Elucidating the Neural Mechanisms Underlying the Contingent Attention Cueing Paradigm

by

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Abstract

Researchers have studied contingent attentional capture for over two decades, and have characterized the behavioural effects; but a complete understanding of the neural mechanisms involved has yet to be developed. This thesis investigated the neural underpinnings of the cue-validity effect in the contingent capture paradigm. Recent research purported to show that observers inadvertently attend to irrelevant cue items that possess a task relevant feature (indexed by the ERP component, the N2pc), and then suppress the location of that cue item in order to respond to the target (indicated by the ERP component the P_D, believed to index suppression). Experiment 1 determined whether the attended cue was in actuality suppressed; whereas, Experiment 2 determined how selection of the cue item affects higher stages of visual processing. Results showed that reaction time costs were due to extraneously cued nontarget information entering working memory, thus delaying target processing on invalid trials.

Keywords: Contingent Attention Capture; Event-Related Potentials (ERPs); N2pc; Contralateral Positivity; SPCN; Attentional Competition

Dedication

I would like to dedicate my thesis to my little man: 2-Bite Brownie Livingstone-Christie.

Whenever I needed a good cuddle, you were there.

I love you little guy!!

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First and foremost, I would like to thank my supervisor, Dr. John McDonald. Thank you for accepting me into your lab, your guidance, ideas and discussions that made this such a rewarding project. Also, thank you for continually pushing me to be a better writer. Sometimes the amount of red was distressing, but the finished product was always worth the effort.

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To the RA's: Alannah, Amanda, Meagan, Daniel, Sam, and Ellen. Thank you for your help collecting all the data for the seemingly endless string of experiments I could think up. I would still be collecting data without you.

To Mom and Jim: you are the best cheerleaders I could ever ask for. Words will never do justice how appreciative I am of you. I love you, and thank you for putting up with me.

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List of Acronyms

Ag/AgCl	Silver/Silver Chloride
CP	Contralteral Positivity
EEG	Electroencephalography
ERP	Event Related Potential
HEOG	Horizontal Electrooculogram
N2pc	Posterior contralateral negativity in the N2 time range
P_D	Distractor Positivity
RT	Reaction Time
SOA	Stimulus Onset Asynchrony
SPCN	Sustained Posterior Contralteral Negativity

Chapter 1. Introduction

An observer's visual system receives a continuous stream of information from the environment. The processing of incoming visual information can be biased towards certain stimuli or locations so that potentially relevant objects can be consciously perceived, acted upon, and remembered. The neural mechanisms that bias processing of sensory information are generally referred to as selective attention mechanisms. These attentional mechanisms can be influenced by several factors, including an observer's goals (top-down control; Egeth & Yantis, 1997) and the properties of the stimulus themselves (bottom-up control; Theeuwes, 1991).

Researchers have employed the cue-target paradigm to investigate top-down and bottom-up control of attention (e.g. Posner, 1980). In the general paradigm, a cue display is used to orient attention to a particular location of the visual field shortly before the appearance of a task-relevant target stimulus or display. Typically, observers are required to detect the target or discriminate one of its features, and to make an appropriate manual response as quickly and as accurately as possible. Researchers employ peripheral cues (i.e. abrupt visual onsets appearing at a potential target location) or a centrally presented symbolic cue to investigate the effects of involuntary (i.e. reflexive) and voluntary shifts of attention, respectively (e.g. Jonides, 1981; McDonald & Ward, 1999; Störmer, McDonald, & Hillyard, 2009; Theeuwes, 1991; Woodman & Luck, 1999; Wright & Ward, 1994; Yantis & Jonides, 1990).

The contingent capture cueing paradigm was developed to determine the precise interplay between bottom-up and top-down control processes mediating shifts of attention to peripheral cues (Folk & Remington, 2006 & 2008; Folk, Remington, & Johnston, 1992; Folk, Remington, & Wright, 1994). In this paradigm, the target appears in a multi-item search display and is defined by a unique feature. Participants must establish an 'attentional set' for that specific feature in order to find the target. Figure 1.1 depicts a

sequence of events on a trial of a contingent-capture cueing task with a colour-defined target (i.e. the red item). In this example, observers might be required to discriminate the shape of the target as quickly as possible (circle vs. square). On each trial, an irrelevant cue display precedes the search display. For the majority of trials, one item within the cue display appears with the same target-defining feature (termed the *match cue*). Typically, observers are faster to respond to targets appearing at the location of a previous match cue (valid trials) than to targets appearing elsewhere (invalid trials). No such validity effect is evident when none of the cue items possess the target-defining feature, even when one of the cue items is highly salient (e.g. an abrupt onset or singleton). This pattern of results indicates that the ability of an irrelevant visual item to capture attention reflexively is contingent upon a match between that item's features and the observer's 'attentional set'.

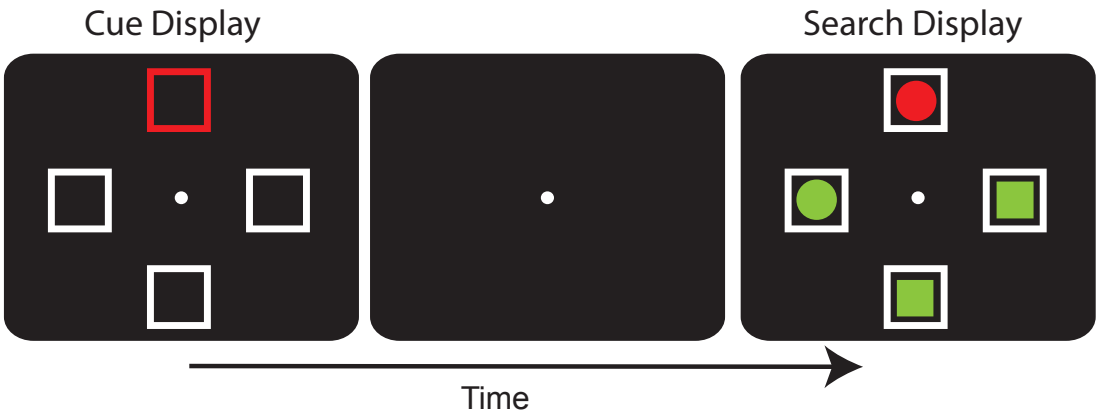

Figure 1.1. Prototypical display used in the contingent attentional capture cueing paradigm where colour (i.e. red) is the target-defining feature.

Recent research has turned to electrophysiological measures to provide converging evidence for the contingent capture of attention. Specifically, the event-related potential (ERP) technique has been used to assess whether selection of a cue item is contingent on a match between its features and the attentional set. Most ERP studies of contingent capture have focused on the N2pc component, which has been associated with attentional selection in visual search tasks (Hickey, Di Lollo, & McDonald, 2009; Luck & Hillyard, 1994; Woodman & Luck, 2003). The N2pc component is a negative ERP difference beginning ~175 ms post stimulus at electrode sites contralateral to a target relative to electrode sites ipsilateral to a target (Hickey, Di Lollo, & McDonald, 2009; Luck

& Hillyard, 1994). Several ERP studies have found that match cues elicit the N2pc, whereas cues that do not match the observer's attentional set do not (Eimer & Kiss, 2008, 2010; Eimer, Kiss, Press, & Sauter, 2009; LeBlanc, Prime, & Jolicœur, 2007; Lien, Ruthruff, Goodin, & Remington, 2008). This pattern of results is consistent with the contingent attention capture hypothesis (Folk et al, 1992 & 1994; Folk & Remington, 2006 & 2008) but not with the perspective that attention capture is completely automatic (Theeuwes, 1994).

