Psychological Research (2009) 73:222–232
DOI 10.1007/s00426-008-0214-y

ORIGINAL ARTICLE

Attentional and anatomical considerations for the representation of simple stimuli in visual short-term memory: evidence from human electrophysiology

Rosalie Perron · Christine Lefebvre · Nicolas Robitaille · Benoit Brisson · Frédéric Gosselin · Martin Arguin · Pierre Jolicœur

Received: 20 December 2007 / Accepted: 28 May 2008 / Published online: 18 February 2009

Abstract Observers encoded the spatial arrangement of two or three horizontal line segments relative to a square frame presented for 150 ms either in left or right visual field and either above or below the horizontal midline. The target pattern was selected on the basis of colour (red vs. green) from an equivalent distractor pattern in the opposite left–right visual hemifield. After a retention interval of 450 or 650 ms a test pattern was presented at fixation. The task was to decide whether the test was the same as the encoded pattern or different. Selection of the to-be-memorised pattern produced an N2pc response that was not influenced by the number of line segments nor by the length of the retention interval, but that was smaller in amplitude for patterns presented in the upper visual field compared with patterns presented in the lower visual field. A sustained posterior contralateral negativity (SPCN) followed the N2pc. The SPCN was larger for patterns with three line segments than for two, was larger for patterns encoded from lower visual field than from upper visual field, and returned to baseline sooner for the shorter retention interval than for the longer interval. These results, and others, provide an interesting and complex pattern of similarities and differences between the N2pc and SPCN, consistent with the view that N2pc reflects mechanisms of attentional selection whereas the SPCN reflects maintenance in visual short-term memory.

R. Perron · C. Lefebvre · N. Robitaille · B. Brisson · F. Gosselin · M. Arguin · P. Jolicœur (✉)
Département de Psychologie, Université de Montréal, C.P. 6128, Succursale Centre-ville, Montreal, QC H3C 3J7, Canada
e-mail: pierre.jolicoeur@umontreal.ca

Introduction

The main goals of the present research were to investigate the neural basis of selective visual spatial attention and of visual short-term memory, and the relationship between these two important cognitive functions in the neurologically intact adult human brain. We used the event-related potential (ERP) method to analyse the electroencephalogram recorded while observers performed a task that required both visual selection based on colour and the encoding and retention of simple visual patterns in the context of a task designed to isolate visual short-term memory (VSTM). The N2pc component was used to index the moment-to-moment deployment of visual spatial attention. The sustained posterior contralateral negativity (SPCN) was used to index encoding and retention in VSTM.

The N2pc is an ERP component that has been argued to reflect the locus of visual spatial attention (Eimer, 1996; Luck & Hillyard, 1994a, 1994b; Luck, Girelli, McDermott, & Ford, 1997). This component is observed typically about 180–270 ms after target onset and is computed by taking the voltage difference between corresponding pairs of electrodes over left and right posterior scalp (e.g., PO7 and PO8), taking into account the hemifield in which attention is deployed. The voltage at the electrode on the contralateral side relative to the attended hemifield is more negative than the voltage at the ipsilateral electrode. Usually, researchers compute an average contralateral waveform by averaging the voltage at the right-sided electrode (e.g., PO8) when attention is deployed to the left with the voltage at the left-sided electrode (e.g., PO7) when attention is deployed to the right. An average ipsilateral waveform is computed by averaging the voltage at the left-sided electrode when attention is deployed to the left with the voltage at the right-sided electrode when attention is deployed to

the right. In a final step, the ipsilateral waveform is subtracted from the contralateral waveform, yielding the N2pc waveform. The name of this component, N2pc, thus signifies a negative-going deflection in the "N2" time range (180–270 ms) that is largest at posterior "p" scalp sites and contralateral "c" to the location of the attended visual item. The designation "N2" is not meant to associate this component with other N2 components, but rather merely indicates the approximate time range of the component (Luck & Hillyard, 1994a).

