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Research Report

Colour perception deficits after posterior stroke: Not so rare after all?

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ABSTRACT

Cerebral achromatopsia is an acquired colour perception impairment caused by brain injury, and is generally considered to be rare. Both hemispheres are thought to contribute to colour perception, but most published cases have had bilateral or right hemisphere lesions. In contrast to congenital colour blindness that affects the discrimination between specific hues, cerebral achromatopsia is often described as affecting perception across all colours.

Most studies of cerebral achromatopsia have been single cases or case series of patients with colour perception deficits. Here, we explore colour perception deficits in an unbiased sample of patients with stroke affecting the posterior cerebral artery ($N = 63$) from the Back of the Brain project. Patients were selected based on lesion location only, and not on the presence of a given symptom. All patients were tested with the Farnsworth D-15 Dichotomous Colour Blindness Test and performance compared to matched controls ($N = 45$) using single case statistics. In patients with abnormal performance, the patterns of colour difficulties were qualitatively analysed.

22% of the patients showed significant problems with colour discrimination (44% of patients with bilateral lesions, 28% with left hemisphere lesions and 5% with right hemisphere lesions). Lesion analyses identified two regions in ventral occipital temporal areas in the left hemisphere as particularly strongly related to impaired performance in colour perception, but also indicated that bilateral lesions are more strongly associated with impaired performance that unilateral lesions. While some patients only had mild deficits, colour perception impairments were in many cases severe. Many patients had selective deficits only affecting the perception of some hues. The results suggest that colour perception difficulties after a PCA stroke are common, and that they vary in severity and expression. In addition, the results point towards a bilateral processing of colour perception with a left hemispheric domination, contradicting previous reports.

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1. Introduction

Colour perception plays a significant role in many aspects of human everyday life. It is vital for the detection and recognition of objects in our surroundings, it is used in non-verbal communication, for example blushed cheeks communicate embarrassment, and it is used to guide decision-making, for example a traffic light switching from red to green. But colours are more than a means for discriminating objects and guiding decision-making. Colours affect humans emotionally and make the world more enjoyable to live in. Despite the importance of colour perception in our lives, our understanding of the cerebral processes underlying colour perception remains limited.

Patients who have a reduced ability to perceive colours due to brain injury, have provided important insights into how colour is processed in the brain and which cerebral areas are involved. Cerebral achromatopsia is characterized by impaired perceptual colour experience following cerebral lesions (Bartolomeo, 2021). The first known cases descriptions of patients with cerebral achromatopsia were described in the late 1800s (Brill, 1882; Steffan, 1881; Verrey, 1888) and since then, many cases have been reported (Bartolomeo et al., 2014; Crognale et al., 2013; Heywood et al., 1994; Heywood et al., 1987; von Arx et al., 2010) (see Bouvier & Engel, 2006 for a review). The extent of the deficit can vary and this has led some authors to differentiate between cerebral achromatopsia to refer to a complete loss of colour perception and cerebral dyschomatopsia to refer to a reduction of colour perception (Carroll & Conway, 2021). Beauchamp et al. (2000) pointed out that a closer look at the literature reveals that the majority of reported cases of cerebral achromatopsia are of reduced colour perception and not a complete lack of colour perception. In this paper we use the term achromatopsia to refer to significant acquired colour perception impairment, regardless of the severity of the deficit.

Reviewing existing cases, the expression of colour perception deficits following brain injury can vary. While some patients complain of seeing everything in grey tones (Bartolomeo et al., 1997; Heywood et al., 1987) other patients describe the world as faded, with colours perceived to some extent but appearing dirty and less bright (Kennard et al., 1995; Meadows, 1974). Acquired colour perception deficits can be constricted to parts of the visual field (one hemifield or in rare cases a quadrant (Bouvier & Engel, 2006; Short & Graff-Radford, 2001). In some cases, the patients are not aware of the deficit prior to assessment (Kölmel, 1988; Short & Graff-Radford, 2001; Verrey, 1888; von Arx et al., 2010). The deficits are in general thought to affect the perception of all colours. This stands in contrast to congenital colour blindness that affects the perception of specific hues. Nevertheless, some cases of achromatic patients have been described with selective difficulties within certain hue categories (Moroz et al., 2016; Rizzo et al., 1993).

The number of case studies of patients with cerebral achromatopsia is sparse compared to other acquired visual deficits (Bouvier & Engel, 2006). So despite the exact prevalence being unknown, acquired colour perception deficits are considered to be rare (Bartolomeo, 2021; Bartolomeo et al., 2014; Beauchamp et al., 2000; Kraft et al., 2014; Meadows, 1974). Achromatopsia rarely occurs without other deficits. The most frequently reported co-occurring deficits are visual field defects, prosopagnosia, and topographagnosia (Bouvier & Engel, 2006).

Verrey (1888) was the first to suggest that lesions involving the fusiform and/or the lingual gyri in the ventral occipito-temporal region can cause colour perception difficulties. This has been confirmed across many case studies (e.g (Bartolomeo et al., 2014; Beauchamp et al., 2000; Damasio et al., 1980; Green & Lessell, 1977; Kentridge et al., 2004; Pearlman et al., 1979; Rizzo et al., 1993; von Arx et al., 2010; Zeki, 1990), and also recently through a group study of patients with unilateral lesions to the ventral stream (Nestmann et al., 2021). A lesion overlap analysis based on a meta-analysis of 92 case reports from the literature revealed a small region in the ventral occipital cortex with high lesion overlap (Bouvier & Engel, 2006), with a majority of lesions affecting the right hemisphere. The authors noted, however, that there was substantial residual colour vision in many cases, despite lesions to the ventral occipital cortex, suggesting that other late visual areas likely also play a role in colour perception. Only a few cases have been reported with pure achromatopsia. Kölmel (1988) reported two patients who suffered from pure homonymous hemiachromatopsia after stroke in the medial occipito-temporal gyrus in the right and left hemisphere respectively and provide some of the strongest evidence of the importance of the medial occipitotemporal gyrus for colour perception. In both cases, the patients' hemiachromatopsia later improved to only involve the upper quadrant of the visual field and were only consistently demonstrated with colour perimetry. Both patients had other visual difficulties in the early stages following their stroke, but these eventually remitted, leaving only the colour perception deficits in the chronic stage. Furthermore, Selim et al. (2021) reported a patient with pure achromatopsia after a unilateral PCA stroke in the left hemisphere. A partial recovery was seen within two days after the stroke and they did not clearly exclude colour agnosia.

