



Spatial characteristics of cerebral polyopia: a case study

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Abstract

A 41-year-old woman showed bilateral monocular polyopia and an incomplete, right-sided homonymous hemianopia following bilateral cerebral strokes confirmed by neuroimaging. She was tested with briefly-presented visual stimuli to determine whether her polyopic images varied with visual field position of stimuli which evoked them. Stimuli close to her scotoma elicited polyopic images at shorter latency and higher probability than did stimuli more distant from it. RS could maintain stable fixation on small stimuli, suggesting that eye movements were not responsible for her polyopia. We discuss the possibility that cerebral polyopia is due to recoding of visual receptive fields in primary visual cortex and that bilateral occipital lesions are a causative factor in the genesis of the disorder. © 1998 Elsevier Science Ltd. All rights reserved.

Keywords: Hemianopia; Occipital lobe; Polyopia; Stroke; Visual field

1. Introduction

Diplopia or 'seeing double', is a relatively common visual complaint typically caused by a misalignment of the two eyes. Rarely, the complaint is monocular, i.e. seen with either eye or a particular eye closed, a condition most often seen with a refractive error or cataract. Monocular polyopia after cerebral damage is an extremely uncommon complaint. It is distinguished from palinopia, another the rare and poorly understood abnormality, in which visual images persist or reappear when the original stimulus is no longer present [1,2]. Since monocular diplopia following cerebral damage is rare, its etiology, characteristics and cerebral pathology are not well described. Occipital lesions, verified either by the entrance site of missile wounds [3,4] or by brain scans [2] appear to be a common feature of polyopia. Visual field defects [2–7] or transient visual hallucinations [8,9] are consistently reported in cases of polyopia. Bilateral cerebral lesions are associated with this disorder in many cases [2–4,6]. There is evidence that poly-

opic images are experienced while viewing stimuli that are simple or complex [7]. Much less, however, is known about the spatial distribution of this disorder, in particular, how polyopia varies with stimulus position in the visual field, a factor that has not been controlled in the studies cited above. In addition, it is not clear whether there is a relationship between direction in which polyopic images appear and the position of visual field defects. Polyopic images forming in the direction of the scotoma and extending into it have been reported in two cases [2,6]. However, the patients described by Bender [3] and by Sobin and Bender [8] reported polyopic images formed in other parts of the visual field.

Since the spatial aspect of cerebral polyopia is a basic feature of the disorder, its systematic study may shed light on the underlying pathophysiology. In the present study we investigated the likelihood of duplicated images and the speed with which they appear when stimuli are presented in different parts of the visual field of a patient with bilateral, monocular polyopia, unilateral visual field defects and bilateral occipital infarctions. Prompted by the observations of Gottlieb [7], we also investigated whether her polyopia varied with the complexity of visual stimuli.

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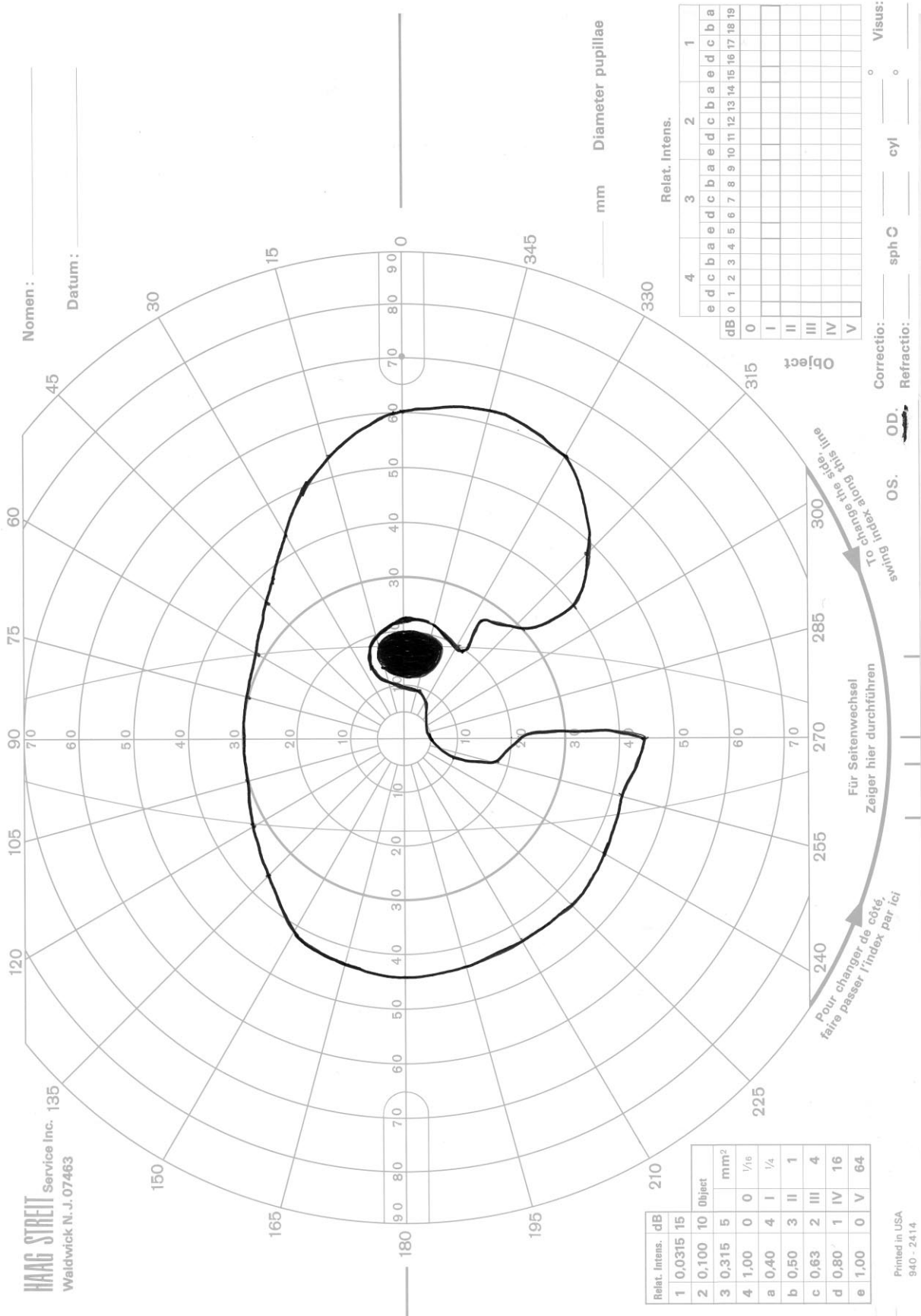


Fig. 1. Goldmann visual fields showing congruous right inferior scotoma.