Despite converging behavioural and ERP evidence for contingent attention capture, it is still unclear how these spatial shifts of attention manifest in the reaction time (RT) effects routinely reported. One view that is consistent with most models of visual selection is that reflexive orienting of attention to the cued location enhances early perceptual processing of subsequent items appearing nearby (e.g. Hopfinger & West, 2005). In the context of the contingent capture paradigm, it would follow that perceptual processing of the target would be enhanced on valid trials relative to invalid trials, thereby biasing selection in favour of the task-relevant search item. Although this view is widely accepted, results of a recent ERP study provided evidence for suppression at the location of a match cue (Sawaki & Luck, 2013). Specifically, a contralateral positivity (CP) that was assumed to reflect suppression (i.e. a distractor positivity, or P_D; Gaspar, Christie, Prime, Jolicœur, & McDonald, in press; Gaspar & McDonald, 2014; Hickey et al., 2009; Sawaki, Geng, & Luck, 2012) was observed after the cue-elicited N2pc. To account for the facilitatory cueing effect (shorter RTs on valid trials), Sawaki and Luck concluded that the cued location was not fully suppressed.

Two timing-centered considerations call into question the conclusion that processing the match-cue location was suppressed in Sawaki and Luck's (2013) study. First, the CP started 400 ms after the onset of the cue display, whereas the P_D typically appears 180 – 250 ms after the appearance of a search display (Gaspar et al., in press; Gaspar & McDonald, 2014; Hickey et al., 2009; Sawaki et al., 2012). Second, the putative P_D onset was actually about 100 ms after the appearance of the subsequent search display. Sawaki and Luck argued that the CP was too early to reflect cortical activity triggered by the target, but because the SOA was fixed, in their study, it is currently not

possible to determine whether the CP was time-locked to the cue display or to the search display.

Consistent with the possibility that the CP was triggered by the search display, several prior ERP studies have demonstrated that orienting attention to the left or right in advance of a bilateral visual target array can modulate the amplitude of the target-elicited P1 component. Specifically, across a variety of paradigms, the target-elicited ERP waveform is more positive contralateral to an attended side than ipsilateral to an attended side in the time range of the P1 (and subsequent N1; Fukuda & Vogel, 2009; Heinze, Luck, Mangun, & Hillyard, 1990; Luck, Heinze, Mangun, & Hillyard, 1990; McDonald, Teder-Sälejärvi, Russo, & Hillyard, 2005; Störmer, McDonald, Hillyard, 2009). Such contralateral positivities have been observed when attention is sustained at a location for several trials (Heinze et al., 1990; Luck et al., 1990), symbolically cued to a location on a trial-by-trial basis (e.g. Mangun & Hillyard, 1991; Sawaki et al., 2012), cued reflexively by peripheral visual transients (Hopfinger & Mangun, 1998), or cued reflexively by sudden peripheral sounds (McDonald et al., 2005; Störmer et al., 2009). These target-elicited CP components have been hypothesized to reflect signal enhancement at early stages of perceptual processing. Thus, it is possible that the CP reported by Sawaki and Luck reflected enhancement of the cued search items rather than suppression of the match cue.

The research described in this thesis was conducted to address two outstanding questions about the contingent capture cueing paradigm. Experiment 1 was designed to test the nature of the contralateral positivity reported by Sawaki and Luck (2013) by varying the cue-target stimulus onset asynchrony (SOA). If the CP reflects cue suppression, it would be time-locked to the onset of the cue display regardless of when the target appeared. Alternatively, if the CP reflects enhancement of the cued search display item, it would be time-locked to the onset of the search display rather than the cue display. To foreshadow the results, the CP was found to be time-locked to the search array. Experiment 2 was designed to track processing of a cued nontarget search item beyond the CP. The main objective of Experiment 2 was to determine whether the cued nontarget was selectively processed at the stage of visual processing indexed by the N2pc.

Surprisingly, the cued nontarget was found to trigger not only the N2pc but a subsequent contralateral ERP negativity associated with item identification and active representation in working memory.

Chapter 2. Experiment 1

2.1. Methods

2.1.1. Ethics

The Office of Research Ethics at Simon Fraser University approved all experimental procedures.

2.1.2. Participants

Forty-nine neurologically healthy observers participated in Experiment 1 after giving informed consent. Data from four participants were excluded from the analysis because their ocular artifacts exceeded our standard laboratory limits (>25% of the trials contaminated by ocular artifacts or averaged horizontal electrooculogram (HEOG) deflections > 3.2 μV; see below). In the end, data from 44 participants (22 in condition 1; 22 in condition 2; 17 men; mean age = 19.5 years; three left-handed) were analyzed. Participants reported normal or corrected-to-normal vision and were screened for colour blindness using the Ishihara colour plates.

2.1.3. Stimuli and procedure

All stimuli were presented on a black background (u' = .280, v' = .360). Participants viewed sequences of two four-item displays. The first display (cue display) contained four filled circles (1.4° radius), whereas the second display (target display) consisted of four unfilled squares (1.4° x 1.4°) that each had one missing side. Stimuli on both displays appeared 1.6° above and below the horizontal meridian and 2.7° to the left and right of the vertical meridian (Figure 1). At the start of each trial, participants were presented with one of two cue displays: a match-cue display or a neutral-cue display. Each item in the match cue and search displays possessed a unique colour: red (u' = .655, v' = .320), green (u' = .313, v' = .633), blue (u' = .142, v' = .045), or yellow (u' = .425, v' = .536); whereas, only white circles (u' = .303, v' = .325) were presented for neutral cue displays.

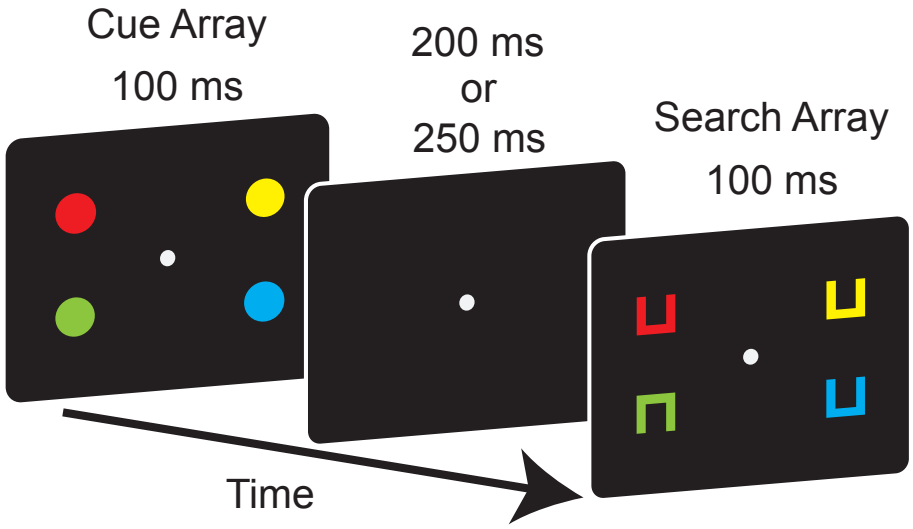

Figure 2.1. Stimulus displays and example trial sequences for Experiment 1.

Trials began with the presentation of a fixation screen for 900 – 1200 ms. The cue display was then presented for 100 ms and was followed by a fixation screen for 200 ms (group 1) or 250 ms (group 2). Following this fixation interstimulus interval (ISI), the search display was presented for 100 ms. The cue-target SOA was therefore 300 ms in condition 1 and 350 ms in condition 2. Participants were instructed to keep their eyes fixated on a central fixation point throughout each trial. The task was to indicate whether the top or the bottom side was missing from a target square. Participants were informed of the target's color at the beginning of each block, and the target color was varied randomly across the blocks. Two-alternative forced choice responses were made within a 2100 ms response window using the left and right buttons of a computer mouse (operated with the right hand). Following one practice block, participants completed 32 blocks of 40 trials for a total of 1280 experimental trials.

Cue displays and search displays were presented in five equally probable sequences (trial types): valid trials (match cue and target at the same location), vertical-invalid (match cue and target at different locations on same side of fixation), horizontal invalid (match cue and target at different locations directly across the vertical meridian), diagonal invalid (match cue and target diagonally opposite locations), and neutral.

