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Acquired colour deficiency in patients with Parkinson's disease

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Abstract

The blue cone pathway is reported to be affected early in Parkinson's disease (PD) and acquired type three (tritan) defects may occur. Sixty-one patients attending a treatment and rehabilitation centre for PD were examined with clinical colour vision tests. Seven of 13 patients, for whom the diagnosis of PD was equivocal or who had other medical conditions, were identified as having tritan colour deficiency. Results for the remaining 44 PD patients were compared with 40 age matched controls. Ten PD patients (22.7%) had tritan defects. Tritan defects were not found in the control group but performance on some tests was age related. We conclude that clinical tests for tritan colour deficiency are unlikely to be helpful in identifying PD. © 1998 Elsevier Science Ltd. All rights reserved.

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1. Introduction

Abnormal colour vision has been reported in several studies of patients with Parkinson's disease (PD). Methods of assessment have included clinical tests, such as the Farnsworth–Munsell 100 hue test (FM 100 hue test), and computerised techniques to measure colour thresholds and colour contrast sensitivity. Significantly higher FM 100 hue error scores have been found in 'de novo' PD patients, compared with age matched controls [1]. Error scores have been found to correlate with duration of the disease and with it's severity according to the Hoehn–Yahr staging and the Unified Parkinson's Disease Rating Scale (UPDRS) [2,3]. The FM 100 hue error scores were also found to decrease significantly when patients, who had been deprived of treatment for 12 h, were re-examined 30 min after receiving individual L-Dopa medication [4]. The FM 100 hue plots generally showed poor overall hue discrimination both before and after treatment. However, Price et al. [3] found that six out of 35 patients obtained a tritan axis of confusion and that two patients obtained a red-green axis. Poor overall hue discrimination was found with the Lanthony desaturated D15 test by Haug et al. [5] although colour contrast sensitivity measurements showed specifically poorer performance along a tritan axis. Tritan contrast thresholds, for the detection of motion, were also found to be significantly raised for both foveal and parafoveal stimuli [6]. Similar results, in which psychophysical tests show tritan deficits and clinical tests demonstrate poor overall hue discrimination, have been reported for patients with advanced diabetic retinopathy and are typical of acquired type three colour deficiency [7].

Dopaminergic neurons degenerate in the retina as well as in the brainstem of PD patients. Short wavelength (blue) receptors are sparse in the central retina and the short wavelength mechanism is particularly vulnerable to acquired damage. In consequence, Buttner et al. [1] have suggested that an acquired type three (tritan) defect may originate at the retinal level as an early diagnostic sign of PD. Colour vision screening, with tritan tests, may therefore be helpful in identifying and monitoring patients with idiopathic PD. The FM 100 hue test is not the test of choice because it is not a dedicated tritan screening test and is relatively time consuming. The aim of the present study is to establish whether other more rapid clinical tests for tritan defects

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are effective in showing acquired colour deficiency in PD patients.

2. Subjects and methods

Colour vision was assessed in 61 patients (mean age 63.5 ± 18.9 years) attending a treatment and rehabilitation centre for patients with Parkinson's disease (the Paracelsus Elena Klinik, Kassel). Medical records of the PD patients were consulted at the end of the study. The results of 13 patients (mean age 72.0 ± 9.9 years) for whom the diagnosis of PD was equivocal or who had other medical conditions, which might lead to acquired colour deficiency, were excluded from the main analysis and test results evaluated separately. These were five diabetic patients, five symptomatic patients not confirmed as PD, two patients with glaucoma and one patient receiving anti-epileptic medication. The results of four male patients with congenital red-green (R/G) colour deficiency were also excluded. Results were analysed for the remaining 44 PD patients, one of whom was aphakic (23 male and 21 female, mean age 68.5 ± 7.7 years).

The examination consisted of a battery of pseudoisochromatic plates and hue discrimination tests able to identify both R/G and tritan defects. Individually each test was much less time consuming and easier to complete than the FM 100 hue test. Pseudoisochromatic tests involve the detection and verbal identification of a coloured figure. In hue discrimination tests the patient arranges small coloured samples into a natural hue sequence. The tests included were as follows:

