

Kanizsa subjective figures capture visual spatial attention: evidence from electrophysiological and behavioral data

Daniel Senkowski^{a,b,*}, Stefan Röttger^c, Sabine Grimm^d, John J. Foxe^{b,e},
Christoph S. Herrmann^f

^a Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany

^b The Cognitive Neurophysiology Laboratory, Nathan S. Kline Institute for Psychiatric Research, Program in Cognitive Neuroscience and Schizophrenia, 140 Old Orangeburg Road, Orangeburg, NY 10962, USA

^c Institute of Psychology and Ergonomics, Berlin Technical University, 10587 Berlin, Germany

^d Institut für Allgemeine Psychologie, Leipzig University, Leipzig, Germany

^e The City College of the City University of New York, New York, NY, USA

^f Institute for Psychology, Otto von Guericke University, Magdeburg, Germany

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Abstract

Figural binding and attention are two important processes that help to perceive the outside world. Binding is necessary to link together the different features of single objects which are represented in a distributed fashion in the brain. Attention serves to focus onto a small subset of incoming information. It is still not clear how exactly these two mechanisms operate and interact. We performed two experiments employing illusory Kanizsa figures (KFs) to investigate the temporal order of figural binding and spatial attention. In a visual search task, subjects had to detect the presence of a KF among distractor stimuli. We found only a slight increase of reaction times when increasing the number of distractors, indicating that KFs popped out and drew the perceiver's attention. In a further event-related potential (ERP) study, we used displays of the search task as non-informative cue for a subsequent target choice-reaction task. Enhanced contralateral negative amplitudes (starting at about 230 ms) over ventral occipital areas were found for cue displays which included a KF. For target stimuli, faster reaction times and enhanced ipsilateral N1 amplitudes over occipito-parietal areas were observed for validly (target presentation inside a KF) as compared to invalidly cued targets (target presentation outside a KF). Furthermore, enhanced contralateral N1 amplitudes were found for invalidly cued targets. It might be that interactions between perceptual closure processing of the ventral pathway and spatial target processing of the dorsal pathway contributed to the present result. We conclude that KFs automatically capture spatial attention when used as visual cues.

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1. Introduction

Figural binding of visual objects and attention are crucial processes affecting the perception of our environment. The 'binding problem' thereby concerns the questions how we link together and integrate the separately represented features of a single object in the correct combination (Treisman, 1998; Treisman & Gelade, 1980). Most studies have exam-

ined binding processes between different dimensions (e.g. color and form) of the same object (Robertson, 2003). The present article, however, focuses on the investigation of processes that are necessary to perceive illusory Kanizsa figures and their relationship to selective spatial attention. In order to perceive its illusory contours, the edges of the inducer disks of a Kanizsa figure must be bound together to form one coherent object (Fig. 1). It has been suggested that figural binding which is necessary for the perception of illusory Kanizsa figures is closely related to processes of selective spatial attention (Driver, Davis, Russell, Turatto, & Freeman, 2001).

* Corresponding author. Tel.: +1 845 398 6618; fax: +1 845 398 6545.
E-mail address: dsenkowski@nki.rfmh.org (D. Senkowski).

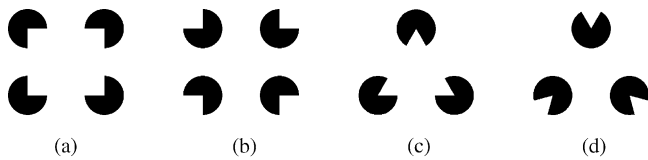


Fig. 1. Examples of frequently used Kanizsa figures and their corresponding distractor stimuli: (a) Kanizsa square, (b) distractor square, (c) Kanizsa triangle, and (d) distractor triangle.

However, it is still not clear how exactly these two mechanisms operate and there are two current models regarding the temporal relation of figural binding and attention.

One model, which we will call the *binding-first model*, proposes that visual binding does not depend on visual spatial attention (Davis & Driver, 1994). Evidence from different visual search experiments using illusory contours such as Kanizsa figures suggests that visual binding is a process that operates in parallel across the visual field (Albert, 1993; Davis & Driver, 1998). It has been reported that the detection of a Kanizsa figure among a varying number of distractor items leads to flat reaction time (RT) slopes smaller than 10 ms/item (Davis & Driver, 1994). This result indicates the pop-out effect of Kanizsa figures, which is presumably the result of a parallel visual search. The findings in behavioral data are well in line with the results of monkey studies, showing that illusory contours are coded already at very early stages in visual processing (areas V1 and V2). This may indicate that object feature binding of illusory contours can occur without spatial attention (Grosf, Shapley, & Hawken, 1993; von der Heydt, Peterhans, & Baumgartner, 1984).

The second model, which we will call the *attention-first model*, proposes that figural binding requires spatial attention. Davis and Driver as well as other authors have been criticized for supplying sufficient information in their search displays to support a parallel search independent of illusory contour presentation (Gurnsey, Humphrey, & Kapitan, 1992; Gurnsey, Poirier, & Gascon, 1996). The critical points were that Davis and Driver (1994) used a prefield to indicate subsequent presentation locations and that the items of the figures were presented at a fixed distance from the fixation point. Further critical points were the use of relatively small set sizes, the large number of practice trials, and the higher luminance gradients inside the target Kanizsa subjective figures as compared to the luminance gradients inside the distractor items. Gurnsey et al. (1996) ran a series of three experiments, in which they tried to control some of these factors. In these experiments, the authors did not find evidence for a pop-out of illusory figures, suggesting that spatial attention may be necessary for illusory contour processing.

The goal of the present article was to investigate the temporal relationship between figural binding of Kanizsa figures and visual spatial attention. To do this, Kanizsa subjective figures among distractor items were used as stimulus material in two experiments. In the first experiment, behavioral data were collected in a visual search paradigm in which the

critical variables that have been described by Gurnsey et al. (1996) were controlled. The second experiment consisted of a visual cueing paradigm in which some of the stimuli of the first experiment were used as cues for a subsequent visual target discrimination task. Event-related potentials (ERPs) and behavioral data were investigated. In accordance with Davis and Driver (1994), we expected to find evidence for the *binding-first model* in both experiments.

2. Experiment 1

In Experiment 1 we investigated whether Kanizsa figures pop-out of visual displays under controlled conditions. The subjects were instructed to detect the presence of a target Kanizsa triangle within a non-symmetric arrangement of inducer disks (Fig. 2a). The independent variable in this experiment was the set size. The set size was manipulated by varying the number of inducer disks. Reaction times (RT) and error rates were measured in response to target trials (presentations that included a Kanizsa triangle) and standard trials (no-Kanizsa triangle). In order to control the critical point mentioned by Gurnsey et al. (1996), we used visual displays which included blank areas at various positions. The luminance of these areas was comparable to the luminance inside the Kanizsa figure (as can be seen in Fig. 2b). Furthermore, some inducer disks in the search displays had the same spatial configuration as the Kanizsa figures, but did not constitute an illusory figure. Furthermore, in contrast to earlier studies, we did not use a prefield to indicate subsequent presentation locations and subjects performed only a few practice trials prior to the experimental runs (Davis & Driver, 1994; Gurnsey et al., 1996). It has been reported that parallel visual search leads to target-present slopes of below 10 ms/item (Davis & Driver, 1994; Enns & Rensink, 1990). Considerably steeper RT slopes/item would support the assumption of a serial search. In accordance with the results of Davis and Driver (1998), as well as Peterhans and von der Heydt (1989), we expected to find a pop-out of the Kanizsa figures, as indicated by shallow RT slopes/item. We propose that such a result would support the *binding-first model*.

2.1. Methods

2.1.1. Participants

Ten right-handed student subjects (25.8 ± 4.2 years, 4 females) participated in Experiment 1. All of them had normal or corrected-to-normal vision and showed no signs of any neurological or psychiatric disorder. Subjects were paid for participating in the experiment and gave written informed consent in accordance with the Declaration of Helsinki.

