

## Short communication

## Measuring palinopsia: Characteristics of a persevering visual sensation from cerebral pathology

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## ABSTRACT

Palinopsia is an abnormal perseverative visual phenomenon, whose relation to normal afterimages is unknown. We measured palinoptic positive visual afterimages in a patient with a cerebral lesion. Positive afterimages were confined to the left inferior quadrant, which allowed a comparison between afterimages in the intact and the affected part of his visual field. Results showed that negative afterimages in the affected quadrant were no different from those in the unaffected quadrant. The positive afterimage in his affected field, however, differed both qualitatively and quantitatively from normal afterimages, being weaker but much more persistent, and displaced from the location of the inducing stimulus. These findings reveal distinctions between pathological afterimages of cerebral origin and physiological afterimages of retinal origin.

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## 1. Introduction

Palinopsia (or ‘visual perseveration’) is a symptom in which images of a visual stimulus persist or recur after its physical disappearance. In the *immediate type* the stimulus is immediately followed by a persistent positive or negative afterimage which gradually fades after a period of time longer than that for normal afterimages [1]. In the *delayed type* an afterimage of a previously seen object reappears after an interval of minutes to hours, sometimes repeatedly for days or even weeks [2]. There are many possible causes for palinopsia, such as drug intoxication, metabolic and psychiatric conditions [3]. In rare situations, palinopsia is caused by a cerebral lesion, usually in the right-hemisphere [1,4,5]. The anatomic correlate of palinopsia is not clear: both occipito-parietal [1,6–8] and occipito-temporal lesions have been implicated [5,9–11], Palinopsia with cerebral lesions is often [2,5] but not always [1,12] associated with visual field defects.

In spite of numerous case reports [1,5–11], the characteristics of the afterimage and the conditions that evoke it are unknown. There have been only two studies in which the characteristics of the afterimage have been investigated [2,13]. In both these studies, the cerebral lesions underlying the palinopsia were not clearly revealed by imaging. Kinsbourne and Warrington [2] concluded that the characteristics of the palinoptic afterimage were similar to the normal after-images experienced by healthy subjects, differing quantitatively rather than qualitatively.

In the present report, we describe the properties of palinopsia in a patient with a known cerebral lesion. Unlike the previously studied patients, in whom palinopsia occurred throughout the visual field [2,13], our patient’s immediate-type palinoptic afterimages were confined to the left inferior quadrant. This allowed a unique comparison between afterimages in the intact and the affected part of his visual field.

In a first set of experiments, we investigated the properties of his negative afterimages in the affected quadrant, in terms of intensity, the strength of the adapting stimulus needed to produce them, and their duration, and compared the results to those of an unaffected quadrant. In a second set of experiments, we quantified his positive palinoptic images along similar lines, measuring the intensity, the strength of the adapting stimulus, and the duration of the afterimage.

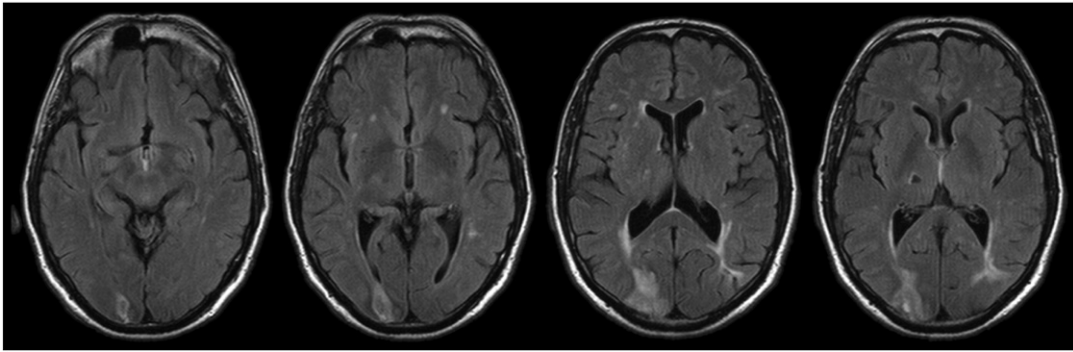
## 2. Case report

At the moment of testing, CQ was a 64 years old right-handed man with no history of psychiatric disorders nor substance abuse. He had had an intra-cerebral hemorrhage five months before testing. At the time he was examined in our laboratory for the first time, he showed normal language, memory and executive function (Table 1). There were no signs of neglect or deficits in visual perception.

