Bias for the Left Visual Field in Rapid Serial Visual Presentation: Effects of Additional Salient Cues Suggest a Critical Role of Attention

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Abstract

Everyday experience suggests that people are equally aware of stimuli in both hemifields. However, when two streams of stimuli are rapidly presented left and right, the second target (T2) is better identified in the left hemifield than in the right hemifield. This left visual field (LVF) advantage may result from differences between hemifields in attracting attention. Therefore, we introduced a visual cue shortly before T2 onset to draw attention to one stream. Thus, to identify T2, attention was correctly positioned with valid cues but had to be redirected to the other stream with invalid ones. If the LVF advantage is caused by differences between hemifields in attracting attention, invalid cues should increase, and valid cues should reduce the LVF advantage as compared with neutral cues. This prediction was confirmed. ERP analysis revealed that cues evoked an early posterior negativity, confirming that attention was attracted by the cue. This negativity was earlier with cues in the LVF, which suggests that responses to salient events are faster in the right hemisphere than in the left hemisphere. Valid cues speeded up, and invalid cues delayed T2-evoked N2pc; in addition, valid cues enlarged T2-evoked P3. After N2pc, right-side T2 evoked more sustained contralateral negativity than left T2, least long-lasting after valid cues. Difficulties in identifying invalidly cued right T2 were reflected in prematurely ending P3 waveforms. Overall, these data provide evidence that the LVF advantage is because of different abilities of the hemispheres in shifting attention to relevant events in their contralateral hemifield.

INTRODUCTION

In many everyday life situations, we are exposed to multiple sources of information that are changing very dynamically, like during car driving or basketball playing. In those situations, we have the impression of being equally aware of stimuli in both, left and right, visual fields. Recent studies contradict this belief showing a large advantage for the left visual field (LVF) whenever stimuli have to be identified in complex tasks, like the two-stream rapid serial visual presentation task (briefly “two-stream RSVP”). In this task, two streams of letters are rapidly presented in the LVF and right visual field (RVF) simultaneously, and two targets, T1 and T2, randomly presented within the streams, have to be identified (Holländer, Corballis, & Hamm, 2005). T1, being a salient stimulus (e.g., a colored letter), is relatively easy to identify. T2, being an inconspicuous stimulus (e.g., a digit in the color of the other letters), is often missed. Crucially, whereas T1 identification is equally good in both visual fields, T2 is more often missed in the RVF than in the LVF (Verleger, Śmigasiewicz, & Möller, 2011; Śmigasiewicz et al., 2010; Verleger et al., 2009, 2010; Scalf, Banich, Kramer, Narechania, & Simon, 2007; Holländer et al., 2005). This LVF advantage is not caused by deviations of fixation toward the LVF (Verleger, Dittmer, & Śmigasiewicz, 2013; Verleger et al., 2009), nor entirely by learned habits of reading direction (Śmigasiewicz et al., 2010), nor by overload of the left hemisphere (LH) with verbal input (Asanowicz, Śmigasiewicz, & Verleger, 2013). A remaining plausible explanation, suggested by Asanowicz et al. (2013), still has to be tested: The LVF advantage in identifying rapidly presented stimuli might be related to dominance of the right hemisphere (RH) in attentional processes, in particular, in stimulus-driven orienting of attention (Shulman & Corbetta, 2012).

The aim of this study is to examine the hypothesized relationship between the LVF advantage and orienting of attention. Our means of testing this relationship was to insert cues that were intended to reflexively attract participants’ attention to one of the two streams: Small red frames were presented briefly before T2 onset, either surrounding the stimuli in one stream or centered at the fixation point. Thus, cues could be presented in the same stream as T2 (valid cues), in the other stream than T2 (invalid cues), or in the center (neutral cues). Center cues were preferred over a no-cue control condition used in pilot studies, to match all conditions for the temporal information provided by the cue, thereby providing a...
baseline of the LVF advantage. We assumed that cues, being salient color-deviant stimuli at unpredictable locations (similar to T1), will automatically capture attention (Jonides, 1981; Posner, 1980). Thereby, demands on orienting of attention should be lowest after valid cues, because then T2 will be presented directly in the focus of attention. If the LVF advantage is because of an imbalance between hemifields in stimulus-driven orienting of attention, then, by attracting attention to the T2 location and thereby compensating for the disadvantage of T2 in the RVF, valid cues should cause a decrease of the LVF advantage. In contrast, demands on orienting of attention should be highest after invalid cues, because then T2 falls outside the current focus of attention. The largest LVF advantage was therefore expected in the invalid cue condition.