2.1.2. Stimuli

A 17" Sony Trinitron Monitor was used for visual stimulation. Stimulus presentation was controlled by the

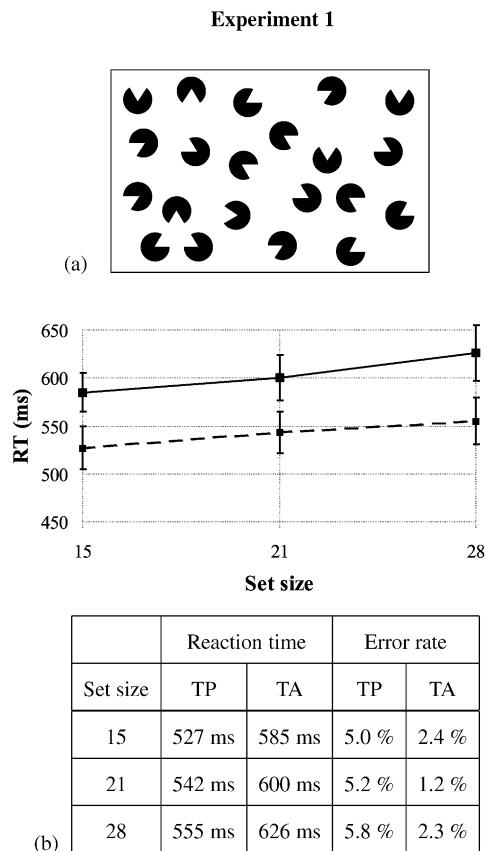


Fig. 2. Panel a: Example for target stimulus in Experiment 1. Arrangement of 21 inducer disks in the visual search display. A target Kanizsa triangle is located in the lower left corner. Panel b: Reaction times and error rates for target present (TP) and target absent (TA) trials are shown. The upper graph show reaction times with standard error of mean for target present (dashed line) and target absent trials (solid line). Flat RT slopes of 2.15 (TP) and 3.15 (TA) ms/item in the experiment indicate a pop-out of Kanizsa figures.

Experimental Runtime System (BeriSoft Cooperation, Germany). The visual angle of the stimulus display was 8° vertical by 12° horizontal. The visual angle of the inducer disks was 1.5° , 1.3° , and 1.1° for set sizes 15, 21, and 28, respectively. Fig. 2a shows a target stimulus of the 21 inducer disks configuration with a Kanizsa triangle in the lower left corner. The number of possible Kanizsa triangle positions varied as a function of set size. Sixteen possible triangles locations were contained in the largest set size.

2.1.3. Procedure

The search displays were presented for 1000 ms at a variable inter-stimulus-interval between 500 and 1500 ms (mean 1000 ms). RT was recorded starting at stimulus onset. Subjects had to indicate by pressing the left or right button (counterbalanced across subjects) whether a target Kanizsa triangle was included or not included in an arrangement of inducer disks. Target probability was 50%. Stimuli were presented in 15 blocks including 30 trials each. A total num-

ber of 150 trials (75 targets, 75 non-targets) was presented for each of the three set sizes. Ten practice trials were presented prior to the experimental session. RTs on error trials, as well as trials with RTs exceeding 2.5 standard deviations of the mean, were excluded from analysis, separately for each subject. The analysis was focussed on the increase of RTs with increasing set size. For this reason, RT differences between the largest and the smallest displays were calculated and divided by the differences of inducer elements. The result of division is the RT slopes/item. Smaller RT slopes/item than 10 ms would indicate a parallel visual search.

2.2. Results

Shorter RTs (541 ms) were found for target present as compared to target absent trials (604 ms). In addition, an increase of RTs with increasing set size was observed (Fig. 2b). The differences of RTs of the 28-element and the 15-element display (28 ms for targets and 41 ms for non-targets) divided by the difference of inducer elements ($28 - 15 = 13$) yielded RT slopes of 2.15 and 3.15 ms/item for target present and target absent trials, respectively.

2.3. Discussion

The search tasks revealed RT slopes per item of around 2–3 ms. Visual search is considered to be parallel when these slopes are shallower than 10 ms/item (Davis & Driver, 1994). Thus, our flat slopes suggest that parallel search strategies were used to detect illusory Kanizsa figures. There is another indication that we are dealing with a parallel search. The RT slopes for target absent trials was not twice as high as RT slopes for target present trials (Wolfe, 1994). However, since an extensive analysis of 1 million search trials yielded a unimodal rather than a bimodal distribution of RT slopes, it has been argued that no clear distinction between parallel and serial search can be made (Wolfe, 1998). Instead, a continuum of search strategies has been suggested. Nevertheless, our RT slopes of around 3 ms/item are clearly within the range of parallel search processes (Davis & Driver, 1994). Therefore, we suggest that the pop-out effect in our experiment supports the assumption that Kanizsa figures can be processed in a parallel visual search. The present Experiment 1 is also comparable with the first experiment (condition 1) reported by Gurnsey et al. (1996). In line with this experiment, our study showed that Kanizsa figures can pop-out of visual displays when they are salient among distractors.

3. Experiment 2

To investigate the relationship between figural binding and spatial attention, we ran a second experiment in which we used electroencephalography (EEG) to measure electric brain

waves evoked by such stimuli. In Experiment 2 we used some of the visual search displays from Experiment 1 as cue displays for a subsequent target choice-reaction task. The cue displays either included or did not include a Kanizsa figure. The Kanizsa figure could occur at one of two fixed positions (horizontal right or left from fixation point) in the display (Fig. 3). Three conditions were compared: (1) A valid cueing condition where the target occurs inside the Kanizsa figure. (2) An invalid cueing condition where the target occurs contralateral to the Kanizsa figure. (3) A no-cue condition where the cue display did not include a Kanizsa figure. Importantly, the Kanizsa figures themselves gave no information about the position of the subsequently presented target and were therefore not relevant for target processing. Since we demonstrated that Kanizsa figures pop-out of the visual displays in Experiment 1, we predicted that Kanizsa figures would lead to an automatic shift of visual spatial attention, even if they were not task relevant. This attention shift should lead to enhanced performance in the valid cue trials as compared to the invalid cue trials and the no-Kanizsa control trials and to differences in the ERPs. Based on previous ERP findings, amplitude effects in response to the laterally presented Kanizsa figures were expected primarily at a latency of about 230 ms after cue display onset over contralateral occipito-temporal scalp regions (Murray et al., 2002). These responses have been previously related to perceptual closure processing (Doniger et al., 2000, 2001). Since the Kanizsa figures were non-informative with relation to the subsequently presented target, such a finding would indicate that illusory Kanizsa figures are automatically processed by the visual system. For the target ERPs we expected to find cueing effects on P1 and N1 amplitudes. Although previous results on target P1 and N1 effects are not totally consistent and seem to be influenced by experimental factors like cue–target interval, cue validity, cue location, and the complexity of the cue and the target stimuli (Eimer, 1994; Fu et al., 2001; Yamaguchi, Tsuchiya, & Kobayashi, 1994), taken together an enhancement of contralateral P1 and N1 amplitudes for validly cued targets can be considered as a well-established finding (Mangun & Hillyard, 1991; Hopfinger, Woldorff, Fletcher, & Mangun, 2001). Thus, if the non-informative Kanizsa figures in the present study automatically capture visual spatial attention, we would expect to find contralateral enhanced P1 and N1 responses for validly cued targets.

3.1. Methods

3.1.1. Participants

Twelve student subjects (25.6 ± 4.5 years, 9 males) participated in Experiment 2. All subjects had normal or corrected-to-normal vision and showed no sign of any neurological or psychiatric disorder. All subjects were right-handed. Subjects were paid for participating in the experiment and gave written informed consent, in accordance with the Declaration of Helsinki.

3.1.2. Stimuli

The center of potential cue and target position was located at a 2.6° visual angle to the left and right of the screen center. The stimulus size was 0.18° visual angle for the target triangle, 1.14° for the inducer disks, and 1.74° for the side length of the illusory Kanizsa triangle. The location of the target stimuli and the Kanizsa figures was on the same height as the fixation dot (as shown in Fig. 3). The background color of all arrangements was white and the color of the inducer disks was black.

3.1.3. Procedure

Fig. 3 shows the procedure of the experiment. Subjects were instructed to look at the center of the screen, which was indicated by a round fixation point and to keep their eyes still during the whole experimental runs. Each trial started with the presentation of a cue display that consisted of an arrangement of 23 randomly distributed inducer disks, which either included a Kanizsa figure (Fig. 3, middle and lower panel), or did not include a Kanizsa figure (Fig. 3, upper panel). In 2/3 of all experimental trials a target triangle, which either pointed to the right or to the left, was presented 290 ms after cue display onset at a fixed right or left location inside the cue display. The inducer disks in the cue displays were configured in a way that the luminance of the displays was balanced. The target location probability of trials including a target was 50% for the right and 50% for the left location. The target was presented for a short time period of 50 ms. Subjects were instructed to indicate the pointing direction of the target triangle by pressing a left button with their right index finger when the triangle pointed to the left, or a right button with their right middle finger when the triangle pointed to the right. The cue display was presented for 670–970 ms (mean 770 ms). After this time period, the cue display disappeared and the fixation point turned into a small ‘+’ or ‘–’ for 500–1100 ms (mean 800 ms), indicating that the response was correct or incorrect, respectively. If a subject did not respond to a target trial within 1000 ms, the fixation point automatically turned into a ‘–’. Using this experimental design, we distinguished three cueing conditions. In valid cue trials, targets were presented inside a Kanizsa figure (Fig. 3, middle panel). In invalid cue trials, targets were presented contralateral to the Kanizsa figure (Fig. 3, lower panel). Finally, no-cue trials did not include a Kanizsa figure (Fig. 3, upper panel). For each of the three cueing conditions 156 target stimuli were presented at both possible target locations, adding up to a total number of 936 trials. The experiment was run in 13 blocks with a length of 72 trials each. Prior to data acquisition, subjects performed 12 practice trials.