Magnetic resonance images of his brain showed extensive focal cerebral gliosis. In the left hemisphere there was mainly damage to white matter in the lateral occipito-temporal region, possibly in the region of area V5 and lateral occipital cortex (LOC). On the right there was cortical damage involving V1 at the occipital pole with white matter extension to the ventricle and underlying early extrastriate V2 and V3 regions (Fig. 1). In the right hemisphere, there is also a thalamo-capsular lesion, likely unrelated to the palinopsia.

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**Fig. 1.** Axial images of the lesion of CQ (T1 weighted MRI). The left side of each slice represents the right side of the brain, and the images proceed from ventral slices on the left to dorsal slices on the right.

When shown an Amsler grid, he reported that elements in the left inferior quadrant were distorted and blurry with either eye viewing. Goldmann perimetry showed an incongruous relative defect in his left inferior quadrant, more prominent in the left eye (Fig. 2). He reported an afterimage in this quadrant, which, like the afterimages he had in daily life, he described as being *a copy of the original image*, instead of a negative afterimage (i.e. with contrast and colors reversed in polarity).

CQ explained that the afterimages in his left inferior quadrant were always present and that he frequently had to close his eyes to remove them when their persistence bothered him. A particularly vivid example was that, when driving through a tunnel, the lights in the tunnel evoked a trail of persisting afterimages (creating a form of phenomenological overlap between polyopia and palinopsia). The afterimages made reading difficult because the words previously fixated would persist. However, CQ was always confident that he could distinguish between real images and afterimages.

Informed consent was obtained prior to the study in accordance with the guidelines of the Helsinki Declaration.

In all experiments described below for both negative and positive afterimages, CQ had to fixate a central fixation point. Eye movements were monitored with an Eyelink1000 video eye-tracking system ([www.sr-research.com](http://www.sr-research.com)) and showed that CQ maintained excellent fixation. Pilot experiments confirmed that CQ only experienced abnormal afterimages with stimuli in the left inferior quadrant. We therefore chose to contrast the right superior (unaffected) and left inferior (affected) quadrants.

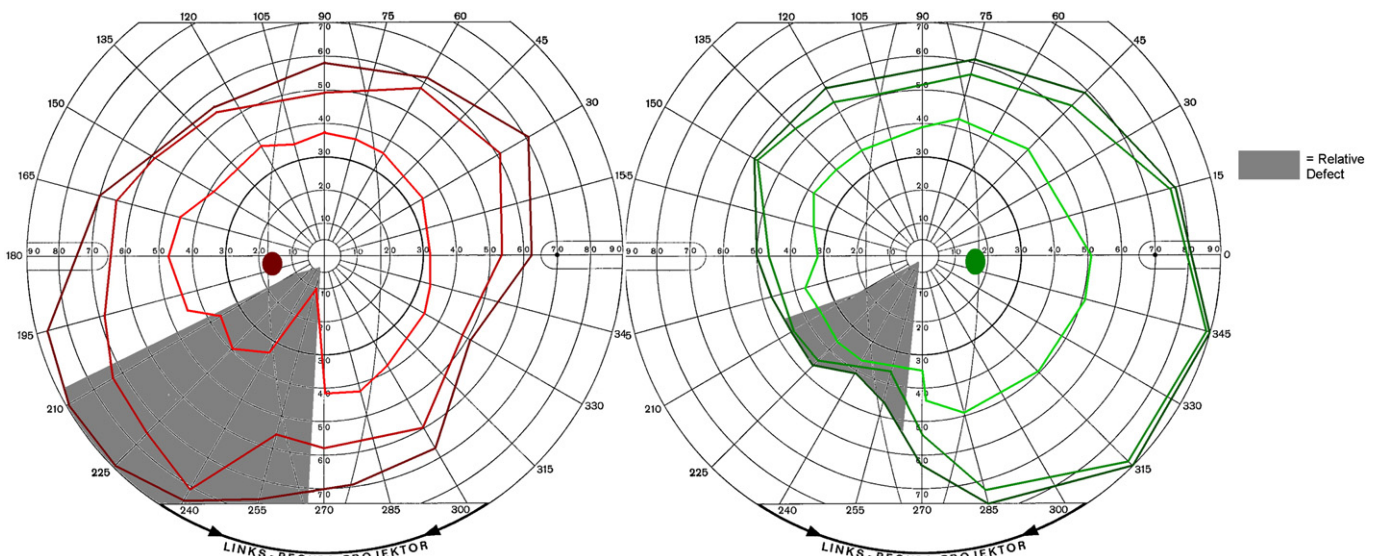
All experiments were performed in a dimly lit room, with lighting constant across Experiments 1–7. In Experiments 1–5, the background of the monitor was black ( $<0.1 \text{ cd/m}^2$ ). Experiments 6 and 7 used more eccentric stimuli projected onto a blank wall.