We tested these hypotheses in two experiments: one behavioral and the second one with the same task but also with electroencephalographic (EEG) measurement of ERPs, providing more direct assessment of dynamics of neural responses to cues and to targets. By being salient and laterally presented stimuli, valid and invalid cues are expected to evoke visual ERPs (P1 and N1) that are larger in the contralateral hemisphere. These cue-evoked potentials are expected to be followed by T2-evoked activity. Previous two-stream RSVP studies have shown that correctly identified T2 stimuli evoked N2pc and P3 components (Śmigasiewicz, Weinrich, Reinhardt, & Verleger, 2014; Verleger, Dittmer, et al., 2013; Verleger, Talamo, Simmer, Śmigasiewicz, & Lencer, 2013; Verleger et al., 2009, 2011). N2pc is a negative deflection recorded at about 200 msec after target onset and measured as the difference between posterior sites PO8 and PO7 contralateral minus ipsilateral to the target (Eimer, 1996; Wascher & Wauschkuhn, 1996; Luck, Fan, & Hillyard, 1993). As N2pc is supposed to reflect attentional selection needed for correct identification of relevant stimuli (Verleger et al., 2009, 2011; Eimer, 1996; Luck et al., 1993), amplitudes and latencies of T2-evoked N2pc are expected to decrease after valid cues. This is because less attention is needed in this case (Sawaki, Geng, & Luck, 2012). On the other hand, the invalid cue condition provides large demands on attention; thus, along with impaired T2 identification, larger T2–N2pc amplitudes and longer T2–N2pc latencies should be observed for invalidly cued T2s, as compared with the valid and neutral cue conditions. Of importance, previous two-stream RSVP studies demonstrated shorter latencies of N2pc evoked by LVF–T2, when compared with RVF–T2, in line with the LVF advantage in accuracy, which was interpreted as reflecting dominance of the RH in target selection (Verleger et al., 2009, 2011). Similar asymmetry in T2-evoked N2pc latency is expected to be replicated in the current study.

P3 is a positive deflection evoked by task-relevant stimuli starting about 300 msec after target onset. Its major component, the P3b, with a maximum at the parietal midline, has been suggested to reflect updating of working memory (Donchin & Coles, 1988) or may indicate activation of stimulus–response links (Verleger, Jaśkowski, & Wascher, 2005) or decisional processes (Kelly & O’Connell, 2013). In the context of RSVP tasks, P3 reflects success in target identification, being evoked with correctly identified T2 and suppressed with incorrectly identified T2 (Krančioch, Debener, & Engel, 2003; Rolke, Heil, Streb, & Hennighausen, 2001). Its amplitude has been shown to correlate with “bottom–up strength” and perceptual distinctiveness of target stimuli (Craiston, Wyble, Chen, & Bowman, 2009; Sergent, Baillet, & Dehaene, 2005), which means that P3 is larger for targets easy to perceive than for targets difficult to perceive. Thus, P3 amplitudes, being larger after left than right T2 as observed in a previous two-stream RSVP study (Verleger et al., 2011), might be related to greater perceptual distinctiveness of left T2 as a consequence of attention being biased to the left hemifield. Importantly, in the context of cueing paradigms, salient task-irrelevant cues that reflexively attract attention were shown to increase P3 amplitude evoked by targets presented in validly cued locations at short cue–stimulus intervals (Ries & Hopfinger, 2011; Hopfinger & Mangun, 1998, 2001). This increase in P3 amplitude was interpreted to reflect that stimuli at cued locations are treated as more important in processing at further stages than stimuli at uncued locations (Hopfinger & Mangun, 1998). Thus, in the current study, apart from replicating larger P3 amplitudes with left T2 than with right T2 (Verleger et al., 2011), we also expected to observe increased P3 amplitudes with validly cued T2s compared with neutrally and invalidly cued ones.

METHODS
Participants
Twenty-nine students from the Jagiellonian University in Krakow (eight men, mean age = 20.4 years, SD = 1.3 years) participated in the first experiment (E1), and 19 students of the University of Lübeck (nine men, mean age = 25.4 years, SD = 2.4 years) participated in the second experiment (E2). Informed written consent was obtained. Credit points were assigned in E1, and €7 per hour were paid in E2. All participants reported normal or corrected-to-normal vision, normal color vision, and no history of neurological disorders. In E2, three participants were rejected from analysis because of systematic eye movements toward T1 (see below for details), leaving 16 participants for analysis. All participants were right handed, with scores in the Edinburgh Handedness Inventory (Oldfield, 1971) of 84.1 (SD = 17) in E1 and 92.5 (SD = 13.9) in E2.

Stimuli and Apparatus
The task is illustrated in Figure 1. The stimuli were presented on the white background of a computer screen, which was a 21-in. TFT screen in E1, driven at 60 Hz, and a
16-in. CRT screen (visible diagonal) in E2, driven with 100 Hz. The screen was situated at about 0.8 m from participants’ faces in E1 and at about 1.2 m in E2, such that 1 cm on the screen spanned 0.75° visual angle in E1 and 0.5° in E2. Stimuli consisted of capital letters of the Latin alphabet and the digits 1–6 in Helvetica font and were displayed in two streams left and right from the fixation cross. The cue was a red frame presented 50 msec before T2 onset surrounding either the left or right letter stream or the fixation cross. Letter stimuli were 11 mm high and maximally 9 mm wide, with their midpoints 16 mm from fixation (1.2° in E1, 0.8° in E2). Fixation was marked by a 3 × 3 mm small cross at the center of the screen. The red frame (the cue) was 18 × 18 mm when surrounding the letter stream and 6 × 6 mm when presented at fixation. In each trial, two targets had to be identified: The first target (T1) was a blue letter (D, F, G, J, K, L), and the second target (T2) was a black digit (1, 2, 3, 4, 5, 6). The distractor set consisted of all letters presented in black font. DMDX software (Forster & Forster, 2003) was used for experimental control in E1; and Presentation software, version 14.1 (Neurobehavioral Systems, Inc., Albany, CA), in E2.