Due to the short cue–target interval of 290 ms, it is likely that later activity to the cue display onset overlaps with responses to the subsequently presented target stimuli. For this reason, the responses to the cue display were measured for trials where no subsequent target was presented. We will refer to these trials as ‘no-target trials’. No-target trials were presented for all the three different cue displays (Kanizsa

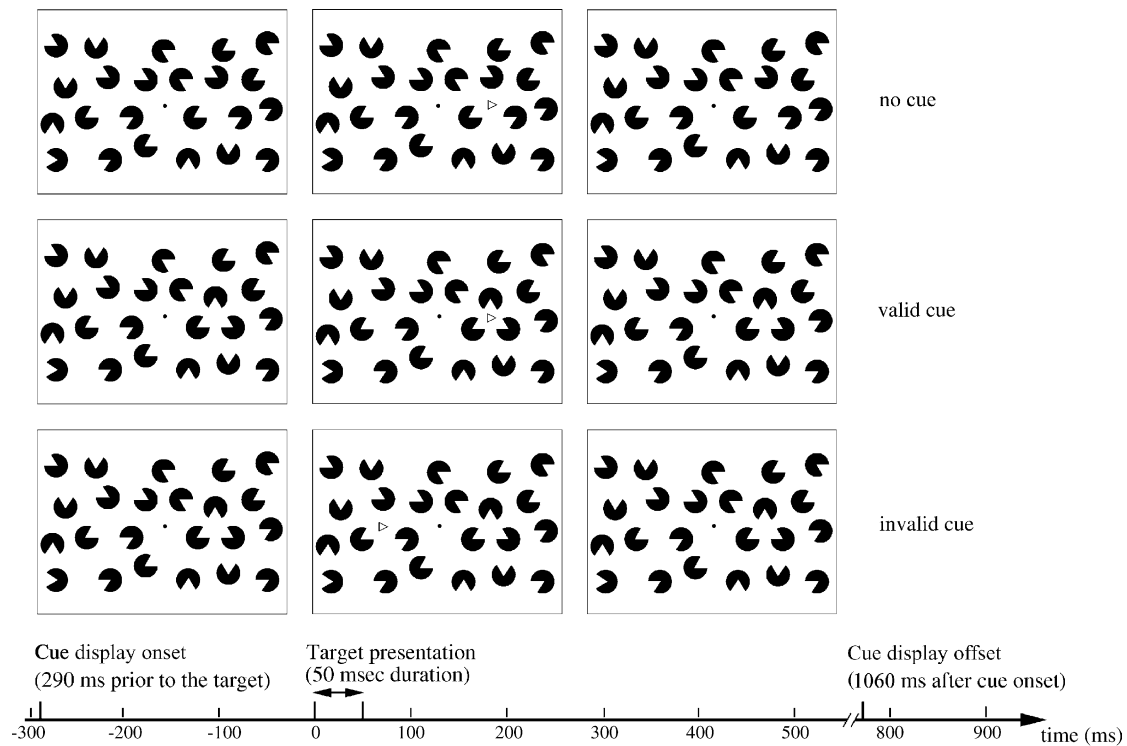


Fig. 3. Procedure of Experiment 2. A cue display, which either included or not included a Kanizsa figure was presented 290 ms prior to the target triangle. The target triangle either appeared inside the Kanizsa figure (valid cue, middle panel), contralateral to the Kanizsa figure (invalid cue, lower panel), or in a cue display that did not contain a Kanizsa figure (no-cue, upper panel). The subjects task was to indicate the direction of the target triangle which could point to either the right or left.

figure left, Kanizsa figure right, and no-Kanizsa). The idea of no-target trials is similar to the idea of ‘no-stim’ events that have been used in various fMRI studies (Burock, Buckner, Woldorff, Rosen, & Dale, 1998; Buckner et al., 1998). ‘No-stim’ events are points in the trial sequence that are randomized as if they were real stimulus events, but during which no stimulus is presented (Busse & Woldorff, 2003). Accordingly, no-target trials in the present study were trials where no target was presented at the time point where targets were presented in regular cue–target trials. In order to eliminate overlapping cue display activity from target ERPs, the ERPs to no-target trials were subtracted from the ERPs to regular cue–target trials. For example, the ERPs to no-target trials with a cue display containing a Kanizsa figure on the left were subtracted from ERPs to the cue display containing a Kanizsa figure on the left with a subsequently presented target on the left. The result of this subtraction is an ERP to a target left that does not contain overlapping activity from the cue display. Thus, the subtraction of no-target trials allowed us to analyze target ERP responses while controlling for the overlapping cue display activity. The effects on target ERP responses that will be described in the result section will be based on these derived target responses where cue-related activity has been subtracted out. For the analyses of ERPs in response to cue displays we used all trials that were identical until the target appeared. For example, the first 290 ms of no-target trials with a Kanizsa figure on the left were combined

with the first 290 ms of all other trials that had a Kanizsa figure on the left side (e.g. valid cue left condition).

Busse and Woldorff (2003) recently showed that omitted stimuli can evoke strong ERP responses of their own if the percentage of these stimuli in the experimental runs is low (e.g. 11 or 22%). However, responses to omitted stimuli were much smaller when a higher percentage of omitted stimuli was included (e.g. 33%). For this reason, a no-target trial probability of 33% was chosen in the present experiment. No-target trials were randomly inserted into the stream of regular cue–target trials (the other 67% of trials). A total number of 156 no-target trials was presented for each of the three different cue displays. Prior to the experiment, subjects were instructed to not respond to no-target trials.

3.1.4. Data acquisition

The EEG was recorded with NeuroScan amplifiers using 124 scalp electrodes mounted in an elastic cap and four additional EOG electrodes (two vertical and two horizontal) to measure vertical and horizontal eye movements. Electrode impedances were kept below 5 k Ω for all channels. Data were recorded referenced to the nose and online band-pass filtered from 0.05 to 100 Hz at a sample rate of 500 Hz. In the offline analyses, data were re-referenced to the algebraic mean of the two mastoid electrodes and filtered with a digital 1 Hz high-pass filter. Averaging epochs for ERP activity lasted from 200 ms before cue display presentation to 500 ms after

target onset. For artifact rejection, trials were automatically excluded from averaging if the standard deviation within a moving 200 ms time interval exceeded $30 \mu\text{V}$ in any one of the scalp and $40 \mu\text{V}$ in any of the EOG channels. After the automatic artifact rejection, all trials were visually inspected and rejected if eye-movement artifacts or electrode drifts were visible. An average number of 396 ± 46 artifact-free cue display trials was entered in the analyses of cue display ERPs. For target ERPs, an average number of 129 ± 16 artifact-free target trials was entered in the analyses. An additional 30-Hz low-pass filter was applied for the presentation of the ERP figures.

3.1.5. Data analyses

The ANOVAs for the behavioral data were calculated for RTs and error rates using the factors Cue Validity (valid cue, invalid cue, no-cue) and Target Location (right, left). Trials with incorrect responses, as well as trials with RTs exceeding 2.5 standard deviations of the mean, were excluded from analyses.