Colour perception deficits have been described following bilateral lesions as well as following lesions restricted to either hemisphere (Bouvier & Engel, 2006; Nestmann et al., 2021). Unilateral lesions usually lead to colour perception losses constricted to one hemifield, whereas full field colour perception difficulties are thought to require bilateral medial occipitotemporal lesions (Barton, 2011; Bouvier & Engel, 2006). Some exceptions have been described. Setälä and Vesti (1994) refer to a patient who, after a unilateral lesion in the anterior inferior right occipito-temporal region, showed a full field colour perception impairment. The patient reported by Selim et al. (2021) also showed full field colour perception impairment after a unilateral lesion in the medial margin of the left occipital and left posterior temporal lobe. A greater number of cases with colour perception deficits have been described following lesions restricted to the right hemisphere than the left hemisphere (Bouvier & Engel, 2006) possibly suggesting a dominance for colour perception in the right hemisphere.

Neuroimaging studies have also identified areas in the ventral occipitotemporal cortex as particularly relevant for colour perception (Beauchamp et al., 2000; Chao & Martin,

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1999; McKeefry & Zeki, 1997; Simmons et al., 2007). Some imaging studies, in line with patient studies, also suggest some degree of lateralization in response to colours in the right hemisphere (e.g. Brewer et al., 2005).

Despite years of research regarding colour perception deficits following brain injury, there are still ambiguities regarding the deficits prevalence and expression, as well as uncertainties regarding the respective roles of the two hemispheres. Here, we take advantage of a unique data set from the Back of the Brain project (Rice et al., 2021) in which 64 patients were recruited based on their lesion location within the areas supplied by the posterior cerebral artery (PCA). All patients were assessed with a large detailed visual perceptual test battery including a colour perception test, and structural imaging scans were performed. By investigating colour perception deficits in this large group of patients that were recruited based on lesion location rather than symptom profile (as is typically done in single case studies), we provide evidence that colour perception deficits are more common than previously assumed, that they are more common following lesions restricted to the left hemisphere than right hemisphere, and that they can vary greatly in their expression.

2. Materials and method

The data were collected as a part of the Back of Brain Project (BoB) (Rice et al., 2021). The aim of the BoB project was to improve our understanding of the cognitive and cerebral architecture underlying face, word and object processing. 64 native English patients with stroke in the areas supplied by the PCA were recruited and assessed with a test battery assessing low-level, intermediate- and high-level visual perception. Patients also underwent structural and functional imaging scans (MRI, functional localizer and Diffusion Tensor Imaging). 46 control participants were also included in the project. The current study provides an analysis of the participant's performance on the colour perception test that was included in the BoB project, and structural imaging of their lesions (T1 MRI). All participants gave written informed consent in line with the declaration of Helsinki, and the BoB project and its experiments was approved by Manchester North West Research Ethics Committee (MREC 01/8/094) and the London Queen Square Research Ethics Committee (UCL; 16/EM/0348). No part of the study procedures or study analyses were pre-registered prior to the research being conducted. We report here how we determined sample size, all data exclusions, all inclusion/exclusion criteria, whether inclusion/exclusion criteria were established prior to data analysis, all manipulations, and all measures in the study. All details regarding patient recruitment, testing, and inclusion/exclusion criteria are reported in Rice et al. (2021). The patient sample size was determined by the number of eligible patients who consented to participate during the two year project period.

2.1. Participants

2.1.1. Patients

An overview of the demographic details for both the patients and the control group can be seen in Table 1. 64 patients (12 women and 52 men) were included in the BoB-project. They were all English-speaking and recruited from either London ($N = 41$) or Manchester ($N = 23$). All patients had lesions resulting from a stroke in the PCA artery. Patients with an isolated cerebellar or thalamic stroke were excluded. Diagnosed psychiatric, developmental or other neurological disorders and head injuries were exclusion criteria as well. The demographics were initially not matched in regard to the subgroup of the different laterality of the brain damage, but they did not differ in terms of age, educational level and time since the stroke (Rice et al., 2021). The average volume of the lesion was 37 cm^3, the smallest one measured .01 cm^3 and the largest 126.14 cm^3. At a group level there was no substantial difference in right-hemispheric and left-hemispheric lesions, regarding the lesion volume and lesion location (see Fig. 1). See Rice et al. (2021) for more details about recruitment and details about the patient group, including scan protocol and lesion identification procedure.

Table 1 – Demographics.

	Patients total	Left hemisphere lesion	Right hemisphere lesion	Bilateral lesion	Controls
N	64	32	23	9	46
Age - years	61 (13)	64 (12)	58 (15)	58 (11)	62 (15)
Gender (M/F)	52/12	26/6	18/5	8/1	22/24
Handedness					
Right handed	57	26	23	8	42
Left handed	6	5	–	1	2
Mixed	1	1	–	–	2
Education - years	14 (3)	14 (3)	14 (3)	14 (4)	15 (2)
Time since stroke - months	42 (50)	42 (48)	42 (59)	40 (29)	–
Lesion volume - cm^3	37 (35)	32 (30)	35 (39)	61 (38)	–
Visual field deficits					
Hemianopia					
Quadrantanopia	32	19	12	–	–
Bilateral deficit	13	8	5	–	–
	9	–	1	8	–

Note. Demographic information for all participants given as mean (SD). Gender, handedness, and visual field deficits are listed as number of patients. Visual fields are coded in mutually exclusive categories; homonymous hemianopia, homonymous quadrantanopia, and bilateral visual field deficit. M = male, F = female.