The N2pc has proven to be a sensitive measure of the locus of visual spatial attention in visual search (e.g., Luck & Hillyard, 1994a, b; Woodman & Luck, 2003), under dual-task conditions, such as the attentional blink (e.g., Dell'Acqua, Sessa, Jolicœur, & Robitaille, 2006; Jolicœur, Sessa, Dell'Acqua, & Robitaille, 2006a, b; Robitaille, Jolicœur, Dell'Acqua, & Sessa, 2007) or the psychological refractory period (e.g., Brisson & Jolicœur, 2007a, b), and under conditions where attention could be captured by distracting peripheral stimuli (e.g., Hickey, McDonald, & Theeuwes, 2007; Kiss, Jolicoeur, Dell'Acqua, & Eimer, 2008; Leblanc, Prime, & Jolicœur, 2008).

In many experiments designed to elicit the N2pc researchers have also found a slower and later wave in the contralateral-minus-ipsilateral waveforms. We will refer to this ERP component as the SPCN (sustained posterior contralateral negativity; e.g., Jolicœur et al., 2006a). Jolicœur et al. (2006a, b) suggested that the SPCN reflects information storage in VSTM, despite the fact that their experiments were not specifically designed to study memory. Jolicœur and colleagues argued that passage through VSTM was required in order for visual stimuli to make contact with mechanisms that exercise cognitive control over subsequent behaviour. As for the N2pc, the SPCN is observed following the visual encoding of a stimulus presented off the vertical midline, either in the left or right visual field. In order to deconfound memory encoding from low-level stimulus differences, the target stimuli in one visual field are presented with an equivalent set of distractor stimuli in the other visual field. Klaver, Talsma, Wijers, Heinze, and Mulder (1999) were the first to argue that the SPCN reflects activity specifically related to encoding and retention in VSTM, a view that received additional support from the elegant work by Vogel and colleagues (Vogel & Machizawa, 2004; McCollough, Machizawa, & Vogel, 2007). Importantly for present purposes, Vogel and colleagues showed that the amplitude of the SPCN increased as the number of target items is increased, reaching a maximum when the number of stimuli to be encoded equalled or exceeded the estimated capacity of VSTM (on a subject-by-subject basis; Vogel & Machizawa, 2004). There is also a strong association between the probability of a correct response and the amplitude of the SPCN in several studies (e.g., Dell'Acqua et al., 2006; Jolicœur et al., 2006a, b; Robitaille et al., 2007).

Given that the N2pc and SPCN are observed following the same contralateral-minus-ipsilateral waveform subtraction, just at different times, one may wonder whether the SPCN is simply additional activity of the same sort as that which generated the N2pc. In other words, is the SPCN a reflection of additional activity due to spatial attention, as opposed to activity associated with VSTM, as some have argued? Jolicœur, Brisson, and Robitaille (2008) examined this issue by varying the number of items to be encoded in a task requiring an immediate response. They found an effect of the number of items on the amplitude of the SPCN and the absence of a similar effect for the N2pc. One goal of the present study was to provide further evidence for the claim that the SPCN is a component distinct from N2pc that reflects primarily activity in VSTM whereas N2pc reflects spatial attention. Thus, one goal was to dissociate these two ERPs. To achieve that goal, we first addressed a possible methodological confound that could provide an alternative, attentional account of the SPCN. Then, we used two different manipulations to link the SPCN to VSTM.

In the work of Vogel and colleagues, the test items were always presented at the same, lateralized location as the memory items (Vogel & Machizawa, 2004; McCollough et al., 2007). Thus, the resulting SPCN could reflect the maintenance of stimuli in VSTM at that particular location, or simply be the result of sustained attention to a lateralised location, resulting in a prolongation of the N2pc. We therefore designed our experiment specifically to discourage subjects from maintaining attention at the location of encoding, by always presenting the test array at fixation instead of at the lateralised memory location. Consequently, the most important spatial location immediately following encoding of the memory items was the region around the fixation point. Sustained attention to the central fixation point could not yield an SPCN, because of the lateralised nature of this component. We note that Klaver et al. (1999) tested memory for polygons either at the same location, or on the opposite side relative to side of encoding and nonetheless found an SPCN, suggesting that anticipating a test shape at the same location on all trials is not necessary in order to observe the SPCN, and one might argue that their procedure would have encouraged subjects to attend to the middle or to spread attention to the entire display in anticipation of the test array. Nonetheless, in 50% of their trials the memory probe was on the same side as the encoded shape, and this may have been sufficient to encourage subjects to maintain an attentional focus on that side (with attention shifting to the other side when required). As such, our procedure provides a stronger test because the test array was never presented at the site of the memory array, providing no reason to maintain attention at

that location, unless part and parcel of the creation and maintenance of a visual memory require continued attention at the spatial locations of encoded items.