**Procedure**

Participants were seated in front of the computer screen with a standard keyboard placed on a table at comfortable working position. Each trial began with the fixation cross displayed at screen center, which remained during the following stimulus series. After 800 msec, the first frame of distractor stimuli appeared for 117 msec in E1 and for 110 msec in E2, immediately followed by the next frame. The entire trial consisted of 13–20 frames. T1 was presented in the 6th, 8th, or 10th frame; and T2, in the third or fifth frame after T1 (lags 3 or 5, amounting to 350 or 583 msec after T1 onset in E1 and 330 or 550 msec after T1 onset in E2), each one on the left or right side accompanied by a letter distractor on the opposite side. T2 was followed by five pairs of distractors. T1 and T2 were randomly selected from the target sets, and distractors were randomly selected with replacement from the distractor set (with a restriction against immediate repetition and against equal characters simultaneously left and right). The cue was displayed 50 msec before T2; thus the cue appeared 67 msec in E1 and 60 msec in E2 after onset of the distractors preceding T2 and remained on the screen until the offset of T2. The cue could be presented around the fixation cross (neutral cue), in the same stream as the following T2 (valid cue) or in the other stream than the following T2 (invalid cue), each in one third of all trials in random order. Thereby, the validity of the spatial cues (i.e., valid or invalid) was 50/50%. At the end of each trial, participants entered their responses on the keyboard: first, the T1 letter on the middle row and then the T2 digit on the number pad, even if the answer was not known. The next trial started immediately after the T2 response. In E2, eye position was measured by means of a remote infrared eye tracker (600 series binocular; Eyegaze LC Technologies, Fairfax, VA) and online feedback by software (Interactive Minds, Dresden, Germany), which communicated with the Presentation program. At the beginning of each trial, fixation was checked by the
program. In case of a deviation of more than 6 mm from vertical midline, a red exclamation mark was presented at midline, inducing shifts of gaze back to fixation.

The relevant independent variables were Cue Type (neutral, valid, invalid) and T2 Side (left, right). This resulted in six experimental conditions that were replicated 96 times in E1 and 120 times in E2, in random sequence, with a break inserted after half of the trials. Before the task proper, some trials were presented in slow motion for practice.

**EEG Recording and Preprocessing in E2**

EEG was recorded with Ag–AgCl electrodes (Easycap, www.easycap.de) from 60 scalp sites, which were eight midline positions from AFz to Oz and 26 pairs of symmetric left and right sites, and from the nose tip. During recording, Fz served as reference, and data were offline rereferenced to the nose tip. The ground electrode was placed at Fpz. Vertical EOG and horizontal EOG were recorded for control of eye movements. Data were amplified from DC to 250 Hz by a BrainAmp MR plus and stored at 500 Hz per channel. Further processing was done with Brain-Vision Analyzer software (version 2.02; Munich, Germany). After rereferencing and low-pass filtering at 20 Hz, segments were defined in each trial for analysis of cue- and target-evoked potentials, starting 100 msec before T1 onset and lasting until 500 msec after T2 for lateralized potentials evoked by cue and T2 and until 700 msec after T2 for the T2-evoked P3 component. Data were referred to the first 100 msec of the segment as baseline and edited for artifacts, by rejecting trials with zero lines, and absolute amplitudes ≥ 100 μV. This artifact rejection procedure was chosen to be conservative enough to allow rejecting segments with blinks and eye movements. Finally, data were referred to the first 100 msec before cue or before T2 onset as baseline and high-pass filtered at 5 Hz for measuring the P3 component. For analysis of cue-evoked potentials, segments were averaged across artifact-free trials irrespective of correctness in the identification of T1 or T2. For analysis of T2-evoked potentials, segments were averaged across artifact-free trials with accurate identifications of both T1 and T2. Averages of differences contralateral–ipsilateral to T1 were formed for horizontal EOG. Any participant’s data were rejected if these averages deviated from baseline by 10 μV within 700 msec after T1 onset, indicating eye movements ≥ 0.7° toward T1. As mentioned in the Participants section, this criterion led to excluding 3 of the 19 participants in E2.

**Data Analysis**

Percentages of trials with correct responses were computed in each condition for T1 as T1-correct relative to all T1-correct trials. For reducing analysis to the essential factors, data were collapsed over T1–T2 lags (three and five frames) and over relative locations of the two targets to each other (T1 and T2 in the same stream or in other streams).

Cue and T2-related ERPs were measured separately for the LH and RH to examine if these stimuli were processed in the same way in both hemispheres. To obtain cue-related activities uncontaminated by constant asymmetries between hemispheres, activity at each hemisphere in the condition with cues on the contralateral side was corrected by subtracting activity at the same hemisphere in the condition with cues on the ipsilateral side, for example, PO8 (left cue) − PO8 (right cue) for the RH and PO7 (right cue) − PO7 (left cue) for the LH (Verleger et al., 2011; Oostenveld, Stegeman, Praamstra, & Oostrom, 2003). The same procedure (with side of T2 rather than side of cue) was performed to obtain T2-evoked potentials. In case of cue-related potentials, this procedure was made separately for valid and invalid cues. The cue-related potential was apparent as negativity in the N1 range (contralateral to lateral cues) and was measured as the largest negative peak 100–220 msec after cue onset. In case of T2-evoked potentials, the subtraction procedure was performed separately for the three cue conditions, two lags, and two possible T1 and T2 locations (same stream or different stream). However, for ANOVA calculations, data were averaged across two lags and two T1 and T2 locations as these two factors were irrelevant to the present purpose. T2-evoked negativity reached its maximum at about 250 msec, which is the N2pc range. Latencies of this N2pc were determined by means of the “50% area” measure, that is, as the time point (relative to T2 onset) that divided the 150- to 300-msec voltage × Time area-under-the-waveform into equal halves (Craaston et al., 2009; Luck & Hillyard, 1990). To obtain reliable estimates of 50% area also in cases where the negative deflection did not span the entire 150- to 300-msec epoch, any positive values in this epoch were set to zero. N2pc amplitude was then measured as mean absolute amplitude of the ±25-msec range around this 50% area latency. Apart from N2pc, T2 evoked also sustained posterior contralateral negativity (SPCN) that was apparent from 300 msec after T2 onset onward, larger at PO7 than PO8. To quantify this SPCN, average amplitudes were measured in two epochs (300–370 and 370–440 msec after T2 onset).