1. A composite pseudoisochromatic test consisting of 13 plates: Seven numeral plates from the Ishihara test for the detection of R/G colour deficiency, the New City University Tritan test (five plates) and the Farnsworth F2 plate. The seven Ishihara plates were the introductory plate, two plates with transformation designs (plates three and six from the 38 plate edition), two plates with vanishing designs (plates 11 and 14) and one protan/deutan classification plate (plate 22). The numerals are of serif design. The City tritan test has five plates with vanishing designs containing geometric symbols [8]. Circles, crosses and triangles are used. Three plates have small colour differences for tritan screening and two plates have large colour differences to identify severe tritan deficiency.
2. The SPP2 plates. All these plates have vanishing designs containing numerals. There are 11 designs for tritan defects and five designs for R/G defects. The numerals have a square box-shaped design similar to that found on computer screens.
3. The Lanthony Tritan Album. The Tritan Album has an introductory plate and five plates, with ranked colour difference steps, for identifying and grading tritan colour deficiency. Errors on two, or more, plates identifies severe tritan deficiency. Each plate has a square of grey dots with violet dots in one corner. The subject has to locate the position of the coloured dots.
4. The T16 test. The T16 test is similar to the Farnsworth D15 test but has more scope for identifying tritan defects. The Munsell colours have value five and chroma of either four, six or eight [9]. Tritan colour deficiency, R/G colour deficiency or poor overall hue discrimination is identified from the results diagram.
5. The Adams desaturated D15 test. The Munsell hues are the same as those of the Farnsworth D15 but the samples have value five and chroma two. Tritan colour deficiency, R/G colour deficiency, or poor overall hue discrimination can be identified from the results diagram.
6. Sahlgren's Saturation test (SST). This test consists of ten colour samples, two greys, four blue-greens and four violets in graded saturation steps. The subject has to select samples which appear to be neither bluish or greenish (grey). The test is scored. An error score of 15 is borderline and a score of 20, or more, is abnormal.

Tests were illuminated with the Richmond 1338R True Daylight Illuminator. The mean illuminance was 250 lux.

Forty age-matched subjects with no ocular or general pathology (mean age 68.0 ± 9.1 years) were examined with the same test battery in the School of Optometry

Table 1
Percentage failure rate of 44 PD patients and 40 age-matched normal subjects on clinical colour vision tests designed to identify tritan colour deficiency

Test	Percentage of Parkinson's disease patients failing	Percentage of normal subjects failing
City University Tritan plates	23	13
SPP2 (Tritan designs)	14	70
Lanthony Tritan Album	16	5
Sahlgren's Saturation Test	16	0
T16	54	15
Adams D15	95	43

Table 2
Test results for four patients with Parkinson's disease with other medical conditions and three symptomatic patients not confirmed as PD who have acquired type three (tritan) colour deficiency

Medical condition	M/F	Age	Tritan plate errors	SPP2 tritan errors	Tritan album errors	SST score	T16 result	Adams. D15 result	Diagnosis
PD and diabetes									
HG	M	67	5[a]	7[b]	5	80	Tritan	Tritan	Severe tritan
PB	M	61	1	1	1	20	Tritan	—	Slight tritan
WK	M	75	2	0	1	10	Tritan	Tritan	Slight tritan
PD and epilepsy									
MM	F	82	5[a]	3[b]	1	20	Tritan[b]	Tritan[b]	Severe tritan
Symptomatic (PD not confirmed)									
GI	F	84	5[a]	8[b]	5	40	Tritan[b]	Tritan[b]	Severe tritan
FV	F	76	3[a]	4[b]	2	10	Tritan[b]	Tritan[b]	Severe tritan
EM	F	59	1	0	0	30	Tritan[b]	Tritan[b]	Slight tritan

—, did not complete.
[a] Also fail F2 plate.
[b] Also R/G errors.

at the University of New South Wales. In this case illumination was provided by screened GE fluorescent daylight tubes with a correlated colour temperature of 5500 K, rated category D when assessed in accordance with C.I.E. Publication 51. The mean illuminance was 240 lux.

Tests were carried out binocularly and normally took between 10 and 15 min to complete. The visual acuity of all the subjects was 6/12 or better.

Verbal test instructions were given in German to patients in the Paracelsus–Elena Klinik and in English at the School of Optometry in the University of New South Wales. Examinations were supervised by the same person in both locations in order to minimise any possible bias in administration.

3. Results

The percentage failure rate for 44 PD subjects and 40 age-matched normals is shown in Table 1. Ninety five percent of patients and 43% of normals made errors on the Adams D15 test. In consequence, the criterion used for the diagnosis of acquired type three (tritan) colour deficiency was that tritan errors must be made on at least two other tests in addition to the D15. The diagnosis of acquired colour deficiency was not therefore based exclusively on failure of hue discrimination tests. Severity of colour deficiency was determined by errors on tritan grading plates and/or the number of errors on the Lanthony Tritan Album. No errors were made on the Ishihara plates except by patients with congenital red-green colour deficiency.