2.1.4. Apparatus

Participants sat approximately 57cm from the display monitor in a sound attenuated and electrically shielded booth under LED lighting. A 24-inch LCD monitor with a screen resolution of 1980 x 1080 pixels was used for stimulus presentation. Stimulus presentation and recording of participants' responses was controlled by Presentation (Neurobehavioral Systems Inc, Albany, CA) from a Windows-based computer. Custom software (Acquire) responsible for EEG acquisition was run from a second Windows-based computer. A 64-channel, 12-bit A-to-D board (PCI 6071e, National Instruments, Austin, TX) housed in the acquisition computer was connected to an EEG amplifier system with high electrode impedance (SA Instruments, San Diego, CA). Sintered Ag/AgCl electrodes mounted in an elastic cap (Electrode Arrays, El Paso, TX) were used to record EEG.

2.1.5. Electrophysiological recording

EEG was recorded from 25 sintered Ag/AgCl electrodes placed at FP1, FPz, FP2, F7, F3, Fz, F4, F8, T7, C3, Cz, C4, T8, P7, P3, Pz, P4, P8, PO7, POz, PO8, O1, Oz, O2, & M1. All EEG signals were referenced to the right mastoid during recording of the EEG. Bipolar electrodes positioned lateral to the external canthi of each eye monitored horizontal eye movements (HEOG). Electrode impedances were kept below 10 kOhms. All signals were recorded with a bandpass filter of 0.01 – 100Hz and a gain of 20,000 using SA Instrument amplifiers and custom Windows software (Acquire). All EEG signals were digitized at 500 Hz.

2.1.6. Data analysis

Artifact rejection and ERP averaging were conducted using ERPLAB (Lopez-Calderon & Luck, 2014). The EEG and HEOG were segmented into 1.25-s epochs, starting 250ms before cue display onset. Epochs containing incorrect responses or ocular artifacts (saccades or blinks) were excluded from further analyses. Saccades and blinks were detected in the HEOG and Fp2 channel, respectively, and were defined as sudden changes in voltage (step functions), over the course of 50 ms, greater than 16 microvolts for a saccade and 35 microvolts for a blink. If any artifact was detected, the epoch was

rejected from subsequent averaging and analysis. We replaced any participants for whom the residual HEOG activity was more than 3.2 μV, meaning that the residual eye movements in the remaining participants was less than 0.2^{o} (Lins, Picton, Berg, & Scherg, 1993; McDonald & Ward, 1999).

EEG and HEOG signals were digitally low-pass filtered with a half-power cutoff at 30 Hz to remove high frequency noise. EEG signals were then digitally re-referenced to the average of the left and right mastoids. ERPs time-locked to the presentation of the cue display and referenced to the cue location were computed from artifact-free trials on which participants correctly identified the target. For each participant, the ERP waveforms were collapsed across left and right visual hemifields and left and right electrode sites to create waveforms recorded contralateral and ipsilateral to the match-cue item. Lateralized ERP difference waveforms were then derived for each trial type by subtracting the ipsilateral waveform from the corresponding contralateral waveform using lateral occipital electrode sites (PO7 and PO8). Negative voltages were plotted upward, such that cue- and target-elicited N2pc components would appear as upward deflections and any contralateral positivities would appear as downward deflections.

The mean amplitudes of the lateralized ERP components of interest were measured at lateral occipital electrodes (PO7/PO8) within time windows selected *a priori* on the basis of prior studies and/or specific hypotheses. All measurements were based on the contralateral-ipsilateral difference waves. The cue-elicited N2pc was measured 175–225 ms post-cue, whereas the target-elicited N2pc was measured 200–300 ms post-target (cf. Sawaki & Luck, 2013). The CP was measured in two time intervals, one that matched Sawaki and Luck's (2013) P_D measurement window (400–450 ms post-cue; 100–150 ms post-target) and one that was delayed by 50 ms to match the difference in SOAs across the two conditions (450–500 ms post-cue; 100–150 ms post-target).

2.2. Results

2.2.1. Behavioural

Median RT data for all trial types is presented in Table 2.1. For both SOA conditions, RTs were shortest on valid trials, intermediate on neutral trials and longest on invalid trials. RTs were subjected to a mixed-model ANOVA with Trial Type as the within subject factor and SOA condition as the between subject factor. The ANOVA revealed a significant main effect of Trial Type: $F_{(4,168)} = 87.1$, $p < .001$. The Trial Type x SOA interaction was not significant ($F_{(1,168)} = 0.23$, $p > .05$), indicating that the pattern of RTs across trial types was similar between SOAs. Planned pairwise comparisons determined that valid trials were significantly shorter than all other trial types for each SOA ($ps < .01$, Bonferroni corrected). Finally, there was no main effect of SOA on RT ($F_{(1,43)} = 4.0$, $p > .05$). RTs follow the cost/benefit pattern reported by Sawaki and Luck (2013), and previous studies using the contingent capture cueing paradigm (e.g. Eimer & Kiss, 2008, 2010; Eimer et al., 2009).

Table 2.1. Experiment 1 median reaction time (RT) and standard error (SE) for each trial type by SOA.

	SOA	
Trial Type	300 ms	350 ms
Valid	551 (13.0)	591 (13.4)
Vertical Invalid	600 (11.2)	640 (14.3)
Horizontal Invalid	598 (11.1)	636 (12.9)
Diagonal Invalid	610 (11.4)	644 (14.0)
Neutral	585 (11.4)	625 (13.8)

2.2.2. Electrophysiology

For this experiment, ERP analyses focused on valid and vertical-invalid trials exclusively. ERPs from horizontal-invalid and diagonal-invalid trials were not analyzed because the components of interest, the CP and target N2pc, are the same polarity and thus are inseparable whenever the match cue and target are on opposite sides of the vertical meridian (cf. Sawaki & Luck, 2013).

The first goal of Experiment 1 was to replicate Sawaki and Luck's 2013 findings using a 300 ms SOA. In the left column of Figure 2.2, the ERP (panels A-C) and difference waveforms (panel D) are depicted for the 300 ms SOA condition. On Valid and vertical-invalid trials, a small match-cue elicited N2pc was evident in the ERP waveforms beginning approximately 150 ms post cue display. A measurement window of 175 – 225 ms (identical to the window used by Sawaki and Luck, 2013) was used to assess mean amplitude of the match-cue N2pc. Valid and vertical-invalid match-cue N2pcs were found to be significantly different from zero (both $ts_{(21)} > 2.80$, $ps < .01$). A contralateral positivity (CP) was observed for both the valid and vertical-invalid trials beginning at approximately 400 ms. Again, using a measurement window identical to Sawaki and Luck's (i.e. 100 – 150 ms post search display), the mean amplitude of the CP was found to be significantly different from zero (both $ts_{(21)} > 3.13$, $ps < .01$). The target-elicited N2pc began ~180 ms after the presentation of the search display. Measured 200 – 300 ms post search display onset, the target N2pc was found to significantly differ from zero (both $ts_{(21)} > 4.68$, $ps < .001$). The neutral cue elicited neither an N2pc nor the subsequent CP (both $ts_{(21)} < .26$, $ps > .05$); however, a target-elicited N2pc was observed beginning approximately 150 ms post search display onset ($t_{(21)} = 5.07$, $p < .001$). This pattern of lateralized activity closely replicated Sawaki and Luck's (2013) ERP findings.