For the analysis of ERPs, baselines were computed in the time interval between 100 and 0 ms prior to cue display onset for each trial and subtracted from the raw data before averaging. To avoid a loss of statistical power that is inherent when repeated measures analysis of variance (ANOVAs) is used to quantify multi-channel EEG data, activity for selected regions of interest (ROIs) was calculated. The time windows and ROIs for the analysis of the ERP data were chosen according to the grand average ERP curves and topographical maps (Figs. 5–10). For the ERPs after cue display onset we calculated the maximum amplitudes for an early (120–160 ms) and a later (230–290 ms) time interval for one left and one right posterior ROI (Fig. 4a). The left ROI was defined using three electrodes over each hemisphere that are located over ventral occipito-parietal scalp regions. For the early and late posterior ROI activity, repeated measures ANOVAs were calculated using the within-subject factors Cue Location (left, right, no-Kanizsa figure) and ROI (right occipito-parietal region, left occipito-parietal region). Next to the posterior activations, a fronto-central positive component in a later time interval (230–290 ms) was investigated at one fronto-central

ROI. This ROI comprises a symmetrically arranged cluster of scalp-sites, spanning the region typically covered by the 10–20 electrodes FCz and Fz (Fig. 4a). For the anterior cue display ERPs an ANOVA using the within-subject factor Cue Location (left, right, no-Kanizsa figure) was calculated with the anterior ROI activity as dependent variable.

For the target ERPs, positive P1 and negative N1 components were observed (Figs. 7 and 9). The posterior topography of the target P1 and N1 components was more dorsal than topography of the late negative ERPs of the cue displays. Two dorsal occipito-parietal ROIs (Fig. 4b) were defined to investigate the maximum of positive amplitudes in a post-stimulus interval between 130 and 160 ms for the P1 component and the minimum of negative amplitudes between 180 and 220 ms for the N1 component. ANOVAs for the target P1 and N1 components as dependent variables were calculated using the within-subject factors Cue Validity (valid, invalid, no-cue), ROI (right occipito-parietal region, left occipito-parietal region), and Target Side (target right, target left).

3.2. Results

3.2.1. Behavior

An ANOVA for the RTs using the factors Cue Validity (valid cue, invalid cue, no-cue) and Target Location (right, left) yielded a significant main effect of the factor Cue Validity ($F(2, 22) = 4.01, P < 0.03$). Post hoc comparisons between the conditions revealed faster RTs for validly cued trials (453 ± 28 ms) as compared to invalidly cued trials (457 ± 30 ms; $F(1, 11) = 5.69, P < 0.04$) and no-cue trials (457 ± 29 ms; $F(1, 11) = 6.34, P < 0.04$). No differences were found between invalidly cued trials and no-cue trials. Furthermore, no significant effects of the factor Target Location were observed. The findings in RTs indicate that the Kanizsa figures captured visual spatial attention which facilitated the processing of the target stimulus. The ANOVA for the error rate yielded no significant differences between validly cued trials ($2.8 \pm 3\%$), invalidly cued trials ($2.6 \pm 2\%$) and no-cue trials ($2.3 \pm 2\%$). No significant effects of the factor Target Location were found.

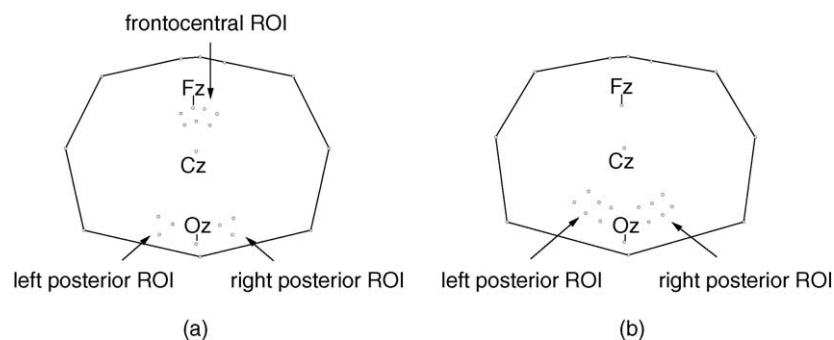


Fig. 4. Definition of regions of interest (ROIs) for the ERP analyses of cue displays (left) and target stimuli (right). (a) ROI definition for cue displays and (b) ROI definition for target stimuli.

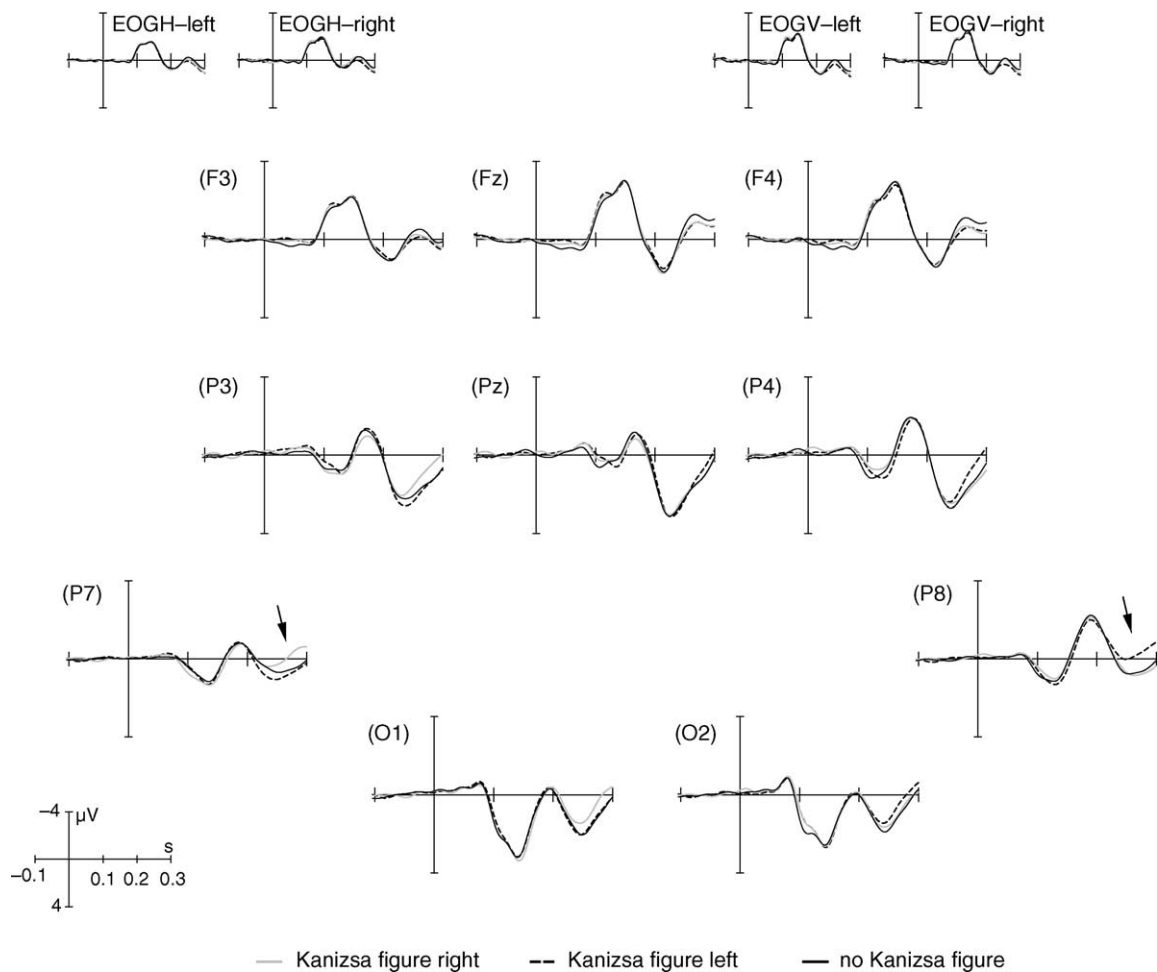


Fig. 5. ERPs averaged across 12 subjects in response to cue displays including a right Kanizsa figure (solid gray line), a left Kanizsa figure (dashed black line) and control displays not containing a Kanizsa figure (solid black line). An enhanced contralateral negativity over posterior electrode sites was found starting after about 230 ms. The electrodes are located close to the named 10–20 electrode site.