3.1. Excluded patients

Seven of the 13 patients excluded from the main analysis were found to have tritan colour deficiency (Table 2).

Three of the five diabetic patients had tritan defects. The defect was severe in one patient (HG) and slight in two. HG's colour deficiency was identified by all six tests. He could not interpret any of the plates in the City test or the Lanthony Tritan Album, and failed eight of the 11 tritan designs in the SPP2 test. Tritan axes of confusion were obtained with the T16 and the Adams D15. A score of 80 was obtained on Sahlgren's saturation test. One diabetic patient passed all the tests and one failed the Adams D15 only.

Three of the five patients with symptomatic disease were found to have tritan defects; one patient had slight colour deficiency and two patients severe colour deficiency. The results for patient GI were similar to those of HG except that errors were made on R/G designs of the SPP2 test in addition to tritan errors and the error score obtained on Sahlgren's test was 40. R/G errors as well as tritan errors were also made on hue discrimination tests. One symptomatic patient passed all the tests and one failed the Adams D15 only.

The patient being treated with anti-epileptic medication (MM) also had severe tritan colour deficiency. MM made tritan errors on all six tests but failed fewer pseudoisochromatic plates than HG and GI. She failed only one plate of the Lanthony Tritan Album and could not interpret three R/G designs as well as three tritan designs of the SPP2 plates. An error score of 20 was obtained with Sahlgren's test. R/G and tritan errors were made on hue discrimination tests.

Table 3
Test results for ten patients with Parkinson's disease diagnosed as having acquired type 3 (tritan) colour deficiency

Patient	M/F	Age	Tritan plate errors	SPP2 tritan errors	Tritan album errors	SST score	T16 result	Adams D15 result	Diagnosis
AB	F	79	3	2	5	15	Tritan	Tritan	Severe tritan
EK	F	78	5[a]	4	0	5	Tritan	Tritan	Severe tritan
AU	M	76	3	1	0	5	Pass	Tritan	Moderate tritan
GB	F	72	3	2	0	20	Tritan	Tritan	Moderate tritan
AG	F	68	2[a]	2	0	10	Tritan[b]	Tritan	Slight tritan
EW	F	61	2	1	0	15	Tritan	Tritan	Slight tritan
JW	F	80	3	0	0	0	Tritan	Tritan	Slight tritan
HM	M	69	1	0	1	20	Pass	Tritan	Slight tritan
HZ	M	67	1	1	0	10	Tritan	Tritan	Slight tritan
WS	M	75	0	0	1	20	Tritan	Tritan	Slight tritan

[a] Also fail F2 plate.
[b] Also R/G errors.

Neither of the patients with glaucoma nor any of the patients with congenital R/G colour deficiency made errors indicating tritan colour deficiency. One patient with glaucoma failed the Adams D15.

3.2. PD patients

Ten of the 44 PD patients (22.7%) were identified as having tritan colour deficiency (Table 3). Two patients had severe colour deficiency, two had moderate deficiency and six had slight colour deficiency. The mean age of these patients was 72.6 ± 4.4 years.

Two patients had severe tritan deficiency but neither failed all the tests. AB failed the tritan screening plates in the City test and could not interpret any of the tritan plates in the Lanthony Tritan Album. She failed three tritan designs in the SPP2 test and obtained tritan results for both hue discrimination tests. A borderline result was obtained with Sahlgren's test. The second patient with severe tritan colour deficiency, EK, failed the F2 plate and both screening and grading tritan plates in the City test. EK failed four tritan plates in the SPP2 test but was able to pass the Lanthony Album, Sahlgren's test and the T16 test. Two patients, AU and GB, made mistakes on all the tests except the Lanthony Tritan Album and were considered to have moderate tritan deficiency. Six PD patients were found to have slight tritan colour deficiency. All these patients failed the Adams desaturated D15 test and two other tests. Five patients failed the City test, five failed the T16, three failed the SPP2, two recorded single errors on the Lanthony Album and two patients failed Sahlgren's test.

Of the remaining 34 PD patients, three patients completed all the tests without error; 22 patients made mistakes on the Adams desaturated D15 and failed one other test, nine patients failed the D15 only.

In total, 41 of the 44 patients with confirmed PD patients failed the Adams D15, 12 patients obtaining a diagnostic tritan result and 29 patients showing poor overall hue discrimination. In comparison 19 patients failed the T16 test, 12 obtaining a tritan result and seven patients showing poor overall hue discrimination. Only four patients obtained tritan results on both tests.