In addition, the P3 component of ERPs evoked by T2 was measured from Pz separately for the three cue conditions (neutral, valid, and invalid), two lags, two spatial relations of T1 and T2 (same stream or different streams), and two sides of T2 (left or right). Similar to T2-evoked N2pc, for ANOVA calculations, data were averaged across the two lags and two T1 and T2 locations. P3 amplitudes and latencies were measured at the most positive peak 250–550 msec after T2 onset.
One participant was rejected from analysis of T2-evoked potentials, because of too many artifacts in the EEG. For the other participants, the minimum number of trials included in the T2-evoked average was 33, and the mean number was 77 ($SD = 20.6$).

ANOVAs were performed on behavioral and P3 parameters with the repeated-measurement factors Cue (neutral, valid, invalid) and Target Side (left, right; with target being T1 or T2 depending on analysis). For T2-evoked N2pc and SPCN, Target Side was replaced by hemisphere (right, left; i.e., PO8, PO7), because of the way these parameters were computed (see above), and Epoch (300–370 and 370–440 msec) was an additional factor in analyzing SPCN. Hemisphere was the only factor in ANOVA on potentials evoked by the lateral cues. Greenhouse–Geisser correction of degrees of freedom was applied to effects of the Cue factor. Corrected $p$ values will be reported, but $\varepsilon$ values will not be indicated, for brevity. Likewise, partial eta-squared will not be explicitly indicated, being easily derived from the reported $F$ values by the formula $\eta^2_p = (F/df)/(1 + F/df)$. IBM SPSS statistics 20 was used. When interactions were significant, their sources were clarified by ANOVAs or $t$ tests on subsets of the data.

**RESULTS**

**Target Identification**

Identification rates of T1 and T2 in both experiments are presented in Figure 2 and are compiled in Table 1. Data from E1 and E2 were entered to one ANOVA, with Experiment as an additional factor, between participants.

**T1**

T1 was correctly identified in 94% of the trials in E1 and in 86% of the trials in E2, $F(1, 43) = 15.5, p < .001$, probably because of the differences in stimulation rate and stimulus size. Identification rates differed neither between T1 sides nor between cue conditions, as could be expected because cues were presented well after T1.

Table 1. Percentages of Correct Identification of T1 (Relative to All Trials) and of T2 (Relative to All T1-correct Trials) in Experiments 1 and 2

<table>
<thead>
<tr>
<th>Cue</th>
<th>Neutral</th>
<th>Valid</th>
<th>Invalid</th>
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<tbody>
<tr>
<td></td>
<td>Left</td>
<td>Right</td>
<td>Left</td>
</tr>
<tr>
<td>T1 (E1)</td>
<td>95 (07)</td>
<td>95 (07)</td>
<td>93 (08)</td>
</tr>
<tr>
<td>T1 (E2)</td>
<td>88 (09)</td>
<td>87 (10)</td>
<td>87 (10)</td>
</tr>
<tr>
<td>T2 (E1)</td>
<td>91 (16)</td>
<td>84 (20)</td>
<td>98 (04)</td>
</tr>
<tr>
<td>T2 (E2)</td>
<td>93 (08)</td>
<td>82 (15)</td>
<td>97 (04)</td>
</tr>
</tbody>
</table>

Values are presented as means ($SD$) across participants.
The LVF advantage was replicated, as reflected by an effect of T2 Side in separate analysis of neutral cues, $F(1, 43) = 44.1, p < .001$. Cue validity had a large effect ($F(2, 86) = 58.0, p < .001$). As expected, this effect was larger on right T2 than on left T2 ($F(2, 86) = 38.3, p < .001$; Cue effect for right T2: $F(2, 86) = 75.8, p < .001$; for left T2: $F(2, 86) = 28.3, p < .001$). Thus, in the valid cue condition, the LVF advantage was smaller than in the neutral cue condition ($F(1, 43) = 43.4, p < .001$), being significant in this pooled analysis across E1 and E2 only (effect of T2 Side with valid cues: $F(1, 43) = 5.6, p = .02$; separately in E1: $F(1, 43) = 1.9, ns$; in E2: $F(1, 43) = 2.6, ns$). Correspondingly, in the invalid cue condition, the LVF advantage was larger than in the neutral cue condition ($F(1, 43) = 22.7, p < .001$; T2 Side effect in the invalid cue condition: $F(1, 43) = 48.0, p < .001$). There were no significant differences between experiments ($F \leq 3.4, p \geq .07$).

**EEG Results**

**Cue-evoked Negativity**

Figure 3A presents the waveforms evoked by lateral (valid and invalid) cues recorded from sites contralateral and ipsilateral to the cue and their difference wave contralateral minus ipsilateral to the cue. The positive peak at about 80 msec after cue onset (P1) and the negative peak at about 130 msec (N1) were larger at contralateral than ipsilateral sites, and increased contralateral negativity extended from the N1 peak onward until about 260 msec. In Figure 3B, this contralateral–ipsilateral difference waveform is displayed separately for valid and invalid cues. The cue-evoked P1 and N1 components were exactly of the same size and latency irrespective of cue validity, suggesting that cues were equally processed no matter whether the following T2 was in the same stream or in the other stream. However, starting about 200 msec after cue onset, activity differed between valid and invalid cue conditions. When cues were valid, with the following T2 presented at the same side as the cue, cue-evoked negativity merged with T2-evoked negativity. When cues were invalid, with T2 presented on the opposite side, cue-evoked negativity was superseded by a downturn to positivity, which might be interpreted as negativity (N2pc) evoked by T2.