As can be seen in the right column of Figure 2.2, similar lateralized deflections were found in the 350-ms SOA condition. As in the 300-ms SOA condition, small match-cue-elicited N2pc deflections were evident in the difference waveforms beginning approximately 150 ms post cue display. These N2pc deflections were significantly different from zero for both valid trials and vertical-invalid trials (both $ts_{(21)} > 3.36$ $ps < .01$) but not for neutral trials ($t_{(21)} = 1.20$, $p > .05$). Paralleling these N2pc results, a CP was observed on both valid and vertical-invalid trials and was significantly different from zero 100 – 150 ms post search display (both $ts_{(21)} > 4.62$, $ps < .001$). The target-elicited N2pc began ~180 ms after the presentation of the search display and was significantly different from zero in the 200–300 ms measurement window (both $ts_{(21)} > 3.91$, $ps < .01$). On neutral trials, the match-cue N2pc and CP were absent (both $ts_{(21)} < 2.01$, $p > .05$), but a target N2pc was present as expected ($t_{(21)} = 4.98$, $p < .001$).

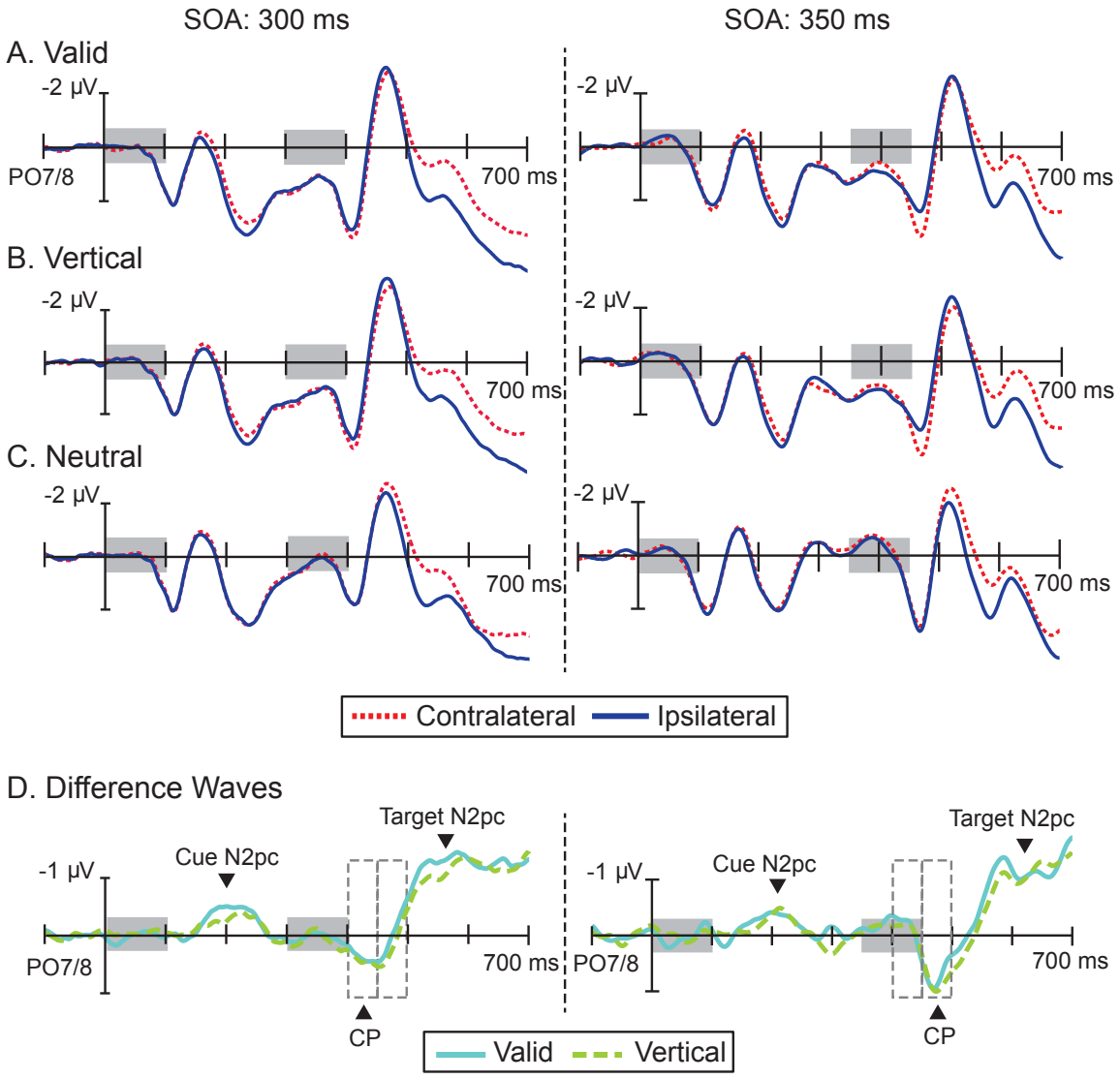

Figure 2.2. **Grand-averaged ERP waveforms time locked to cue arrays in Experiment 1. Waveforms are plotted contralateral or ipsilateral to the target, which appeared in the same visual hemifield as the match cue on valid and vertical-invalid trials. The left-most and right-most grey boxes indicate the timing of the cue array and search array, respectively. (A) Valid trials. (B) Vertical-invalid trials. (C) Neutral trials. (D) Contralateral-minus-ipsilateral difference waves for valid trials and vertical-invalid trials.**

Two converging methods were employed to assess the timing of the CP. First, the mean amplitude of the difference waveforms for each SOA, collapsed across valid and vertical-invalid trials, was measured in two consecutive time windows: 400 – 450 ms and

450 – 50 ms. The mean amplitudes were submitted to a mixed-model ANOVA, with Time Window as the within subject factor and SOA as the between subject factor. A significant Time Window x SOA interaction was present ($F_{(1,42)}$ = 27.0, p < .001), indicating that different patterns of amplitude effects were observed in the two SOA conditions. As displayed in Figure 2.3, the CP was larger in the early measurement window than in the late window for the 300-ms SOA condition, while the CP was larger in the late window than in the early window for the 350-ms SOA condition. A significant main effect of Time Window was found ($F_{(1,42)}$ = 5.16, p < .05), but no significant main effect of SOA was found ($F_{(1,42)}$ = .33, p > .05). Second, the onset latency of the CP was compared across the two SOAs. The onset latency was operationally defined as the 50% fractional peak latency of the positive deflection (in the contralateral-ipsilateral difference wave); this was measured for valid and vertical-invalid trials, separately for each SOA condition. Following convention, jackknife sub-averages were computed in order to extract the 50% fractional peak latency (Miller, Patterson, & Ulrich, 1998). From these sub-averages, estimates of individual-subject latencies were computed (Smulders, 2010) and entered into a mixed-model ANOVA with Trial type (valid & vertical-invalid) as a within subject factor, and SOA as a between subject factor. Critically, the CP was found to onset 49 ms earlier in the 300-ms SOA condition than in the 350-ms SOA condition, resulting in a significant main effect for SOA ($F_{(1,42)}$ = 7.3, p < .01). Specified in relation to the onset of the cue display, the CP onset at 402 ms and at 451 ms for the 300-ms and 350-ms conditions, respectively. Specified in relation to the onset of the search display, the CP onset at 102 ms and 101 ms in the two conditions. In other words, the CP was time-locked to the search array rather than the cue display. Neither the Trial Type main effect nor the Trial Type x SOA interaction were found to be significant ($F_{(1,42)}$ = .01, p > .05; $F_{(1,42)}$ = .14, p > .05).

