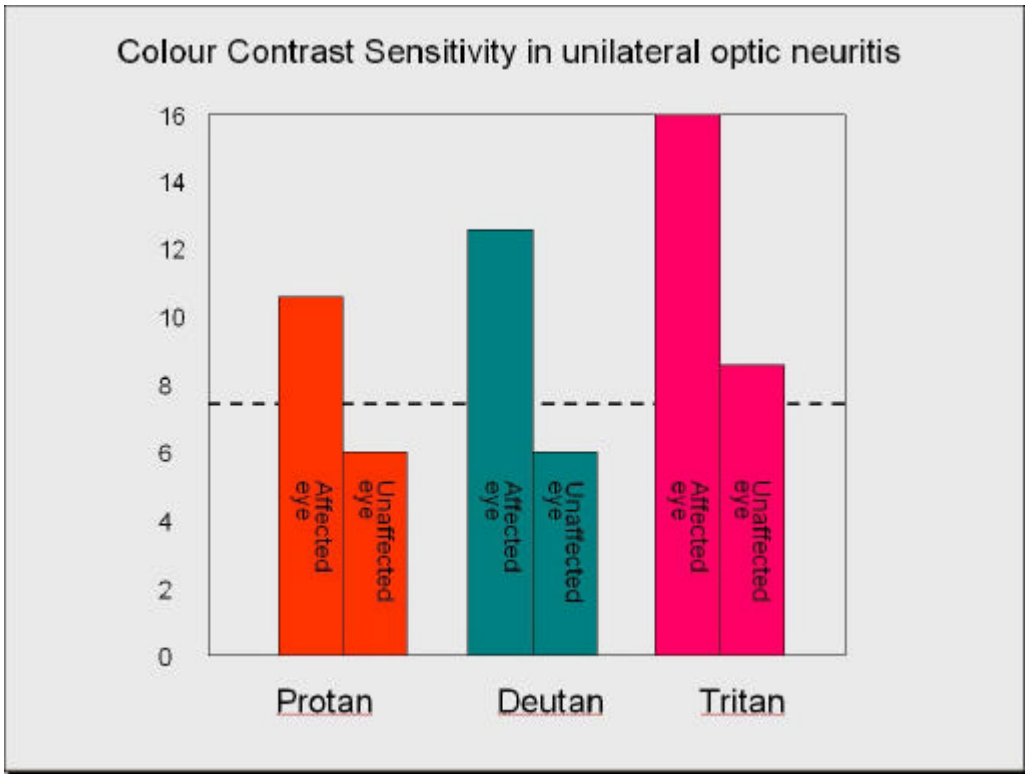


Figure 6.
Changes in colour contrast thresholds in optic neuritis. All colour axes have equal elevations.

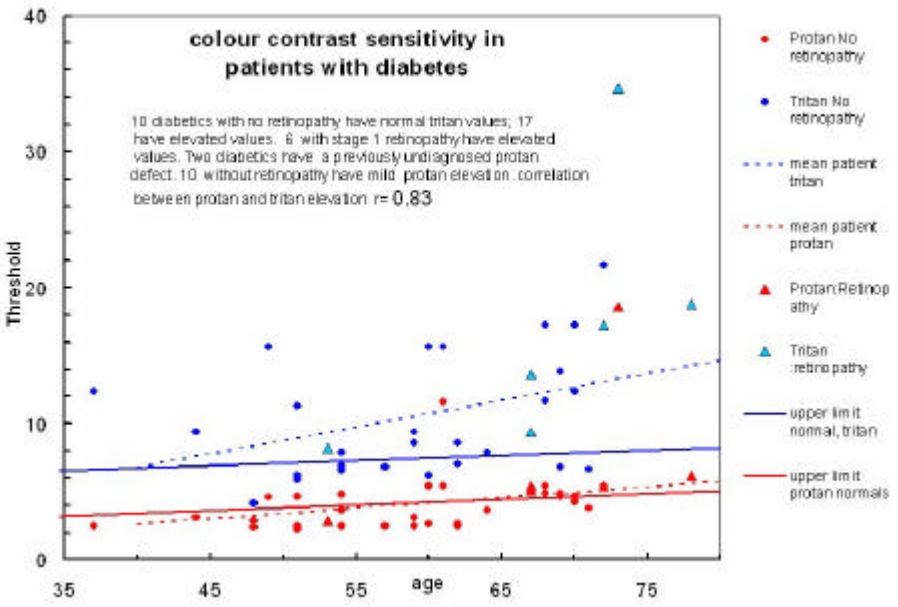
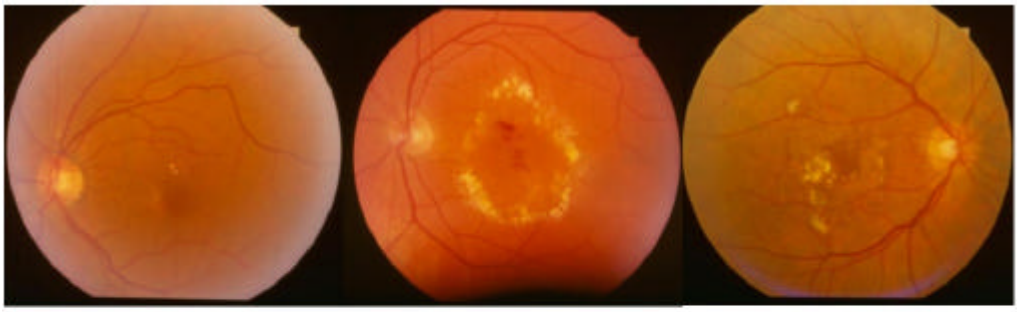


Figure 7.
Tritan and protan threshold in type II diabetics as a function of age. The upper limits of normal are indicated.
Circles, patients with normal fundi (grade 0). Triangles, patients with mild background diabetic retinopathy, (grade 1). Changes in tritan threshold are seen very early. In patients with more severe retinopathy than those shown here, protan threshold are also raised.



JW 63 years	VA = 1.2	AW 71 years	VA = 1.0	DB 69 years	VA = 0.3
Thresholds for large letters (6.5°)					
protan	tritan	protan	tritan	protan	tritan
5.3%	9.1 %	5.7 %	19.9 %	65.6%	not seen
Thresholds for small letters (1.5°)					
protan	tritan	protan	tritan	protan	tritan
7.9%	32.7%	7.9%	88.1%	not seen	not seen

(colours indicate abnormality in SDs above normal mean: green, 0-1 , orange 1.5-2 , Red > 2)

Figure 8.
Three fundus photographs of patients with varying degrees of ARM, and the ChromaTest results. Even the presence of a few drusen increases threshold, and alerts the tester, but not until colour vision is greatly reduced does the patient have any symptoms. Routine testing can easily pick up marked changes (centre panel), before treatment is required. Thresholds more than 2 standard deviations from normal mean are underlined. A suspicious threshold is circled. The text is coloured to indicate the colours in the standard report (fig. 5)