We also used two strategies to link the SPCN with VSTM. First, we manipulated the number of items to be encoded and retained in the memory task. The stimuli consisted of two or three small horizontal bars in a square frame, illustrated in Fig. 1. Based on previous research (e.g., Jolicoeur et al., 2008; Vogel & Machizawa, 2004), we anticipated that the amplitude of the SPCN would be greater for three bars than for two bars, to the extent that observers encoded them as distinct objects within the frame. At the same time we could determine whether the N2pc was also affected by this manipulation. Here we expected that the N2pc would likely not vary in amplitude as a function of the number of items to be encoded. This pattern of results—greater SPCN amplitude for three bars than two, with no difference for N2pc—would constitute a dissociation of SPCN from N2pc in terms of the effects of the quantity (or perhaps complexity) of the information to be encoded and retained in VSTM.

The second way of providing a link between retention in VSTM and the SPCN was to vary the duration (450 vs. 650 ms) of the retention interval (the time between the termination of the memory array and the onset of the test

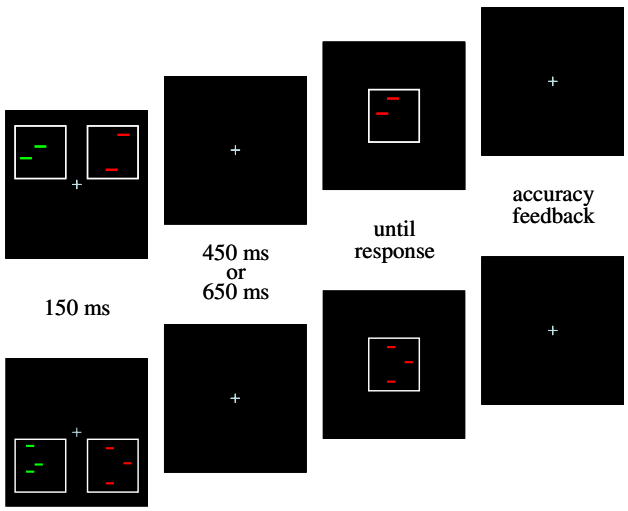

Fig. 1 Illustration of the stimuli and trial sequence. The *upper sequence* shows a trial in which stimuli to be memorised were presented in the upper visual field and consisted of two red bars (half of the subjects encoded *red* items and *half green* items). Stimuli to be encoded could be presented to the left or right. From left to right, the memory array was presented for 150 ms, followed by a retention interval of either 450 or 650 ms during which the fixation cross was visible. The probe array was presented centered at fixation and remained on the screen until the observer responded (same or different). Immediately after the response we provided accuracy feedback (+ or −) at fixation. The *lower sequence* illustrates a trial in which memory items (three *red bars*) were presented in lower visual field. See text for further details

array). On the assumption that observers would maintain a representation of the memory array only as long as necessary, we expected that the SPCN would return to baseline earlier (by about 200 ms) for the shorter retention interval than for the longer one, some time after the presentation of the test array. The N2pc should not be affected by the retention interval because this manipulation occurred after the usual time range of the N2pc.

