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Impaired perception of simultaneous stimuli in a patient with posterior cortical atrophy: an attentional account

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ABSTRACT

We assessed visuospatial abilities in PCA. Sequential display of two simple geometric figures enhanced detection and discrimination relative to simultaneous display (Exps 1 & 2). Comparing edges of a single object enhanced discrimination relative to comparing edges of two separate objects, consistent with object-based attention (Exp. 3). Recognition of complex line drawings was spared for a single object but disrupted by an attention-grabbing small circle (Exp. 4). A covert orienting task showed difficulty disengaging from previous locations and attentional bias toward the right visual field (Exp. 5). These findings shed light on the role of visual attention in perceptual awareness.

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KEYWORDS

Simultanagnosia; Balint's syndrome; perceptual awareness; consciousness

Over the years, the relation between perceptual awareness and visuospatial attention has fascinated researchers and clinicians alike (Balint, 1909; Fernandez-Duque, Grossi, Thornton, & Neville, 2003; Pitts, Martinez, & Hillyard, 2012; Poppelreuter, 1917–1990; Rees & Lavie, 2001; Schurger, Cowey, Cohen, Treisman, & Tallon-Baudry, 2008; Simons & Rensink, 2005; Vuilleumier & Rafal, 2000). In neuropsychology, deficits in perceptual awareness related to attention have been described most often in hemispatial neglect syndrome and in dorsal simultanagnosia. In dorsal simultanagnosia, the patient is said to be aware of only one object at a time. For example, the patient is able to name line-drawings of common objects when presented one at a time, but when presented with two figures, above and below fixation, the patient typically reports only one item.

As in the case of hemispatial neglect, dorsal simultanagnosia has sometimes been explained as a deficit in the *disengagement* of visuospatial attention. As evidence, object recognition increases dramatically when the presentation of the drawings alternates every second (Pavese, Coslett, Saffran, & Buxbaum, 2002). Presumably, the offset of one figure releases attention from its location thus allowing attention to be redeployed to the second figure, and vice versa. On the other hand, covert orienting studies in simultanagnosic patients have yielded mixed results. In one case, the peripheral cue was ineffective in directing attention to either visual field (Verfaellie, Rapcsak, & Heilman, 1990). Other studies have reported normal orienting in dorsal simultanagnosia (Coslett & Saffran, 1991; Rizzo & Robin, 1990). Methodological differences across studies and differences in patient selection criteria may account for this diversity of findings (Coslett & Chatterjee, 2003).

Dorsal simultanagnosia is caused by bilateral damage to posterior parietal cortex, and that often co-occurs with other

parietal deficits such as optic apraxia and optic ataxia (i.e., Balint's syndrome) (Coslett & Chatterjee, 2003; Wolpert, 1924). It can be brought about by acute events such as strokes as well as by progressive diseases such as dementia. A member of the latter group is Posterior Cortical Atrophy (PCA), a syndrome characterized by insidious onset of visual dysfunction. PCA has similar histopathology as Alzheimer's disease (AD) including plaques and tangles (Crutch et al., 2017; Galton, Patterson, Xuereb, & Hodges, 2000; Tang-Wai et al., 2004), and thus it is sometimes referred to as atypical Alzheimer's disease. Despite this similar histopathology, PCA differs from typical AD on the cortical distribution of the affected areas. In typical AD, the atrophy starts in mid-temporal areas, giving rise to episodic memory deficits. In PCA, the atrophy starts in occipito-parietal regions and gives rise to a varied set of visuospatial impairments (Chechlacz et al., 2012; Kas et al., 2011). These deficits may include simultanagnosia, optic ataxia (misreaching), apraxia, environmental disorientation, acalculia, apperceptive agnosia, or alexia (Crutch et al., 2017; Delazer, Karner, Zamarian, Donnemiller, & Benke, 2006). At early stages of PCA, memory and language functions are relatively spared, unlike what happens for typical AD and in other variants of atypical AD such as primary progressive aphasia (Caine & Hodges, 2001).

An important question about dorsal simultanagnosia refers to its phenomenological experience: Is the patient truly unaware of the second object and its features, or is he merely unable to recognize the object as such? We address this question in Experiments 1 and 2. Simultanagnosia also offers an opportunity to assess predictions made by object-based attention theories. To explore this, in Experiment 3 we ask whether it is easier to compare the edges of a single object than to compare two separate objects. Furthermore, in Experiment 4 we ask whether the recognition of complex

line drawings is spared for a single object, but disrupted following the onset of an attention-grabbing centrally displayed distractor. Finally, in Experiment 5 we explore whether the perceptual failure stems from a deficit in *disengaging* attention from its previous location or from a *visuospatial bias* toward certain parts of the visual field. Altogether, we aim to shed light on the mechanisms and phenomenology of simultanagnosia in PCA.

Case description and clinical assessment of visuospatial attention

RD, a right-handed retired management consultant with a MBA education, was 68-year-old at the time of testing. He sought medical opinion 4 years earlier due to concerns with visuospatial abilities. At the time of the initial visit, the patient reported getting disoriented in familiar surroundings such as the neighborhood where he had grown up. At times, he would be looking for an item such as a set of keys on the desk to later realize it had been lying in front of him all along. He also expressed difficulties with numbers, as when using a banking machine or his credit card. Over the course of 4 years, he had gradually developed other problems, such as word-finding difficulty and forgetfulness.

At the time of testing, the clinical assessment revealed left visual field *extinction*: when bilateral stimuli were displayed, the patient reported seeing only the right visual field stimulus. This was true whether the bilateral stimuli was displayed in the upper or the lower quadrant, and it occurred despite the spared detection of unilateral stimuli on either side of the visual field. Importantly, *extinction* was limited to the visual domain, as bilateral tactile or auditory stimuli were detected. Paper and pencil tasks revealed *neglect*: there was a rightward bias of 10–16% in the *line bisection* task, and lines in the left side were missed in the *line cancellation* task. Neglect and extinction were confirmed using a covert orienting task (Posner, Walker,

Friedrich, & Rafal, 1984); misses were most common for left visual field targets and after a cue to the invalid location (see Exp. 5). In other words, there were both a spatial-attention bias and a deficit in disengagement attention from the cued location.¹

When presented with two overlapping objects in the center of his visual field (e.g., a pair of spoons), RD was often able to report both objects. Thus, his simultanagnosia was not evident in this clinical measure. In the neurological exam, RD demonstrated normal pursuit and saccadic ocular movements, and full visual fields. His visual acuity with corrective lenses was adequate (20/40, as measured by the Freiburg Visual Acuity Test) (Bach, 1996). His color vision and depth perception were normal. RD mis-reached when targets were presented at non-foveal vision (optic ataxia), but his reach was spared when allowed to view the item directly.