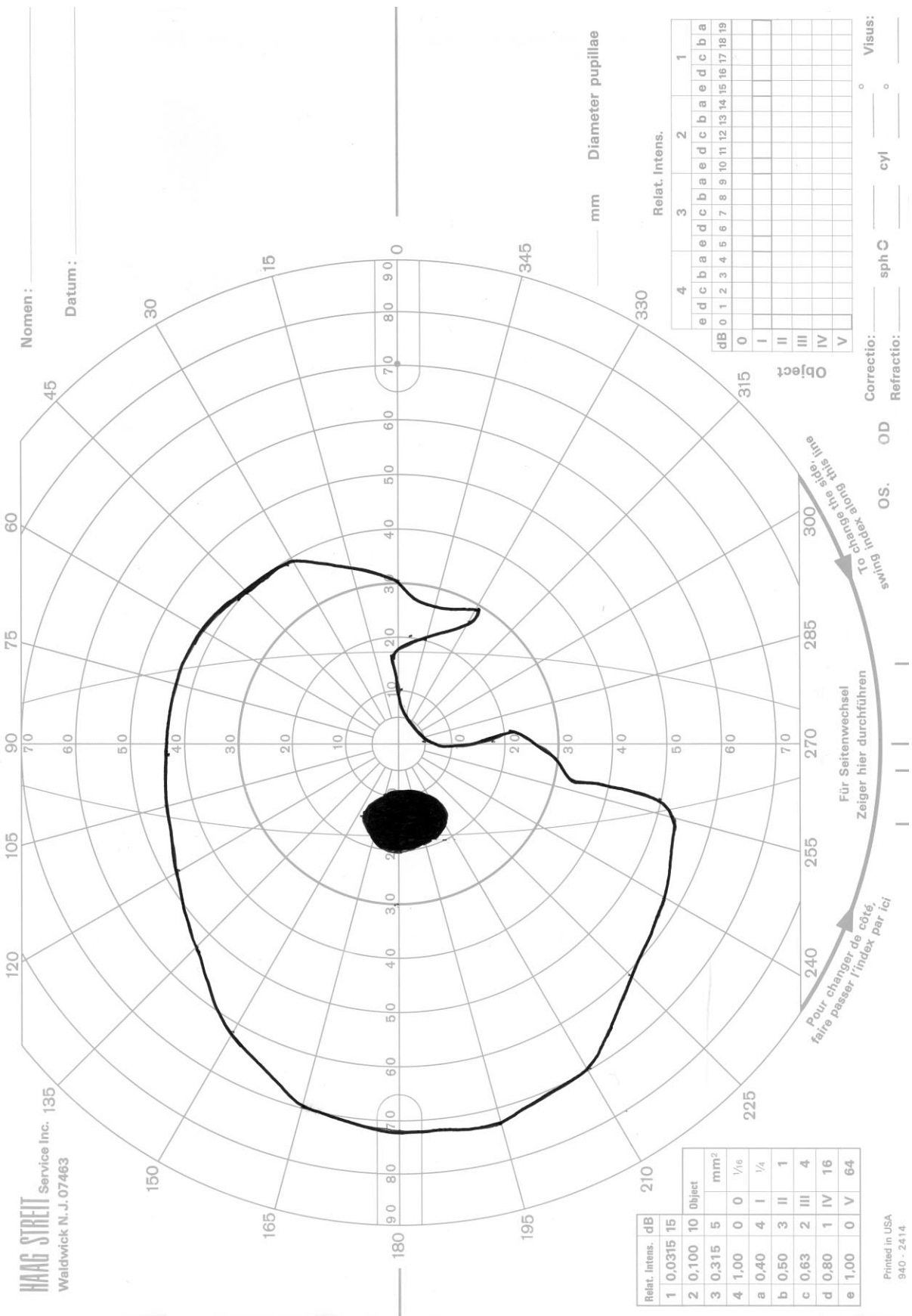


Fig. 1. (Continued)

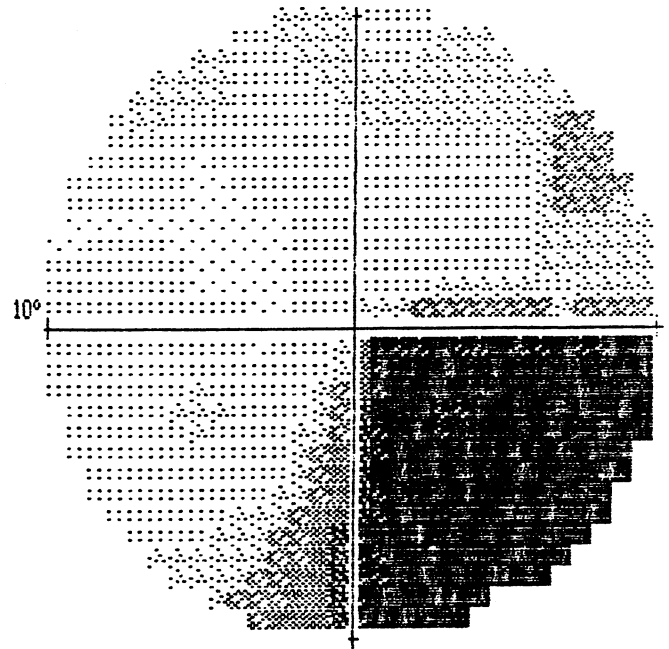
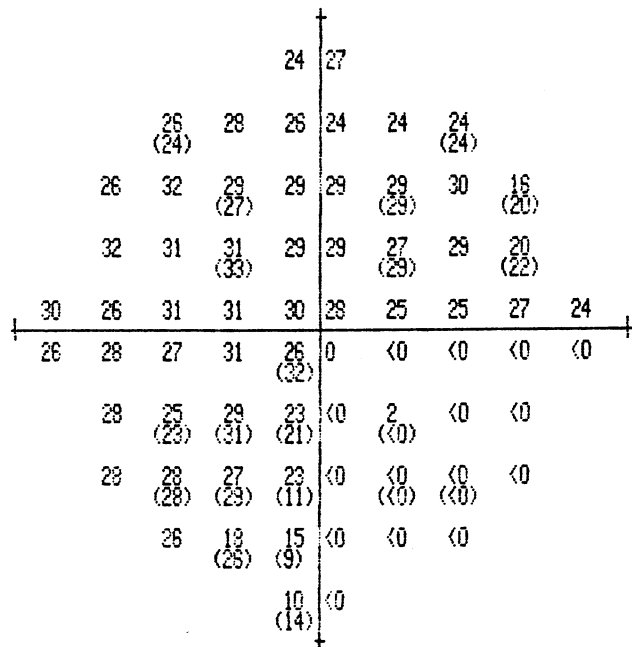


Fig. 2. Humphrey central 10-2 threshold test showing congruous right inferior scotoma.

2. Case history

RS, a 41-year-old, right handed woman, was initially seen on 25 November 1992 for evaluation of polyopia. On 2 November 1992 she had a cholecystectomy complicated by pancreatitis and severe nausea and vomiting. She was treated with nasogastric drainage, Phenergan™, Dramamine™, meperidine and phenobarbitol for 4 days. She recalls getting up to go to the bathroom and passing out during this time. When the medications were withdrawn, she complained of seeing ten copies of objects she looked at; they appeared in a row located down and to the right of the fixated object.