Figure 3C shows cue-evoked contralateral–ipsilateral differences separately for the two hemispheres (see Methods for details). Cue-evoked negativity peaked earlier at the RH (recorded from PO8, 157 msec) than at the LH (recorded from PO7, 185 msec), $F(1, 15) = 20.1, p < .001$. The difference in amplitude was not significant ($F(1, 15) = 2.1, p = .20$).
T2-evoked Negativity

Figure 4 presents the waveforms contralateral and ipsilateral to T2 in the three cue conditions, separately for short and long temporal distances between T1 and T2 (lags 3 and 5). As can be seen, waveforms consistently were more negative contralateral than ipsilateral to T2 at 150–300 msec after T2 onset. This effect appeared to be delayed after invalid cues. The same contralateral and ipsilateral waveforms, averaged across the two lags between T1 and T2, are displayed in Figure 5A, together with the difference waves (contralateral minus ipsilateral to T2) for all three cue conditions. After neutral cues, N2pc is clearly seen as the negativity between 150 and 350 msec after T2 onset. In the invalid cue condition, after early positivity and negativity evoked by the cue (cf. previous section), a second negative peak is evoked by T2, presumably being the N2pc. In the invalid cue condition, the early positivity and negativity evoked by the cue are mirrored with respect to the valid cue condition because, here, the cue was presented on the other side than T2 (such that the difference waveform is ipsilateral minus contralateral to the cue), followed by a clear negative N2pc peak at around 250 msec evoked by T2.

N2pc. In Figure 5B, all three difference waves (T2-contralateral minus T2 ipsilateral) from Figure 5A are plotted in one graph for direct comparison. Evidently, N2pc was evoked earlier after valid cues than after neutral cues and later after invalid cues than after neutral cues (Cue Validity: $F(2, 28) = 10.7, p = .002$; valid vs. neutral: $F(1, 14) = 9.2, p = .01$; invalid vs. neutral: $F(1, 14) = 5.9, p = .03$). T2-evoked negativity (pooled across cues) separately at the RH (PO8) and LH (PO7) is plotted in Figure 5C. Latencies did not significantly differ between PO8 and PO7 (Hemisphere: $F(1, 14) = 3.0, p = .1$).

In contrast to N2pc latency, there were hemispheric differences in N2pc amplitude, modulated by cue validity (Cue × Hemisphere: $F(2, 28) = 5.4, p = .01$): N2pc was equally large at PO8 and PO7 with neutral cues ($F(1, 14) < 0.01, ns$), tended to be larger at the LH than at the RH with valid cues ($F(1, 14) = 3.9, p = .07$), and was smaller at the LH than at the RH with invalid cues ($F(1, 14) = 7.0, p = .02$). These relationships are illustrated in Figure 6, where T2-evoked negativity is split into potentials evoked at the RH (PO8) and the LH (PO7) separately for the three cue validity conditions. As can be seen, these amplitude modulations were probably because of merging and dissociation of cue- and T2-evoked potentials. To detail, in the valid cue condition, activity started to differ between RH and LH already at around 50 msec after T2 onset (which is 100 msec after cue onset), because of the fact that left cues evoked N1 earlier at the RH than right cues at the LH. In consequence, the first negative peak (around 70 msec after T2 onset) at the RH (PO8, black line) is evoked by the cue, and the second negative peak (at around 180 msec after T2 onset) is presumably the N2pc component evoked by T2. In contrast, at the LH, cues evoked the N1 component later, and therefore, N1 merged with T2-evoked N2pc (PO7, gray line). Because the cue-evoked N1 component tended to be larger at the LH than at the RH, the N2pc component evoked by T2, by being merged with N1, tended to be larger at the LH than at the RH. In the invalid cue condition, where cues were in the stream opposite to T2, cue-evoked activity is mirrored with respect to the valid cue condition. Thus,
cue-evoked N1 is on the positive side, again larger at the LH than at the RH, making the following N2pc evoked by T2 start from a more positive voltage level at the LH than at the RH and, in consequence, reducing the peak amplitude of N2pc at the LH.

SPCN. Figures 5C and 6 show that, from 300 msec onward, there was negative sustained activity at the LH, whereas this activity turned to the positive side at the RH. This SPCN was measured in two consecutive 70-msec epochs, from 300 to 440 msec. Overall, SPCN was more negative at PO7 than at PO8 (Hemisphere: F(1, 14) = 10.5, p = .005), generally in the first epoch (Epoch × Hemisphere: F(1, 14) = 5.3, p = .04; effect of Hemisphere in Epoch 1: F(1, 14) = 11.1, p = .005; in Epoch 2: F(1, 14) = 8.5, p = .01) and in the second epoch still with neutral and invalid cues (Epoch × Hemisphere × Cue: F(2, 28) = 13.5, p < .001; effects of Hemisphere at Epoch 2 after neutral cues: F(1, 14) = 7.4, p = .02; after invalid cues: F(1, 14) = 5.5, p = .03; after valid cues: F(1, 14) = 0.2, ns).