RD was severely impaired in drawing simple figures, such as a hexagon or a house (see Figure 1), and was completely unsuccessful in the judgment of line orientation test, scoring 0 out of 30 (Benton, Hamsher, Varney, & Spreen, 1983). When asked to name both global and local elements of compound hierarchical letters, RD showed local capture identifying only the local elements.²

RD's verbal comprehension was relatively good, as assessed by the Western Aphasia Battery. His score in the Cornell depression scale was within normal limits (4/38). Although his nonvisual skills were less impaired than his visual skills, RD did have memory deficits, which are not uncommon in this type of patient as the disease progresses. He scored more than two standard deviations below normal in California Verbal Learning Task, and he was impaired in the digit span task (see Table 1).

Neuroimaging: A structural MRI revealed bilateral atrophy extending to parietal and occipital lobes, most pronounced in the right hemisphere. Similarly, a SPECT (single-photon emission computed tomography) revealed hypo-perfusion extending

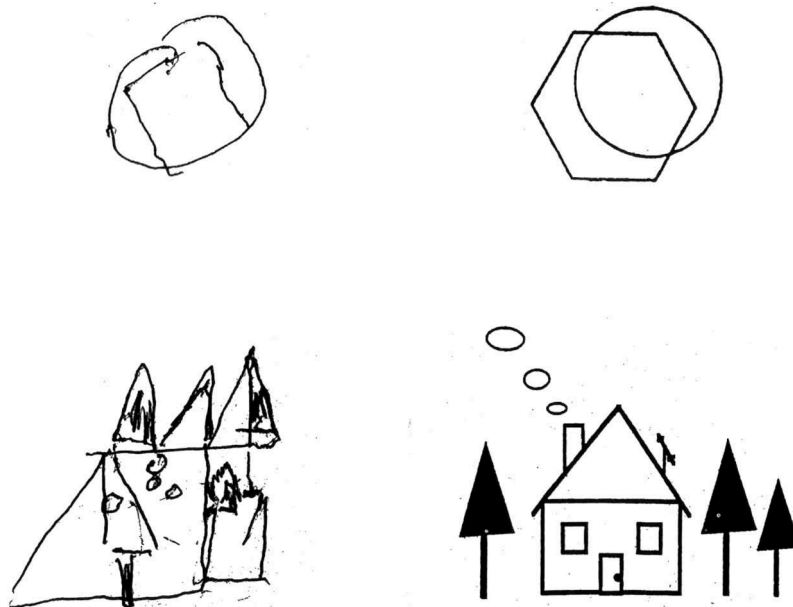


Figure 1. RD's drawings of figures in the Birmingham object recognition battery.

Table 1. Demographic, neuropsychiatric and neuropsychological information.

| | Max Score | Healthy Controls | RD |
|-------------------------|-----------|------------------|------|
| MMSE | 30 | 28.7 (1.5) | 27 |
| DRS | 144 | 140.5 (2.8) | 110 |
| WAB comprehension | 10 | 10 (.1) | 9.75 |
| Rey Copy | 36 | 33.3 (2.6) | 4.5 |
| Line Orientation | 30 | 25.9 (2.7) | 0 |
| Semantic Fluency | | 20.5 (4.7) | 11 |
| Verbal Fluency (FAS) | | 47.4 (14.8) | 28 |
| Forward digit span | 12 | 9.2 (2.0) | 6 |
| Backward digit span | 12 | 7.4 (2.3) | 2 |
| CVLT | | | |
| Short Delay Free Recall | 16 | 10.5 (3.3) | 3 |
| Short Delay Cued Recall | 16 | 11.6 (2.5) | 3 |
| Long Delay Free Recall | 16 | 11.2 (3.3) | 2 |
| Long Delay Cued Recall | 16 | 11.6 (3.2) | 2 |

MMSE, Mini-Mental State Examination; DRS, Mattis Dementia Rating Scale, WAB, Western Aphasia Battery; Rey Copy: Rey-Osterreith Complex Figure; CVLT, California Verbal Learning Test. The "Healthy Controls" group consists of 47 age-matched healthy volunteers who had undergone a full neuropsychological test in our clinic (1 standard deviation in parentheses). Forward digit span max score of 12 requires holding 8 numbers (10 = 7 numbers, etc.). Backward digit score max 12 = 7 numbers (10 = 6, etc.).

from the posterior parietal cortex to the lateral occipital cortex and posterior temporal regions, more pronounced in the right hemisphere (see [Figure 2](#)).

Experiment 1: object discrimination

In Experiment 1, we asked whether the simultaneous presentation of two simple geometric figures would lead to impaired shape discrimination. To answer this question, we compared sequential and simultaneous presentations of two concentric shapes.

Method

Equipment

Stimuli were displayed on the laptop's 14-in. monitor, which was set to a screen resolution of 1024 × 768 pixels. The timing of the stimulus display and data collection were managed using E-prime, a commercial experiment application. The same equipment was used in all the other experiments.

Stimuli

In each trial, two concentric geometric figures (square, ring) of different sizes (large, small) and colors (black, white) were displayed in the center of the screen against a gray background (see [Figure 3](#)). In a given trial, the figures were always of a different color and size; in half the trials they were of the