There is a final way in which the SPCN and the N2pc could be distinguished. Luck et al. (1997) found that N2pc amplitude was smaller when the items in the search array were in the upper visual field compared to equivalent items in the lower field. One account of this difference is based on the way in which receptive fields of visually-sensitive cells project to primary and secondary visual cortex depending on the position of the stimuli in visual space. Stimuli in the upper visual field project from the retina to more ventral portions of the primary visual cortex (not to be mistaken with the ventral stream) whereas stimuli in the lower visual field project from the retina to more dorsal portions of the primary visual cortex (Kandel, Schwartz, & Jessell, 2000; Sereno et al., 1995). This anatomical distinction is maintained, although less so, in other (later) visual areas. One interpretation of the effect of visual fields on the amplitude of the N2pc is that projections to neural generators in more dorsal portions of visual cortex are closer and/or perhaps oriented more optimally to produce large potentials at typical posterior electrode sites such as PO7 and PO8. In contrast, stimulation of the upper visual field would lead to the activation of more distant (and perhaps less optimally oriented) generators in visual cortex, leading to smaller voltages at occipital electrodes. Another account that has been proposed hinges on the notion that attentional mechanisms receiving projections from the upper versus lower visual fields are different, with higher spatial resolution for the lower visual field than or the upper visual field (He, Cavanagh, & Intriligator, 1996). Based on several psychophysical results differentiating performance for stimuli in the upper versus lower visual field, but not for adaptation believed to occur in V1, He et al. (1996) argued that an attentional filter acts beyond the primary visual cortex, in one or more higher visual cortical areas, with different filter characteristics for upper and lower visual field inputs. The evidence suggests a functional asymmetry in the allocation of the attention in favour of the lower visual field (better spatial resolution in lower visual field compared to upper), and perhaps this is the result of an anatomical difference between projections from V1 to the dorsal versus ventral paths in early visual processing. This could be taking place very locally, rather than at the scale of the larger ventral–dorsal distinction associated with the what–where distinction, although, for example, Maunsell and Newsome (1987) found more

numerous projections between early visual areas to parietal region for the lower visual field than the upper visual field.

Although we cannot yet determine whether the difference in the amplitude of the N2pc for upper versus lower visual field stimuli is due to simple anatomical differences in terms of direct projections from the retina to early visual cortex (ventral vs. dorsal projections for V1 and V2; Luck et al., 1997) or to more complex wiring differences across cortical areas representing upper and lower visual fields in terms of connections to other brain regions, implementing different attentional filtering properties (He et al., 1996), this is not critical. We know of no experimental work that has examined the effects of upper versus lower visual field encoding on the SPCN, and presumably on the representation of objects in VSTM. This was one of the key manipulations in the present work. Stimuli to be encoded in VSTM were presented either in the upper or lower visual hemifield. We expected this manipulation to have a large impact on the amplitude of the N2pc (replicating results of Luck et al., 1997), and we sought to discover whether this manipulation would have any effect on the SPCN. If the SPCN is not affected by the upper/lower field manipulation but the N2pc is, as expected, then we would conclude that the N2pc and SPCN are generated by different neural generators because this would constitute an obvious neurophysiological dissociation. If both the N2pc and SPCN are affected, in similar ways, by the upper/lower visual field manipulation, however, then the interpretation would be more complex. Given the convincing prior evidence for a functional difference between these components (corroborated further by results of the present work), we might conclude that the same neural generators produce the observed activity (N2pc and SPCN), but that they perform different functions at different times. We will discuss these possibilities at greater length in "Discussion" of the article.

Methods

Participants

Twenty-three undergraduate student volunteers were tested and paid for their participation. They reported no history of neurological problems, normal or corrected-to-normal acuity, and normal colour vision. Signed informed consent was provided by each participant prior to participation. Ten subjects were excluded because their electroencephalogram (EEG) included too many artefacts (as explained in details in the electrophysiological recording section); leaving 13 subjects (9 females, 4 males, ranging from 20–26 years in age). All females and one male were right-handed.

Stimuli

The stimuli were displayed on a 17-in. colour cathode-ray tube (CRT) controlled by a microcomputer running E-prime 1.04 software. The stimuli were two outline white squares (two frames), presented at the same time, each containing horizontal bars. The center of each frame was located 3.0° above or below and 3.2° on the left and right of the fixation point (at a distance of 57 cm from the computer screen). Each frame subtended a visual angle of 5.0°. A fixation point (0.2°) was present at the center of display. The layout is illustrated (not to scale) in Fig. 1.

In a given trial, the two frames were presented in the upper visual field or the lower visual field, randomly from trial to trial. Each frame in a trial contained the same number of bars, two or three (varied randomly from trial to trial). The total number of pixels in two- and three-bar displays was the same and thus bars in two-bar and three-bar displays had different lengths, but the overall stimulus energy was the same. The position of each bar in each frame was randomly generated at run time, under following constraints: the bars did not have the same position, did not go outside of the frame, or overlap other bar(s), the vertical distance between two bars had a minimum of 0.02°.