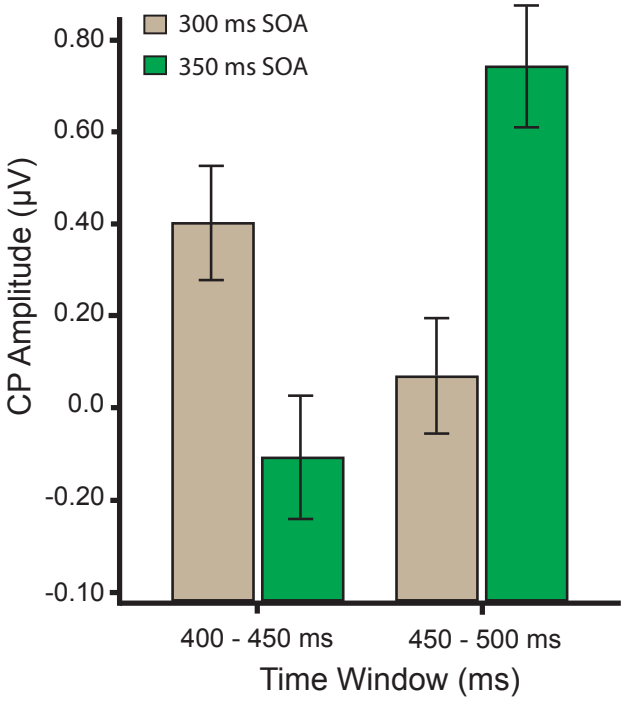

Figure 2.3. Mean amplitude of the contralateral positivity (CP) as a function of stimulus onset asynchrony (SOA) and measurement window.

2.3. Experiment 1 Discussion

The behavioural and electrophysiological results from Experiment 1 are broadly consistent with past research. As expected, RTs were shorter on valid trials than on the various invalid trial types. Consistent with several ERP studies of contingent capture, the match cue elicited an N2pc, thereby confirming that observers selectively attended to the cue item that shared the target's colour. Finally, following presentation of the search display, a robust target N2pc was measured, indicating that observers selectively attended to the target search item.

The main objective of Experiment 1 was to determine whether the CP was time-locked to the cue display or to the search display. The results were clear-cut: the CP was found to be time-locked not to the cue display but to the subsequent search array. Consequently, it is highly unlikely that the CP is an index of cue suppression, as proposed by Sawaki and Luck (2013). Given the similarities between the CP observed here and those previously attributed to attention-induced enhancement of target visual processing

(Fukuda & Vogel, 2009; Luck et al., 1990; McDonald et al., 2005; Störmer et al., 2009), it is concluded that the CP reflects signal enhancement at the location of the (attended) match cue. This signal-enhancement account offers a straight forward explanation for the usual cue-validity effect on behavioural performance: when the match cue and target appear at the same location (valid trials), processing of the task-relevant search item is boosted, thereby speeding responses. By contrast, when the match cue and target appear at different locations (invalid trials), selection of the match cue leads to a boost of one of the non-target search items. Presumably, boosting the perceptual processing of a nontarget either delays search for the target or otherwise slows target identification once that item is located. The purpose of Experiment 2 was to elucidate the neural mechanisms involved in processing the cued nontarget on invalid trials.

Chapter 3. Experiment 2

In Experiment 1 it was difficult to track processing of the cued nontarget beyond the CP because of overlapping lateralized ERP components associated with the target. As discussed in chapter 1, the target was found to elicit a large N2pc starting ~200 ms after the appearance of the search display. The target and cued nontarget always appeared at lateral locations (either on the same side or opposite side); therefore, it is not possible to confidently ascribe any part of the observed ERPs to one stimulus or the other (although, as the name suggests, the target N2pc was assumed to be primarily associated with the target search item). Because the target N2pc is computed by comparing contralateral and ipsilateral ERP waveforms to targets in a lateral visual field, no target N2pc will be elicited when the target appears on the vertical meridian (Eimer & Grubert, 2014; Eimer, Kiss, & Nicholas, 2011; Hickey et al., 2009; Hickey, McDonald, & Theeuwes, 2006; Woodman & Luck, 1999). On these midline-target trials, lateralized activity observed after the CP will reflect processing of the cued non-target item.

In Experiment 2, lateralized ERP activity associated with the cued-nontarget (and the target on lateral-target display configurations) was tracked to determine what happens after a match cue inadvertently enhances the early perceptual processing of an irrelevant search item. Two hypotheses were considered. First, the cued nontarget may be actively supressed to prevent that item from being attended. If the cued nontarget was actively suppressed, a P_D should be found contralateral to that item in the conventional P_D time range (250 – 350 ms; e.g. Gaspar & McDonald, 2014; Gaspar et al., in press; Hickey et al., 2009). Second, top-down control might not prevent the cued nontarget item from "capturing" attention. In this case, the cued nontarget should elicit an N2pc rather than a P_D.

3.1. Methods

3.1.1. Participants

Thirty-five new observers participated after providing informed consent. Data from six participants were excluded using the same criteria as in Experiment 1, thereby leaving 29 participants in the final sample (eight men, mean age = 19.5, two left-handed).

3.1.2. Apparatus

The apparatus was identical to Experiment 1.

3.1.3. Stimuli and Procedure

Stimuli were similar to those in used in Experiment 1, with the exception that the cue and target displays contained two additional items four degrees above and below fixation (six items in total; see Figure 3.1). The colours of the six items were now sampled without replacement from a set of six colours, including orange (u' = .514, v' = .383), purple (u' = .306, v' = .132), and the four colours from Experiment 1. The SOA between cue display and search display was 350 ms.

Participants completed 24 blocks of 48 trials, for a total of 1152 trials. A sixth trial type was added to the original five from Experiment 1. Specifically, trials with a lateral match cue and target above or below fixation were designated as midline-target trials. Each trial type was presented with equal probability (16.7%).

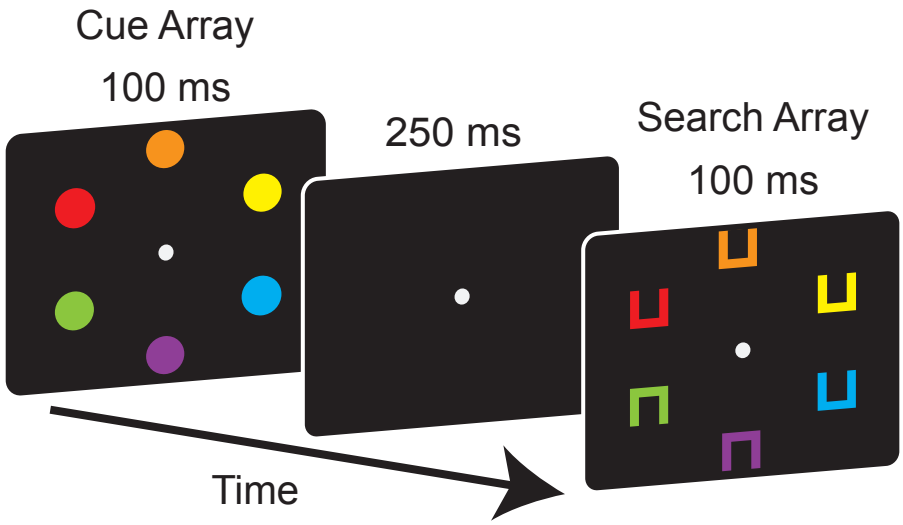

Figure 3.1. Stimulus displays and example trial sequences for Experiment 2.

3.1.4. Electrophysiological Recording and Analysis

EEG recordings and ERP grand averaging procedures were identical to Experiment 1. ERPs in Experiment 2 were time-locked to the presentation of the search display. A symmetric mapping method in Event Related Potential Software System (University of California, San Diego, CA) was used to assess the topography of the lateralized ERP activities of interest. That is, the contralateral-ipsilateral voltage differences measured at symmetric left and right electrodes (e.g., PO7/8) were assigned to each electrode in a given electrode pairing, while voltages measured at midline electrodes were set to zero.

3.2. Results

3.2.1. Behaviour

Behavioural results for Experiment 2 were similar to that reported in Experiment 1. That is, RTs were shorter on valid trials than on all other trial types (see Table 3.1). A one-way ANOVA confirmed a significant main effect of Trial Type, $F_{(5,140)} = 28.8$, $p < .001$. Bonferroni pairwise comparisons indicated that RTs for valid trials were shorter than all other trial types (all *ps* < .05), and RTs on midline-target trials were longer than RTs on all

other trials types (all *ps* < .05). The longer RT on midline-target trials is likely related to the fact that the match cue never appeared on the midline and thus never primed observers for a midline target.