T2-evoked P3

T2-evoked P3 amplitudes were larger with valid cues than with neutral and invalid cues (Figure 7A; F(2, 28) = 12.5, p < .001; comparison between valid and neutral cues: F(1, 14) = 24.1, p < .001; valid and invalid cues: F(1, 14) = 13.8, p = .002; neutral and invalid cues: F(1, 14) = 0.2, ns) and were larger when evoked by left T2 than by right T2 (Figure 7B; F(1, 14) = 13.3, p < .001). Figure 7C suggests that this T2-side effect might have been largest after neutral cues, but the interaction was not significant, F(2, 28) = 1.1, p = .3.

The negative deflection visible with neutral cues at about 250 msec after T2 onset (Figure 7A and 7C), which was maximum at occipital sites, might be an N1 evoked by offset of these centrally located cues, which occurred 110 msec after T2 onset. The lateral (valid and invalid)
cues evoked an earlier negative deflection, at about 180 msec; therefore, the P3 component started to turn in positive direction earlier with lateral than neutral cues. These negative deflections were also less negative with left cues (black solid and light blue lines) than with right cues (gray and blue lines), and therefore, P3 turned in positive direction even earlier with left cues. Importantly, the P3 component peaked earlier when evoked by invalidly cued right T2 than in all other conditions (Cue: $F(2, 28) = 6.1, p = .01$; Cue × T2 Side: $F(2, 28) = 6.6, p = .01$; cue effect for T2 left: $F(2, 28) = 0.7, ns$; Cue effect for T2 right: $F(2, 28) = 8.5, p = .002$; cue effect for right T2 in valid vs. neutral cue conditions: $F(1, 14) = 1.6, ns$; in invalid vs. neutral cue conditions: $F(1, 14) = 6.4, p = .02$; in invalid vs. neutral cue condition: $F(1, 14) = 18.9, p = .001$), and so only in the invalid cue condition P3 latency was earlier when evoked by right T2 than by left T2 (T2 Side effect for invalid cues: $F(1, 14) = 10.5, p = .01$; for neutral cues: $F(1, 14) = 0.8$; for valid cues: $F(1, 14) = 0.9$). This premature P3 latency seems to be be-cause both of the relatively early onset of the P3 component after left cues and of its quick return to baseline. Because invalidly cued right T2 had the lowest T2 identification rate in behavioral data, this feature of P3 probably reflects the low perceptual availability of right T2 in this most difficult condition.

**DISCUSSION**

The LVF advantage in two-stream RSVP has been replicated multiple times in different laboratory settings (Asanowicz et al., 2013; Verleger et al., 2010, 2011; Holländer et al., 2005). It has proven remarkably stable so far, being only slightly influenced by habits of reading direction (Śmigasiwicz et al., 2010), nonverbal and facial stimulation (Asanowicz et al., 2013), positive and negative priming (Verleger, Śmigasiwicz, Michael, & Niedeggen, 2012), or change in mutual inhibition between hemispheres (Śmigasiwicz et al., 2014). However, previous experiments did not test the hypothesis of unequal abilities of both hemispheres in attentional processes, although attention actually might be a critical factor producing this bias. In two experiments, we studied the relationship between attention and the LVF advantage, combining the two-stream RSVP task with spatial cueing (Posner, 1980). We hypothesized that the LVF advantage is related to dominance of the RH in stimulus-driven orienting of attention (Corbetta & Shulman, 2002). Salient cues were inserted in the stimulus streams to attract attention exogenously toward or away from T2. We expected that these salient cues would modulate the LVF advantage, reducing it with valid cues and increasing it with invalid cues.

**T2 Identification Rates**

Both experiments confirmed these expectations: T2 identification improved with valid cues and declined with invalid cues as compared with neutral cues, which confirms the efficiency of the cue manipulation. Moreover, the LVF advantage was present across all cue conditions, but its magnitude depended on cue validity: The LVF advantage largely decreased with valid cues and further increased with invalid cues (both because of larger changes in the identification of right than left T2) as compared with neutral cues. Similar effects have been reported, although much smaller in size, in standard cueing tasks (Asanowicz, Marzecová, Jaśkowski, & Wolski, 2012; Evert, McGlinchey-Berroth, Verfaillie, & Milberg, 2003). These results confirm a major role of attention in producing the LVF advantage. To detail, with neutral cues where the onset of T2 triggers allocation of attention to its hemifield, attention is directed faster, or more efficiently, to the LVF than to the RVF. This “pull” of attention to the RVF may sometimes be too weak, so T2 will not be identified. With salient spatial cues, attention is drawn to one hemifield before T2 onset; therefore, T2 falls into the focus of attention with valid
cues and outside the focus of attention with invalid cues. Thus, stimulus-driven orienting of attention initiated already by the cues compensates for the weaker pull of attention by RVF stimuli when attention is drawn to the correct stream and further impedes identification of right T2 when attention is drawn to the incorrect stream because then the demands on orienting of attention are even larger.