This occurred both at distance and near and with monocular as well as binocular viewing. She would see multiple copies several seconds after she opened her eyes or changed fixation. She described one episode lasting several hours during which she looked at her hands and saw approximately 100 little hands.

A CT of the head done at this time showed bilateral occipital infarctions, left greater than right. MRI done 2 months later showed left greater than right occipital volume loss (see Fig. 3). Three weeks after onset of her symptoms, she described seeing three or four duplicated images of fixated objects. On neuro ophthalmologic examination, best-corrected visual acuity was 20/25 in each eye and color vision was mildly reduced bilaterally. Stereo vision, pupillary reaction, biomicroscopic examination and fundoscopic examination were all normal. Extraocular movements were full and she was orthophoric. Goldmann visual field testing showed a

congruous right, inferior quadrantanopsia (see Fig. 1). This was also seen on Humphrey automated perimetry with a 10-2 program (see Fig. 3). We did not look specifically for subtle changes, such as fluctuations, obscurations or distortions in the appearance of objects in the patient's left visual field, nor did she spontaneously report any.

Over the next 3 years, her visual acuity improved to 20/15 in each eye, color vision became normal and the visual field defect remained unchanged. Her polyopia persisted during this period. She developed migraine headaches that responded to amitriptyline therapy.

3. General methods

3.1. Apparatus and materials

RS was tested in four sessions while viewing two-dimensional stimuli in a two-field tachistoscope (Gerbrands, model T10-9). The fixation mark (a star, approximately 0.5° in diameter) was presented in one field; all other stimuli were presented in the second field. Each field was 8° square. Stimuli were drawn with black ink on white poster board. The luminance of the field displaying the fixation mark was 11.9 cd/m^2 ; that of the field displaying the stimulus was 11.5 cd/m^2 . The timing of exposure durations of 1 s or less was controlled by motor-driven cam, accurate to approximately 2%. Exposure durations longer than 1 s were controlled manually by the experimenter.



Fig. 3. T1 weighted MRI showing bilateral occipital infarcts, left greater than right.

3.2. General procedures

RS was instructed to place her face against a cushioned support on the tachistoscope and to look at the fixation mark located in the center of the field at the beginning of each trial. Only RS's right eye was used for viewing; this put her scotoma in the larger temporal field rather than in the smaller nasal field. The experimenter then gave her a signal that the stimulus would soon appear. Approximately 1–3 s later, the experimenter operated a switch that extinguished the first field and simultaneously exposed for a predetermined period of time the second field containing the stimulus. If the stimulus was eccentric to fixation, a fixation mark identical to the one in the first field was also present in the second field. If a stimulus was presented at fixation, no fixation mark was present. RS was instructed to continue fixating the fixation mark, or, in its absence, to fixate the centered stimulus. At the end of each trial, she was asked to describe the stimulus and any replicated images of it, which could take the form of a duplicated (i.e. single) image or multiple duplications of the stimulus. She had no difficulty telling the difference between the physical stimulus and replicated images of it, because the latter were delayed in relation to the stimulus. RS was instructed to make the following judgements: (a) only the stimulus was present; (b) a

duplicate image of the stimulus began to form but was not completed; (c) a completely formed duplicate image was present; (d) multiple images of the stimulus were present. RS was tested on four occasions: 11 August 1993, 27 April 1993, 28 September 1994 and 24 March 1995.

3.3. Statistics

Analyses involving more than one factor and their interactions were performed with repeated-measures analyses of variance (ANOVAs). Differences between means were analyzed with *t*-tests (two-tailed); differences between frequencies were analyzed with the χ^2 statistic (two-tailed). Probability levels associated with multiple comparisons were adjusted by Ryan's Bonferroni correction factor [10].

4. Specific procedures and findings

The tests conducted in the first session were designed to explore the effects of three factors on RS's replication of visual stimuli: stimulus complexity, stimulus position in the visual field and duration of stimulus exposure.

4.1. Session 1

4.1.1. Procedures

A solid circle, 0.5° in diameter, was tachistoscopically presented either at central fixation or 2° extrafoveally in each quadrant (midway between the horizontal and vertical meridians) at six exposure durations; 5.0, 2.0, 1.0, 0.9, 0.8, 0.5 s. The circle was presented three times at each of the 30 combinations of exposure duration and visual field position. In addition, the following stimuli were presented at fixation: A solid vertical bar, 3° in height and 0.5° in width, four line drawings of a single object (truck, clothes pin, flower, racoon), all approximately 2° in vertical and horizontal extent, and a scene showing a person being robbed, approximately 4° in vertical and horizontal extent. Each of these stimuli was presented three times at each of the following durations: 5, 2 and 1 s. The order in which different stimuli and the visual-field position of the circles were presented was randomized at each exposure duration, which was successively reduced from 5.0 to 0.5 s (see Table 1).

4.1.2. Results

RS reported replicated images of the circle on 95% (86/90) of the trials when it was presented, averaged over the five visual-field positions (see Table 1). These images, like those she reported when viewing objects outside the testing situation, appeared successively and to the right of the physical stimulus. Although the frequency with which she reported replicated stimuli did not change markedly with stimulus position, her reports of replication were somewhat (although not significantly) more frequent when the circle appeared to the right of fixation than when it appeared to the left of fixation or at fixation. She also reported multiple images of the circle when it was presented in the lower right quadrant with significantly greater frequency (61%, 11/18) than she did when it was presented in the lower left quadrant (11%, 1/18) ($\chi^2(1) = 7.56$, $P < 0.01$), in the upper right quadrant (11%, 1/18) ($\chi^2(1) = 7.56$, $P < 0.01$) or at fixation (0/18; frequency too low to statistically analyze). Furthermore, she reported that the multiple images of the circle presented in the lower right quadrant were formed rapidly and successively, and appeared as a train of images eventually extending all the way to the lower right corner of the stimulus field (5.7° from fixation). Exposure duration did not alter the frequency with which RS replicated the circle; her reports of replication were as frequent at the shortest exposure (0.5 s) as they were at the longest (5 s).

RS also reported replicated images of the vertical bar and of other objects exposed at all durations. The only exceptions to this finding were on one presentation each of the clothespin and truck. When viewing the robbery scene, she reported multiple images only of the dollar

sign (approximately 0.5° in extent), which was centered on fixation and prominently displayed on the bill that the man is handing to the robber.

4.2. Session 2

In this session, we investigated whether RS's tendency to replicate stimuli was dependent upon; (a) the eccentricity of stimuli from central fixation; (b) the direction of the stimulus relative to central fixation; and (c) the role of eye movements in her polyopia. To investigate the first two factors, we varied the distance from central fixation of small stimuli presented on the horizontal meridian, to the left and right of fixation. To investigate the third factor, we recorded RS's horizontal eye movements while viewing a small stimulus on a computer screen in order to determine; (a) whether there was a relationship between RS's polyopia and the direction of her eye movements, as previously suggested [3,7]; and (b) whether she was able to maintain central fixation when there was no fixation stimulus present, a procedure employed in the following two sessions. Eye movements could not be recorded while using the tachistoscope because of its design.