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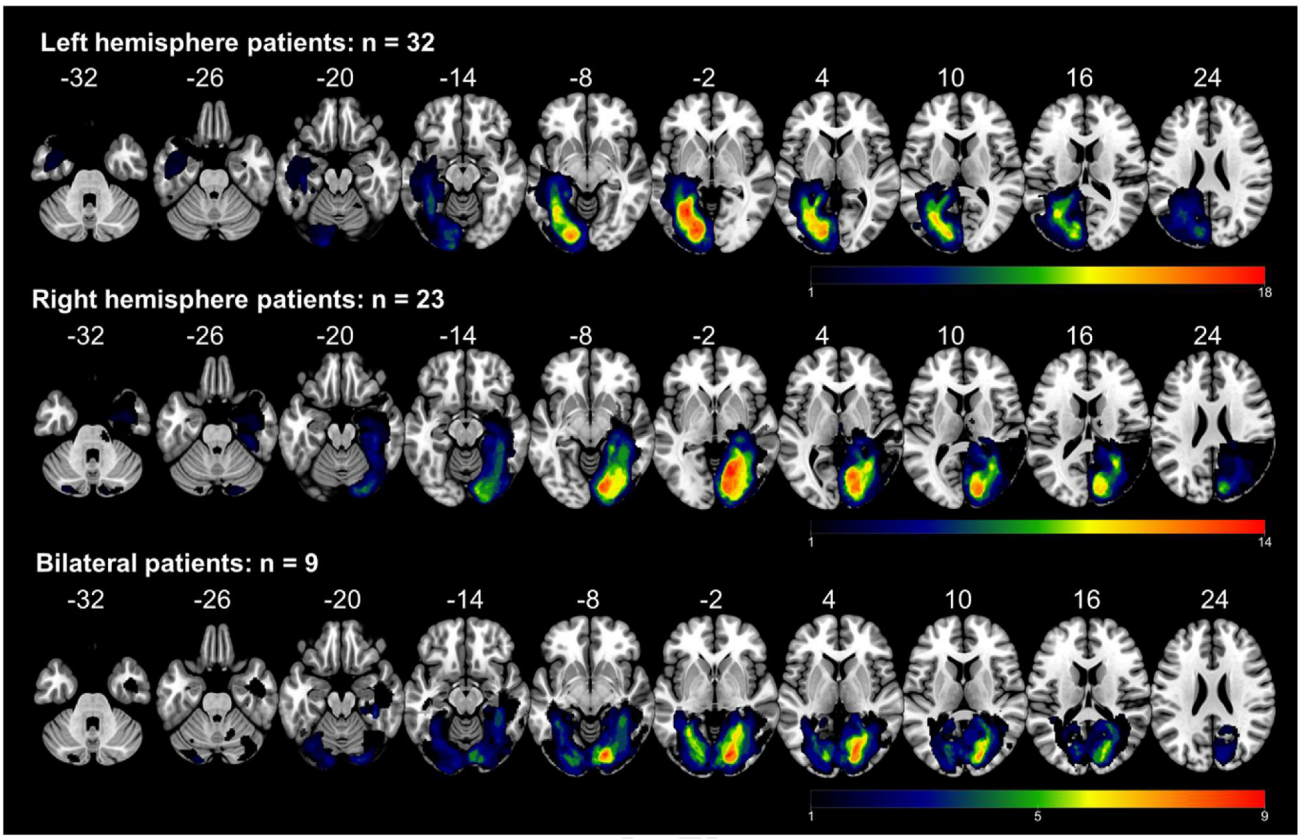

Fig. 1 – Lesion Overlap for each Patient Subgroup in BoB. Illustration from Rice et al. 2021. Note. The warmer the colours, the greater the lesion overlap.

2.1.2. *Control group*

A control group of 46 participants (22 men and 24 women) was included. The controls were either family members of the patients or recruited in the local community in the two locations (Rice et al., 2021). The mean age of the controls was 62 ranging from 26 to 84 years ($SD = 15$). The control group had no prior medical history with developmental, neurological or psychiatric diagnoses or challenges. They were asked if they were colourblind and were excluded if they reported yes. 3 controls were excluded from the BoB project based on difficulty with face processing, reading or an abnormal brain scan. In respect to the educational level, the level in the control group was mildly, though significantly, higher than that of the patient group ($M = 15$ years, $SD = 2$; $t\,(108) = 2.56$, $p = .012$). The control group underwent the same test-battery as the patients, except for tests for which normative data was available (see Robotham, et al. 2021 for details about the test-battery).

2.2. *Materials - Farnsworth D-15 Dichotomous Colour Blindness Test (D-15)*

The Farnsworth D-15 Dichotomous Colour Blindness Test (D-15) was used to assess colour perception (Farnsworth, 1947). D-15 is an arrangement test developed by Farnsworth (1947). The test consists of 16 coloured caps. One cap is placed as a pilot cap in the left end of a box and the subject is asked to arrange the 15 colour caps in order of their hue in a gradual progression from left to right. The patient's arrangement of the caps is then registered and can be drawn into a circular diagram for a visual interpretation of the performance. If there is an impaired hue-discrimination, the diagram reveals so-called "axes of confusion" for the administrator to interpret. These axes are also called crossovers and are lines that go across the diagram depending on the colours that have been confused. Specific patterns of crossovers provide information about the type of colour perception deficit the participant is exhibiting. In congenital colour blindness, axes typically seen in protanopia go from blue/green to red, in deuteranopia from green to purple and in tritanopia from yellow to blue. With the D-15 it is possible to detect difficulties with colour discrimination and investigate the participants' specific colour discrimination patterns. It was included in the test battery for the BoB project as it is short and easy to administer, and suitable for patients with brain injuries who easily get fatigued. The test only requires the use of the central visual field, and patients are allowed to move the caps around freely, thereby minimizing effects of visual field defects on performance.

2.3. *Testing procedure*

The procedure followed the D-15 test manual from Munsell Colour closely to ensure uniformity (X-Rite, n.d.). The BoB-project used ceiling light in the test room (not particularly bright) and ensured that the light did not reflect on the caps.

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The test was administered to the patients on the second day of the three non-consecutive days of testing. The administrator sat across the table from the participant. The pilot cap was positioned in the left end of the tray (left of the patient), and the other caps were randomly arranged in the lid with the colour side up. The test was carried out under binocular viewing. The administrator asked the participant to:

"Select the cap which is the closest possible match to the pilot (point to the pilot cap) and place it just to the right of the pilot cap here (administrator points). Please do not touch the coloured surfaces of the caps. Now select the closest colour match to the cap you just selected and place it to the right of it. Continue doing this until you have built the whole sequence of colours".

At the end of each test, the order was recorded on a scoring sheet. Apart from a perfect first try, where all the caps were placed flawlessly in order from 1 to 15, the subject was asked to repeat the test a second time under the same conditions. A few participants with a perfect first try were mistakenly tested a second time. Since this was not in line with the manual procedure, only the first attempt was scored. There was no time limit for task completion. One patient (PM009) did not perform a second attempt despite a very poor first attempt. The participant was so frustrated by the task that they did not want to repeat it. For this participant, the first attempt that was recorded as the participant's score. Participants were allowed to wear glasses, contact lenses and hearing aids.

2.4. Exclusion criteria

Patients and controls who reported congenital colour blindness were excluded, since the interest was only in an acquired abnormality in colour perception.

2.5. Behavioural analysis

2.5.1. Scoring of D-15

The scoring method suggested by Bowman (1982) was used as the primary scoring method. It is a quantitative scoring method that has been applied successfully for acquired colour perception deficits (Atchison et al., 1991). The method is a mathematical colour difference formula, which is used to calculate a colour difference score between any two coloured caps (see Bowman (1982) for more details about the formula). The colour difference scores between the laid caps are summarized when the participant has finished the sequence, so one full cap arrangement gives one single measure of error. This measure of error is the total colour difference score (TCDS). The bigger the colour differences between the adjacently laid caps, the higher the TCDS and the more severe the colour discrimination problem is. The TCDS thus gives a quantitative indication regarding the type and number of cap transpositions made by the subject. A perfect arrangement gives the lowest possible TCDS of 116.9 (Bowman, 1982). The colour difference scores between each cap are shown in Supplementary Table 1. The Bowman scoring method has several advantages. It provides a single quantitative performance severity measure for each participant, which makes the participants' performance easily comparable. The score takes into consideration the difference in how much each cap differentiates in colour from the next cap. In addition, the participants are not punished more than once if they, after placing a wrong cap, proceed by arranging the following caps in reverse order (e.g. cap 1, 2, 3, 10, 9, 8). The two first authors scored the test individually and compared their results afterwards. In cases in which the scores differed, the test was scored again until agreement was reached. The participants with an imperfect first performance had two scores, since they, according to the procedure, repeated the test a second time. Their lowest (i.e., best) performance score was chosen for the analysis. For participants who performed perfectly the second time it was assumed that a colour perception deficits was not the cause of their imperfect first performance. For participants with errors in both trials, it was assumed that their lowest performance was a more accurate indication of their colour perception abilities. In both cases, a conservative estimate was considered the best choice in order to avoid too many false positives.