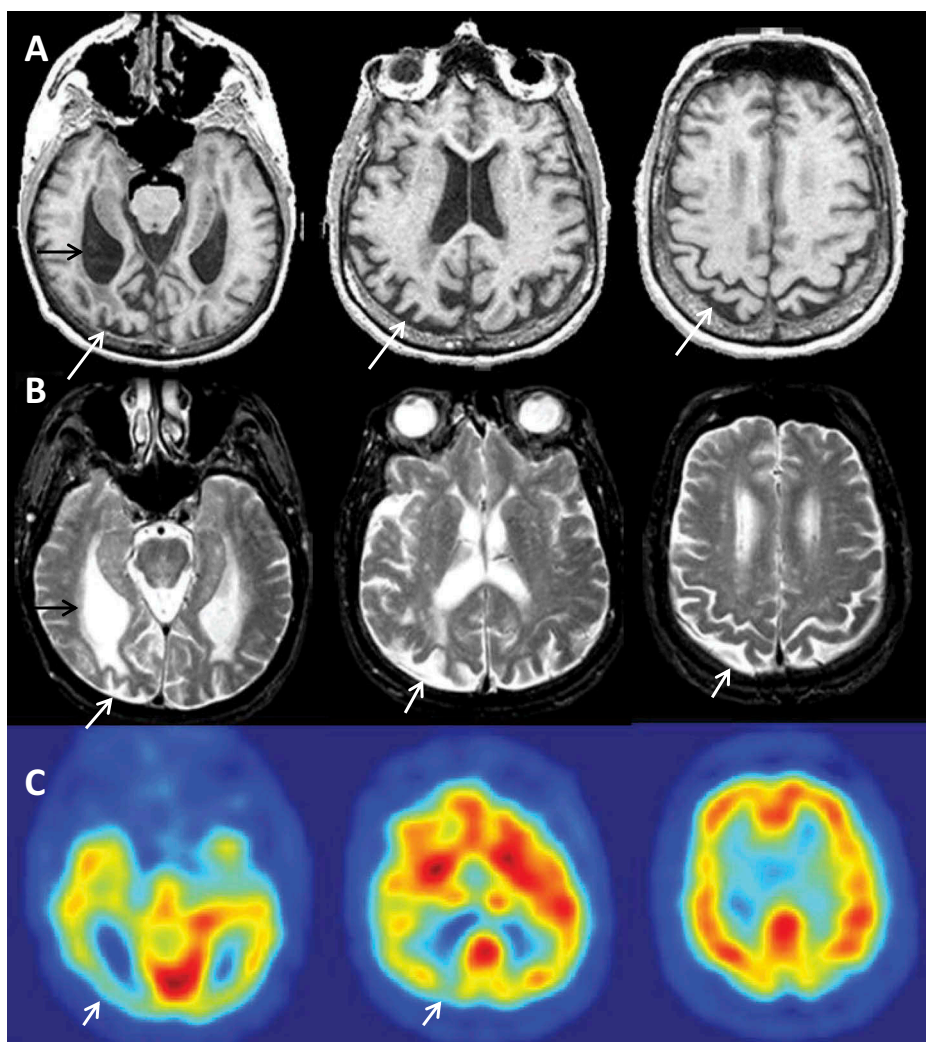


Figure 2. (a) Axial T1-weighted and (b) T2-weighted MRI from the inferior to superior brain sections illustrating more enlarged right lateral ventricle (black arrows) and widened sulci in the right parietal and occipital lobes (white arrows), indicative of more severe right parieto-occipital atrophy than the left. (c) SPECT images corresponding to the brain sections of the MRI show more pronounced hypo-perfusion in the right parieto-occipital region (white arrows).

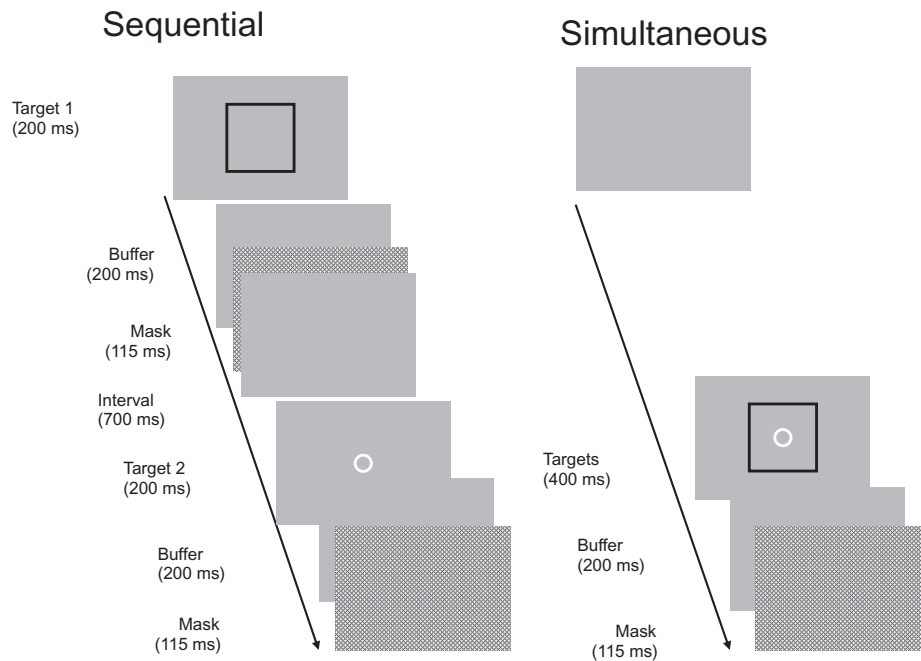


Figure 3. Sample stimulus and procedure used in Experiment 1. The left side of the figure depicts the timeline for sequential presentation of stimulus. The right side depicts the simultaneous presentation of stimuli.

same shape and in the other half, they had a different shape. The big square was 8.5 cm wide (9.6° VA) and the big circle had a 9 cm diameter (10.2° VA), while the small square was 1.2 cm wide (1.4° VA) and the small circle had a 1.5 cm (1.7° VA) diameter. For all figures, the borders were 1.5 mm wide.

Procedure

Figure 2 illustrates the timeline for each trial. In the simultaneous condition, both figures were displayed at the same time for 400 ms. In the sequential condition, each figure was displayed for 200 ms, followed by a 115 ms mask. The stimulus onset asynchrony (SOA) between figures was 1015 ms. The order of figure presentation in the sequential condition was counterbalanced for size, color and shape. Patient RD reported verbally whether the figures were of the “same” or “different” shape, and the experimenter entered this response in the computer by using a keyboard that sat on the experimenter’s lap and was connected to the laptop. In this and all other experiments, RD sat about 50 cm away from the screen. Healthy adults performed at ceiling levels (>98% accuracy) in this task, even with target displays as short as 15 ms.³

There were two blocks of 48 trials, each preceded by 12 practice trials; patient RD was encouraged to take a break between blocks. In the first block, the figures were displayed sequentially; in the second block, they were displayed simultaneously.