4.2.1. Procedures

In the first part of the session, RS was presented with black circles of 0.5 and 0.25° diameter on the horizontal meridian and exposed for 3.0 s along with a fixation mark in the tachistoscope. The center of the 0.5° circle was located 0.375° to the left and right of the fixation stimulus (its inner border was next to the fixation stimulus). It was also presented with its center 1.0 , 1.5 and 2.0° to the right of fixation and 0.67 and 1.0° to the left of fixation. The 0.25° circle was presented 1.0 and 1.5° to the right of fixation and 0.5 , 0.67 and 1.0° to the left of fixation. These stimulus positions were chosen on the basis of RS's reports in an exploratory series of stimulus presentations, following which each of the combinations of stimulus size and position were presented three times in a randomized order. The total number of presentations of each combination of stimulus size and position varied between seven and thirteen. Otherwise, the testing procedures were the same as those described above (see Section 3.2). RS was instructed to report after each stimulus exposure whether she perceived replications of the target stimulus (i.e. the circle), or the fixation stimulus, or both, and the direction of the replicated images relative to the stimulus.

In the second part of this session, RS's horizontal (right) eye position was monitored by infrared scleral reflection with an Eye-trac (model 210) eye-movement recording system with a resolution of 0.25° and accuracy of 1° . The voltage output of the horizontal detector was linear over a range of 15° lateral to the centered position of the eye. The eye position signal was digi-

Table 1
RS's frequencies, percent responses to single circle session 1. Stimulus position^a

Exposure (s)	Stimulus position																								
	Fix			Right upper quadrant			Right lower quadrant			Left upper quadrant			Left lower quadrant			Total									
	S	DF	M	S	DF	M	S	DF	M	S	DF	M	S	DF	M	S	DF	M							
5.0	0	1	0	0	1	2	0	0	0	3	0	0	0	3	0	0	1	2	0	2	11	2	0		
2.0	0	3	0	0	0	3	0	0	0	0	3	0	0	0	3	0	0	0	3	0	0	6	3	3	
1.0	0	1	2	0	0	1	1	1	0	0	3	0	0	1	0	2	0	2	0	1	0	5	3	7	
0.9	0	1	2	0	0	1	2	0	0	1	0	2	0	3	0	0	0	3	0	0	0	9	4	2	
0.8	0	0	3	0	0	3	0	0	0	1	0	2	1	2	0	0	0	2	1	0	0	1	8	4	2
0.5	1	2	0	0	0	1	2	0	0	2	0	1	0	1	2	0	0	3	0	0	0	1	10	4	1
Totals	1	10	7	0	0	10	7	1	0	4	3	11	1	10	5	2	0	11	6	1	0	91	(left)		
Percent replications	94			100 (right)																					

S, stimulus only; DF, duplicate image forming; D, completely duplicated image; M, multiple images (two or more).

^aAll eccentric stimuli centred 1° from fixation.

tized at a sampling rate of 500 Hz by a computer (Zenith Superport 286 computer) which stored the eye position signals. During testing, RS was seated with her chin in a chin rest facing the computer screen, which was located approximately 24 cm in front of her. Horizontal eye position was monitored and recorded while RS was instructed to look at stimuli presented at the center of the computer screen, which measured 20.5 cm in width and 15.5 cm in height.

The first eye-movement recording test was performed to determine whether RS's eyes tended to drift predominantly in one direction (left or right) while she was attempting to maintain fixation. She was instructed to continuously fixate the center of a 1° outline circle for as long as it was present (5 s) on the screen. The circle was presented 12 times. She also was asked to report whether or not she saw polyopic images of the circle. The predominant direction of eye-position drift during fixation was defined as the direction in which the eye-position signals changed monotonically in at least 75% of the time period during which she looked at the circle without blinking. In the second test, RS's fixation of a central stimulus and saccades to peripheral visual targets were recorded. She was instructed to fixate a small cross, 1° in diameter, when it was presented at the center of the computer screen. After she fixated within 2° of the cross for 300 ms, the target stimulus appeared 10° to the left or right of the fixation mark, vertically centered on the screen. The target was a white rectangle 0.5° in height and 0.5° in width. RS was instructed to move her eyes to the target as soon as it appeared. Targets were presented 32 times to each side; the order in which they were presented on the two sides was randomized. Saccade latencies (the time from the appearance of the target to initiation of a saccade) were estimated by examining the stored voltage signals with the aid of a program that displayed the time (in ms) from appearance of the target to saccadic initiation, estimated with a moveable cursor. Latencies shorter than 100 ms, which might be due to anticipation of the peripheral target rather than its appearance, were not included in the analyses.

4.2.2. Results

On all tachistoscopic trials, RS reported completely formed polyopic images (for the most part, duplications) of the 0.5 and 0.25° stimuli presented left and right of fixation. In all cases, she reported that the images were completely formed. In addition, she consistently reported replicated images of stimuli that were presented at more eccentric positions on the right than on the left side of the central fixation stimulus (see Table 2). This asymmetry is seen more precisely with the smaller than with the larger diameter stimuli. For the 0.25° stimuli, the limit of her replicated images was 1° to the right of fixation, but only 0.5° to the left of

fixation. Furthermore, she reported that duplicate images of the target stimulus always appeared to the right of the stimulus. This was also the case for images of the fixation stimulus, which she reported on all trials.

During eye position recording, RS reported that she saw duplicate or multiple images of the outline circle, whose center she was instructed to fixate, each time it was presented. These images, like those of other stimuli, appeared to the right of the stimulus. On six of the 12 occasions on which the circle was presented, blinking interrupted the eye position signal too frequently to determine the direction in which her eyes drifted. Of the remaining six occasions, 14 epochs that were sufficiently long (at least 600 ms) to examine drifts in eye position. Six epochs occurred prior to any blinks; eight occurred after one or more blinks. The mean duration of these epochs was 1.1 s (S.D. = 0.42; range = 0.68–2.93 s). The predominant direction of eye position drift was to the right on five epochs and to the left on nine epochs, a difference that was not significant. The mean range of these drifts was 0.68° (S.D. = 0.46); there was no difference between the ranges of leftward and rightward drifts. RS fixated to the right of the center of the circle on five of the 14 epochs and to the left on the remaining nine epochs. However, these deviations from the true center of the circle were small: The average maximum deviation of RS's gaze from the center of the circle was 1.72° (S.D. = 0.31). Similarly, when she was tested for visually-guided saccades, RS's maximum deviation of gaze from the center of the fixation mark averaged 1.52° (S.D. = 0.71). The latencies and amplitudes of her saccades were in the normal range and symmetric.