2.5.2. Analysis of the abnormality in performance

Our main aim was to test whether the patients performed significantly worse than the control group on D-15. We used Crawford and Howell (1998) modified *t*-test to determine whether a patient's performance was abnormal compared to the performance of the control group. The control group performances were treated as a sample statistic and not as a population parameter, avoiding overestimating the abnormality of performances (Crawford & Howell, 1998). The significance test was run through the computer program: "SingslimsES.exe", available at Crawford's homepage: https://homepages.abdn.ac.uk/j.crawford/pages/dept/The program provides, beside a t-value and a *p*-value, an estimate of the abnormality of an individual performance, confidence limits of the estimate of abnormality, as well as point and interval estimates of the effect size (zcc) (Crawford et al., 2010; Crawford & Garthwaite, 2002; Crawford & Howell, 1998).

2.5.3. Analysis of colour patterns in the performance

For a deeper exploration of achromatopsia, the pattern of performance of patients with abnormal scores was analyzed to identify what problems with colour discrimination they showed. The analysis built on the two first authors' inspections and interpretations, which is common in arrangement tests (Moroz et al., 2016). It was based on five colour categories (Blue, Green, Yellow, Red, and Purple) and the number of major crossovers. Identifying major crossovers is a method that is commonly used to identify colour perception impairments on the D-15 test and is considered here as an additional indication of colour discrimination severity. A major crossover was defined as a difference of more than three between the two laid caps, which is the commonly used definition (Hovis et al., 2004). The patterns were also analysed and compared to typical patterns of congenital colour blindness.

2.6. Lesion analysis

As part of the BoB-project, a high-resolution T1 weighted structural scan was acquired for each patient, including 260 slices covering the whole brain with TR = 8.4 ms, TE = 3.9 ms, flip angle = 8°, FOV = 240 × 191mm2, resolution

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matrix = 256 × 206, voxels size = .9 × 1.7 × 0.9mm3. Automated outlines of the area affected by stroke were generated using (Seghier et al., 2008) modified segmentation-normalisation procedure (run using SPM12). This procedure is designed for use with brain-injured patients and identifies areas of lesioned tissue in order to optimise fitting lesioned brains to standard MNI space. Using this procedure, there were four patients whose small lesions could not be identified. For these patients, a neurologist (APL) manually traced the lesions using a semi-structured lesion identification technique. The binarised lesion image was used to create the lesion overlap map in Fig. 1, and was the basis for the lesion analyses described below (see Rice et al., 2021 for full details).

To quantify the lesions across the patient group, a mask of the PCA territory in the left and right hemisphere was derived from the Harvard Oxford atlas (Desikan et al., 2006) and the John Hopkins White Matter atlas (Hua et al., 2008). The PCA mask consisted of the occipital pole, supracalcarine and intracalcarine cortex, lingual gyrus, parahippocampal gyrus (posterior, anterior), fusiform cortex (occipital, temporal occipital, posterior, anterior), inferior temporal gyrus (anterior, posterior, temporal occipital), lateral occipital cortex (inferior, superior), the cuneal cortex and the thalamus. The white matter tracts of the forceps major/splenium, inferior longitudinal fasciculus, and inferior fronto-occipital fasciculus were also included.

The proportion of overlap between each patient's lesion and the left and right PCA mask was calculated. The proportion of lesion overlap for each patient was then used to calculate: (1) Total lesion volume (the sum of left + right PCA overlap); (2) the presence of a unilateral vs. bilateral lesion, coded as either 1 (unilateral lesion) or 2 (bilateral lesion); and (3) the percent overlap with each of the PCA-ROIs specified above (see Supplementary Table 3a for full list of included regions).

2.6.1. *Anatomical analysis*

Looking first at lesion lateralization in the patients with impaired performance, we assessed the proportion of patients in each lesion group (left, right, bilateral) showing significant impairment on the D-15 colour test. Secondly, we tested the hypothesis that significantly impaired patients as a group had larger lesion than the unimpaired patients using an independent sample *t*-test. Finally, to assess the importance of specific subregions of the PCA territory (listed above) for performance on D-15, we performed a forward stepwise linear regression using SPSS (version 28). The proportion of damage in each of the constituent PCA ROI and white matter tracts were included as independent variables, as well as the total lesion volume and the presence of a unilateral vs bilateral lesion. At each step, variables were added based on *p*-values and a *p*-value threshold of .05 was used to set a limit on the total number of variables included in the final model.

3. Results

3.1. *Scoring of D-15*

None of the control participants reported to be congenitally colourblind (exclusion criteria). One patient (PL536) was excluded due to reported congenital colour blindness. Hence, the patient group ended up consisting of 63 participants. The detailed scoring of each participant can be found in Supplementary Table 2. The mean TCDS for the patient group was 137.9 (SD = 41.2), ranging from 116.9 to 332.1. Of the 63 patients, 24 had a performance indicating possible problems with colour discrimination (see Table 2 for patient scores). The mean score for this group was 171.9 (SD = 51.7). The rest of the patients (*N* = 39) had at least one perfect performance on D-15 and were therefore considered to have preserved colour discrimination. While 38 control participants had a perfect performance, eight made mistakes on the task (TCDS of CL818 = 394.4, CM309 = 142.5, CM311 = 139.3, CM321 = 149.2, CM327 = 173.7, CM328 = 177.5, CM333 = 149.2 and CM334 = 125.4). Control participant CL818 scored 394.4. This score represents such a severe outlier (see Supplementary Figure 1 for distribution of scores) that we decided to exclude the control from the analysis based on Chauvenet's criterion (Barnett & Lewis, 1994). Control CL818 performed 6.3 SD from the mean of the control group when their own score was included in the data (Mean = 128.12; SD = 42.54). As such, CL 818 must be a considered an extreme outlier that would skew the data to such a degree that true deficits in the patient group would not be detected. Following the exclusion of CL 818's data, the mean TCDS of the remaining 45 control participants was 122.2 (SD = 14.26).