Results & discussion

RD’s ability to discriminate the figures in a 2AFC was much higher in the sequential display (87.5%, 42/48) than in the simultaneous display (45.8%, 22/48), Fisher’s exact test, $p < .001$, consistent with an inability to recognize two figures at the same time. The RT data showed the same pattern. We limited the analysis to

correct trials, and excluded an outlier trial from the simultaneous condition. The median response to simultaneous presentation was significantly slower than to sequential presentation (1600 ms vs 347 ms), $t(61) = 4.97$, $p < .001$.

In sum, patient RD was unable to discriminate two simple geometric shapes simultaneously displayed. One question that Experiment 1 could not answer was whether this deficit was due to a failure of shape discrimination or a failure in conscious perception. In other words, was the patient merely unable to discriminate the two different shapes or was he truly unable to “see” the second object (i.e., perceptually unaware of it)? We addressed this question next.

Experiment 2: object detection

In Experiment 2, we asked whether patient RD was aware of only one of the object when two objects were simultaneously displayed. To answer this question, we instructed RD to report the number of items he saw (one, two); in half of the trials, two figures were displayed simultaneously while in the other half only one figure was displayed.

Method

The stimuli were identical to Experiment 1 with the sole exception that for half the trials only one figure was displayed. For these single figure displays, the shape color and size of the figure varied randomly, with an equal number of trials for each stimulus type per block. For the two-figure displays, the figures were always displayed simultaneously. In any given trial, the two figures were always of a different color and size; in half the trials they were of the same shape and in the other half they had a different shape.

For each block, the one-figure and the two-figure displays were randomly distributed. There were three blocks of 48 trials each. In the first block, the stimulus was displayed very briefly (50 ms), in the second block it was displayed for 400 ms and in the third block, it was displayed for 1500 ms. Healthy adults were tested on 100 ms displays and performed at ceiling levels (100% accuracy).

Results & discussion

RD's performance was at chance for short (54%, 26/48) and medium display durations (50%, 24/48), exact binomial calculation, $P_{(\geq 26 \text{ out of } 48)} = .33$. In other words, even when the stimulus was displayed for 400 ms, RD was unable to report whether there were one or two items. Performance was above chance only when the stimulus was displayed for 1500 ms, and even then it was far from perfect at 79% accuracy, exact binomial calculation, $P_{(\geq 38 \text{ out of } 48)} < .001$. The increase in accuracy during long displays may have stemmed from an increase in the strength of the perceptual signal. Alternatively, the longer duration might have allowed RD's attention to wander to other regions of the screen.

In any case, RD's deficit was not merely an inability to discriminate two shapes but rather a true deficit of perceptual awareness.

Summary of Experiments 1 & 2

Experiment 1 revealed that RD could not discriminate two simple shapes when they appeared simultaneously, despite the objects' differences in size and color. Experiment 2 showed that this was a deficit of perceptual awareness rather than merely a deficit in object recognition. The stimuli we used in those experiments were simple geometric figures, with lines that did not cross at any time. This separates our experiments from the typical clinical assessments of simultanagnosia, such as the presentation of two overlapping objects and the Poppelreuter figure in which the lines of overlapping objects often cross paths.

The simplicity of the stimuli argues against the hypothesis that object recognition *per se* was at the root of the patient's problem; instead, it seems far more likely that a deficit in spatial

attention was at the core of the patient's deficits in object recognition (Exp. 1) and object awareness (Exp. 2).

In the next three experiments, we moved beyond the phenomenological description to start exploring the role of attention.

Experiment 3: object-based attention

In this experiment, we explored whether the patient's failure to discriminate between two simultaneously displayed shapes stemmed from a deficit in object-based attention. More precisely, we asked whether our patient had an easier time comparing the edges of a single object than comparing two separate objects.

Object-based theories of attention postulate that the physical properties of the stimulus enable the visual system to extract object representations pre-attentively, based on Gestalt principles such as spatial proximity, contour, or color (Lavie & Driver, 1996). After features have been integrated into objects, attention is directed to candidate objects for further processing. Thus, the limitation in attentional capacity is on the number of separate objects that can be perceived at once. In the words of a leading proponent of this view: "it is whole objects that are neglected, not spatially determined parts of objects; and the objects that are neglected may occupy the same spatial coordinates as an object that is seen" (p. 128) (Rafal, 2001).

Object-based theories of attention predict that extinction in dorsal simultanagnosia will be evident when stimuli are coded as separate objects but less so when coded as parts of the single object representation (Barton, Malcolm, & Hefter, 2007). Experiment 3 tested this hypothesis. We reasoned that if the deficit stemmed from a difficulty at attending two objects at the same time, performance should improve when the two objects were perceptually merged into one. To facilitate such perceptual grouping, in Experiment 3 the two figures and the surface in between them were displayed in a uniform color white, and the size difference between inner and outer figure was reduced. These changes gave the stimulus the appearance of a single object with internal and external edges. In some trials, those edges matched (e.g., two circular edges, as in a doughnut) and other trials, they did not (e.g., a circular inner edge and square outer edge) (see Figure 4).

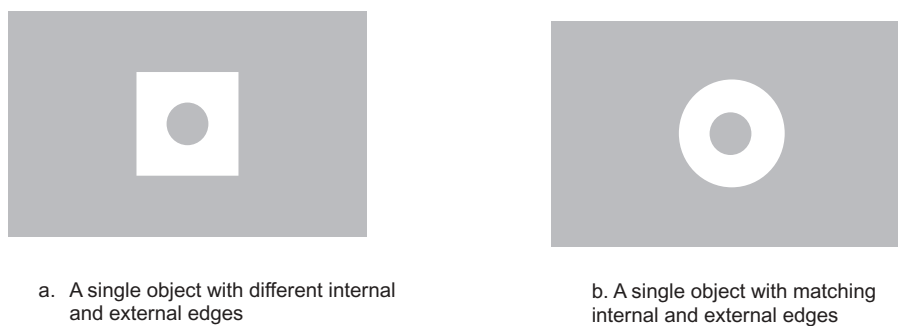


Figure 4. Sample stimuli used in Experiment 3. (a) A *Single Object* display in which the internal and external edges of the figure do not match, and (b) A *Single Object* display in which the edges do match. The *Two-Objects* displays were similar to the simultaneous displays of Experiment 1 (see Figure 3).