4.3. Session 3

In this session, tests were undertaken to further investigate the likelihood and speed with which RS repli-

Table 2
RS's responses to stimuli session 2

Target size	Target position	Target stimulus duplicated?
0.5°	Adjacent right	Yes
	1° right	Yes
	1.5° right	Yes
	2° right	No
	Adjacent left	Yes
	0.67° left	Yes
0.25°	1° left	No
	1° right	Yes
	1.5° right	No
	0.5° left	Yes
	0.67° left	No
	1.0° left	No

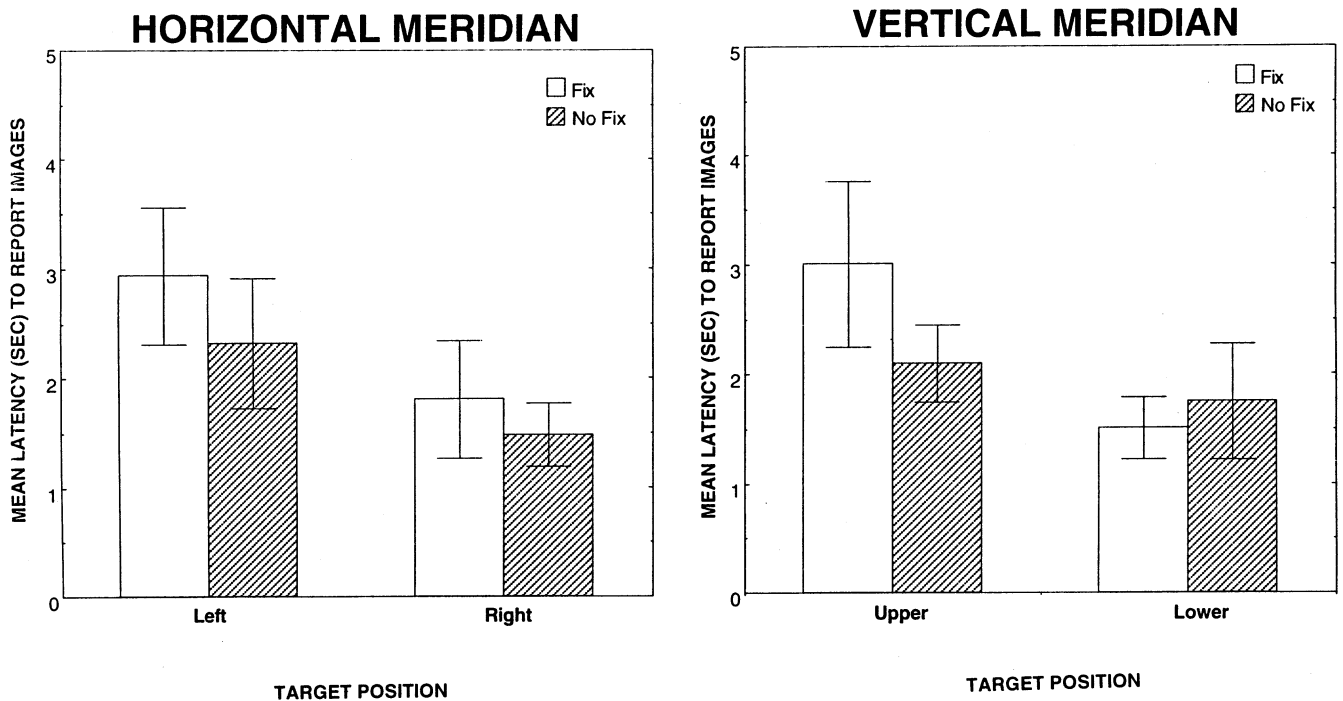


Fig. 4. Mean latencies (s) to report polyopic images of target stimuli presented to left and right of fixation on horizontal meridian and of stimuli presented above and below fixation on vertical meridian, with and without fixation stimulus (session 3). Vertical bars = S.E.M.

cated images of target stimuli depending on their position in the visual field. To this end, we presented visual stimuli in varying positions on the horizontal and vertical meridians and on diagonals within each quadrant. In addition, we investigated the role of the fixation stimulus in her reports of duplicated images of stimuli. Because we were concerned that varying stimulus duration (which had no effect in session 1), in addition to these two parameters, would require a large number of trials and so fatigue the subject, we estimated the latency of her image formation by the speed of her manual response indicating when she saw replicated images.

4.3.1. Procedures

The target stimulus, a 0.25° black circle, was presented tachistoscopically four times at 0.5 , 1.0 , 1.5 and 2.0° eccentricity on the horizontal and vertical meridians and on the four diagonals. The fixation stimulus was present with target stimulus on half the trials and absent on the remaining trials when the target stimulus was absent. RS was instructed to maintain fixation on the fixation stimulus (an X) when it was present prior to and during target displays, and, when it was not present with the target stimulus, to maintain fixation in the center of the display. She also was instructed to quickly release a switch with her right index finger as soon as she saw replications of the target stimulus. Release of the switch stopped a timer, accurate to 0.01 s, started by exposure of the stimulus field, thus provid-

ing a measure of latency of image formation. Release of the switch also extinguished the target stimulus. Otherwise, the procedures were the same as those used in previous sessions.

4.3.2. Results

Because of the limited number of trials, the effects of stimulus eccentricity and of fixation stimulus could not be examined in the same analyses. Therefore, RS's latencies to report replication of the target stimulus were submitted to two sets of repeated-measures ANOVAs. In the first set, the effect of presenting a fixation stimulus along with the target stimulus was analyzed; in the second, the effect of stimulus eccentricity was analyzed. The effects of stimulus position on the horizontal and vertical meridians and diagonals were examined in both sets of analyses.

In the first analysis (see Fig. 4), RS's mean latency for duplicating right-sided targets (1.7 s) was significantly less ($F_{1,7} = 10.677$, $P = 0.014$) than for left-sided targets (2.7 s). When no fixation stimulus was present, RS's mean latency for reporting replication of the circle was marginally less (1.9 s) than her mean latency (2.4 s) when a fixation stimulus was present ($F_{1,7} = 3.613$, $P = 0.099$). The interaction of the fixation factor and direction of target on horizontal meridian was not significant.

In the next analysis, which included stimulus position on the vertical meridian and the fixation factor (see Fig. 4), RS's latency to report images of stimuli below

fixation (mean = 1.7 s) were significantly shorter ($F_{1,7} = 11.182$, $P = 0.012$) than her latencies to report images of stimuli above fixation (mean = 2.6 s). Whereas there was no main effect of fixation stimulus for stimuli on the vertical meridian, the interaction of the fixation factor and stimulus position on the vertical meridian was significant ($F_{1,7} = 8.142$, $P = 0.025$). This significant interaction was due to the marginally significant elevation of latencies associated with a fixation stimulus for upper-field target stimuli ($t_{14} = 2.07$, $P = 0.06$) with no effect of fixation stimulus for lower-field target stimuli (see Fig. 4).

