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Editorials

The Farnsworth-Munsell 100 hue test and optic neuritis

'Optic neuritis' is not a well defined disease as much as it is a sometimes poorly defined syndrome. Generally the term is used to describe optic nerve dysfunction associated with a wide range of aetiologies including, but not limited to, generalised demyelinating disease. Moreover, it needs to be emphasised that in the individual patient with the first episode of unilateral optic neuritis no clinical, laboratory, or neuroradiological test can predict consistently whether the patient will develop generalised demyelinating disease. One must wonder, therefore, if the study of Ménage and coworkers, which appears in this issue of the journal, might not be more precisely stated to be that of 'optic neuritis' rather than 'demyelinating optic neuritis' since it is not a long term outcome study.

Be that as it may, it is an excellent study that addresses the question of whether colour vision testing is useful in initial evaluation and subsequent follow-up examinations of the patient with 'optic neuritis'. In a previous study by Fleischman and coworkers¹ it was concluded that subjective visual complaints correlated better with deficits in contrast sensitivity than they did with other measures (colour vision, perimetry, stereoacuity, light brightness, pupillary reaction, and disc appearance). Nevertheless, this study found that 57% of recovered eyes with optic neuritis had deficits of colour vision when tested with standard pseudochromatic Ishihara colour plates, and the Farnsworth-Munsell 100 hue test. The results of Ménage and coworkers are comparable. They found that 53% of their patients had a colour vision defect when tested at the 6 month follow-up examination.

What is most striking in both of these studies is that the traditional notion that a red/green disturbance is to be expected in this clinical setting cannot be verified. In the study of Fleischman *et al.*, of 20 eyes with abnormal FM-100 hue scores, four demonstrated a tritan axis, one a deutan axis, and 15 were non-specific. The findings of Ménage and colleagues are even more provocative. They demonstrated that 'few patients demonstrating any recognisable pattern of colour defect at any of the three examinations.' In addition, in any given patient the colour defect could change from one examination to the next.

One might ask, therefore, why should a clinician even bother to test colour vision in the optic neuritis patient? I

agree with Ménage *et al.* that the usefulness of colour vision testing may be primarily in assessing clinical improvement in these patients. If that is the case the more quantitative Farnsworth-Munsell 100 hue test is preferable to the more qualitative colour vision tests. There can be no doubt that once a diagnosis of optic neuritis has been established the patient becomes anxious about the outcome. Both the physician and the patient are concerned about the acute visual loss as well as the potential for long term development of generalised demyelinating disease. Ménage and coworkers' data suggest that improvement in the Farnsworth-Munsell 100 hue test may be most helpful in reassuring the patient that the optic nerve dysfunction is improving even at a time when other clinical tests of optic nerve function may be equivocal.

One word of caution needs to be voiced here about the implications of colour vision abnormalities detected in the fellow eye of patients with optic neuritis. In the past, a defect of optic nerve dysfunction in the fellow eye of a patient with optic neuritis has been used as evidence that the patient has multiple sclerosis. The results of the optic neuritis treatment trial in the United States does not lend support to this conclusion.² In 457 patients with acute unilateral optic neuritis, fellow eye abnormalities were found for visual acuity in 13.6%, contrast sensitivity in 15.1%, Ishihara plates in 19.9%, F-M 100 in 21.2%, and threshold perimetry in 47.9%. Most of these defects improved over several months. Moreover patients with abnormalities in the fellow eye in the absence of a history of previous optic neuritis were no more likely to have clinical or magnetic resonance imaging evidence of multiple sclerosis than patients with normal fellow eyes at the time of initial diagnosis. These findings suggest that once again our understanding of optic neuritis is at best rudimentary. This is a disorder that deserves continuing investigations and periodic re-evaluations.

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1 Fleischman JA, Beck RW, Linares OA, Klein JW. Defects in visual function after resolution of optic neuritis. *Ophthalmology* 1987; **94**: 102-5.

2 Katz BJ, Coppersmith MJ, Beck RW, Kleary P. Fellow eye visual deficits in acute unilateral optic neuritis: experience of the optic neuritis treatment trial. *Ophthalmology* 1992; **99** (suppl): 109.