Method

Stimuli & procedure

The big square was 6.8 cm wide (7.7° VA) and the big circle had a 8.2 cm diameter (9.3° VA), while the small square was 4.4 cm wide (5.0° VA) and the small circle had a 5.2 cm diameter (5.9° VA). There were three blocks of 48 trials each, and in each trial, the target stimulus was displayed for 400 ms, followed by a 115 ms mask. In blocks 1 and 3, the stimulus was a single object with internal and external edges. For those two blocks, RD and healthy adults were instructed to report whether the edges of the figure matched or not. Healthy adults performed at ceiling levels (98% accuracy), even with target displays of 100 ms (i.e., one quarter of the duration of the displays shown to RD). In block 2, there were two objects, much like in the simultaneous condition of Experiment 1, except that now the figures were closer in size and always of the same white color. RD was instructed to report whether the two figures were the same or different shape. Each of the three blocks was preceded by four practice trials.

Results & discussion

In block 2, RD was at chance (52.1%, 25/48) at discriminating two simultaneously displayed figures, a result that replicates the findings from Experiment 1. In blocks 1 and 3, RD had to discriminate the edges of a single figure, and his performance was better than chance⁴ [59%, 57/96; exact binomial calculation, $P_{(\geq 57 \text{ out of } 96)} = .04$], albeit far from perfect. This better-than-chance performance in edge discrimination gives support to the prediction, stemmed from object-based attention theories, that extinction is ameliorated when stimuli are coded as parts of a single object.⁵

Experiment 4: attentional cueing to a central location

In Experiment 4, we further explored the role of attention, this time by looking at the effect of attentional cueing on recognition of more complex objects (i.e., Snodgrass line drawings). More precisely, we asked whether the presence of an attention-grabbing small circle at the center of the figure would disrupt object recognition. Such a finding would support our contention that RD's deficit in perceptual awareness could be traced to his deficit in visual attention.

Method

Stimuli & procedure

Twenty-four line drawings from the Snodgrass and Vanderwart corpus served as stimuli (Snodgrass & Vanderwart, 1980). Half of these were drawings of animals.⁶ There were 24 trials per session, with all items displayed once each session in a random order. The drawings were approximately 3.5 cm x 5 cm (4° x 5.7° VA) in size. Each trial started with a 700 ms small fixation cross. Next, the figure was displayed for 300 ms, after which a mask appeared. RD was instructed to report verbally whether the stimulus was a line drawing of an animal or not. Three sessions were administered across different days. In sessions 1 and 3, the procedure was as just described. In session 2, the fixation cross was replaced

by a sequence of four black circles, of decreasing size. The first circle was the largest (1.5 cm), and each subsequent circle was smaller than preceding one (1.1 cm, 0.6 cm and 0.4 cm). Each circle was displayed for 100 ms, followed by a 100 ms interval, which gave the impression of a shrinking flickering object. The smallest circle remained on the screen until response, with the Snodgrass figure appearing behind it. Only a very small part of the figure was occluded by it. Healthy adults were tested in the attention-grabbing circle condition using target displays of 150 ms (i.e., half of the duration of the displays used for RD) and performed flawlessly (100% accuracy).⁷

Results

When the figure was displayed alone, the performance was relatively good, with 89% accuracy (43/48). The presence of an attention-grabbing circle significantly reduced accuracy to 62.5% (15/24), Fisher's exact test, $p < .01$. In other words, the presence of a circle at the center of the screen biased attention toward the center of the screen and away from the line drawing, preventing its conscious recognition. This suggests that RD's perceptual deficit was mediated at least in part by a deficit in visual attention.

Experiment 5: attentional cueing to a peripheral location

In Experiment 5, we used a covert orienting paradigm to further explore the attentional mechanisms underlying RD's deficit in visual awareness. The patient was asked to maintain eye fixation at the central fixation point and respond to the onset of a lateralized target. The target was preceded by a cue that informs the participant about the location of the target. We used a central predictive cue (80/20) and a simple detection response. With this design, we assessed the extent to which the presence of the central cue disrupted target detection in the periphery, and whether such disruption was dependent on cue validity (valid, invalid) and/or on target location (left, right). In neglect patients, two main findings have been reported (Posner et al., 1984). Consistent with a *disengagement deficit* mechanism, performance is worst when the target is displayed opposite to the cued location (i.e., invalid trials). Consistent with a *visuospatial bias* mechanism of neglect, performance is worst when the target is displayed in the contra-lesional field. We tested both of these mechanisms in our analysis. We also tested a group of healthy adults to confirm they had no problem detecting the target.

Method

Stimuli

Two white rectangular boxes were displayed against a gray background at all times (Figure 5). These boxes were displayed 6.85 cm (7.8° VA) left and right of fixation, as measured from the center of the screen to the center of the box. Each box was 5.2 cm wide and 4.2 cm high, with lines that were one pixel wide. The central cue was a triangle that measured 1 cm in its horizontal axis (1.1° VA), and pointed left or right depending on the

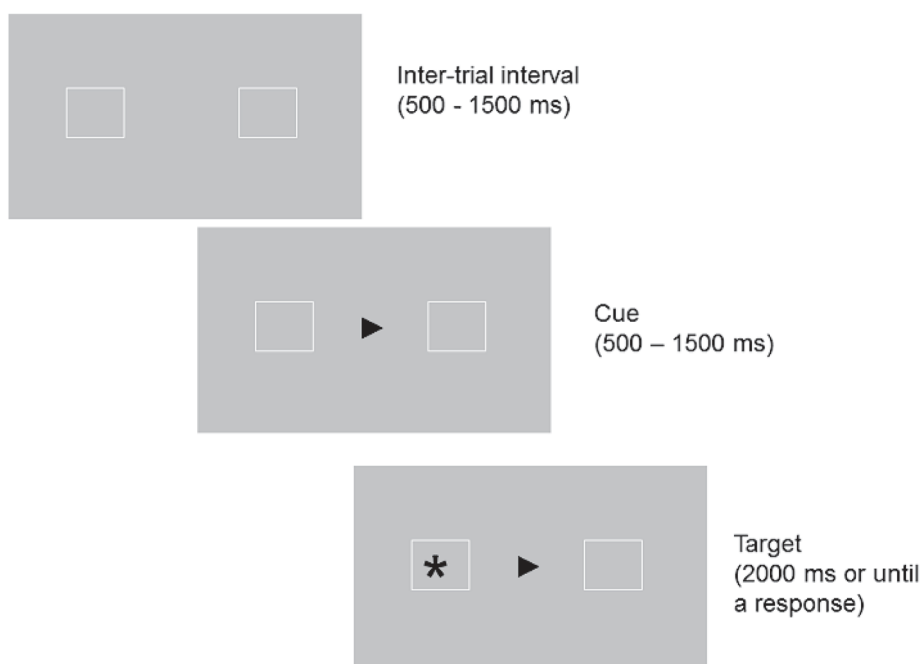


Figure 5. Example of an invalid trial in the covert orienting task (Exp. 5).

trial. The target was a black asterisk, 1.5 cm in diameter, which was displayed at the center of one of the rectangular boxes.