The target was defined by bar colour: red for half of the participants and green for other half, counterbalanced across subjects. The green and red bars were always in opposite visual field. The relevant side changed randomly from trial to trial. The colours of the bars were approximately equiluminant to equate their early sensory responses. The luminance of the stimuli was measured with a Minolta CS-100 chroma meter. The luminance of the green colour was 26 cd/m^2 (CIE xy coordinates of $x = 0.297$, $y = 0.579$; Wyszecki & Stiles, 1982), red was 26 cd/m^2 ($x = 0.391$, $y = 0.282$), the fixation point and frame were 26 cd/m^2 ($x = 0.276$, $y = 0.271$) and luminance of the background was 0.70 cd/m^2 ($x = 0.249$, $y = 0.271$).

Procedure

The participant was seated at a distance of 57 cm from the computer screen in a dark electrically shielded chamber. The space bar on the keyboard was pressed to start each trial. After trial initiation, a fixation point appeared in the center of the screen followed by the memory stimulus array. As illustrated in Fig. 1, the memory array consisted of a 150 ms symmetric bilateral display of two white frames—one containing green bars; the other containing red bars. After the offset of the visual display, a delay of 450 ms [short-retention condition; 600 ms, stimulus onset asynchrony (SOA)] or 650 ms (long-retention condition; 800 ms SOA) was imposed, followed by the presentation

of a probe display at fixation. The retention interval (450 or 650 ms) varied randomly from trial to trial. The fixation point remained visible during the retention interval.

The memory probe array was a white frame with bars in the target colour. Participants were instructed to decide whether probe bar positions were identical (in the positions of all bars) or different from the memory array, as fast as possible, while making as few errors as possible. When different, the probe display varied from the memory display in the position of one bar. When different, one bar was moved by a minimum of 20 pixels, (visual angle of 0.7°) but remained inside the white frame. The trials in which a bar position was changed represented 50% of trials (randomly throughout the experiment). Participants responded with the right hand using a specific key on the numeric keypad of a computer keyboard when target and probe were identical (the key was "1") and another key when they were different (the key was "2"). Accuracy feedback was provided at the end of each trial. A plus sign appeared when participant made a correct response and a minus sign when they made an error. Participants pressed the space bar on the keyboard to start the next trial. Each subject performed one practice block of 64 trials followed by 15 experimental blocks of 64 trials for a total of 960 experimental trials. Participants were required to maintain fixation on the centrally located point throughout each trial. They were instructed to blink and move their eyes only between trials.

EEG recording and data analysis

EEG recordings were made with a Biosemi Active-two system, with 64 active Ag-AgCl scalp electrodes positioned using the International 10-10 system: Fp1, Fp2, Fpz, AF3, AF4, AF7, AF8, AFz, F1, F2, F3, F4, F5, F6, F7, F8, Fz, FC1, FC2, FC3, FC4, FC5, FC6, FCz, FT7, FT8, C1, C2, C3, C4, C5, C6, Cz, T7, T8, TP7, TP8, CP1, CP2, CP3, CP4, CP5, CP6, CPz, P1, P2, P3, P4, P5, P6, P7, P8, P9, P10, Pz, PO3, PO4, PO7, PO8, POz, O1, O2, Oz, Iz. In addition, activity was also recorded at the two mastoids. The EEG was algebraically re-referenced to the average of the left and right mastoids during post-recording analyses. The electrooculogram (EOG) was recorded with active Ag–AgCl electrodes placed at the left and right canthi (HEOG) and above and below the left eye (VEOG). The horizontal electrooculogram (HEOG) was obtained by subtracting the signal at the left electrode from the signal recorded at the right electrode. The vertical electrooculogram (VEOG) was obtained by subtracting the signal at the electrode above the left eye from the signal at the electrode below the left eye. For three participants, the vertical electrooculogram (VEOG) was obtained by subtracting the signal at FP1, above the left eye, from the signal at the electrode below the left eye. The signals were amplified, low-pass filtered with a cut-off frequency of 67 Hz, and digitized at 256 Hz during the recordings.