Table 3.1. Experiment 2 median RT and standard error (SE) for each trial type.

Trial Type	RT
Valid	596 (12.0)
Vertical Invalid	645 (12.5)
Horizontal Invalid	638 (12.4)
Diagonal Invalid	652 (12.3)
Neutral	664 (15.0)
Midline Target	686 (10.4)

3.2.2. Electrophysiology

Figure 3.2 presents ERPs (A-C) and difference (D) waveforms, time-locked to the search display, for valid, vertical-invalid and midline-target trials. As in Experiment 1, the search array was found to trigger an early CP and a subsequent N2pc contralateral to the cued/target side on valid and vertical-invalid trials. In addition, a sustained posterior contralateral negativity (SPCN) was observed following the target N2pc. This latter component has been linked to stimulus identification and is hypothesized to be an index of the active maintenance of information in working memory (Jolicoeur, Brisson, & Robataille, 2008). The ERP waveform for the new midline-target trials also show the CP contralateral to the cued side of the search display. Critically, the ERP to midline-target displays revealed a lateralized selection negativity following the CP. Because the midline target cannot elicit lateralized activity, these lateralized components are hypothesized to index attentional processing of the cued nontarget.

To understand the mechanisms involved with processing the cued-nontarget item, the CP on midline-target trials was compared to the CP on valid and vertical-invalid trials. Upon visual inspection, the CP in the midline-target waveform appears to have a similar onset and mean amplitude to the CP in the valid and vertical-invalid waveform. The mean amplitude of the CP, measured 100 – 150 ms, was significantly different from zero for all three trial types (all $ts_{(28)} > 5.5$, *p*s < .001) and not significantly different from each other

($F_{(2,56)}$ = 0.9, p > .05). Across the three trial types, the onset of the CP did not differ; however, the *offset latency* of the CP (as measured by the 50% fractional peak amplitude following the peak) was found to be significantly later on midline-target trials ($F_{(2,56)}$ = 40.1, p < .001), suggesting that the initial enhancement of the cued-nontarget continues into the N2pc time range.

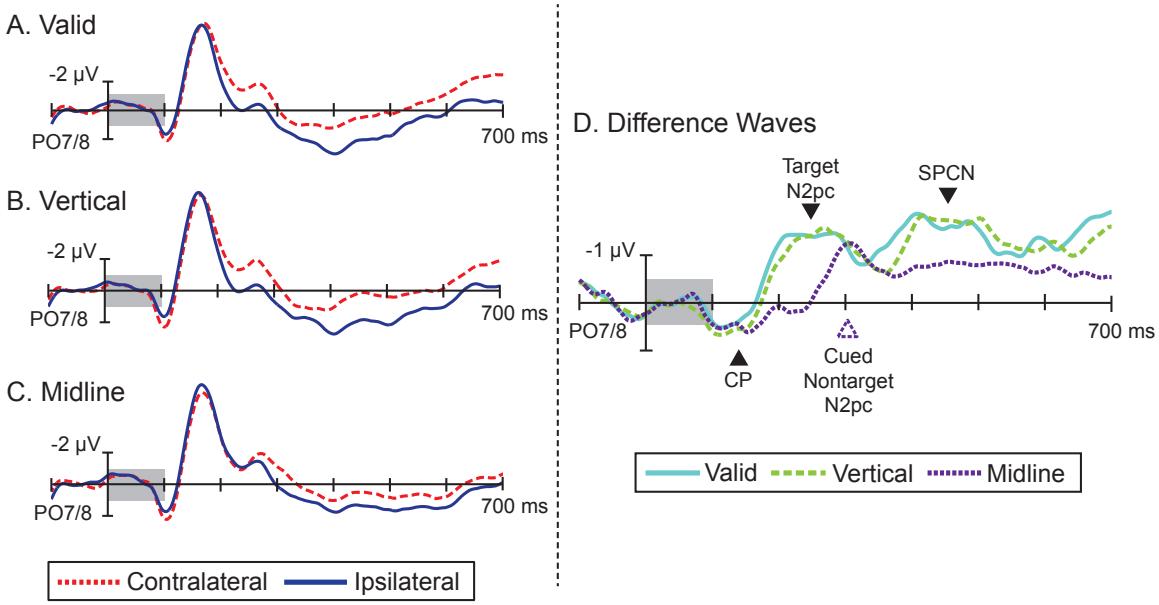

Figure 3.2. **Grand-averaged ERP waveforms from Experiment 2 time locked to search arrays in Experiment 2. Waveforms are plotted contralateral or ipsilateral to the match cue, which appeared in the same visual hemifield as the match cue on valid and vertical-invalid trials. The grey box indicates the timing of the search array. (A) Valid trials. (B) Vertical-invalid trials. (C) Midline-target trials. (D) Contralateral-minus-ipsilateral difference waves for valid trials, vertical-invalid trials and midline-target trials.**

The next comparison was made between the target N2pc on valid and vertical-invalid trials and the somewhat later contralateral negativity on midline-target trials. On valid and vertical-invalid trials, a large target N2pc was observed in the expected time window of 180 – 300 ms. Using the same time window from Experiment 1 (200 – 300 ms), the mean amplitude was extracted and compared against zero. Both valid and vertical-invalid target N2pcs were found to be significantly different from zero (both $ts_{(28)}$ > 5.0, ps < .001). On midline target trials, a negativity contralateral to the cued-nontarget item was evident between 250 – 350 ms after the onset of the search display. Topographical maps of the valid and midline-target difference waves were plotted to determine if the late

selection negativity had the same scalp distribution as the target N2pc (Figure 3.3). Apart from the 50ms delay, the cascade of voltage distributions was nearly identical between the two trial types. This was taken as evidence that the late selection negativity on midline-target trials was an N2pc to the cued nontarget. Therefore, on midline-target trials, the cued-nontarget N2pc was quantified as the mean amplitude of the contralateral-minus-ipsilateral difference wave in the 250–350 ms time window. The cued-nontarget N2pc was found to be significantly different from zero ($t_{(28)}$ = 4.3, p < .001), with a 50% fractional peak latency of 277 ms (SE = 5.1).

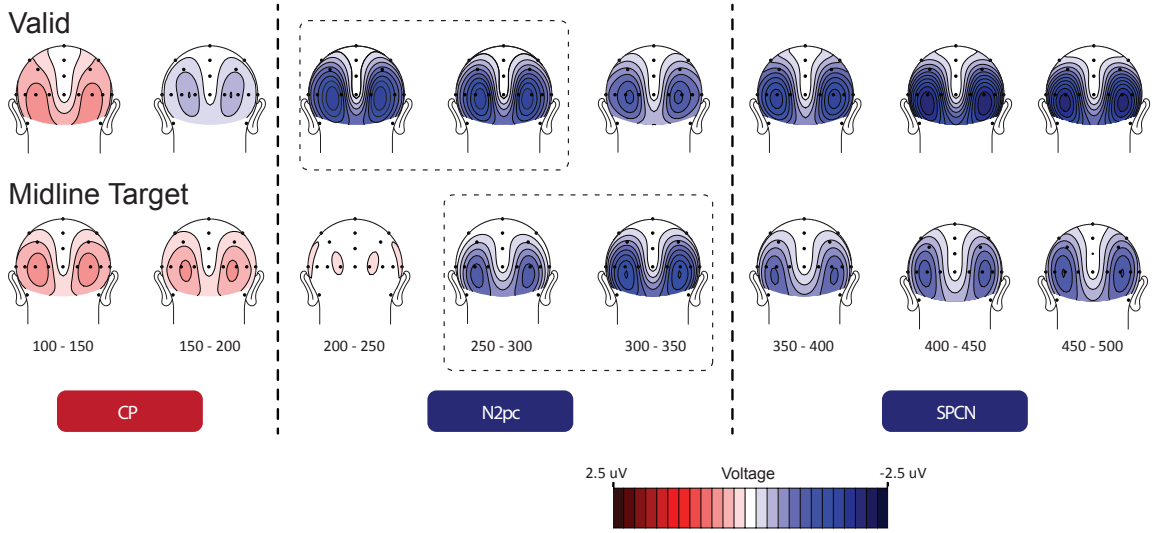

Figure 3.3. Topographical maps of contralateral-ipsilateral voltage differences time-locked to the onset of the search display, plotted separately for valid trials and midline-target trials using conventional symmetric-mapping methods (see Methods section). Dashed grey boxes denote the measurement windows for the target N2pc and cued-nontarget N2pc.