This assumed relationship between the LVF advantage and orienting of attention may be expressed in terms of the model of attention stated by Corbetta and Shulman (2002; see Shulman & Corbetta, 2012, for a recent revision). The model assumes the existence of separate dorsal and ventral frontoparietal attention networks. The dorsal network alone is involved in endogenous orienting of attention (the orienting to relevant locations before target appearance). During stimulus-driven orienting, which involves detecting a target or other potentially relevant stimuli that occur outside the current focus of attention, the ventral network is activated in addition to the dorsal network (Shulman & Corbetta, 2012). Crucially, the dorsal network is organized bilaterally, whereas the ventral network is strongly lateralized to the RH, with crucial nodes localized in the right TPJ and the right ventral pFC (Shulman et al., 2010; Ticini, de Haan, Klose, Nägele, & Karnath, 2010). Thus, during two-stream RSVP, where there is a constant competition for attention between stimuli from both VFs (Desimone & Duncan, 1995), the information from LVF might be favored in this competition. This is because the RH might have direct or faster access to the information from the LVF, whereas RVF information has yet to be relayed through the corpus callosum to be processed in the right ventral network (cf. the callosal relay model of functional hemispheric lateralization; Zaidel, 1987; Moscovitch, 1986) or RVF information is processed in the LH but less effectively than in the information from LVF in the RH (cf. direct access model; Weems & Reggia, 2004; Zaidel, 1987). Therefore, T2 is more often missed in the RVF than in the LVF.

Problematic for our interpretation of the LVF advantage as reflecting unequal abilities of both hemispheres in stimulus-driven attention is the present effect of some slight but significant LVF advantage in the valid cue condition. This is because, only in this condition, the orienting to T2 was not required, and thus, no stimulus-driven attention was involved that might have prioritized the information in the LVF. This small remaining LVF advantage might result from faster attracting of attention by left than right salient cues (cf. below) or from a ceiling effect reached in the identification of left T2. The identification of left T2 improved from neutral to valid cues by 5.5% only, in contrast to 13% for right T2. Thus, left T2 might actually have had no more room to improve. Whereas the first interpretation is in accordance with right-lateralized stimulus-driven orienting of attention as the mechanism

Figure 7. T2-evoked potentials recorded from Pz, showing the P3 component. A presents P3 evoked in the three cue conditions—neutral cues (gray line), valid cues (black solid line), and invalid cues (black dashed line)—together with maps of activity distribution over the scalp at the positive peaks of these waveforms (150, 450, and 570 msec for neutral, valid, and invalid cues, respectively). Scale of the maps is from 8 μV (white) to −8 μV (black). B presents Pz potentials evoked separately by left and right T2 (averaged across all three cue validity conditions). C presents Pz potentials evoked by left and right T2 separately at the three cue validity conditions.
of the LVF advantage, the latter one is neutral with respect to this interpretation. This might be further investigated by making the task more difficult at baseline (neutral cue condition), such that ceiling effects become less probable.

**ERPs Evoked by the Cue**

Valid and invalid cues, as lateralized stimuli without symmetrical fillers in the other hemisphere, evoked larger P1 and N1 components in the contralateral hemisphere (P1pc and N1pc). These components might be related to perceptual processing of cues. On the other hand, as cues drew attention effectively, N1pc might also be related to attentional processing, similar to N2pc (Schneider, Beste, & Wascher, 2012; Verleger, Zurawska vel Grajewska, & Jaśkowski, 2012; Wascher, Schatz, Kuder, & Verleger, 2001; Valle-Inclán, 1996). Interestingly, this N1pc was evoked earlier in the left hemisphere than in the right hemisphere. This asymmetry might be again related to stimulus-driven orienting of attention realized by the right-lateralized ventral network. In a study using non-informative cues, Natale, Marzi, and Macaluso (2010) have shown that the ventral network responds more strongly to task-irrelevant stimuli that share visual features congruent with the current task set (cf. Folk, Remington, & Johnston, 1992). In our experiment, it may reasonably be argued that the red cues attracted attention only for the reason that color deviance was task-relevant for identifying T1. According to the dimension-weighting account, prior knowledge about targets is dimension specific rather than feature specific (Müller, Reimann, & Krummenacher, 2003), which leads to treating the red color (cue) as deviant similar to the blue one (T1). Moreover, participants were not pressed to selectively attend to blue (T1) deviants and suppress red deviants (cf. Jiao et al., 2013; Eimer, Kiss, Press, & Sauter, 2009) because red cues were helpful not only for valid trials but also in neutral and even invalid trials, as temporal alerting cues (Van der Lubbe, Keuss, & Stoffels, 1996).

In line with the above argument on speed differences between hemispheres, this left–right cue asymmetry in N1pc latency might explain why, in the valid cue condition, there was still an LVF advantage in T2 identification, although no attention orienting toward T2 was required: Left cues attracted attention more quickly to their stream than right cues, and therefore, left T2 was still better identified than right T2.

**ERPs Evoked by T2**

The efficiency of the cue manipulation was also confirmed when T2-evoked ERPs were studied. It was expected that these ERPs would reflect left–right asymmetries of attentional shifts induced by the cues, that is, in ANOVA terms, interactions of Cue Validity × T2 Side. In fact, clearest asymmetries of cue effects were obtained for P3 latencies and for SPCN that followed T2-evoked N2pc. Similar effects were found for N2pc amplitudes but might actually be caused by overlap of preceding cue-evoked asymmetries.

**N2pc**

As predicted, the N2pc component evoked by T2 was modulated by cue validity: Valid cues decreased and invalid cues increased the latency of T2-evoked N2pc as compared with neutral cues. This confirms that T2 was selected faster when attention had already been attracted to its location and was selected slower when attention had to be redirected after invalid cues.