3.2. *Analysis of abnormality*

Using Crawford and Howell (1998) *t*-test, one-tailed, the scores for 14 of the 24 patients were significantly different from the control group, all with $t > 1.95$, $p. \leq .02884$, zcc>1.971 (see Table 2), representing 22% of the patients included in the study. See Table 2 for single case statistics. The mean TCDS of this group was 200.32 (SD = 49.1), with scores ranging from 150.3 to 332.1. The age of the 14 patients ranged from 34 to 87 years with an average of 65.9 years (SD = 13.7) and was thus similar to the entire patient group. 13 of them had visual field defects (8 with homonymous hemianopia, 2 with quadrantanopia and 3 bilateral). As a group, the patients with significantly abnormal scores performed significantly worse that the group of unimpaired patients, who had a TCDS group mean of 120.0 (SD = 6.9), $t(13.135) = 5.878$, $p < .001$. The difference between group means was 80.33 with a 95% CI [50.83, 109.82].

3.3. *Colour discrimination patterns in the abnormal performances*

The specific colour discrimination problems and background information for the patients with significantly impaired D15 scores are summarized in Table 3. While no two patients had identical patterns of impairment, there were some similarities. Some made small mistakes mixing one or two caps (See Fig. 2a (PL505)) and others made larger mistakes and major crossovers (see Fig. 2b (PM009). All patients showed difficulties with the red-purple section in the cap arrangement, although to different extents. The green caps were confused by almost all patients and they were either confused with other green caps or with red, yellow or blue caps. Confusions were rare between the yellow caps but yellow was confused

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Table 2 – Analysis of abnormality (using Singslims.ES.exe.).

Subject	Performance score	Significance Test[a]		Estimated Percentage of the Normal Population obtaining a lower Score than the Case[b]		Estimated Effect Size (z_{cc})[c]	
		t	P (one-tailed)	Point	(95% CI)	Point	(95% CI)
PL503	**245.6**	**8.56**	***p*.<.001***	**100.00**	**(100.00 to 100.00)**	**8.654**	**(6.826 to 10.475)**
PL505	**166.7**	**3.08**	**.00175***	**99.83**	**(99.19 to 99.99)**	**3.121**	**(2.405 to 3.830)**
PL506	127.1	.34	.36779	63.22	(51.63–73.98)	.344	(.041–.643)
PL511	127.7	.38	.35234	64.77	(53.21–75.39)	.386	(.081–.687)
PL513	130.2	.56	.29089	70.91	(59.62–80.87)	.561	(.244–.873)
PL515	**166.1**	**3.05**	**.00196***	**99.80**	**(99.11 to 99.99)**	**3.079**	**(2.371 to 3.779)**
PL518	**185.5**	**4.39**	***p*.<.001***	**99.99**	**(99.97 to 100.00)**	**4.439**	**(3.467 to 5.405)**
PL526	**277.3**	**10.76**	***p*.<.001***	**100.00**	**(100.00 to 100.00)**	**10.877**	**(8.591 to 13.156)**
PL527	**170.2**	**3.33**	***p*.<.001***	**99,91**	**(99,54to 100.00)**	**3.366**	**(2.603 to 4.122)**
PL529	**208.8**	**6.01**	***p*.<.001***	**100.00**	**(100.00 to 100.00)**	**6.073**	**(4.772 to 7.368)**
PL530	125.4	.22	.41269	58.73	(47.10–69.81)	.224	(-.073 to .519)
PL538	**188.7**	**4.61**	***p*.<.001***	**100.00**	**(99.99 to 100.00)**	**4.663**	**(3.646 to 5.674)**
PL541	**183.8**	**4.27**	***p*.<.001***	**99.99**	**(99.96 to 100.00)**	**4.320**	**(3.371 to 5.26)**
PM001	**163**	**2.83**	**.00350***	**99.65**	**(98.59 to 99.98)**	**2.861**	**(2.194 to 3.52)**
PM002	**189.5**	**4.67**	***p*.<.001***	**100.00**	**(99.99 to 100.00)**	**4.719**	**(3.691 to 5.741)**
PM004	140.9	1.30	.10069	89.93	(81.79–95.61)	1.311	(.907–1.70)
PM006	130.2	.56	.29089	70.91	(59.62–80.87)	.561	(.244–.873)
PM009	**332.1**	**14.56**	***p*.<.001***	**100.00**	**(100.00 to 100.00)**	**14.719**	**(11.638 to 17.79)**
PM011	**176.9**	**3.80**	***p*.<.001***	**99.98**	**(99.86 to 100.00)**	**3.836**	**(2.982 to 4.683)**
PM015	127.1	.34	.36779	63.22	(51.63–73.98)	.344	(.041–.643)
PM018	142.6	1.42	.08206	91.79	(84.35–96.73)	1.431	(1.009–1.844)
PM019	**150.3**	**1.95**	**.02884***	**97.12**	**(92.82 to 99.32)**	**1.971**	**(1.463 to 2.470)**
PM024	142.4	1.40	.08410	91.59	(84.07–96,62)	1.417	(.997–1.828)
PM025	127.1	.34	.36779	63.22	(51.63–73.98)	.344	(.041–.643)

Note: Table lineup inspired from Crawford et al. (2010). One-tailed *t*-test, *significant at *p*.<.05.
[a] From Crawford and Howell (1998).
[b] From (Crawford & Garthwaite, 2002).
[c] From (Crawford et al., 2010).

with other colours. More than half of the impaired patients had trouble with the blue category but with varying severity. Five patients showed a pattern of an overall confusion in all hue categories (PL503, PL526, PL529, PM002 and PM009) possibly indicating a larger colour discrimination problem. Their scores ranged from a TCDS of 189.5–332.1. The five patients were also among those with the highest number of major crossovers. However, the major crossovers made by patient PM002 were different from the others, because they did not cross the diagram (see Fig. 2c). This could indicate a less severe colour discrimination impairment than in the other four patients as it was the more closely associated hues that were confused.

Three patients with abnormal TCDS had no major crossovers in their cap arrangement, also suggesting milder colour discrimination impairments. 7 out of 14 patients did not show mistakes resulting in a pattern that divided the diagram in approximately two halves as seen in congenital colour blindness. However, some patients showed a tendency towards tritanopic-like patterns. The closest performance to display this tritan pattern was patient PL503 (see Fig. 2e).

3.4. *Lesion analysis*

28.1% of patients with a left hemisphere lesion performed abnormally (9 out of 32 patients). In contrast, only one (PL505) out of 22 patients with a right hemisphere lesion showed an abnormal performance (less than 5% of this group). Patients with bilateral lesions had the highest incidence of colour discrimination difficulties (44.5%, four out of nine).