3.2.2. Event-related potentials to the cue displays

The ERPs and topographical distribution of the responses to the cue displays are shown in Figs. 5 and 6. The ANOVA for the early posterior negativity (minimum of amplitudes in a time interval between 120 and 160 ms) revealed no significant effects of the factors Cue Location (left, right, no Kanizsa figure) and ROI (right occipito-parietal region, left occipito-parietal region). However, the ANOVA for the late time window (maximum of amplitudes in a time interval between 230 and 290 ms), using the same factors as for the early time window, yielded a significant main effect of Cue Location ($F(2, 22) = 4.93, P < 0.02$) and a significant interaction Cue Location \times ROI ($F(2, 22) = 13.28, P < 0.001$). The most positive amplitudes were found when the cue display did not contain a Kanizsa figure ($3.22 \mu\text{V}$). In contrast, amplitudes were less positive when the cue display contained a Kanizsa figure on the left ($2.70 \mu\text{V}$) or on the right side ($2.73 \mu\text{V}$). Note that the less positive amplitudes for displays containing a Kanizsa figure most likely reflect an additional negative activation (Doniger et al., 2001). Due to the significant interaction between the factors Cue Location \times

ROI, further ANOVAs were calculated separately for the left and the right ROIs using the factor Cue Location. For the left ROI, a significant effect was found ($F(2, 22) = 14.36, P < 0.0001$), indicating that late posterior amplitudes were less positive when a Kanizsa figure was presented on the right side ($2.22 \mu\text{V}$) as compared to the left side ($2.99 \mu\text{V}$) and as compared to the no-Kanizsa cue displays ($3.07 \mu\text{V}$). A different result was found for the right posterior ROI. At the posterior right ROI, less positive amplitudes were observed when Kanizsa figures were presented at the left side ($2.40 \mu\text{V}$) as compared to Kanizsa figures that were presented at the right side ($3.24 \mu\text{V}$) and as compared to no-Kanizsa displays ($3.38 \mu\text{V}$) ($F(2, 22) = 6.29, P < 0.01$). To summarize these results, positive amplitudes in a time interval between 230 and 290 ms were significantly reduced contralateral to the location of the Kanizsa figure. Since the Kanizsa figures were not task relevant, this finding indicates that the figures were automatically processed by the visual system. The temporo-occipital topography of these effects furthermore suggests a crucial involvement of the ventral visual processing stream (Fig. 6). Next to the two posterior ROIs, a central anterior ROI

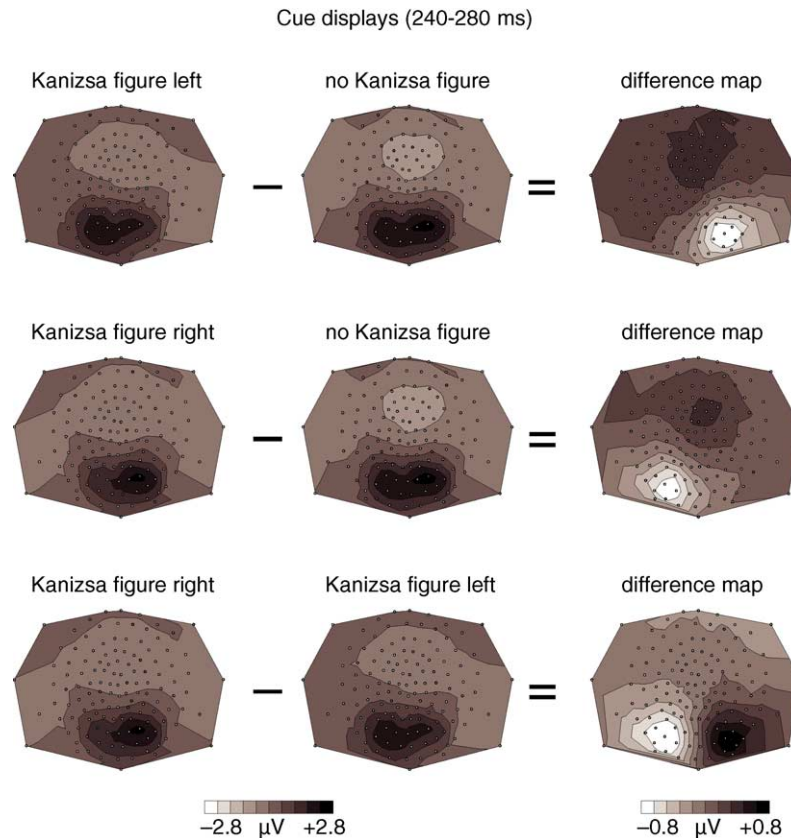


Fig. 6. Experiment 2: Topographical maps of cue display ERPs. Topographies of event-related fields for a time interval between 240 and 280 ms after cue display onset. The figure shows an enhance posterior negativity (compared to no Kanizsa control displays) contralateral to the location of the Kanizsa figure. This negativity is probably related to perceptual closure processing.

was investigated for the later time interval (230–290 ms) using the within-subject factor Cue Location. This ANOVA, however, did not reveal a significant result.

3.2.3. Event-related potentials to the target stimuli

The ERPs and topographies of the responses to the target stimuli are shown in Figs. 7–10. ANOVAs were calculated for two posterior ROIs for the time window of the P1 (maximum of amplitudes in a time interval between 120 and 160 ms) and the N1 (minimum of amplitudes in a time interval between 180 and 220 ms) components using the within-subject factors Cue Validity (valid, invalid, no-cue), ROI (right occipito-parietal region, left occipito-parietal region), and Target Side (target right, target left). For the early amplitudes of the P1 component a significant interaction between the factors ROI \times Target Side was found ($F(1, 11) = 10.31, P < 0.01$). For this reason, ANOVAs for targets that were presented in the right and left hemifield were calculated separately. For targets presented in the left hemifield a significant effect of the factor ROI was found, indicating that P1 amplitudes were more positive at the right ($1.21 \mu\text{V}$) as compared to the left ROI ($0.27 \mu\text{V}$; $F(1, 11) = 8.75, P < 0.02$). No significant effects of the factor Cue Validity were observed. The ANOVA for P1 amplitudes in response to targets that were presented at

the right hemifield also revealed a significant effect of the factor ROI ($F(1, 11) = 5.4, P < 0.05$), indicating higher P1 amplitudes at the left ($0.73 \mu\text{V}$) as compared to the right ROI ($0.06 \mu\text{V}$). Again, no further effects were observed. Next, occipito-parietal target N1 amplitudes were analyzed in an ANOVA using the same within-subject factors as for P1 amplitudes. In this ANOVA, a significant interaction between the factors Target Side \times ROI was found ($F(1, 11) = 21.1, p < 0.0008$), indicating that N1 amplitudes contralateral to the target location were more negative than N1 amplitudes ipsilateral to the target (target location left: right ROI = $-6.70 \mu\text{V}$, left ROI = $-4.86 \mu\text{V}$; target location right: right ROI = $-4.49 \mu\text{V}$, left ROI = $-7.89 \mu\text{V}$). In addition, a significant interaction between the factors Cue Validity \times Target Side \times ROI was found ($F(2, 22) = 4.96, P < 0.02$). Further ANOVAs were calculated separately for targets presented in the left and right hemifield using the factors Cue Validity and ROI. For targets that were presented in the left hemifield a significant effect of the factor ROI was found, indicating higher N1 amplitudes on the right ($-6.70 \mu\text{V}$) as compared to the left ROI ($-4.86 \mu\text{V}$; $F(1, 11) = 9.87, P < 0.0094$). Furthermore, a significant interaction between the factors Cue Validity \times ROI was found ($F(1, 11) = 21.1, P < 0.0008$). Interestingly, N1 amplitudes at the left ROI were more negative for