A smaller number of PD patients made errors on pseudoisochromatic plates. Ten patients made mistakes on screening plates of the City test and two patients failed the F2 plate. Seven patients made errors on the Lanthony Tritan Album and seven patients were classified as having abnormal colour vision with Sahlgren's Saturation Test. Six patients made tritan errors on the SPP2 test. The aphakic patient made errors on the D15 and T16 tests.

3.3. Normal subjects

Only eight of the 40 normal subjects examined passed all the tests without error. All these subjects were under 63 years of age. The remaining subjects made random errors on at least one test. These errors tended to be 'design specific' and related to the visual task needed for the test. The SPP2 numerals were found to be much more difficult for the (British) normal subjects to identify verbally compared with the (German) PD patients. This is possibly due to the greater familiarity with boxed numeral shapes used in German literature. Some subjects in both groups found it consistently difficult to name triangles compared with circles or crosses. No subject was considered to have tritan colour deficiency. Twenty-eight subjects (70%) made errors on the SPP2 plates which included R/G as well as tritan errors. No subject failed two other tests in addition to the SPP2. Seventeen subjects (43%) made errors on the Adams D15 test but in only six cases were these diagnostic tritan errors. Six subjects made errors on the T16 test. Five subjects made a single error on tritan screening plates and two made a single error on the Lanthony Tritan Album. No normal subject failed, or was borderline, on Salhgren's test.

4. Discussion

The present results confirm those of Kupersmith et al. [10] that R/G colour deficiency is not identified with pseudoisochromatic plates in PD. Identification of tritan colour deficiency in older patients is masked by physiological changes in the eye media with age. The present results show that age related errors can be anticipated on both the SPP2 plates and the Adams D15. This is partly attributed to the use of desaturated colours with small colour differences. Haug et al. [5] found that PD patients showed poor hue discrimination with the Lanthony desaturated D15 test (Munsell value eight and chroma two). The Lanthony test has very desaturated colours and is difficult for young normal subjects to complete without error at the illumination levels used in this study. The Adams test presents a more appropriate level of difficulty. Although 95% of PD patients failed the Adams test, 43% of normals also failed. The T16 test is designed specifically to identify tritans and contains more saturated colours than the Adams test. The T16 was failed by 54% of PD patients and by 15% of normals. Neither test is therefore very efficient in separating PD patients and normal age matched controls. Older subjects often found the visual task of ordering colours difficult to comprehend initially and manipulating small samples caused particular problems for PD patients with tremor. Tremor tended to increase with the concentration needed to arrange the colours. Pseudoisochromatic tests are easy to understand and tritan tests which employ moderate or large colour differences, such as the City tritan test and the Lanthony Album, were found to be less affected by age. However isolated design specific errors, depending on the familiarity of the contained figure, were made by both PD patients and normals. The figure designs contain numerals in two different fonts, as well as geometric symbols, and some subjects preferred to draw over the design rather than attempt a verbal identification.

Of the 61 patients examined eight were found to have either severe or moderate type three (tritan) colour deficiency. Two of these patients had additional pathology and the diagnosis of PD was equivocal in two. These results suggest that between 7 and 13% of PD patients, undergoing treatment, have significant tritan colour vision deficits. In all ten PD patients (22.7%) have some degree of aquired colour deficiency. Most patients received medication at intervals throughout the day and it was not possible to co-ordinate the colour vision examination to take place either immediately before medication or at a set interval following medication. In consequence it was not possible to assess the effect of treatment.

Acquired type three (tritan) deficiency is often characterised by poor overall hue discrimination. In this case both tritan and R/G diagnostic errors may occur and, if desaturated colours are used, it is difficult to distinguish pathological and physiological changes with age. Results obtained with tests based on different design principles, employing a variety of visual tasks, are not necessarily expected to agree precisely. Individual clinical tests tend to have either low sensitivity or low specificity for identifying acquired colour deficiency in PD patients. All the tests included in the present study have potential for identifying acquired type three (tritan) colour deficiency confirmed by the results obtained by PD patients with other medical conditions. The results for three patients (particularly HG) were typical of severe acquired colour deficiency. In contrast only two of 44 PD patients, without other pathology, had severe acquired colour deficiency. The absence of clear tritan colour deficiency in PD patients strongly suggests that previously reported colour vision deficits originate in the visual pathway rather than at the retinal level and it is unlikely that clinical tests have a useful role in monitoring the effects of treatment in PD.

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