In relation to the patterns of deficits illustrated in the previous section, there were some noteworthy observations. The five patients with the highest TDCSs and mistakes involving all hues had either left hemisphere lesions ($n = 3$; PL503, PL529, and PM002) or were affected bilaterally ($n = 2$; PL526 and PM009). These two patients with bilateral damage had the highest number of major crossovers, as well as the highest TDCSs of all patients (see Fig. 3).

The mean lesion volume of patients with significantly impaired performance was 64.9 cm^3 (SD = 36.9) (range: 8 cm^3–126.14 cm^3) and was significantly larger ($t(61) = 3.604$, $p < .001$) than the mean lesion volume of the unimpaired patients (mean = 29.2; SD = 31.5). The difference between group means was 35.71 with a 95% CI [15.90, 55.53]. Interestingly, patients with high TDCS scores did not all have large lesion volumes (see Fig. 3).

Based on these results, total lesion volume and lesion laterality (unilateral vs. bilateral) were entered as independent variables along with the proportion of damage in each constituent PCA ROI (see Supplementary Table 3a) and included in a step-wise regression with D-15 scores as the dependent variable. A forward stepwise logistic regression model was able to reduce this to the following 5 variables that could be specifically related to performance on D-15 ($R = .773$,

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Table 3 – Colour Discrimination patterns and Background Information of the 14 Patients with an Abnormal Performance.

Patients	Colour Discrimination Patterns				Background information			
	TCDS score	Colour confusions	Major Crossovers	Problems involve all Colour Categories	Handedness	Age	Lesion lateralization	Size of Lesion (cm^3)
PL503	245.6	Blue & Green Green & Yellow Yellow & Red/Purple Red & Purple	3	Yes	RH	87	L	91.35
PL505	166.7	Green Green & Yellow Red & Purple	0	No	RH	69	R	126.14
PL515	166.1	Green Green & Yellow Red	0	No	RH	60	L	43.30
PL518	185.5	Blue Yellow & Red/Purple Red & Purple	2	No	LH	52	Bilateral	51.82
PL526	277.3	Green & Blue Red/Purple & Yellow Green & Red/Purple Blue & Red/Purple	4	Yes	RH	65	Bilateral	66.78
PL527	170.2	Blue/Green Yellow & Red	0	No	RH	76	L	28.32
PL529	208.8	Blue, Green & Yellow Blue & Red Red & Purple	2	Yes	RH	74	L	46.52
PL538	188.7	Green Green and Red Yellow and Purple	2	No	RH	83	L	48.64
PL541	183.8	Blue & Green Purple & Blue	1	No	RH	34	Bilateral	76.74
PM001	163	Yellow Red & Purple	2	No	RH	66	L	94.30
PM002	189.5	Blue & Green Green & Yellow Red & Purple	2	Yes	RH	51	L	90.27
PM009	332.1	Red, Green, Yellow & Blue Blue & Green Red, Yellow & Purple	5	Yes	RH	65	Bilateral	122.28
PM011	176.9	Blue & Green Green & Red/Purple Red & Purple	1	No	RH	71	L	14.63
PM019	150.3	Red & Purple	1	No	RH	70	L	8.00

Note. Color categories = Blue (from cap P to cap 3), Green (from cap 4 to cap 7), Yellow (from cap 8 to cap 10), Red (from cap 11 to cap 12) & Purple (from cap 13 to cap 15). RH: Right hand, LH: Left hand, L: Left hemisphere, R: Right hemisphere.

R^2 = .597): lesion in left occipital fusiform gyrus (beta = .251, t (57) = 2,267, p = .027), lesion in left temporal occipital sub-region of the inferior temporal gyrus (beta = .357, t (57) = 3.296, p = .002), lesion laterality (unilateral vs bilateral lesion) (beta = .327, t (57) = 3.691, p < .001), lesion in right thalamus (beta = .400, t (57) = 3.887, p < .001) as well as lesion in right posterior inferior temporal gyrus (beta = −.222, t (57) = −2.177, p = .034). Lesion size did not come out as having a significant relation to performance in this more complex analysis. The full results of the regression analysis are available in Supplementary Tables 3a, 3b and 3c. The summary statistics for each ROI for impaired and unimpaired patient groups are available in Supplementary Table 4. Visualisations of lesion overlap for the impaired patient group and the group of patients with perfect performance on D-15 respectively, as well as a subtraction plot, are presented in Supplementary Figure 2.

4. Discussion

It has been assumed that the prevalence of acquired achromatopsia is low but 14 out of the 63 patients (22%) in this study showed abnormal colour-sorting performance after a PCA stroke. Although the BoB-sample was a convenience sample and not consecutively recruited, and thus might not be entirely representative of PCA patients, the results suggest that colour perception problems occur more frequently than previously assumed. The high number is particularly interesting, as there is a chance that some patients with deficits

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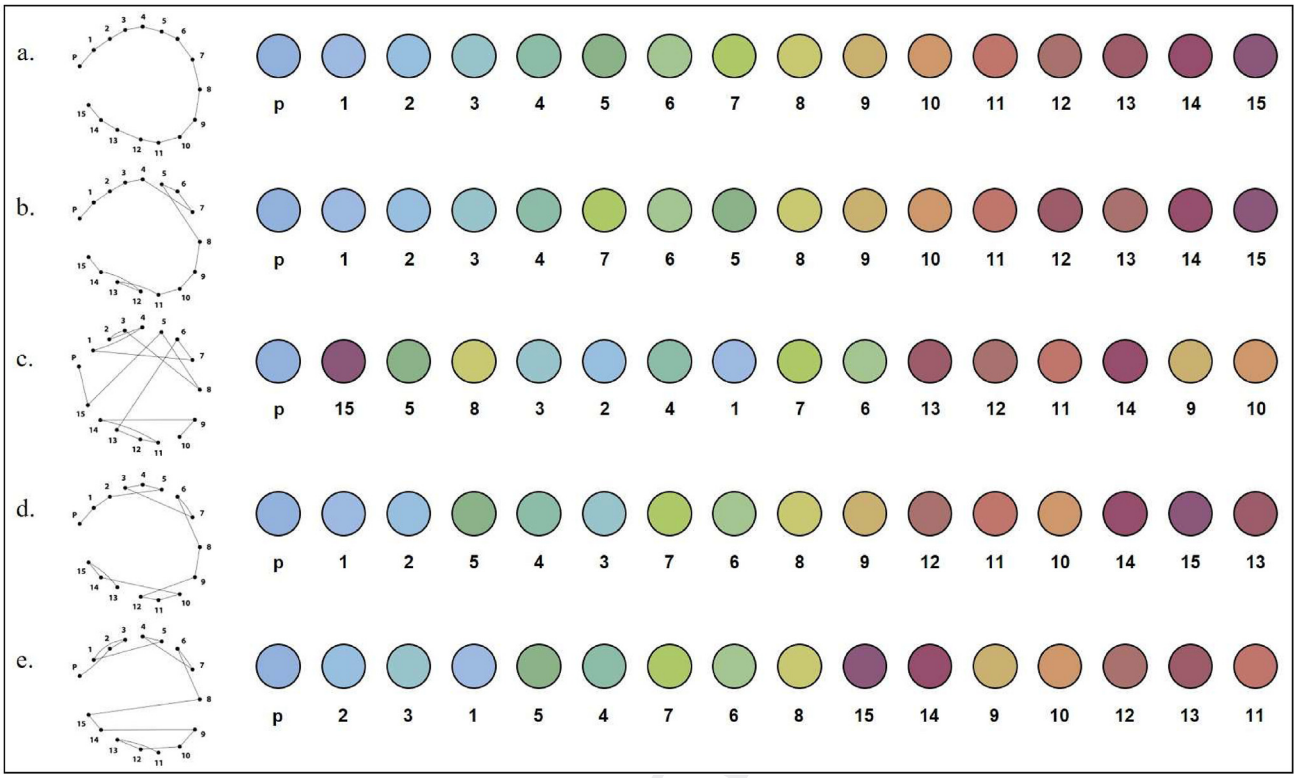