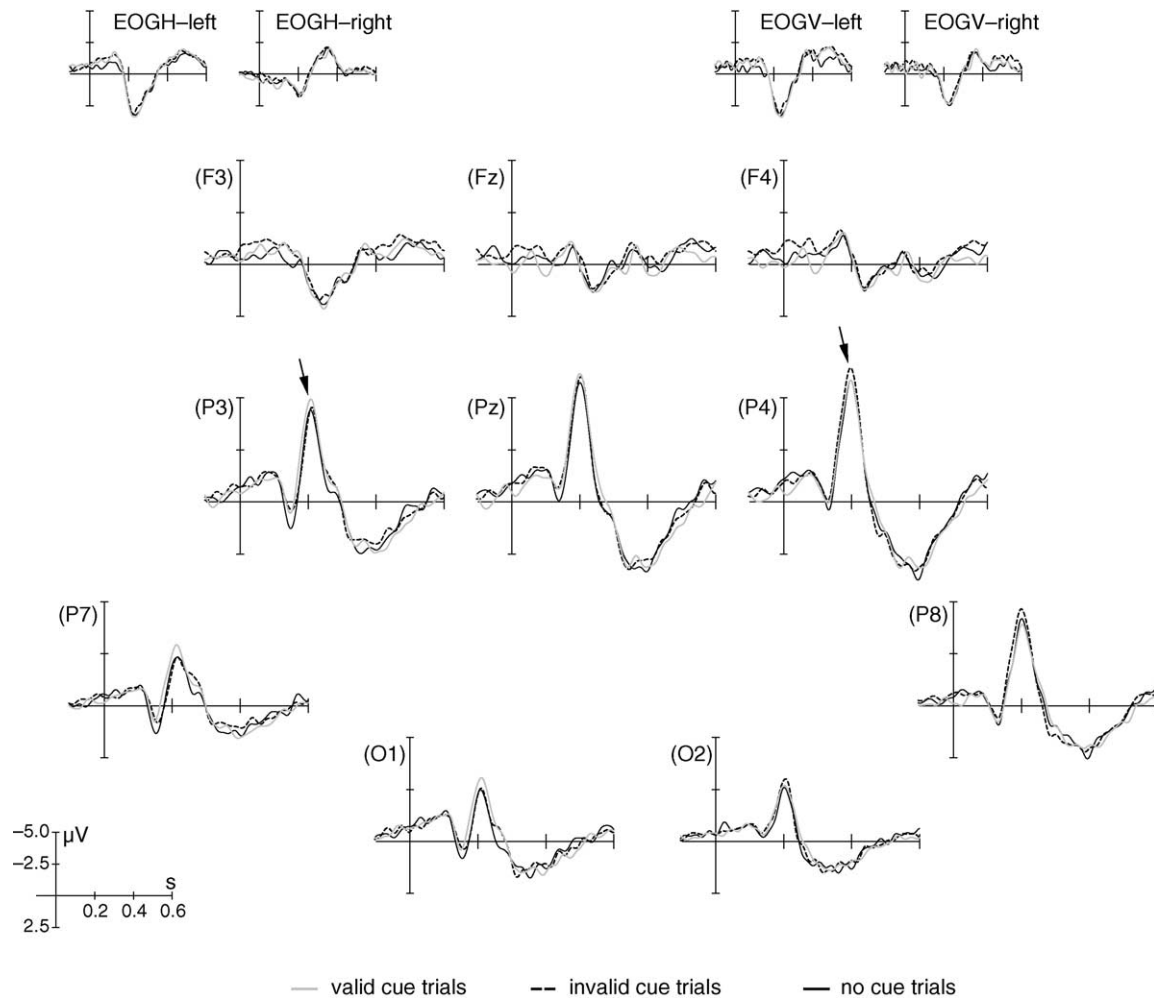


Fig. 7. Experiment 2: ERPs in response to target presentation left. ERPs averaged across 12 subjects in response to target presentation left. The solid gray line represents validly cued trials, the dotted black line invalidly cued trials and the solid black line no-cue control trials. Enhanced posterior N1 amplitudes (at about 190 ms) were found contralateral to the target location for invalidly cued trials. In contrast, posterior ipsilateral (left side) N1 amplitudes were more negative for validly cued trials. No significant differences between the three cueing conditions were found for the target P1 component.

validly cued trials ($-5.30 \mu\text{V}$) as compared to invalidly cued trials ($-4.70 \mu\text{V}$) and no-Kanizsa control trials ($-4.60 \mu\text{V}$). For the right ROI, more negative N1 amplitudes were observed for invalidly cued trials ($-7.24 \mu\text{V}$) as compared to validly cued trials ($-6.34 \mu\text{V}$) and no-Kanizsa control trials ($-6.53 \mu\text{V}$). For the targets presented in the right hemifield, a significant main effect of the factor ROI was found ($F(1, 11) = 16.11, P < 0.002$). Target N1 amplitudes were more negative contralateral to the target location ($-7.89 \mu\text{V}$) as compared to ipsilateral target N1 amplitudes ($-4.49 \mu\text{V}$). No further significant effects with relation to the factors Cue Validity were found in the ANOVA for targets presented in the right hemifield. However, comparing Figs. 8 and 10 shows that the ERP differences between the different cueing conditions for targets presented in the left and right hemifield have a very similar pattern: N1 amplitudes to invalidly cued trials were more negative at contralateral electrode sites whereas N1 amplitudes to validly cued trials were more negative at ipsilateral electrode sites.

3.3. Discussion

Shorter RTs for valid cue as compared to no-cue and invalid cue trials indicate a visual spatial attention shift towards the Kanizsa figure. A behavioral facilitation for validly cued targets as compared to invalidly cued targets has been reported in various studies using cue–target intervals shorter than 300 ms (Anllo-Vento, 1995; Hopfinger, & Mangun, 1998). For longer cue–target intervals this reaction time facilitation usually disappears and RTs to validly cued targets can even be slower than RTs to invalidly cued targets. This effect has been called inhibition of return (IOR, Posner & Cohen, 1984). The present data, however, do not indicate any effects of IOR in RTs. The main effect for the current cue–target interval of 290 ms was a facilitation of RTs for valid as compared to invalid and no-Kanizsa control trials. This indicates that attention was automatically shifted towards the non-informative Kanizsa figure. Interestingly, the shift of attention did not result in longer RTs to invalidly cued trials.

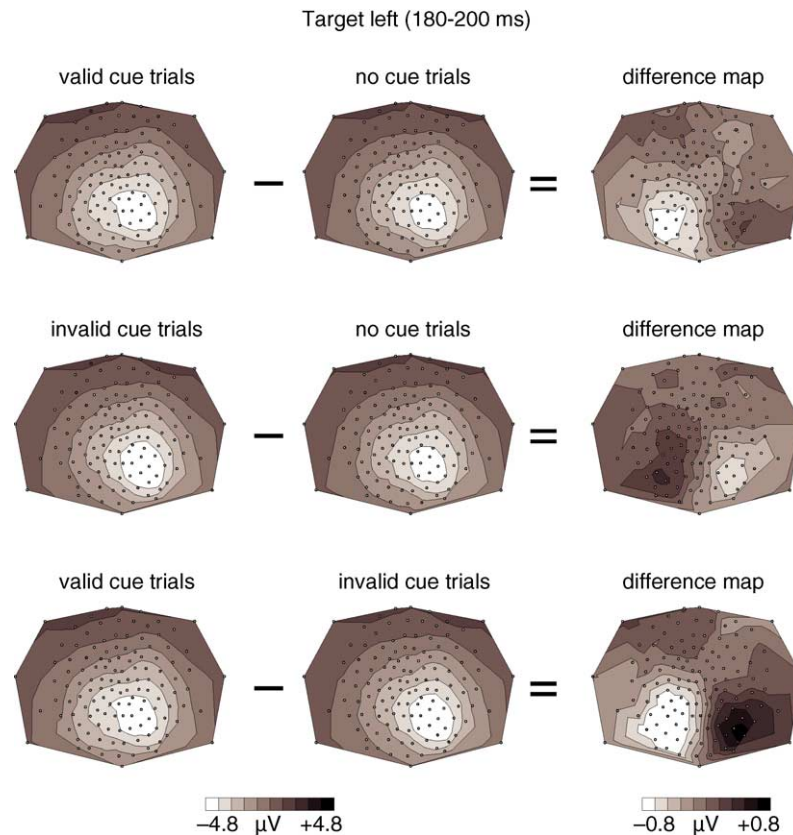


Fig. 8. Experiment 2: Topographical maps of target left trials. Topographies of event-related fields for left target trials for a time interval between 180 and 200 ms. The figure shows an enhanced posterior ipsilateral negativity for valid as compared to no-cue control trials (upper panel). The middle panel shows a slightly enhanced negativity contralateral to the Kanizsa figure for the invalid as compared to valid trials. The lower panel shows the comparison between valid and invalid trials.

The RTs of these trials did not differ from the no-Kanizsa control trials, suggesting that the Kanizsa figure did not affect the processing of invalidly cued targets. Low error rates between 2 and 3% suggest that the task was relatively easy to perform. It may be that the non-informative Kanizsa figures lead to an amplification of spatial attention at the location of the figures. Since the number of possible Kanizsa figure and target locations was limited, it is likely that these locations already had an increased attentional monitoring before the cue displays were presented. It has recently been demonstrated that spatial attention can be divided between separated locations over extended time periods (Müller, Malinowski, Gruber, & Hillyard, 2003). However, the amplification of spatial attention at the location of the Kanizsa figure did not affect RTs when the target was presented outside the Kanizsa figure.