Procedure

Each trial began with a cue and was followed by a target after a variable delay between 500 and 1500 ms.⁸ Cue and target remained on display until response, or for a maximum of 2 secs. RD was instructed to respond by pressing the spacebar with his right hand each time he detected a target. Inter-trial interval was randomized between 500 and 1500 ms.

Two sessions were administered across separate days. Each session had 2 blocks of 106 trials; each block lasted approximately 6–7 min, and RD was encouraged to take a break between blocks. In each block, there were 80 valid trials (i.e., target at cued location), 20 invalid trials (target at the opposite location), and 6 catch trials (no target). Type of cue (valid, invalid) was crossed with target location (left, right). For example, for half of the 20 invalid trials in each block, the target appeared in the left visual field, and for the other half, it appeared in the right visual field.

We emphasized to RD the importance of maintaining eye fixation at the center cue while trying to detect the peripheral target. RD had no difficulty maintaining fixation, but on the few occasions in which he broke fixation, he was reminded of the instructions.

Results

Healthy adults had no difficulty detecting the target (100% detection rate) and showed the typical orienting effect, with faster responses for valid trials (333 ms) than for invalid ones (373 ms), $t(1, 5) = 2.8, p = .04$ (Fernandez-Duque & Black, 2006; Fernandez-Duque & Posner, 1997).

Table 2 illustrates the percentage of trials in which RD failed to detect a target.⁹ Failure in detecting targets was larger in the left visual field (52%, 104/200) than in the right visual field (17%, 34/200), $\chi^2 = 54, p < .0001$. This is consistent with a visuospatial bias toward the right visual field. Failure detecting a target also grew larger following an invalid cue (50.0%, 40/80) than following a valid cue (30.6%, 98/320), $\chi^2 = 10.6, p = .001$. This finding is consistent with a deficit in disengaging attention from the cued location. Furthermore, these two main effects are over-additive, as revealed by a significant interaction in a log-linear analysis, $G^2 = 68.4, p < .0001$. In other words, the disengagement cost was disproportionately large when attention was cued to the right and the target appeared in the left visual field.

In sum, Experiment 5 revealed RD's difficulties in perceiving a lateralized target, and highlighted the roles of attentional disengagement and visuospatial bias in that deficit.

General discussion

The five experiments reported here assessed the visuospatial abilities of a patient with posterior cortical atrophy. Experiments 1 and 2 demonstrated that the detection and discrimination of two simple geometric figures was greatly impaired when the figures were displayed simultaneously, but relatively spared when the figures were displayed sequentially. In Experiment 3, comparing the edges of a single object led to better discrimination than comparing two separate objects. In Experiment 4, the

Table 2. Rate of failure at target detection in the covert orienting task of Experiment 5.

| | Left Visual field | Right Visual field |
|---------|-------------------|--------------------|
| Valid | 46.8% (75/160) | 14.4% (23/160) |
| Invalid | 72.5% (29/40) | 27.5% (11/40) |

recognition of complex line drawings was spared for a single object but disrupted by an attention-grabbing small circle. Finally, Experiment 5 revealed both a deficit *disengaging* attention, as well as a *bias* of visual attention toward the right visual field.

It is clear from our results that perceptual awareness – not just perceptual discrimination – was impaired in this PCA case. It is also evident that lack of attention was a key contributor to such perceptual unawareness: when a complex object figure (e.g., a Snodgrass line drawing) was displayed alone, the patient had no difficulty recognizing it; but the inclusion of a simple attentional cue at the center of such an object was sufficient to render it almost unrecognizable (Exp. 4). Even with simple geometric figures, the presence of a second object was sufficient to often render one of the objects invisible (Exp. 2). The simplicity of the figures and the lack of line crossing between objects suggest it is unlikely that object recognition *per se* was at the root of the patient's problem; instead, our results favor an explanation based on the role of spatial attention.

As previously reported, we also found evidence of unilateral neglect and extinction in PCA (Andrade et al., 2010; Silveri, Ciccarelli, & Cappa, 2011; Zilli & Heilman, 2015). These deficits were evident in paper and pencil tasks (i.e., *line bisection* task, and *line cancellation* task) as well as in the covert orienting task (Exp. 5). For the latter, misses were most common for left visual field targets and after a cue to the invalid location, suggesting that the patient had both a spatial-attention bias and a deficit in disengaging attention from the cued location.

In patients with hemispatial neglect due to stroke, perceptual awareness of a contra-lesional stimulus is sometimes extinguished by the presence of a competing stimulus (Rafal, 1994). In those cases, the deficit is caused by a lesion to the right temporoparietal junction that impairs the *disengagement* of attention, and thus prevents its reorienting toward a new location (Corbetta, Kincade, Lewis, Snyder, & Sapir, 2005). There is also a *rightward spatial bias* in neglect, which stems from a functional imbalance between the competing orienting systems in left and right posterior parietal cortices (Cohen, Romero, & Farah, 1994). These two mechanisms act together to deprive of attention the contra-lesional visual field. As a consequence, a stimulus displayed to that side is extinguished from perceptual awareness.

Our findings suggest that the same mechanisms may be at play in the perceptual deficits brought about by posterior cortical atrophy. Whether the “neglect-like” deficit to lateralized stimuli (Exp. 5) and the lack of awareness to centrally displayed items (i.e., simultanagnosia) share a common underlying process remains an open question. However, the findings from Experiment 4, showing that an attention-grabbing object at the center of the screen disrupts perception of additional figures, lend support to the idea that a common deficit in attention disengagement may lie at the core of both deficits. More generally, our study extends previous reports of unilateral spatial neglect in PCA (Andrade et al., 2010) to include situations in which attention is centrally deployed, as in experiments 1–4, when the distracting and target stimuli were displayed concentrically at the center of the screen. In Experiment 4, drawing attention away from the target was sufficient to render the target invisible, even in the absence of spatial lateralization.