In the analysis of stimuli presented on the four diagonals (see Fig. 5), RS's latency to report replication was significantly shorter ($F_{1,7} = 35.451$, $P = 0.001$) for right-sided than for left-sided targets. However, this effect apparently was due to the significant interaction of the lateral (left vs right) and vertical (upper vs lower) factors ($F_{1,7} = 17.474$, $P = 0.004$). This interaction resulted from very short latencies for stimuli on the right lower diagonal compared to latencies for stimuli on the other three diagonals. Consequently, the mean latencies of responses to right lower stimuli and upper stimuli were reliably different ($t_{14} = 9.25$, $P < 0.0005$), but for the left diagonals, the mean latencies associated with lower and upper stimuli did not differ. Presence versus absence of the fixation stimulus was not significant for stimuli presented on the diagonals. However, the interaction of the fixation factor with the vertical position factor (above vs below fixation) was significant ($F_{1,7} = 10.086$, $P = 0.016$),

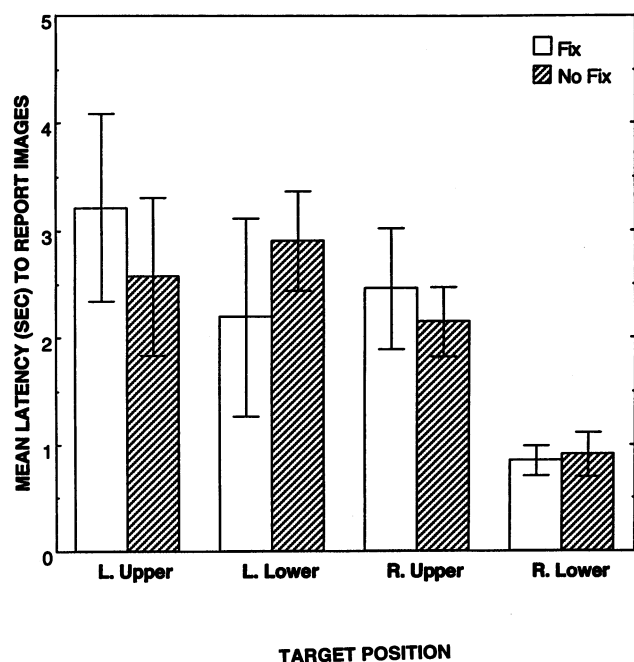


Fig. 5. Mean latencies (s) to report polyopic images of target stimuli presented on diagonals within each quadrant, with and without fixation stimulus (session 3). Vertical bars = S.E.M.

due to marginally shorter latencies without a fixation stimulus compared to latencies associated with the presence of the fixation stimulus for targets on the upper diagonals ($t_{14} = 1.83$, $P = 0.08$), but no effect of the fixation stimulus when targets appeared on the lower diagonals.

Eccentricity effects were analyzed by combining the two stimulus positions near central fixation (0.5 and 1.0°) and the two stimulus positions further from central fixation (1.5 and 2.0°). When stimuli were presented on the horizontal meridian (see Fig. 6), RS's latencies to duplicate near stimuli were marginally shorter than her latencies to duplicate far stimuli ($F_{1,7} = 3.649$, $P = 0.098$). As noted above, her latencies to report replication of right-sided stimuli were significantly shorter than left-sided stimuli. The interaction of eccentricity and position on the horizontal meridian was not significant. For stimuli on the vertical meridian (see Fig. 6), latencies for near stimuli were significantly shorter than latencies for far stimuli ($F_{1,7} = 9.712$, $P = 0.017$). As described above, latencies for lower-field stimuli were reliably shorter ($F_{1,7} = 24.144$, $P = 0.002$) than latencies for upper-field stimuli. The interaction of stimulus eccentricity and upper versus lower position was not significant. With regard to stimuli presented on the diagonals (see Fig. 7), the eccentricity factor was statistically insignificant. However, the eccentricity by side (left vs right) interaction approached significance ($F_{1,7} = 4.586$, $P = 0.069$). This interaction was due to shorter latencies for near than for far stimuli on the left but not on the right side. In contrast, for stimuli on lower right diagonal, there was a reversal of this effect: Latencies for far stimuli were marginally less than latencies for near stimuli ($t_7 = 1.93$, $P = 0.062$).

4.4. Session 4

The first goal of this session was to further compare RS's polyopia when stimuli were presented in the lower right quadrant and when they were presented in the lower left quadrant. The second goal was to examine further her reports of multiple images in her scotoma. The third goal was to further explore the role of the fixation stimulus on the speed with which she reported replicated images of stimuli presented to the left and right of fixation on the horizontal meridian.

4.4.1. Procedures

As in session 3, RS was instructed to quickly release a manual switch when she saw replicated images of the target stimulus, a 0.25° black circle, presented in the tachistoscope for 5 s. The stimuli were presented ten times at 2, 4 and 6° from fixation on the lower left and right diagonals, along with the central