The first effects of Kanizsa figures in the ERP were found at about 230 ms after cue display onset (Figs. 5 and 6). ERP amplitudes contralateral to the location of the Kanizsa figure were more negative as compared to ERPs in response to no-Kanizsa control cue displays. The responses to Kanizsa figures were similar to an enhanced occipito-temporal negativity that has been described by Doniger and colleagues during the processing of fragmented images (Doniger et al.,

2000, 2001). In addition, a further study has reported a similar modulation of the visual ERP after lateral presentation of Kanizsa figures (Murray et al., 2002). The more negative amplitudes which started at about 230 ms post-stimulus have been termed N_{c1} which involves negativity that is associated with perceptual closure processing (Doniger et al., 2000). In line with the assumption that occipito-temporal cortices are involved in perceptual closure processing is our finding of enhanced negative amplitudes contralateral to the Kanizsa figure. Moreover, the temporo-occipital topography of the effect indicates a crucial involvement of the ventral visual pathway. It has been previously shown that the processing of illusory Kanizsa figures activates ventral occipito-temporal brain areas (Hirsch et al., 1995; Kruggel, Herrmann, Wiggins, & von Cramon, 2001; Mendola, Dale, Fischl, Liu, & Tootel, 1999; Murray et al., 2002). For this reason, we suggest that the present ERP effects to the Kanizsa figures mainly reflect processing of the ventral pathway, which is known to be the main pathway in visual feature object processing (Ishai, Ungerleider, Martin, Schouten, & Haxby, 1999). We did not find differences between the displays for the earlier ERPs at a latency between 130 and 160 ms. Positive amplitudes over lateral posterior scalp areas and early negative ampli-

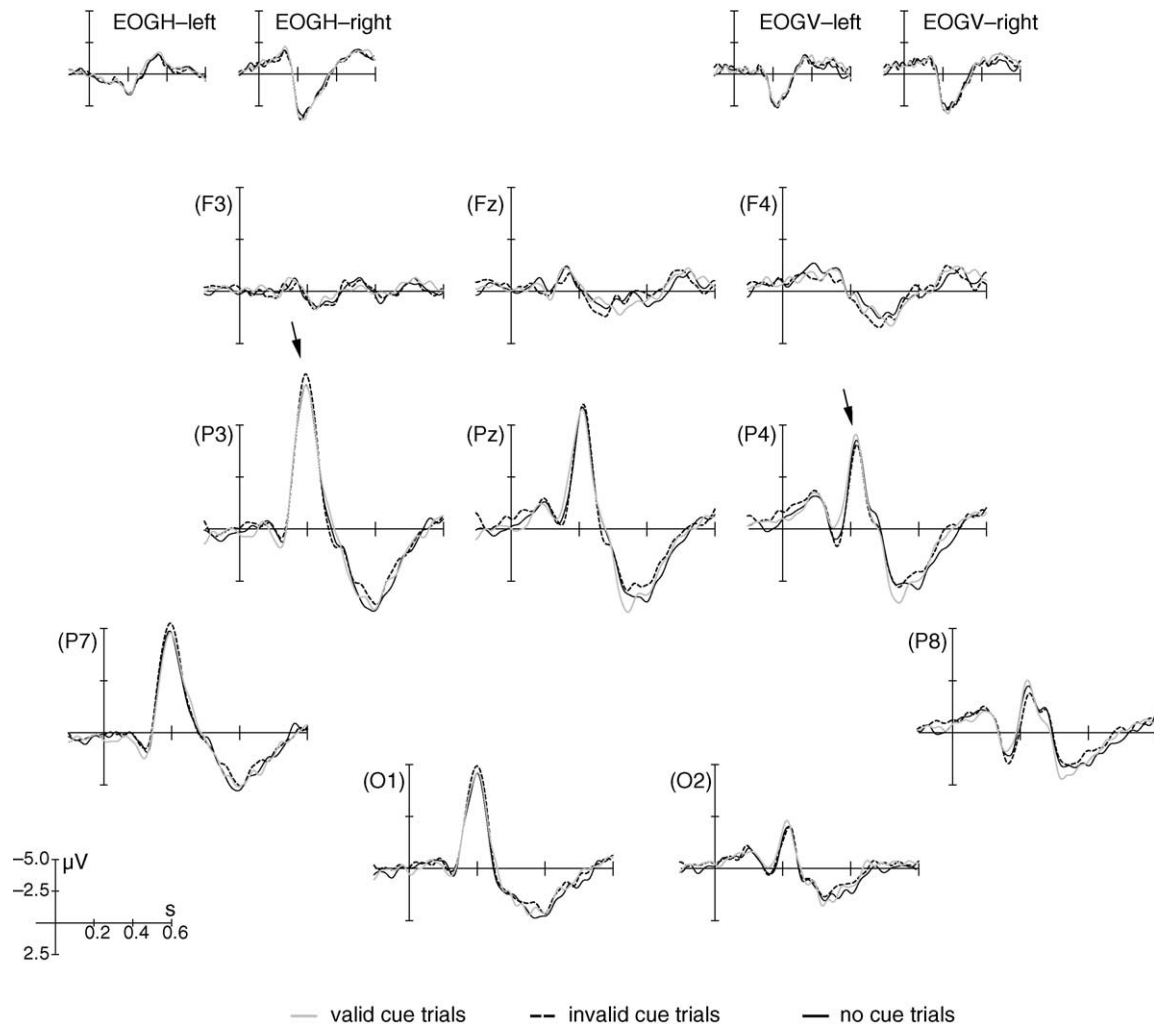


Fig. 9. Experiment 2: ERPs in response to target presentation right. ERPs averaged across 12 subjects in response to target presentation right. The solid gray line represents validly cued trials, the dotted black line invalidly cued trials and the solid black line no-cue control trials. Posterior N1 amplitudes (at about 190 ms) were more negative contralateral to the target location for invalidly cued trials. The posterior ipsilateral (right side) N1 amplitudes, however, were slightly more negative for validly cued trials. Although these differences did not reach statistical significance, the activity pattern is similar to those observed for target left trials. No significant differences between the three cueing conditions were found for the target P1 component.

tudes over central anterior areas did not differ between the cue displays. The posterior positivity probably reflects the visual P1 component which has been related to spatial attention (Heinze & Mangun, 1995; Woldorff et al., 1997). Since the generators of this component are located in the extrastriate cortices (Mangun, Buonocore, Girelli, & Jha, 1998), we suggest that initial early responses of these areas did not contribute to the later effects of the cue displays and to the subsequent cueing effects on target N1 amplitudes. Furthermore, we did not find ERP differences over anterior scalp regions, suggesting that the three cue displays were processed by these areas in a comparable way.

As a main result for the target stimuli, we found ipsilaterally enhanced target N1 amplitudes for valid as compared to invalid and control trials. The effects on target N1 amplitudes were stronger when targets were presented in the left hemifield. Furthermore, a slight increase of contralateral N1 amplitudes was observed for invalidly cued trials. Most stud-

ies investigating target N1 amplitudes for cue–target intervals longer than 300 ms report enhanced contralateral target N1 amplitudes for validly cued trials (Hillyard, Vogel, & Luck, 1999; Mangun, & Hillyard, 1991). For this reason, our findings differ somewhat from our prediction that contralateral N1 amplitudes should be enhanced for validly cued trials. However, the findings of studies using cue–target intervals shorter than 300 ms are more inconsistent than the findings of studies using longer cue–target intervals (Fu, Fan, Chen, & Zhuo, 2001; Luck et al., 1994). For example, similar cueing effects on N1 amplitudes as in the present study have been reported before by Fu et al. (2001) who investigated cue–target effects in two EEG studies. Fu and colleagues reported smaller contralateral and enhanced ipsilateral N1 amplitudes for validly as compared to invalidly cued target trials. The cues in these studies were presented laterally, similar to the present study, and the cue–target intervals were also short, ranging from 100 and 300 ms. The authors suggested that