In all experiments, trials were blocked by location. In Experiments 1–5, stimuli were presented at  $135^\circ$  counterclockwise from vertical (left inferior) or  $45^\circ$  clockwise from vertical (right superior), at an eccentricity of  $6.3^\circ$  of visual angle. In Experiments 6 and 7, stimulus location was adjusted according to CQ's experience and instructions.

### 3. Negative afterimages (Experiments 1 to 3)

A negative afterimage is the phenomenon where exposure to a visual stimulus leads to an afterimage of opposite polarity (e.g. perceiving an illusory black spot after exposure to a white spot). Such afterimages are normal, and are believed to arise at the level of the retina [e.g. [14]]. To exclude the possibility that CQ's abnormal afterimages were mediated by abnormalities in his negative afterimage, we started by assessing his negative afterimages.

**Experiment 1.** This experiment measured the intensity of his negative afterimage using a nulling procedure [15]. CQ viewed for 12 s an adaptor that was a white spot with an intensity following a Gaussian distribution, with a standard deviation of  $1.6^\circ$  of visual angle, and a peak luminance of  $12.4 \text{ cd/m}^2$ . After an inter-stimulus interval of 1 s, a probe stimulus that was another white spot of the same size was



**Fig. 2.** Goldmann perimetry of visual fields of the patient. Perimetry showed an incongruous relative defect in his left inferior quadrant.

shown for 2.5 s, accompanied at its onset by an alerting auditory beep. CQ's task was to indicate whether he perceived this white probe spot.

The luminance of the probe spot was varied using a 1-up, 1-down adaptive staircase method [see [16]] that converged after eight reversals at the Point of Subjective Equality (the nulling point). The luminance of the probe spot was changed by a factor of 1.4 until the third reversal, and by a factor of 1.1 until the eighth reversal, the point at which the staircase terminated. The initial peak luminance of the probe was 12.4 cd/m<sup>2</sup>. The stronger the negative afterimage generated by the adapting spot, the brighter the probe spot would have to be to 'null' the afterimage. Hence the point of subjective equality is a measure of the strength of the afterimage.

**Experiment 2.** This experiment measured the duration of exposure needed to create an afterimage. For this we used the same nulling concept as above. CQ again had to indicate whether the probe spot was perceived, but now the procedure varied the duration of the adaptor using a 1-up/1-down adaptive staircase, rather than the luminance of the probe spot. The duration of the adaptor was changed by a factor of 1.4 until the third reversal, and by a factor of 1.1 until the eighth reversal, the point at which the staircase terminated. The initial duration of the adaptor was 1 s. In this experiment, the peak luminance of the adaptor was kept constant at 30.0 cd/m<sup>2</sup>, and that of the probe spot kept constant at 23.9 cd/m<sup>2</sup> (these values were based on pilot experiments on author CP). The point at which the staircase converged indicates the duration of the adaptor at which the afterimage nulled the white probe spot 50% of the time. This experiment reveals the time the adaptor needs to be present to produce an afterimage.

**Experiment 3.** This simply measured the duration of his negative afterimage. CQ was instructed to press a button when the black afterimage (occurring after adaptation to the white spot) was gone. Thus no probe spot was involved. The adaptor spot had a peak luminance of 58.3 cd/m<sup>2</sup>. We measured five durations for each of the two locations.