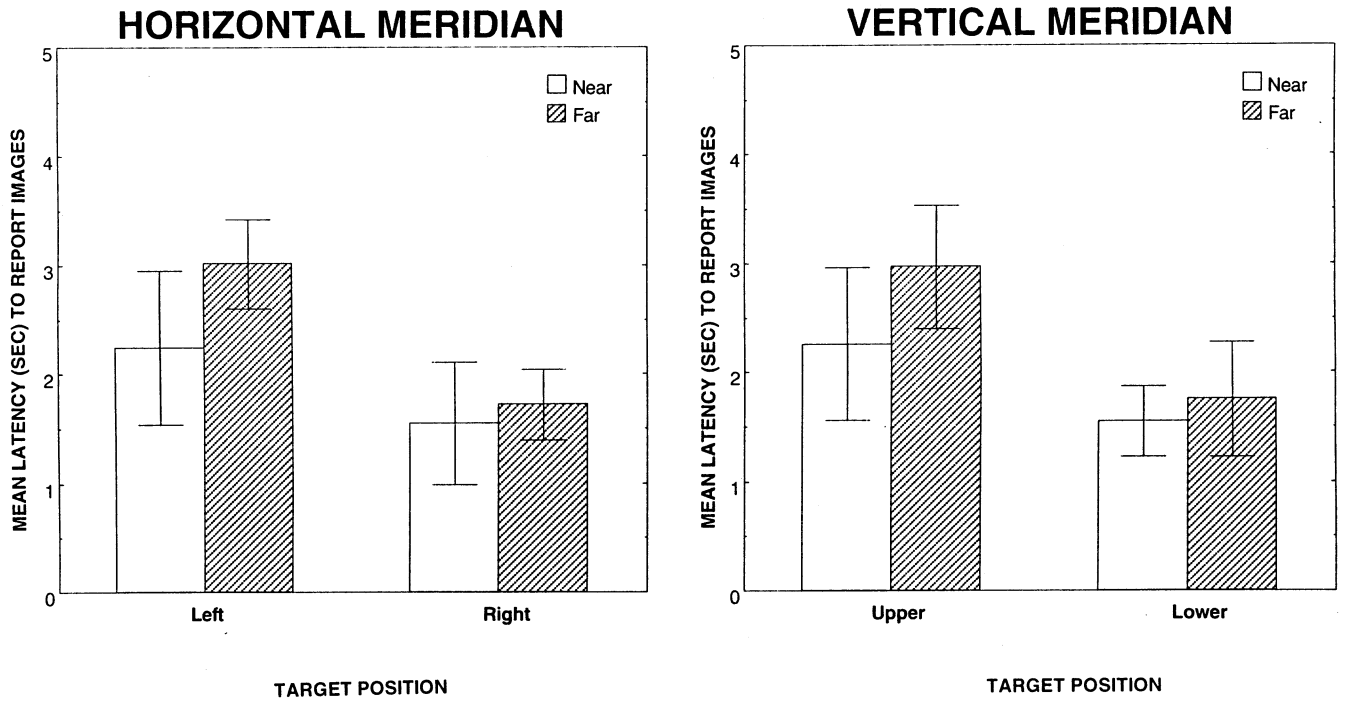


Fig. 6. Mean latencies (s) to report polyopic images of target stimuli presented to left and right of fixation on horizontal meridian and of stimuli presented above and below fixation on vertical meridian, at near (0.5 and 1.0°) eccentricities and at far (1.5 and 2.0°) eccentricities (session 3). Vertical bars = S.E.M.

fixation stimulus. Stimuli also were presented on the horizontal meridian, 1 and 2° to the left and right of the fixation stimulus. At each of these four positions, stimuli were presented ten times accompanied by the fixa-

tion stimulus and ten times without it. The order of presentation of stimuli on the lower diagonals and on the horizontal meridian was counterbalanced to reduce order effects (e.g. fatigue). In the first and last blocks of (30) trials, stimuli were presented five times at each eccentricity on the lower left and right diagonals. In the second and third blocks of (40) trials, stimuli were presented five times at each eccentricity to the left and right, on the horizontal meridian.

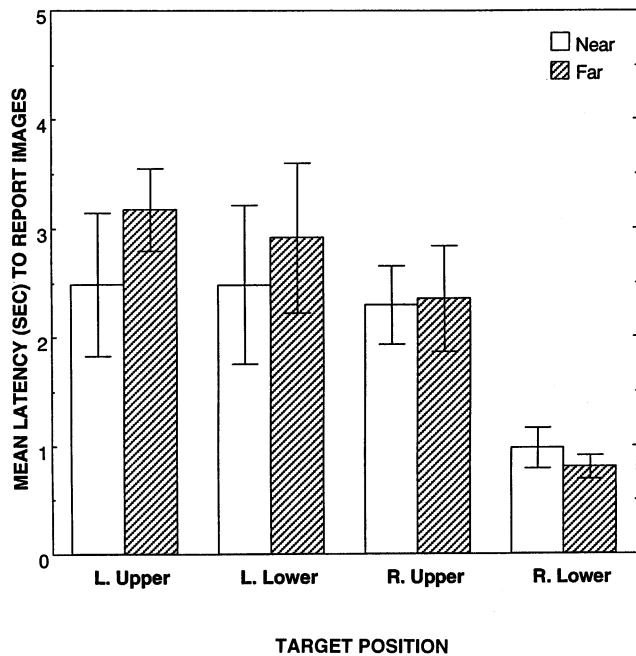


Fig. 7. Mean latencies (s) to report polyopic images of target stimuli presented on diagonals within each quadrant at near (0.5 and 1.0°) eccentricities and far (1.5 and 2.0°) eccentricities (session 3). Vertical bars = S.E.M.

Table 3
Latencies(s) of RS's reports of replicated images to stimuli on lower diagonals

Trial	Right (°)			Left (°)		
	2	4	6	2	4	6
1	NR	1.45	NR	1.7	2.7	0.9
2	NR	NR	NR	1.7	1.5	1.8
3	NR	2.1	NR	1.3	1.9	2.4
4	2.7	2.5	NR	1.9	2.4	2.2
5	NR	2.7	NR	1.9	2.5	2.3
6	NR	1.9	NR	1.8	1.2	3.1
7	NR	2.1	NR	1.9	1.8	3.1
8	NR	2.2	NR	1.7	1.7	1.4
9	NR	2.1	NR	1.7	1.7	3.3
10	NR	1.5	NR	1.9	1.9	3.1
X		2.1		1.7	2.0	2.1
S.D.		0.4		0.2	0.5	0.7

NR, No report of stimulus or replicated images.

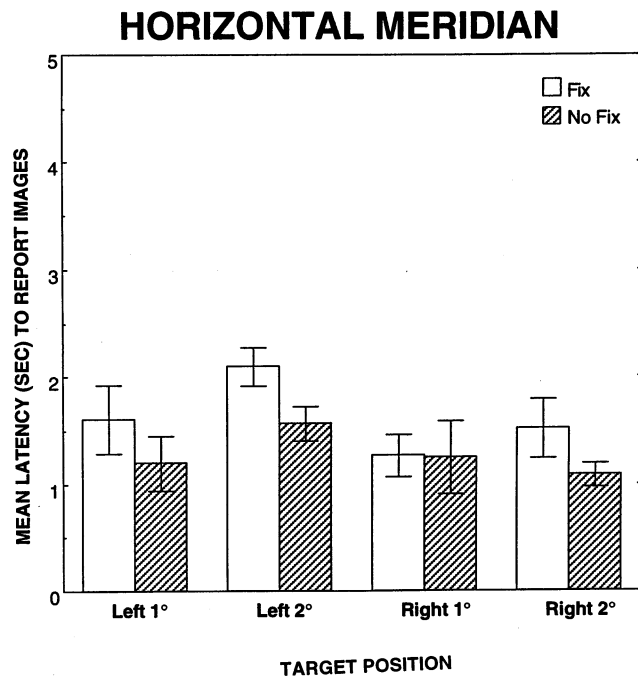


Fig. 8. Mean latencies (s) to report polyopic images of target stimuli presented on the horizontal meridian to left and right of fixation at 1.0 and 2.0° eccentricities (session 4). Vertical bars = S.E.M.

4.4.2. Results

RS reported seeing stimuli presented on the lower right diagonal 10/30 times (see Table 3). On eight of the nine trials on which the stimulus was presented 4° to the right of fixation, she also reported a streak of images extending all the way to the lower right corner of the display area. She reported the same phenomenon when stimuli were located 2° to the right of fixation, but never when stimuli were presented to the left of fixation. An ANOVA of the latencies with which she reported replications of stimuli presented on the lower left diagonal disclosed a marginal effect of eccentricity ($F_{2,18} = 2.692$, $P = 0.082$). Her latencies were marginally shorter for 2° than for 6° eccentric stimuli on the lower left diagonal ($t_{18} = 1.921$, $P < 0.10$). No other comparisons between mean latencies for duplicating stimuli on the lower left diagonal reached significance. RS's latencies to report replications of stimuli on the lower right and left diagonals 4° from fixation were not significantly different from each other (see Table 3).