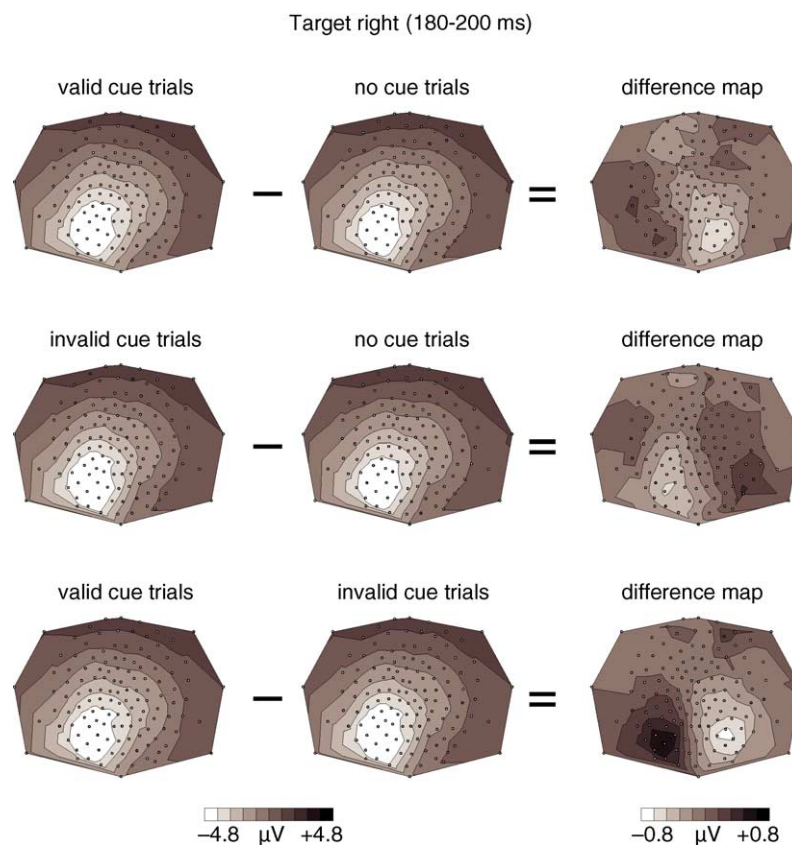


Fig. 10. Experiment 2: Topographical maps of target right trials. Topographies of event-related fields for left target trials for a time interval between 180 and 200 ms. The figure shows similar topographical effects as for the target left trials. N1 amplitudes ipsilateral to the target position were found for the contrast between valid as compared to no-cue control trials (upper panel). In contrast, a slightly enhanced negativity was found contralateral to the Kanizsa figure for the invalid as compared to valid trials (middle panel). The lower panel shows the comparison between valid and invalid trials.

their findings indicated a functional differentiation between the P1 and N1 component. This assumption is supported by another study in which the effects of a non-informative cue on target ERPs were systematically investigated (Hopfinger and Mangun, 1998). Although Hopfinger and Mangun found enhanced P1 amplitudes for validly as compared to invalidly cued trials for a cue–target interval between 34 and 234 ms, they did not report amplitude modulations for the N1 component. While the cue display effects on later ERP amplitudes fit well with previously reported findings (Doniger et al., 2000), the effects on target N1 amplitudes might be related to a number of design-specific parameters. For example, we used fixed 290 ms cue–target interval, non-informative laterally presented illusory figures as cues, briefly presented small targets, and a small number of possible target locations. However, the ERP effects on the target N1 component together with the facilitation effects in the RTs indicate that the Kanizsa figure within the cue display influenced the processing of the subsequently presented target stimulus. Furthermore, the cue display effects on ERPs support the idea that Kanizsa figures are automatically processed by the visual system.

In contrast to the target N1 amplitudes, we did not find significant effects between valid, invalid and control trials for the

target P1 component. Interestingly, various previous studies using a peripheral cue and a cue–target interval shorter than 290 ms have reported an enhancement of the P1 component for validly as compared to invalidly cued targets trials (Doallo et al., 2004; Fu et al., 2001; Hopfinger, & Mangun, 2001). The lack of cueing effects on target P1 amplitudes in the present study also might be related to factors that differ between the present study and previous examinations. A fixed cue–target interval of 290 ms was used in the present experiment. Doallo et al. (2004) reported an enhancement of contralateral P1 amplitudes for validly cued targets at a fixed cue–target interval of 100 ms. However, the authors found the opposite effect (enhanced P1 amplitudes for invalid cue trials) when they used a fixed cue–target interval of 300 ms. Similarly, Hopfinger and Mangun (2001) reported cueing effects on the P1 amplitude for a short cue–target interval of 34–234 ms, whereas they did not find cueing effects on P1 amplitudes for a longer cue–target interval of 566–766 ms. Other factors that differed between the present study and previous investigations were the physical properties of the cue stimuli. We used complex visual cue displays that either contained or did not contain an illusory Kanizsa figure, whereas in most previous studies, a more basic cue (e.g. a short blinking (extinguishing) of four dots) was used (Hopfinger & Mangun, 1998). The first

ERP differences between the cue displays in the present study were found after about 230 ms contralateral to the location of the Kanizsa figure and were still observed when the target is presented. This indicates that the ventral visual pathway is involved in perceptual closure processing at the time-point of target presentation. It might be that the closure processing in the ventral pathway interacts with the early target processing in the dorsal pathway. Further studies are necessary to investigate possible interactions between perceptual closure processing of cue displays and spatial attention effects on early target ERP amplitudes in more detail.

4. General discussion

We investigated the relationship between attentional processes and visual object feature binding in two different experiments. Both experiments showed that Kanizsa figures pop-out within an arrangement of similar distractor items. The results of Experiment 1 support the *binding-first model* by showing that Kanizsa figures can be detected in a parallel search by the visual system, even when factors like luminance and configuration are controlled. In Experiment 2, a possible cueing effect of non-informative Kanizsa triangles within the figural display arrangement of Experiment 1 was investigated. The results of this experiment demonstrate that Kanizsa figures can act as a visual spatial cue for a subsequently presented target. In order to perceive Kanizsa subjective figures, binding processes are necessary. We suggest that the later contralateral ERP effects of the Kanizsa figures in the cue displays are related to perceptual closure processing of these figures (Doniger et al., 2000). The topography of this effect suggests a crucial involvement of the ventral visual processing stream which has been previously shown to be the main pathway of visual feature object processing (Ishai et al., 1999). However, the topography of the effect differed from the topography of target N1 amplitudes. The processing of the laterally presented targets showed an activity pattern over the dorsal visual pathway. It has been described before that this pathway is crucially involved in the processing of visual spatial attention (Woldorff et al., 2002). Therefore, Experiment 2 gives further evidence that binding processes in the case of Kanizsa figures can precede rather than follow an attention shift.

An alternative explanation for pop-out effects of Kanizsa figures could be a low-spatial-frequency blurring of the figure (Ginsberg, 1975). We cannot exclude the possibility that low-spatial-frequency blurring might contribute to the present results. However, in a series of four experiments, Davis and Driver (1998) explicitly investigated whether the pop-out effect of Kanizsa figures remains when factors like the low-spatial-frequency blurring and the grouping of aligned edges were controlled (e.g. by using black crosses instead of inducer disks to form an illusory figure). Indeed, the pop-out effect of Kanizsa figures was found even if these factors were controlled. Thus, Davis and Driver concluded that

Kanizsa figures can be coded in parallel as occluding surfaces. Another factor that may have influenced the results of Experiment 2 is the limited number of possible target locations (two different locations). It is likely that these two positions had an increased attentional monitoring that may have made the Kanizsa figures at these locations more salient. However, as demonstrated in Experiment 1, Kanizsa figures popped out of visual search displays even if the number of inducer disks, and correspondingly, the number of target locations were systematically varied. Further studies should address whether Kanizsa figures also capture visuo-spatial attention when targets can appear at more than two possible locations. A further factor that might have contributed to the present results is the collinearity of aligned edges within the Kanizsa figures. ERP amplitudes in response to Kanizsa figures and non-illusory control stimuli with similar collinear aligned edges have been investigated in previous studies by Herrmann and Bosch (2001) and Murray et al. (2004). For example, Herrmann and Bosch (2001) showed that enhanced ERP amplitudes in response to Kanizsa figures cannot be explained by collinearity. We thus argue that in the present study our stimuli also elicited figural binding processes which led to the perception of Kanizsa illusory figures.

There is also evidence that feature binding processes involve processing in higher cortical areas of the parietal lobe (Robertson, 2003), showing that attentional top-down processes are closely related to feature binding mechanisms, as suggested, for example, by the *Feature Integration Theory* (Treisman & Gelade, 1980). Thus, both bottom-up and top-down processes have been associated with feature binding, showing that the *binding-first model* and the *attention-first model* do not exclude each other. Interestingly, several studies supporting the *attention-first model* reported attention effects on feature binding of different dimensions of an object like color, form, and location (Friedman-Hill, Robertson, & Treisman, 1995; Treisman, 1998). In contrast, evidence for the *binding-first model* came frequently from figural object binding processes within one dimension (e.g. the binding of inducer disks as demonstrated in the present work). This suggests that different types of binding might be related to spatial attention in different ways.

In summary, the present work revealed evidence for the assumption that Kanizsa figures can be processed parallel across the visual field. The behavioral and ERP results of Experiment 2 furthermore suggest that the processing of a laterally presented Kanizsa figure influences the processing of a subsequently presented target stimuli. This indicates that Kanizsa figures can automatically capture visual spatial attention.

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