#### 4. Results

**Experiment 1** showed that the intensity needed to null the afterimage was 12.5 cd/m<sup>2</sup> (s.d. 0.9 cd/m<sup>2</sup>) in the unaffected right superior quadrant and 10.2 cd/m<sup>2</sup> (s.d. 2.0 cd/m<sup>2</sup>) in the affected left inferior quadrant. A paired sample *t*-test revealed that the strength of CQ's negative afterimage did not differ between the affected and unaffected quadrants ( $t(3) = 1.6, p = 0.2$ ).

**Experiment 2** showed that the adaptor needed to be present for 3.7 s (s.d. 0.9 s) to null a white probe spot in the right superior quadrant, and for 3.0 s (s.d. 0.3 s) in the left inferior quadrant. Again, a paired *t*-test showed that there was no difference between these two quadrants ( $t(3) = 2.3, p = 0.1$ ).

**Experiment 3** revealed that the mean duration of CQ's negative afterimages was 11.3 s (s.d. 2.9 s) in the right superior quadrant and 15.9 s (s.d. 4.0 s) in the left inferior quadrant. The difference between the results of the two quadrants was not significant ( $t(4) = -1.9, p = 0.1$ ).

#### 5. Positive afterimages (Experiments 4–7)

Experiments 1–3 show that CQ's negative afterimages at the affected region of the visual field were no different in strength (Experiments 1–2) and duration (Experiment 3) from those at his unaffected region. In the experiments below, we assessed CQ's positive afterimages parametrically. To be able to distinguish positive from negative afterimages, we chose to use a green adapting stimulus, so it would be easy for him to label his perceived afterimage. A nulling procedure as we used to assess his negative afterimage could not be used, because CQ stated that his positive afterimages were displaced

with respect to the stimulated location. Therefore, we devised other experimental methods to investigate CQ's positive afterimage.

**Experiment 4.** Whereas Experiment 1 measured the intensity of the negative afterimage, in this experiment we assessed the intensity of the positive afterimage. CQ matched the appearance of the afterimage with that of a probe stimulus presented at fixation. The probe stimulus was presented in either the left inferior or right superior quadrants, at the same locations used in Experiment 1–3. The probe presented at fixation was a green Gaussian spot (standard deviation of 1.6 deg, luminance = 4.1 cd/m<sup>2</sup>,  $u' = 0.1, v' = 0.6$  in CIE 1976 color space) of the same size as the adaptor. As in Experiment 1, an adaptor spot was presented for 12 s (with same luminosity and hue as the initial value of the probe spot). One second later, the probe stimulus appeared at fixation, accompanied by a beep. CQ's task was to indicate whether the probe was brighter than the afterimage produced by the adaptor. The intensity of the probe presented at fixation was varied using a 1 up/1 down staircase. The luminance of the probe was changed by a factor of 1.5 until the third reversal, and by a factor of 1.1 until the eighth reversal, the point at which the staircase terminated. The point at which the staircase converged indicates the intensity of the probe stimulus at fixation that matched the intensity of CQ's afterimage.

**Experiment 5.** This measured how intense the adaptor had to be to produce a positive afterimage. The size of the spots was also identical to those used in Experiments 1–4. In this experiment we varied between trials the luminance of a green adaptor, again using a 1 up/1 down adaptive staircase. The luminance of the adaptor was changed by a factor of 1.4 until the third reversal, and by a factor of 1.1 until the eighth reversal, the point at which the staircase terminated. The initial luminance of the adaptor was 4.1 cd/m<sup>2</sup> ( $u' = 0.1, v' = 0.6$  in CIE 1976 color space). The adaptor's duration was kept constant at 12 s. After an interval of 1 s following the disappearance of the adaptor, an auditory beep sounded, signaling to CQ that he had to indicate whether or not he saw a *green afterimage*. The point at which the staircase converged indicates the adaptor luminance at which CQ reported a positive aftereffect in 50% of the trials.