While these conclusions are solid, some of our other conclusions are more tentative and await further confirmation. Among them are the findings from Experiment 3 in support of an object-based attentional mechanism: by transforming two objects into one, we enhanced the recognition of the unchanged geometric contours. However, the effect was rather modest and thus should be interpreted cautiously.

In sum, our five experiments aimed to shed light on the mechanisms and phenomenology of simultanagnosia in PCA. The neuropsychology of visual awareness is an area of research where theorizing abounds but empirical evidence is often scarce, due to the limited number of patients suitable for testing. By designing and testing new experimental paradigms with very simple and well controlled stimuli, we have also aimed to contribute to overcome this limitation.

Notes

1. Both neglect and extinction are common in patients with posterior cortical atrophy, with previous research showing that two-thirds of PCA patients showed neglect in paper and pencil tasks such as the line bisection task (Andrade et al., 2010).
2. Although researchers initially thought that in simultanagnosia there was always a local bias, more recent studies have shown that it is also possible to get patients to ignore the local level and only report the global level (Dalrymple, Kingstone, & Barton, 2007; Huberle & Karnath, 2006).
3. Accuracy for the simultaneous and the sequential displays were very similar (99.0% vs. 98.3%) in this group of healthy adults ($n = 6$). As a more sensitive measure of performance, we also computed mean response time. Responses to the simultaneous display were as fast as responses to sequential display (537 ms vs. 550 ms) $t(1, 5) = .89$, $p = .45$. This suggests that in the healthy brain, recognition of the simultaneously displayed figures was not any more difficult than recognition of the sequential display (we excluded from the RT analysis 2.5% of trials made of anticipatory responses (RT < 150 ms), outliers (RT > 2500 ms), and errors).
4. There was no significant difference between blocks 1 and 3 (60%, 58%).
5. The same logic has been applied in studies of extinction in patients with hemispatial neglect. That is, object-based attention theories predict that stimulus properties that facilitate selecting of two items as a single object (e.g., collinearity, closure, surface uniformity) should ameliorate extinction. Supporting this view, a white square in the contra-lesional field of a neglect patient is less likely to be extinguished from awareness when paired with another white square than when paired with a black circle (Gilchrist, Humphreys, & Riddoch, 1996). This reduction of extinction is not due to similarity *per se*, as in the absence of bottom-up grouping object similarity leads to increased extinction (Ricci & Chatterjee, 2004). In another example, a bracket is extinguished less easily when paired with a second bracket than when paired with a circle (Ward, Goodrich, & Driver, 1994). Presumably, bottom-up grouping processes based on similarity, closure and symmetry allow the pair of brackets to be selected as a single object. This suggests that attention is allocated to the object representation, rather than to its constituent features.
6. *Animals*: bird, cow, deer, dog, fish, horse, lion, pig, rabbit, snake, turtle, rat. *Non-animals*: anchor, basket, bell, candle, chair, desk, flower, fork, hat, shirt, shoe, plane.
7. Besides showing perfect accuracy, healthy adults ($n = 6$) also exhibited fast response times (Median RT = 493 ms; Standard Deviation: 388 ms) Such good performance is consistent with previous literature showing that 150 ms of exposure is sufficient for normal observers to reach ceiling performance in object categorization tasks (Grill-Spector & Kanwisher, 2005).

8. The rationale for using a variable cue-target SOA was to discourage RD from relying on time as a cue for reporting the target.
9. Only target trials are reported in the main text. There were also a total of 24 catch trials. RD made a false alarm only in one catch trial, following a leftward cue.

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Disclosure statement

No potential conflict of interest was reported by the authors.

Data availability statement

The data described in this article are openly available in the Open Science Framework at <https://doi.org/10.17605/OSF.IO/4CBTV>.