During post-recording analysis, a 5 Hz, 48 db Hz low-pass filter was applied to the HEOG and VEOG waveforms, while a filter with a 0.1 Hz, 12 db high-pass was applied to all electrode waveforms. A derivation of more than 50 μV over 100 ms in the VEOG was considered as an eye blink. A derivation of more than 35 μV within a period of 200 ms (>2°, Hillyard & Galambos, 1970; Lins, Picton, Berg & Scherg, 1993; see also Luck, 2005; for a review), in the HEOG was considered as an eye movement. Trials with incorrect responses, eyes blinks, or eyes movements, were removed during post-recording analysis. The remaining trials were submitted to a final inspection, where a derivation of more than 100 μV over 200 ms during a trial was considered an artefact, and trials with such artefacts were removed from the average for this electrode.

Participants were excluded from behavioural and electrophysiological analyses if the trials with incorrect responses, eyes blinks, eyes movements, and artefacts represented more than 40% of the trials. For each participant and each condition we also computed an average HEOG waveform for trials on which the memory items were on the left and an average for trials on which the memory items were on the right. Participants were also excluded if the average HEOG waveform for left or right trials exceeded residual 3.0 μV, corresponding to an eye movement of about 0.2° in the direction of the to-be-encoded items (Hillyard & Galambos, 1970; Lins et al., 1993; see Luck, 2005; for a review). Eight participants were excluded because of eye movements toward the memory items exceeding 0.2° and one participant was excluded because eyes blinks contaminated too many trials. One participant was excluded from the analysis because a technical problem resulted in the loss of more than 50% of this subject's data.

In most analyses we segmented the continuous EEG starting 200 ms prior to, and ending 1,100 ms after, the onset of the memory array. The EEG at each electrode was baseline corrected by subtracting the mean voltage during the 200 ms pre-stimulus period from the voltage during the entire segment. The EEG from artefact-free trials was averaged, separately for each condition, as detailed below. However, evaluation of the effects of the retention interval manipulation required longer segments (−200 to 1,800 ms, relative to the onset of the memory array). Unfortunately, many subjects had a tendency to blink, or move their eyes, towards the end of the trials, and lengthening the segments caused the rejection of a very large proportion of trials for several subjects. To deal with this issue, we analysed the data differently for the analyses focusing on the

manipulation of the duration of the retention interval. For these analyses only, after removing trials contaminated by eye movements, we did not reject trials with blinks, but instead corrected the EEG using the Gratton–Coles algorithm (Gratton, Coles, & Donchin, 1983), which enabled us to retain a sufficient number of trials. Although we prefer to reject trials contaminated with ocular artefacts, here we adopted a simple correction procedure and compared the waveforms obtained in analyses in which artefact trials were rejected with the waveforms obtained after the application of the correction procedure. The general appearance of the waveforms was not changed, suggesting that the application of the ocular artefact correction procedure did not distort the waveforms significantly. No formal test was used to test the effect of the ocular correction. As will be described in full details below, the N2pc and the SPCN are lateralized components resulting from the subtraction of activation measured on one side from the activation measured on the other side of the scalp. Eye blink artefacts, however, are bilateral. Therefore, even if the correction did not completely remove eye blinks, and residual artefacts were still present in the posterior sites that are of interest in this paper, it would not have an effect on the N2pc or the SPCN. Indeed, the bilateral residual activation would be cancelled in the subtraction of one side from the other.

The N2pc and SPCN waveforms were obtained by subtracting the average ipsilateral waveform from the average contralateral waveform, for each lateralized electrode pair, using the following formula:

$$V_{\mathrm{N2pc,SPCN}} = \frac{1}{2}\left(V\mathrm{left}_{\mathrm{Stim-right}} + V\mathrm{right}_{\mathrm{Stim-left}}\right) - \frac{1}{2}\left(V\mathrm{left}_{\mathrm{Stim-left}} + V\mathrm{right}_{\mathrm{Stim-right}}\right),$$

where $V\mathrm{left}_{\mathrm{Stim\text{-}right}}$ is the voltage at a left-sided electrode (e.g., PO7) for trials on which the to-be-encoded bars were in the right visual field.