**Experiment 6.** CQ reported that the palinopsia in daily life was stronger the more eccentric the stimuli were in his left inferior quadrant. Therefore, we performed a variant of Experiment 3, assessing the duration of the afterimage, using an image projected onto a wall at a distance of 220 cm from CQ, rather than displayed on a computer monitor. First, we asked CQ to indicate the location where he believed his afterimages to be maximal, which was at an eccentricity of 14° of visual angle, along the diagonal in the left inferior quadrant, or 135° counterclockwise from vertical. Next, we assessed the duration of his positive afterimage at this location. For two trials, we presented an adaptor that was a green Gaussian spot, with increased size (standard deviation of 2.5° of visual angle), peak luminance 8.41 cd/m<sup>2</sup>, ( $u' = 0.169, v' = 5.43$ ) for 12s. The luminance of the background was 4.0 cd/m<sup>2</sup>. CQ's task was to report via a key press when the positive afterimage had disappeared.

**Experiment 7.** As CQ reported that his abnormal afterimages were not located at the same position as the original image, we mapped the spatial offset of his afterimage. For this, we presented an adaptor for 12 s at an eccentricity of either 14.4° or 18.9° of visual angle. In the three trials for each location, CQ had to indicate where the afterimage was perceived, by verbally guiding a pencil held by the experimenter, who marked on the wall where the after image was reported.

#### 6. Results

**Experiment 4.** CQ did not perceive a positive afterimage in the right superior quadrant in either this or the following experiments.

**Table 1**  
Performance of CQ on standard neuropsychological tests (5 months post-stroke).

Cognitive domain	Neuropsychological test	Performance	Percentile/decile	
Language	Boston naming test	175/180	93rd percentile	
	Verbal fluency			
	A	21	90–95th percentile	
	N	21	95th percentile	
	Semantic fluency	38	72nd percentile	
Working memory	Token test	21/21		
	Digit span	11	75th percentile	
Memory	RAVT immediate	3/6/12/9/12	9th decile	
	RAVT delay	9	8th decile	
	RAVT recognition	30/30	No abnormalities	
	Rey delay	14.5	35–50th percentile	
Executive functions	BADS-NL rule shift cards	4		
Visual perception	Judgment of line orientation	27/30	72nd percentile	
	Facial recognition test	45/54	33–59th percentile	
	Corvist			
	Symbol acuity	33/36		
	Shape discrimination	4/4		
	Size discrimination	2/2		
	Shape detection	4/4		
	Hue detection	4/4		
	Scattered dot counting	4/4		
	Fragmented numbers	4/4		
	Face perception	8/8		
	Crowding	4/4		
	Rey copy	33.25	50th percentile	
	Neglect	Star cancellation	54/54	

For the left inferior location, a probe stimulus at fixation of 0.41 cd/m<sup>2</sup> (s.d. 0.1 cd/m<sup>2</sup>) matched that of the positive eccentric afterimage perceived by CQ.

**Experiment 5.** The luminance of green needed to generate a positive aftereffect in the left inferior quadrant was 1.0 cd/m<sup>2</sup> (s.d. 0.9 cd/m<sup>2</sup>). Thus, a light of very small intensity is enough to produce a positive afterimage.

**Experiment 6.** The duration of CQ's positive afterimage at the left inferior location was 121 s (s.d. 3.5 s).

**Experiment 7.** When the adapting stimulus was presented at an eccentricity of 14.4° of visual angle along the diagonal meridian, thus corresponding to 10.2° left and 10.2° down from fixation, the positive afterimage occurred 13.7° (s.d. 1.5) left and 20.5° (s.d. 0.1) down from fixation (Fig. 3). An adapting stimulus at an eccentricity of 18.9° of visual angle, 13.3° left and 13.3° below fixation, produced an afterimage at 21.5° (s.d. 0.7) left and 20.3° (s.d. 1.7) below fixation. These results confirm a displacement of the afterimage to a more eccentric location in the affected quadrant.