References

- Andrade, K., Samri, D., Sarazin, M., de Souza, L. C., Cohen, L., de Schotten, M. T., & Bartolomeo, P. (2010). Visual neglect in posterior cortical atrophy. *BMC Neurology*, *10*, 68.
- Bach, M. (1996). The Freiburg visual acuity test—automatic measurement of visual acuity. *Optometry and Vision Science*, *73*, 49–53.
- Balint, R. (1909). Seelenlähmung des “Schauens”, optische Ataxia, räumliche Störung der Aufmerksamkeit. *Monatsschrift für Psychiatrie und Neurologie*, *25*, 51–81.
- Barton, J. J., Malcolm, G. L., & Hefter, R. L. (2007). Spatial processing in Balint syndrome and prosopagnosia: A study of three patients. *Journal of Neuro-ophthalmology*, *27*, 268–274.
- Benton, A. L., Hamsher, K., Varney, N. R., & Spreen, O. (1983). *Contributions to neuropsychological assessment*. New York, NY: Oxford University Press.
- Caine, D., & Hodges, J. R. (2001). Heterogeneity of semantic and visuospatial deficits in early Alzheimer’s disease. *Neuropsychology*, *15*, 155–164.
- Chechlacz, M., Rotshtein, P., Hansen, P. C., Riddoch, J. M., Deb, S., & Humphreys, G. W. (2012). The neural underpinnings of simultanagnosia: Disconnecting the visuospatial attention network. *Journal of Cognitive Neuroscience*, *24*, 718–735.
- Cohen, J. D., Romero, R. D., & Farah, M. J. (1994). Mechanisms of spatial attention: the relationship of macrostructure to microstructure in parietal neglect. *Journal of Cognitive Neuroscience*, *6*, 377–387.
- Corbetta, M., Kincade, M. J., Lewis, C., Snyder, A. Z., & Sapir, A. (2005). Neural basis and recovery of spatial attention deficits in spatial neglect. *Nature Neuroscience*, *8*(11), 1603–1610.
- Coslett, H. B., & Chatterjee, A. (2003). Balint’s syndrome and related disorders. In T. E. Feinberg & M. J. Farah (Eds.), *Behavioral neuroscience and neuropsychology* (2nd ed., pp. 325–336). New York, NY: McGraw-Hill.
- Coslett, H. B., & Saffran, E. (1991). Simultanagnosia. *To See but Not Two See*. *Brain*, *114*, 1523–1545.
- Crutch, S. J., Schott, J. M., Rabinovici, G. D., Murray, M., Snowden, J. S., van der Flier, W. M., & Fox, N. C. (2017). Consensus classification of posterior cortical atrophy. *Alzheimer’s & Dementia*, *13*, 870–884.
- Dalrymple, K. A., Kingstone, A., & Barton, J. J. (2007). Seeing trees OR seeing forests in simultanagnosia: Attentional capture can be local or global. *Neuropsychologia*, *45*, 871–875.
- Delazer, M., Karner, E., Zamarian, L., Donnemiller, E., & Benke, T. (2006). Number processing in posterior cortical atrophy—A neuropsychological case study. *Neuropsychologia*, *44*, 36–51.
- Fernandez-Duque, D., & Black, S. E. (2006). Attentional networks in normal aging and Alzheimer’s disease. *Neuropsychology*. doi:10.1037/0894-4105.20.2.133
- Fernandez-Duque, D., Grossi, G., Thornton, I. M., & Neville, H. J. (2003). Representation of change: Separate electrophysiological markers of attention, awareness, and implicit processing. *Journal of Cognitive Neuroscience*, *15*, 491–507.
- Fernandez-Duque, D., & Posner, M. I. (1997). Relating the mechanisms of orienting and alerting. *Neuropsychologia*. doi:10.1016/S0028-3932(96)00103-0
- Galton, C. J., Patterson, K., Xuereb, J. H., & Hodges, J. R. (2000). Atypical and typical presentations of Alzheimer’s disease: A clinical, neuropsychological, neuroimaging and pathological study of 13 cases. *Brain*, *123*, 484–498.
- Gilchrist, I., Humphreys, G. W., & Riddoch, M. J. (1996). Grouping and extinction: Evidence for low-level modulation of selection. *Cognitive Neuropsychology*, *13*, 1223–1256.
- Grill-Spector, K., & Kanwisher, N. (2005). Visual recognition: As soon as you know it is there, you know what it is. *Psychological Science*, *16*, 152–160.
- Huberle, E., & Karnath, H. O. (2006). Global shape recognition is modulated by the spatial distance of local elements—evidence from simultanagnosia. *Neuropsychologia*, *44*, 905–911.
- Kas, A., de Souza, L. C., Samri, D., Bartolomeo, P., Lacomblez, L., Kalafat, M., ... Sarazin, M. (2011). Neural correlates of cognitive impairment in posterior cortical atrophy. *Brain*, *134*, 1464–1478.
- Lavie, N., & Driver, J. (1996). On the spatial extent of attention in object-based visual selection. *Perception & Psychophysics*, *58*, 1238–1251.
- Pavese, A., Coslett, H. B., Saffran, E., & Buxbaum, L. (2002). Limitations of attentional orienting. Effects of abrupt visual onsets and offsets on naming two objects in a patient with simultanagnosia. *Neuropsychologia*, *40*, 1097–1103. doi: S0028393201001397.
- Pitts, M. A., Martinez, A., & Hillyard, S. A. (2012). Visual processing of contour patterns under conditions of inattention blindness. *Journal of Cognitive Neuroscience*, *24*, 287–303.
- Poppelreuter, W. (1917–1990). *Disturbances of lower and higher visual capacities caused by occipital damage: With special reference to the psychopathological, pedagogical, industrial, and social implications*. Clarendon Press. Maidenhead, Berkshire, United Kingdom. ISBN 978-0-19-852190-7.
- Posner, M. I., Walker, J. A., Friedrich, F. J., & Rafal, R. D. (1984). Effects of parietal injury on covert orienting of attention. *The Journal of Neuroscience*, *4*, 1863–1874.
- Rafal, R. (2001). Balint’s syndrome. In M. Behrmann (Ed.), *Handbook of Neuropsychology* (Vol. 4, pp. 121–141). New York, NY: Elsevier Science.
- Rafal, R. D. (1994). Neglect. *Current Opinion in Neurobiology*, *4*(2), 231–236.
- Rees, G., & Lavie, N. (2001). What can functional imaging reveal about the role of attention in visual awareness? *Neuropsychologia*, *39*, 1343–1353. doi: S0028-3932(01)00122-1.
- Ricci, R., & Chatterjee, A. (2004). Sensory and response contributions to visual awareness in extinction. *Experimental Brain Research*, *157*, 85–93.
- Rizzo, M., & Robin, D. A. (1990). Simultanagnosia: A defect of sustained attention yields insights on visual information processing. *Neurology*, *40*, 447–455.
- Schurger, A., Cowey, A., Cohen, J. D., Treisman, A., & Tallon-Baudry, C. (2008). Distinct and independent correlates of attention and awareness in a hemianopic patient. *Neuropsychologia*, *46*, 2189–2197. doi: S0028-3932(08)00083-3.
- Silveri, M. C., Ciccarelli, N., & Cappa, A. (2011). Unilateral spatial neglect in degenerative brain pathology. *Neuropsychology*, *25*, 554.
- Simons, D. J., & Rensink, R. A. (2005). Change blindness: Past, present, and future. *Trends in Cognitive Sciences*, *9*, 16–20. doi: S1364-6613(04)00293-1.
- Snodgrass, J. G., & Vanderwart, M. (1980). A standardized set of 260 pictures: Norms for name agreement, image agreement, familiarity, and visual complexity. *Journal of Experimental Psychology. Human Learning and Memory*, *6*, 174–215.
- Tang-Wai, D. F., Graff-Radford, N. R., Boeve, B. F., Dickson, D. W., Parisi, J. E., Crook, R., ... Petersen, R. C. (2004). Clinical, genetic, and

- neuropathologic characteristics of posterior cortical atrophy. *Neurology*, *63*, 1168–1174.
- Verfaellie, M., Rapcsak, S. Z., & Heilman, K. M. (1990). Impaired shifting of attention in Balint's syndrome. *Brain and Cognition*, *12*, 195–204.
- Vuilleumier, P. O., & Rafal, R. D. (2000). A systematic study of visual extinction. *Between- and Within-field Deficits of Attention in Hemispatial Neglect*. *Brain*, *123*, 1263–1279.
- Ward, R., Goodrich, S. J., & Driver, J. (1994). Grouping reduces visual extinction: Neuropsychological evidence for weight-linkage in visual selection. *Visual Cognition*, *1*, 101–129.
- Wolpert, I. (1924). Die Simultanagnosie-Störung der Gesamtauffassung. *Zeitschrift für die Gesamte Neurologie und Psychiatrie*, *93*, 397–415.
- Zilli, E. M., & Heilman, K. H. (2015). Allocentric spatial neglect with posterior cortical atrophy. *Neurocase*, *21*, 190–197.