Fig. 2 – Colour diagrams and arrangements. a. Normal performance. b. PL505. c. PM009. d. PM002. e. PL503.

have gone undetected in our sample. Indeed, some patients with a known and detected colour perception impairment (using more advanced methods) have been reported to perform normally or close to normal on a variety of standardised colour vision tests (Bouvier & Engel, 2006; Koyama et al., 2006; Nestmann et al., 2021; Rizzo et al., 1993; Simunovic, 2016; Siuda-Krzywicka & Bartolomeo, 2020). The true number of cases with colour perception deficits in our sample could therefore be higher than reported here. There

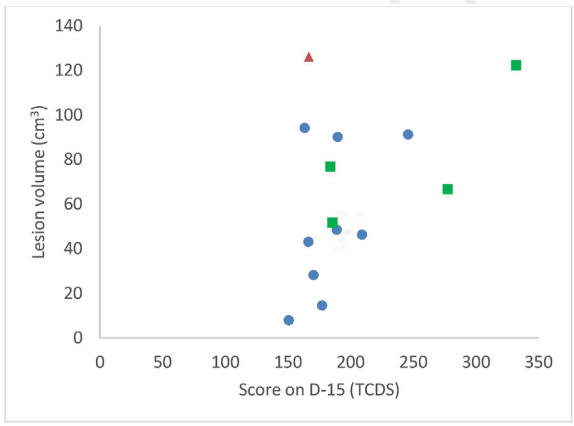

Fig. 3 – Lesion volume and TDCS score for significantly impaired patients. Patient with right hemisphere lesion illustrated with red triangle, patients with left hemisphere lesion illustrated with blue circles and patients with bilateral lesion illustrated with green squares.

can be several explanations for the high rate of colour perception deficits in this sample. This study is, as far as we know, the first to investigate colour perception deficits in a large group of patients selected on the basis of their lesions location in the PCA. Most research on colour perception deficits comes from single-case studies of patients recruited based on symptoms. However, such studies typically recruit patients with severe colour perception deficits and risk excluding milder cases. Patients with colour perception deficits after stroke do not always have insight into their difficulties (Green & Lessell, 1977; Short et al., 2011; Siuda-Krzywicka & Bartolomeo, 2020; von Arx et al., 2010). A study by Kraft et al. (2014) reported a difference between self-report of colour perception deficits (4.7%) and measured impairments (8.9%) suggesting that many lack insight in their colour perception impairments. Patients who lack insight are unlikely to be recruited for a study about colour perception difficulties based on symptoms. Another reason for an underestimation of colour perception deficits in the literature could be a lack of detection in the clinics as asking about colour perception problems is not a standard part of history taking and since clinicians rarely include a colour perception test in their standard assessment battery. Furthermore, it is plausible that the some patients do not report their colour difficulties because they have other symptoms that affect their lives more severely. All the above likely contribute to an underestimation of colour perception deficits in the clinic and in the literature. The patients in the BoB-sample have lesions varying in size and location in the areas supplied by the PCA, and have a variety of visual perceptual deficits. Therefore, we

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cannot exclude the possibility that other visual and cognitive impairments such as visual field deficits, could have contributed to some of the patient's poor performance on the D-15. However, as patients were tested minimum 9 months post stroke and as the D-15 is performed in free vision and not timed, we expect that most patients would be able to compensate for their visual field impairments. Supporting this, a number of patients with visual field deficits did not perform abnormally on the D-15.

While the control group with 21 men and 24 women was relatively balanced with regards to gender, the patient group consisted predominantly of men (52 men and 12 women). This is of relevance as congenital colorblindness is more common amongst men (approximately 8%) than women (approximately .4%). A similar gender balance between the control group and patient group would therefore have been ideal. Congenital colour blindness was an exclusion criteria for participating as a control in the study and only few control participants (3 men and 4 women) made mistakes on the D-15, and those who did, had relatively low scores (a part from CL818, see Supplementary Material for details). In addition, only one of the 52 male patients and none of the 12 female patients included reported having congenital colour blindness. Using a *Chi-square for goodness of fit*, we found that the number of male patients in our sample who were impaired on the colour perception test (14 in all: 1 self-reported and 13 with significant deficit on D-15) is significantly higher than expected based on estimates of 8% prevalence of colour blindness in the general population (χ^2 (1, $n = 52$) = 24.91, $p < .01$). Thus, it is highly unlikely that the difference in gender balance between controls and patients can explain the core findings of the present study. While it is possible that there were patients with unreported congenital colour blindness in the sample, it is more likely for most of our patients that their colour perception deficits were caused by a lesion.

The performance of the 14 patients with significant impairment ranged widely both in severity and expression, from full distortion of the colour space to problems with discriminating the finer colour nuances. Thus it seems that problems with colour discrimination after stroke in the back of the brain are quite diverse. Nevertheless, some colour hues proved more difficult than others, particularly discrimination in the red/purple hues and in the green hues as well. A prominent discussion in the literature has been whether all colours appear diluted in achromatopsia or whether selective difficulties are possible (Moroz et al., 2016). Some patients in this study did not show discrimination problems involving all hue categories (see Table 3). A few cases also had patterns with tritanopia-like errors, which are in line with other studies that reported selective difficulties in acquired cases of achromatopsia (Adachi-Usami et al., 1995; Moroz et al., 2016; Rizzo et al., 1993). Thus, some patients with impaired colour perception are still able to discriminate some colours. This result contradicts the most popular view in the literature; that acquired colour deficits are general/nonspecific. It is possible that achromatic patients with selective colour perception difficulties often remain undetected, which could explain why only a few studies have reported selective hue impairments. There is also a possibility that all hues are indeed affected but to different degrees, and that the D-15 is just not sensitive enough to detect milder deficits, making it look like some patients only have problems with some hues. Whether it is possible to have selective hue impairment remains a question for future research to investigate. Such a study would benefit using FM-100, which has more caps in the same hue but with different lightness and chroma, or D-15 followed with its desaturated counterpart.