The N2pc was quantified by computing the mean voltage in a window of 180–270 ms relative to the onset of the memory array. The SPCN was generally quantified by computing the mean voltage in a window of 450–600 ms from the onset of the memory array. However, there were a number of other measures used to highlight various aspects of the SPCN, and these are described in "Results".

Results

The analyses are reported in two sections: behavioural and electrophysiological results. Only the trials with a correct response were analysed in reaction time and electrophysiological results.

Table 1 Mean accuracy (%) and mean response time (ms) for each target position (upper vs. lower visual field), each memory load (2 vs. 3 segments), and each retention interval (450 vs. 650 ms)

Retention interval	Memory load	Position in visual field			
		Lower visual field		Upper visual field	
		Accuracy	RT	Accuracy	RT
450 ms	2	83	630	82	636
	3	82	643	84	639
650 ms	2	83	617	84	630
	3	81	635	83	635

Behavioural results

Both success rate and reaction time were analysed. For each measure we performed an ANOVA in which position (upper vs. lower field), memory load (two vs. three bars), and retention interval (450 vs. 650 ms) were treated as within-subjects factors. Mean accuracy and mean reaction time are shown in Table 1. The only statistically significant effect was the main effect of number of items in the analysis of response times; the mean RT was 628 ms when verifying memory for 2 bars and 638 ms when verifying memory for 3 bars, $F(1, 12) = 11.92$, $\mathrm{MSE} = 225.8$, $p < 0.01$. All other Fs were smaller than 2.20, while all Fs for accuracy data were smaller than 2.13. These results suggest a slightly longer process of comparison when more bars were to be retained in memory.

Electrophysiological results

Initial electrophysiological analyses examined mean amplitudes measured for the six posterior lateralized electrode pairs: (O1, O2), (PO3, PO4), (P3, P4), (P5, P6), (P7, P8), and (PO7, PO8). The N2pc and SPCN had a maximum voltage at (PO7, PO8), and so we performed all further analyses for the waveforms measured at these sites.

Figure 2 (top panel) shows the ipsilateral and contralateral waveforms for the electrodes PO7 and PO8 for all conditions and the bottom panel shows the grand average difference N2pc/SPCN waveforms at PO7/PO8 for all conditions. It is clear that the experiment produced a clear-cut N2pc followed by a long-lasting SPCN. The impact of our experimental manipulations on these components is examined in detail in the following paragraphs.

The mean N2pc and SPCN amplitudes were submitted to a three-way ANOVA where component (N2pc vs. SPCN), memory load (2 or 3) and visual field (upper vs. lower) were all within-subject factors. While we have no specific hypothesis for the effect of visual field on the SPCN, we expect stimuli presented in the lower visual field to yield a larger N2pc than stimuli presented in the upper