Given that attentional selection of the cued nontarget was not actively prevented (via suppression), the next goal was to determine whether information about the cued nontarget entered working memory alongside target information, as indexed by the SPCN. As expected, the target was found to elicit an SPCN on valid and vertical-invalid trials (Figure 3.2D). This SPCN was found to be significant in a conventional 400 – 500 ms time window (both $ts_{(28)}$ >7.2, ps < .001). Critically, in the same time window, the cued nontarget was found to elicit an SPCN on midline-target trials ($t_{(28)}$ = 4.6, p < .001).

Figure 3.4 displays ERPs from horizontal-invalid and diagonal-invalid trials together with ERP results from the new midline-target trial (Figure 3.4D) As discussed in Experiment 1 (and in Sawaki and Luck, 2013), the CP triggered by the cued nontarget and the N2pc triggered by the target sum linearly to produce a larger negative peak that spans the CP and N2pc time ranges when the cue and target appear on opposite sides of fixation (Figure 3.4C). Following this combined peak, a positive deflection is evident in the difference waveform between 270 – 370 ms. Upon initial inspection, this peak appears as a target positivity (P_T; also called a target P_D) that reflects active termination of target processing (Jannati, Gaspar, & McDonald, 2013; Sawaki et al., 2012). This component was found to be significant in the typical P_T time window of 290 – 340 ms (Sawaki et al., 2012) on both horizontal-invalid and diagonal-invalid trials (both $ts_{(28)} > 2.45$, $p < .05$). However, a different interpretation emerges once the ERPs from horizontal-invalid and diagonal-invalid trials are compared with the ERPs from the midline-target trials. In figure 3.4D, the contralateral-ipsilateral difference waveforms is re-plotted relative to the match cue's location so that the early combined CP/N2pc appears as a positive peak and the putative P_T becomes a negative peak. Plotted in this way, it is apparent that the timing and amplitude of the P_T match the timing and amplitude of the N2pc elicited by the cued nontarget on midline-target trials. Statistical analyses confirmed that the 50% factional peak latencies and the mean amplitudes in the 290–340 ms measurement window were indistinguishable across horizontal-invalid, diagonal-invalid and midline-target trial types (amplitudes: $F_{(2,56)} = 1.86$, $p > .05$; latencies: $F_{(2,56)} = 2.3$, $p > .05$). Based on these results, it is concluded that the post-N2pc deflection observed on horizontal-invalid and diagonal-invalid trials reflects the relatively late N2pc elicited by the cued nontarget rather than a positivity elicited by the target itself.

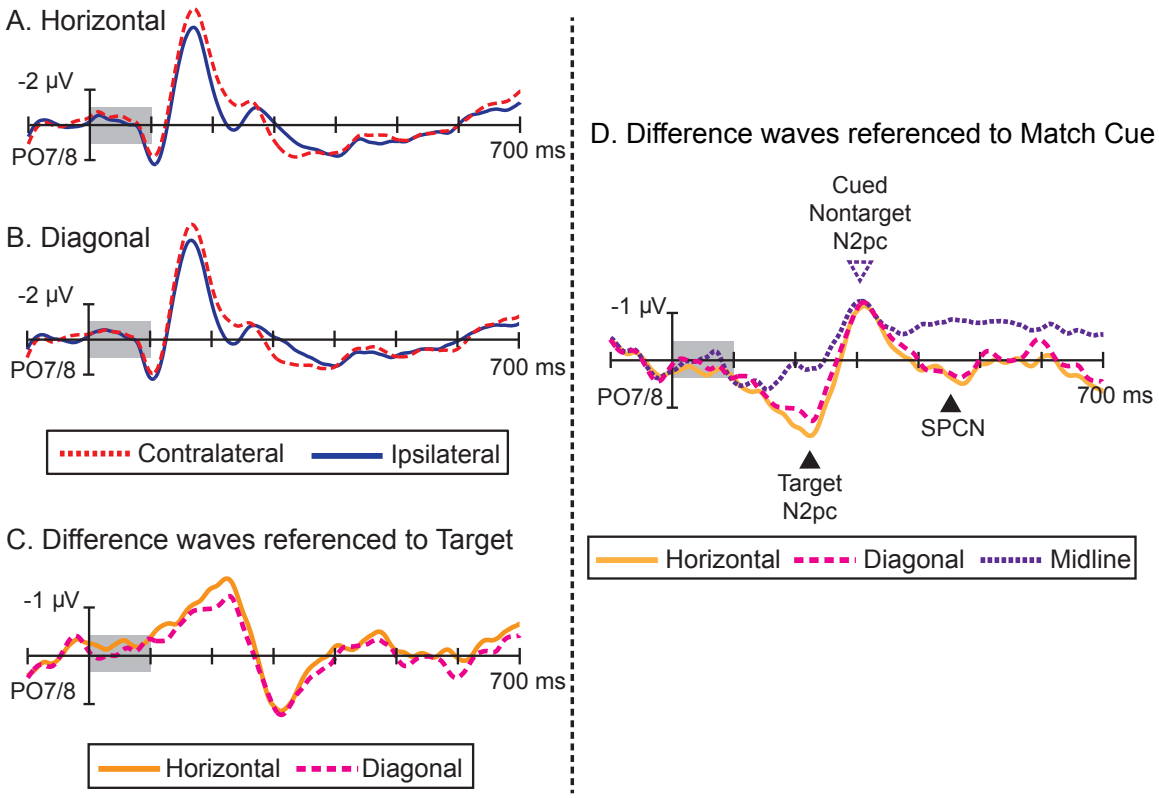

Figure 3.4. **Grand-averaged ERP waveforms from Experiment 2 for invalid trials where the cue and target appear on opposites sides of fixation, time-locked to search arrays in Experiment 2. Waveforms are plotted contralateral or ipsilateral to the match cue, which appeared in the same visual hemifield as the match cue on valid and vertical-invalid trials. The grey box indicates the timing of the search array. (A) Horizontal-invalid trials. (B) Diagonal-invalid trials. (C) Contralateral-minus-ipsilateral difference waves referenced to the target location, for horizontal-invalid trials and diagonal-invalid trials. (D) Contralateral-minus-ipsilateral difference waves referenced to the location of the match cue, for horizontal-invalid, diagonal-invalid and midline-target trials.**

The apparent absence of the SPCN on horizontal-invalid and diagonal-invalid trials can be explained by summation of the SPCNs elicited by the target (isolated on neutral trials) and the opposite-field cued nontarget (isolated on midline-target trials). Based on the isolated target and cued-nontarget SPCN amplitudes (see Table 3.2), one would predict that the SPCN on horizontal-invalid and diagonal-invalid trials would be ~0.43 μV. This predicted value closely approximates the observed SPCN amplitude on horizontal-invalid and diagonal-invalid trials, which was ~0.40 μV. This ERP additivity also accurately

accounts for the variability in SPCN amplitude on trials with same-side cue and target. Based on the logic of additivity, the SPCN observed on valid and vertical-invalid trials would be expected to be larger than the SPCN observed on neutral trials. From the isolated target and cued-nontarget SPCN amplitudes, one would predict that the SPCN would be ~2.31 μV when the cue and target appeared on the same side. This predicted value closely approximates the observed SPCN amplitudes on valid and vertical-invalid trials, which averaged to 2.33 μV.