Colour perception deficits were more common in our sample following bilateral lesions (45%) than following unilateral lesions (19%). The regression analysis also suggests that patients are more likely to have colour perception impairments following bilateral lesions than unilateral lesion. Interestingly, it seems like the distribution of the lesion (bilateral versus unilateral) is more important than total lesion volume for performance on the D-15 in our study as evidenced by the simple main effect of lesion volume (those with colour perceptual deficits vs. those without) disappearing as an important factor from the multivariate analysis, where it is subsumed by combinations of damage across PCA sites. In addition, our study indicates that colour perception impairments were far more common in the group of patients with unilateral left hemisphere lesions (28%) than in the group of patients with unilateral right hemisphere lesions (5%). In line with this result, the two regions of interest that came out as being most strongly related to poor performance on D-15 were both in the left hemisphere (left occipital fusiform gyrus and left inferior temporal gyrus). Taken together, these findings suggest that while colour perception is likely supported through bilateral system, the left hemisphere may play a more critical role in colour perception than the right hemisphere. This contradicts the majority of prior studies, which have suggested a right hemispheric dominance. Some of these studies may suffer from a selection bias that could, at least partially, account for this discrepancy. Several of the patients reported in the literature were initially recruited for prosopagnosia studies, or studies where prosopagnosia were investigated alongside with achromatopsia (e.g., Bouvier & Engel, 2006). As prosopagnosia is more common following bilateral or right hemisphere lesions (Rossion, 2014), this could have led to more patients with colour perception impairments after a right hemisphere lesion being reported in the literature. Our study avoided this bias since recruitment was based on lesion location and not symptom profile. The current study is, however, not the first to point to a possible left hemispheric dominance in colour perception. A PET-study by Zeki et al. (1991), in which 9 neuro-typical individuals were examined while reacting to visual stimuli, drew the same conclusion: Their findings showed an activation of both hemispheres, when the subjects saw colours, but the statistical analysis revealed a lateralization of function in the left hemisphere. Lueck et al. (1989) also found a greater activity in the left than right hemisphere in four neuro-typical subjects looking at coloured displays, regardless of the subject's handedness. A functional imaging study investigating colour perception in macaque monkeys has also pointed to a left lateralization of colour processing (Lafer-Sousa & Conway, 2013). The two patients in our study with the highest TCDS score both had bilateral lesions, but the two patients with the third and fourth highest score had unilateral lesions in the left hemisphere (L503 and L529). They performed badly across all colour

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categories with two and three major crossovers. Our data are not able to discern between full and partial visual field colour perception deficits but it seems unlikely that these two patients only suffer from hemiachromatopsia due to their bad performances. A left-hemisphere dominance would explain why these two patients with a unilateral left-hemispheric lesion showed difficulties typically observed following bilateral lesions.

According to the lesion analysis, lesions in the left occipital fusiform gyrus, the temporal-occipital portion of the left inferior temporal gyrus, the occipital portion of the right inferior temporal gyrus and the right thalamus are particularly strongly related to significantly poor performance on the D-15 in our sample. In our analysis, lesions in the posterior portion of the fusiform gyrus in the left hemisphere came out as being most strongly associated with poor performance on the D-15. This is interesting as the human V4-complex, commonly known as the colour center of the human brain, is considered to be located in the posterior part of the fusiform gyrus (Bartels & Zeki, 2000; Carroll & Conway, 2021). Our analysis suggests that the inferior temporal gyrus, that is directly adjacent to the fusiform gyrus, also may play a role in color perception. This could however simply reflect that a thromboembolic stroke will rarely affect the fusiform gyrus alone but will often also affect adjacent regions (bystander effect). The lateral geniculate nucleus in the thalamus is known to play an important role in receiving information coming from the ganglion cells in the retina and projecting the information to the primary visual cortex. Signals related to colour vision are known to also be transmitted through the lateral geniculate nucleus and could explain why lesions in thalamus were identified in our analysis as significantly associated with color perception impairments. While patients with significant impairments in color perception did on average have larger lesions than those without impairments, once the level of area specific damage was taken into account, total lesion size was no longer significantly related to TCDS score.

5. Conclusion

We report the colour perception performance of 63 patients with stroke in the areas supplied by the PCA, recruited on the basis of lesion location rather than cognitive or perceptual symptomatology. The results suggest that colour perception deficits are common following stroke in the back of the brain, and that the deficits can vary in severity and in expression.

Our data strongly suggest that acquired brain injury can lead to selective colour perception impairments affecting the perception of some colours and not others. In the sample, colour perception impairments were most common after bilateral lesion, and more common after lesions to the left hemisphere than the right hemisphere, suggesting that colour perception is likely supported by a bilateral yet asymmetric system that relies more strongly on the left hemisphere. In addition, analysis of the underlying anatomical regions pointed to lesions in the left occipital fusiform gyrus and the left inferior temporal gyrus as being most strongly related to poor performance on a colour perception test. These findings are particularly interesting as they challenge the majority of prior patient studies, which have pointed to a possible right hemispheric specialization. The contrast in findings could, at least partially, be explained by the recruitment method used in the current study that was based on lesions location rather than symptom profile, leading to a more representative sample. The areas in the left hemisphere that were identified as particularly relevant for colour perception in this study do however correspond to the areas in the right hemisphere that have been reported in prior studies.

Considering the high prevalence of patients with colour perception impairments in our sample, we recommend that clinicians include questions about perceiving colours during history taking with patients who have stroke in the areas supplied by the PCA. Formal assessment of colour perception should be performed in cases where colour perception impairment is suspected. The D-15 is a useful screening tool for this purpose as it is time-efficient. Ideally, however, a desaturated version of the same test should also be included in assessment to improve sensitivity (Birch, 1985). When difficulties are detected, we recommend an in-depth assessment of the patient's colour perception using additional colour perception tests such as the FM-100.

Data accessibility

The conditions of our ethics approval do not permit public archiving of anonymised imaging data. Readers seeking access to the data should contact the corresponding author Prof. Randi Starrfelt (randi.starrfelt@psy.ku.dk). Access will be granted to named individuals in accordance with ethical and data sharing procedures governing the reuse of sensitive data, and a formal data sharing agreement approved by legal consultants at University of Copenhagen must be signed by both parties. Requestors must have the necessary infrastructure to receive and store the data securely.

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Declaration of competing interest

The authors declare no competing interests.

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Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cortex.2022.12.001.

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