When a target stimulus was presented on the horizontal meridian, RS consistently reported both the target and replicated images of it (see Fig. 8). An ANOVA of her latencies to report replicated images, including eccentricity (1 vs 2°), side (left vs right) and presence versus absence of fixation stimulus, was performed. Latencies to report images of right-sided stimuli (mean = 1.3 s) were shorter ($F_{1,8} = 2.098$, $P = 0.003$) than latencies to report images of left-sided stimuli (mean = 1.7 s). In addition, 1° eccentric stimuli elicited

images at shorter latencies ($F_{1,8} = 12.834$, $P = 0.007$) than did 2° eccentric stimuli. Also, the presence of a fixation stimulus was associated with longer latencies than its absence ($F_{1,8} = 17.484$, $P = 0.003$). None of the two-way interactions between factors attained significance. However, the three-way interaction was statistically significant ($F_{1,8} = 7.849$, $P = 0.023$), due to the absence of a fixation effect associated with 1° eccentric stimuli on the right side (see Fig. 8).

5. Discussion

The major findings of the present study may be summarized as follows. (1) RS reported polyopic images of two-dimensional drawings of varied complexity that fell at or near to the fovea (first session). (2) Her polyopic images always appeared to the right of and below the stimulus, i.e. in the part of the visual field where her scotoma was located; her multiple images formed a train that successively appeared rightward and downward (all sessions). (3) Stimuli to the right of fixation, compared with those to the left, elicited images with a greater frequency (session 1), at shorter latencies (sessions 3, 4) and at greater eccentricity when presented on diagonals (sessions 2 and 3). (4) RS's multiple images of stimuli presented at or to the right of fixation extended into her scotoma (first and fourth sessions). (5) A stimulus at fixation slowed the appearance of polyopic images (sessions 3 and 4). (6) There was no relationship between RS's direction of eccentric fixation, or direction of eye drift, and her reports polyopic images.

The finding that polyopic images were elicited by stimuli varying in complexity is consistent with and extends the findings of Gottlieb [7], whose patient reported polyopic images of dots, graphic characters and geometric figures. RS's report of polyopic images of complex objects she fixated suggests that the abnormal process underlying her disorder was not limited to lower-order visual areas in the occipital lobe, where simple features such as borders and angles are coded [11], and which were infarcted in her left hemisphere. Rather, it would appear that her abnormality involved some interaction between lower-order visual areas with higher order visual areas in the temporal lobe that are thought to code whole objects [12].

Bender [3] expressed the view that eye movements play a key role in the genesis of polyopic images. According to this view, defective fixation resulting from hemianopia increases the small involuntary eye movements that accompany normal fixation [13]. Bender [3] suggested that these eye movements lead to the development of false maculae, i.e. new retinal and corresponding cortical regions that code for central vision. Polyopic images, by this account, occur when the invol-

untary eye movements accompanying fixation lead to stimulation of the true and the acquired maculae. Reports of facilitation of monocular diplopia or polyopia by eye movements [3,7] are consistent with this interpretation. Abnormal fixation might also explain why the absence of the fixation mark tended to speed the development of RS's images of eccentric stimuli.

However, it is unlikely that RS's polyopic images could be explained by this hypothetical mechanism. While reporting polyopic images formed to the right of a fixated circle (session 2), RS's eyes did not, as one would expect *ex hypothesi*, move to the right more than to the left. Furthermore, the finding that RS showed stable fixation of small stimuli makes it difficult to attribute her polyopic streaks, which extended 5.6° (the distance from central fixation to the lower right corner of the display), to a series of abnormal eye movements of that amplitude, which were not detected during examinations. A similar argument was made by Meadows [6], whose patient fixated within 1° of a stimulus while experiencing polyopic images separated from the stimulus by approximately 4° .

The finding that RS's polyopic images appeared in the portion of the visual field close to her scotoma has been reported by several investigators [2,6,7], although the images were located elsewhere in other cases [3,5,8]. Several other findings (summarized above) suggest that proximity of stimuli to RS's scotoma was a significant factor affecting her polyopia.

A possible pathophysiological mechanism for cerebral polyopia involves reorganization of receptive fields of neurons close to the damaged area of visual cortex. This possibility is suggested by findings that soon after parafoveal retinal lesions that deprive a region of the striate cortex of visual input, the receptive fields of neurons near the boundary of the deprived cortical region enlarge and expand into nearby regions of the visual field [14]. These findings suggest that polyopic images may be due to altered coding of contour information by neurons nearby the lesioned area. According to this view, after a focal lesion of neurons in striate cortex (or a retinal lesion depriving these neurons of visual input), the receptive fields of nearby healthy neurons now code information about contours of objects normally coded by the missing (or disconnected) neurons, resulting from the growth of thalamic or intracortical connections. However, these neurons would still carry the same information about retinal location that they did prior to the lesion. The finding that receptive fields extend into the part of the visual field normally mediated by the lesioned retina [14] might explain why after a lesion involving primary visual cortex, polyopic images extended into the scotoma of our patient and of case 1 of Kömpf et al. [2]. In order to test the hypothesis that altered coding of contour information is responsible for polyopia, one

would need to record from cortical neurons coding stimuli at fixation and those near the edge of a previously produced lesion. According to the hypothesis, one would expect that after the lesion, the latter neurons would tend to discharge to the same contour features that excite the former neurons and not those features that fall in their receptive fields. Assuming that recoding of visual receptive fields following a deprivation of visual input to striate cortex is not an uncommon phenomenon, how would this hypothesis account for the rarity of cerebral polyopia? It is possible that this disorder is rarely reported because questions as to its presence are rarely asked in visual examinations following cerebral injury, or because the images are not sufficiently salient for patients with these injuries to noticed or be concerned about them. Of course, when speculating about the neural basis of monocular polyopia of central origin, one should not exclude the possibility that it is not a single disorder and consequently, that it may be due to more than one pathophysiological mechanism.

Another interpretation of polyopia (but one that does not exclude the recoding hypothesis) involves the observation that the disorder is frequently associated with bilateral cerebral lesions [2–4,6,9]. Bilateral lesions may have been present in cases lacking brain scans [5,8]. It is also possible that bilateral lesions were present in cases with brain scans indicating only unilateral lesions [7]. CT is relatively insensitive to the presence of cerebral lesions [15], especially if they are small; thus, it would not be unreasonable to assume that some cases diagnosed as unilateral cerebral disease by CT scans in fact had bilateral lesions.

Whatever the nature of the pathophysiology underlying polyopia, it must be different from that responsible for palinopia, in which visual images continue or reappear intermittently, episodically or paroxysmally (see Section 1 for reviews). Furthermore, whereas polyopic images apparently are coded in retinotopic co-ordinates, palinopic images in some patients are apparently coded in non-retinotopic (i.e. ego-centric) co-ordinates, especially when the images are reported to be presented weeks, months or even years after the original stimulus was experienced [16]. Recently it has been suggested that palinopia is due to abnormal activity of mechanisms responsible for short-term visual memory [17], which may utilize non-retinotopic co-ordinates.

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