In contrast to latencies, N2pc amplitudes did not differ between the three cue conditions, although smaller N2pc after valid cues and larger N2pc amplitude after invalid cues as compared with neutral cues were expected, because less attention is needed to select T2 after valid cues and more after invalid cues (cf. Sawaki et al., 2012; Jaśkowski, van der Lubbe, Schlotterbeck, & Verleger, 2002). However, as might be inferred from Figure 5B, N2pc had its onset (around 150 msec) at different voltage levels because of the preceding cues: It started at baseline after neutral cues, at the negative side (about −1.8 μV) after valid cues, and at the positive side (about 1 μV) after invalid cues. Relative to these onset levels, the increase in N2pc amplitude was smallest after valid cues and largest after invalid cues. From this point of view, also the predictions concerning N2pc amplitude were confirmed.

Cue validity modulated amplitude differences of N2pc between RH and LH, especially when T2 followed invalid cues: N2pc at the LH was much smaller than N2pc at the RH. This outcome might be surprising at first sight, because T2-evoked N2pc was predicted to increase after invalid cues, as more attention must be allocated to target selection. However, as was argued above, in the Results section, this outcome might have been largely caused by overlap of preceding cue-evoked asymmetries.

Unlike previous studies (Verleger et al., 2009, 2011), N2pc was not evoked earlier by left T2 than by right T2. Possible explanations for this lack of replication of the right–left asymmetry of N2pc latency might be confounds from the cueing procedure and low signal-to-noise ratio in the neutral cue condition. Nevertheless, based on multiple replications of this asymmetry in our laboratory, the current results appear to be an exception.

**SPCN**

Across all cue conditions, from 300 msec after T2 onset onward, N2pc evoked at the LH stayed on a negative level, whereas N2pc evoked at the RH turned positive. This difference was generally apparent until 370 msec after T2 onset and then disappeared for the valid cue condition, mainly because activity in the RH became again negative at about 360 msec. This large sustained negativity in the LH is presumably the SPCN. SPCN has been
observed to follow the N2pc component both when targets had to (Jolicœur, Brisson, & Robitaille, 2008; Dell’Acqua, Sessa, Jolicœur, & Robitaille, 2006; Vogel & Machizawa, 2004) and did not have to be memorized (Lefebvre, Dell’Acqua, Roelfsema, & Jolicœur, 2011; Mazza, Turatto, & Caramazza, 2009; Waschkuhn et al., 1998) and might be interpreted as continued processing of the target. Thus, there might be more sustained negativity at the LH because right T2 needs longer processing. In contrast, N2pc evoked at the RH was followed by some positivity that presumably belongs to the P3 component. This asymmetry of SPCN may reflect not only that attention is more slowly oriented toward RVF than LVF, as reflected by latency differences of cue-evoked N1pc, but also that right-side targets need more time to be fully processed, although processing of left T2 might already pass to the next step in the processing chain.

Interestingly, this asymmetry between hemispheres was modulated by cueing. The asymmetry ended at about 400 msec after T2 onset in the valid cue condition and only at about 450 msec in the two other cue conditions. This might mean that the extended activity needed by the LH for processing right-side T2 was not needed to the same extent when attention had already directed to the T2 stream before T2’s onset. Thus, this asymmetry may directly reflect a deficit of the LH in shifting attention. A possible mechanism for this earlier end of SPCN asymmetry after valid cues is that N2pc was generally evoked earlier after valid cues than after the two other cues and that, thereby, further target processing could already begin earlier and, consequently, would end earlier.

**P3**

As predicted from previous literature on cueing (Hopfinger & Mangun, 1998), T2-evoked P3 was larger after valid cues than after neutral and invalid cues. Confirming previous evidence (Verleger et al., 2011) and in accordance with accounts of P3 amplitude in terms of perceptual distinctiveness (Craiston et al., 2009; Sergent et al., 2005), P3 was larger when T2 was left than when T2 was right. Thus, left T2 might be processed as more relevant or might have larger perceptual distinctiveness resulting from the attentional bias to the LVF. Importantly, the P3 component peaked earlier in the condition with left cue–right T2 than in any other condition, which resulted from two factors. First, the onset of P3 was earlier with lateral cues than with neutral cues. Second, P3 started to return to baseline earlier in the left cue–right T2 condition than in other conditions, thereby reaching its maximum quickly after P3 onset. It appears plausible to relate these short latencies to the low identification rate of T2 in this condition. To detail, this quick return to baseline of P3 evoked by invalidly cued right T2 might be related to the fact that the process of target identification was impeded because of low perceptual availability of the target.

**Conclusion**

In summary, through modulating the LVF advantage by cue validity and thereby engaging stimulus-driven orienting of attention, the LVF advantage in identifying targets in RSVP was shown to be related to an LVF advantage in the bottom–up organization of attention. Probably, this LVF advantage in shifting attention toward salient stimuli is related to the lateralization of the attentional system, as described in Shulman and Corbetta’s (2012) model. This interpretation is further supported by the present ERP results. Faster shifting of attention to the LVF was apparent for the salient task-irrelevant cues, as reflected in cue-evoked N1pc latencies. Therefore, targets in the LVF were perceived easier, indicated by larger P3 amplitudes after left T2, which resulted in better target identification. In contrast, because of attention shifting slower to the RVF, right T2s needed more processing (SPCN after right T2), and their identification was particularly impeded after invalid cues because of low perceptual distinctiveness (smaller P3 amplitudes after right T2 and shorter P3 latencies after invalidly cued right T2). As these processes sometimes failed, right T2 was often missed and incorrectly reported.

**Acknowledgments**

This work was supported by Grant Ve110/15-1/2 awarded from the Deutsche Forschungsgemeinschaft to R. V. as part of the network PAK270 “Neuro-cognitive mechanisms of conscious and unconscious visual perception” and by Grant 2012/05/D/HS6/03563 awarded from the National Science Centre of Poland to D. A.

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