Table 3.2. Absolute value SPCN mean amplitude and standard error by Trial Type.

Trial Type	SPCN Amplitude
Valid	2.41 (0.29)
Vertical Invalid	2.25 (0.32)
Horizontal Invalid	0.40 (0.41)
Diagonal Invalid	0.39 (0.41)
Neutral (isolated target)	1.37 (0.25)
Midline target (isolated cued nontarget)	0.94 (0.23)

3.3. Experiment 2 Discussion

Experiment 2 was conducted to determine how the cued-nontarget is processed following the reflexive attentional shift to the match cue. One of two attentional mechanisms was hypothesized to occur: active suppression of the match cue, which would yield a P_D in the ERP waveform, or attentional selection of the cued nontarget, which would produce an N2pc in the ERP waveform. The electrophysiological results were consistent with the latter hypothesis and further revealed that the cued nontarget was not only attended (as indexed by an N2pc) but was actively represented at the stage of stimulus identification (as indexed by an SPCN).

Chapter 4. General Discussion

The purpose of Experiment 1 was to elucidate whether the CP reported by Sawaki and Luck (2013) indexed suppression of the match cue or enhanced processing of the cued item in the search display. In Experiment 1, the cue-target SOA was varied in order to track which stimulus display elicited the CP. When the onset of the search display was delayed by 50 ms, the onset of the CP was delayed by 50 ms as well. This result strongly indicates that the CP was time-locked to the search display. Contralateral positivities beginning approximately 100 ms after display onsets have been reported in the electrophysiology literature and have been ascribed to reflect attentional enhancement of incoming sensory signals (e.g. Fukuda & Vogel, 2009; Heinze et al., 1990; Luck et al., 1990; McDonald et al., 2005; Störmer et al., 2009). Thus, in the present study, the CP most likely reflects attentional facilitation of incoming sensory signals for search items at the cued location. On valid trials, this facilitation resulted in shorter RTs; however, on invalid trials, it was still unclear how enhancement of cued-nontarget items resulted in longer RTs.

The purpose of Experiment 2 was to track and understand the neural mechanisms involved in processing the cued nontarget on invalid trials. To do this, target items were placed on the vertical meridian to remove lateralized activity specific to target processing, which enabled measurement of lateralized activity associated with processing of the cued nontarget. The results indicate that following selection of the target item (indexed by the target N2pc), observers attend to and process the cued nontarget (indexed by the cued-nontarget N2pc and subsequent SPCN). Examination of the SPCN from all trial types provided further supporting evidence that information regarding the cued nontarget enters working memory. Cued-nontarget information then competes with task-relevant target information for attentional resources, resulting in longer RTs on invalid trials.

On the basis of these findings, it is hypothesized that two sources of attentional bias contribute to the RT cost/benefit in the contingent capture cueing paradigm (Figure 4.1). First, an *attentional-set* bias (depicted by the filled yellow circle) facilitates selection of items containing the task-relevant feature. Electrophysiologically, this attentional-set bias leads to the N2pc to the match cue and subsequent search target. Second, following

selection of the match cue, a temporary *spatial bias* (the dashed purple circle) is established that enhances early perceptual processing of subsequent items appearing at that location. This enhanced early perceptual processing is indexed by the CP. On valid trials, when the two sources of bias enhance selection and processing of the target item, RTs are shortest. On invalid trials, when the two sources of bias enhance selection and processing of different search items (the target and one of the nontargets), competition is created at higher stages of visual processing and RTs are longer as a result.

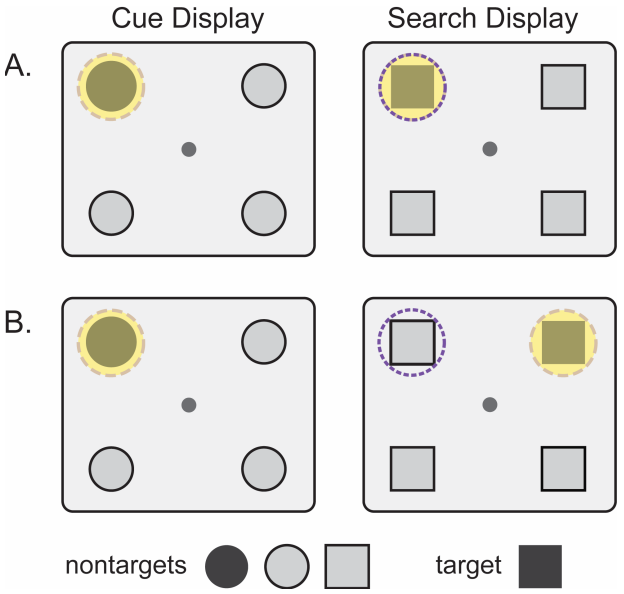

Figure 4.1. Hypothetical processes following contingent attention capture by a cue-array item that possesses a target-defining feature (match cue). Filled yellow circles represent areas of enhanced processing due to an attentional set bias, and unfilled purple circles represent areas of enhanced processing due to a cue-induced spatial bias. (a) Depiction of a valid trial, on which both sources of bias promote selection of the target search item. (b) Depiction of an invalid trial, on which the processing of the cued search nontarget is boosted, thereby increasing competition and neural ambiguity during search.

The present findings contribute to the long-standing debate over serial and parallel modes of visual selection. Serial visual selection occurs when items are individually selected and inspected; whereas parallel visual selection occurs when all relevant items are selected concurrently (Desimone & Duncan, 1995; Itti & Koch, 2005; Theeuwes, 2010; Treisman & Gelade, 1980). Previous ERP studies have demonstrated that when a task requires close inspection of multiple *potential* target items, each item is selected and inspected sequentially, thus leading to sequential and non-overlapping N2pc components

(Woodman & Luck, 1999, 2003). However, recent studies have shown that when a task requires rapid inspection of multiple target items (presented either sequentially or simultaneously), each item of interest is selected in parallel, as indexed by concurrent N2pc components (Eimer & Grubert, 2014; Grubert & Eimer, 2015). The present ERP findings support parallel selection and identification of the cued nontarget and target in the contingent-capture cueing paradigm, indexed by overlapping N2pc components to the two items.

One avenue of future research would be to examine how individual differences in visual working memory (VWM) capacity impact the present electrophysiological and behavioural results. Research has demonstrated that VWM capacity is related to the ability to suppress distracting information (Gaspar & McDonald, 2014; Gaspar et al., in press). Do individuals who have a higher working memory capacity experience less capture by the match cue (indexed by a smaller cue N2pc)? Are these individuals less likely to select the cued nontarget for processing, thus decreasing the amount of extraneous information in working memory on invalid trials? How is the RT cost/benefit different for high capacity individuals? It is hypothesized that high-capacity individuals may not experience any less capture by the match cue, but would be able to disengage attention from the cued nontarget more quickly (possibly preventing the cued nontarget from eliciting an N2pc).

Research examining individual differences in cognitive functioning could be extended to include subclinical populations, for instance individuals who have sustained a concussion. Several studies have demonstrated that VWM capacity is related to overall cognitive functioning (Johnson et al., 2013; Fukuda et al., 2010). Severely concussed individuals routinely report having cognitive impairments, such as an inability to focus (e.g. Guskiewicz et al., 2003; McCrea et al., 2003). Testing concussed individuals using the contingent capture cueing paradigm would allow for assessment of both bottom-up and top-down processes. It is possible that once the attentional set is in place, concussed individuals may have a difficult time recovering from the reflexive shift to the match cue (likely measured by a larger and longer lasting cue N2pc). Concussed individuals may also have difficulty recovering from the spatial bias induced by the match cue in order to select the target on invalid trials, possibly indexed by an N2pc to the cued nontarget first with a subsequent N2pc to the target. This cueing paradigm could lend considerable

insights into how the concussed brain functions, as electrophysiological changes in selective visual attention have been largely uncharacterized.

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