## 7. Discussion

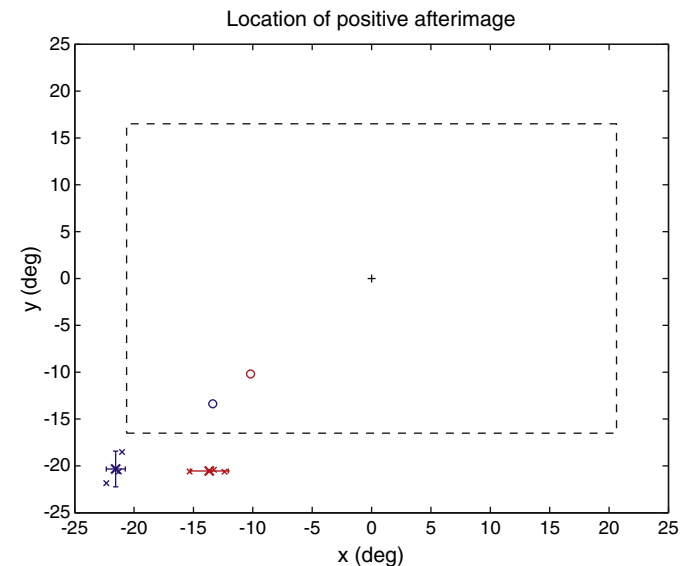
We studied the properties of palinopsia due to a known cerebral lesion, in which positive afterimages were confined to a region in the lower left quadrant. The results of Experiments 1–3 show that the negative afterimages in CQ's affected quadrant were no different from those in his unaffected quadrant. The experience of an immediate-type palinopsia is therefore not necessarily accompanied by distortion of normal adaptive phenomena, in particular when the palinoptic images are positive rather than negative afterimages. The aim of Experiment 4–7 was to provide objective measures of CQ's positive palinoptic afterimages, concerning their intensity, duration and location. The results show that the palinoptic afterimage could be evoked by an adaptor of relatively low intensity (1.0 cd/m<sup>2</sup>). CQ was able to match the intensity of his positive afterimage with a real physical stimulus, showing that his palinoptic afterimage was

about 10% of the brightness of the stimulus inducing it. This contrasts with the negative afterimages in his affected and unaffected quadrants, which were almost as strong as the stimuli inducing these. Importantly, his positive palinoptic afterimages lasted for over two minutes, 7 to 10 times as long as his negative afterimages, and were systematically displaced from the inducing location, in distinct contrast to the highly retinotopic nature of normal negative afterimages.

Palinopsia has previously been explained by damaged connections that prevent inhibition of signals to neural systems subserving vision [2]. This inhibition is necessary to remove a stimulus from visual awareness once it is no longer present in the visual environment. Kinsbourne and Warrington [2] stated that release from this inhibition results in overactivity of the visual system, which causes the observed positive visual phenomenon. The spatial displacement of the afterimage from the inducing stimulus location in our case suggests that on occasion this loss of inhibitory connections may result in overactivity of neighboring neurons in the visual cortex. The existence of these inhibitory signals has also been proposed in explanations of saccadic suppression, in which the brain cuts off the processing of blurry images during an eye movement [17].

There are two possible mechanisms for the lack of inhibitory signals in the visual cortex. First, the neural areas responsible for sending inhibitory signals to the visual cortex may be damaged, resulting in the sustained visual experience of the stimulus. Second, the relative field defect at the location of the afterimage may prevent or reduce transmission of the visual information required to generate the inhibitory signals. Regardless, the location of the brain lesions in CQ appears to implicate lower visual areas as responsible for these inhibitory mechanisms.

The current study aimed to quantify the visual afterimage in a patient with palinopsia caused by brain damage. So far, most studies have merely described the phenomenon without providing quantitative details about the afterimage. Our results concur in part with previous conclusions that palinoptic afterimages show quantitative differences with normal afterimages [2,13]: CQ's palinoptic afterimages were inducible by weaker stimuli, fainter, but lasted longer than his own normal negative afterimages. However, besides being positive rather than negative afterimages, they also showed



**Fig. 3.** Perceived locations of CQ's positive afterimage in visual angle. The + indicates the location of the fixation cross; the dashed line the limits of the projected background. Adapting probes were presented at the location of the red and green circle. Crosses indicate the location of the perceived afterimages (red for the red adaptor location, blue for the blue adaptor location). The small crosses indicate the perceived locations at individual trials, the large crosses and error bars the average and standard deviations respectively.

qualitative differences from normal phenomena, particularly in the systematic spatial displacement of the afterimage in relation to the inducing stimulus. Hence these results indicate distinctions between pathologic afterimages of cerebral origin and physiologic afterimages of retinal origin.

### Conflict of interest

There is no conflict of interest.

### Acknowledgments

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