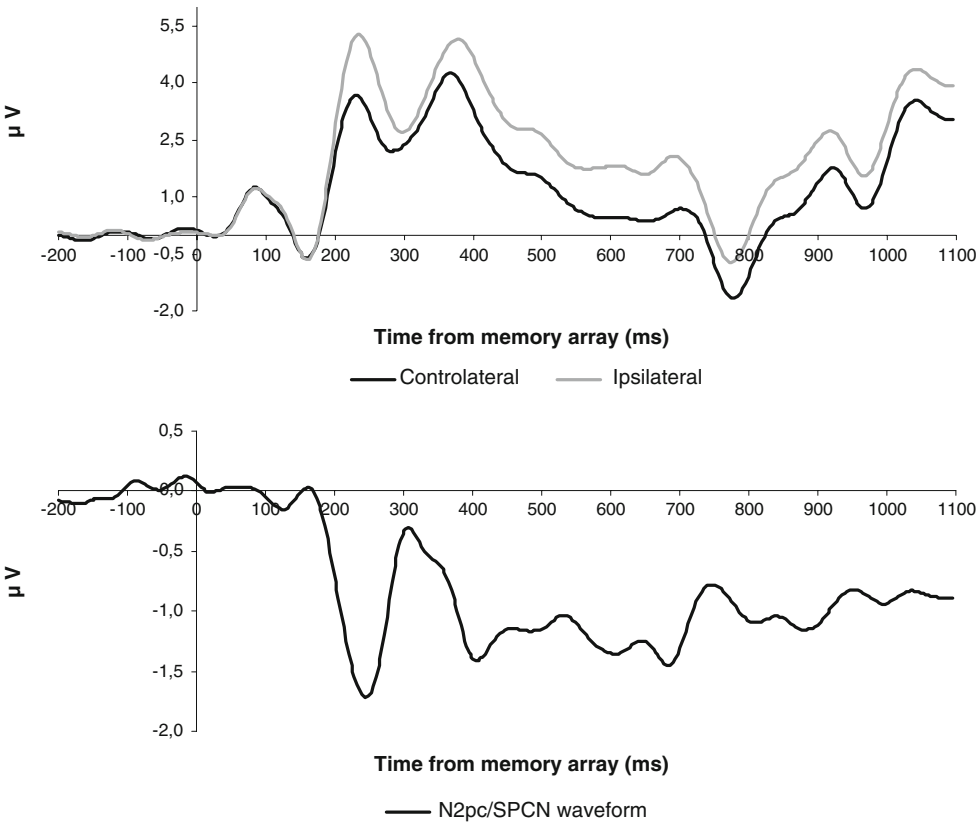

Fig. 2 *Top panel* grand average contralateral and ipsilateral ERPs, over all conditions, timelocked to the onset of the memory array, at electrode pair (PO7, PO8). *Bottom panel* grand average N2pc/SPCN subtraction waveforms (contralateral minus ipsilateral) for electrode pair (PO7, PO8)

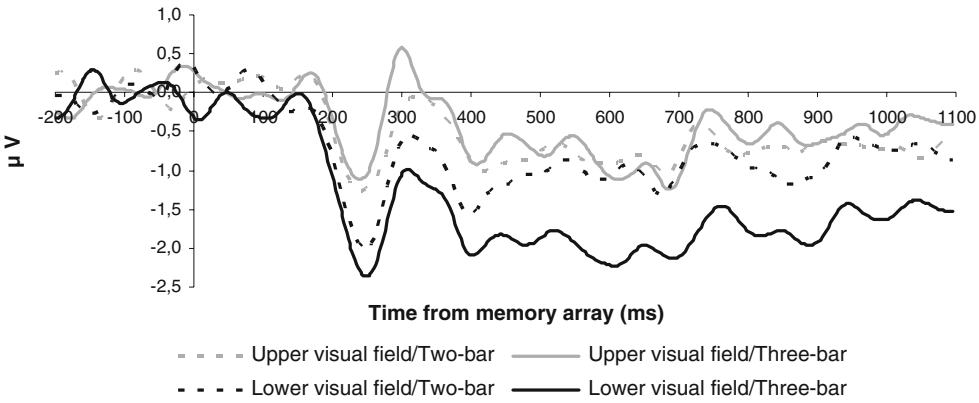

Fig. 3 Grand average N2pc/SPCN waveforms for memory arrays with two or three items encoded from the upper or lower visual field. The data shown was filtered (12 Hz, 24 dB/o) for aesthetic purposes. Note that the analyses were computed without that filter

visual field. We also expect that a higher memory load will create a greater SPCN, but that factor will not have an effect on the size of the measured N2pc, therefore producing at least an interaction between component and memory load. As can be anticipated from the observation of Fig. 3, the ANOVA yielded a three-way interaction, $F(1, 12) = 5.01$, $\mathrm{MSE} = 11$, $p < 0.046$. There was no main effect of component, $F(1, 12) = 0.01$, $\mathrm{MSE} = 2.62$, $p > 0.972$, nor memory load, $F(1, 12) = 1.6$, $\mathrm{MSE} = 0.81$, $p > 0.229$. Since we are mostly interested in the differential effect of load and visual field on the N2pc and SPCN, we decomposed the interaction separately for the N2pc and the SPCN.

N2pc

As we expected, there was no main effect of memory load on the N2pc, $F(1, 12) = 0.07$, $\mathrm{MSE} = 0.67$, $p > 0.800$. However, there was a main effect of visual field, $F(1, 12) = 25.59$, $\mathrm{MSE} = 0.29$, $p < 0.001$) stimuli in the lower visual field (-1.51 μV) generating a larger N2pc than stimuli presented in the upper visual field (-0.75 μV).

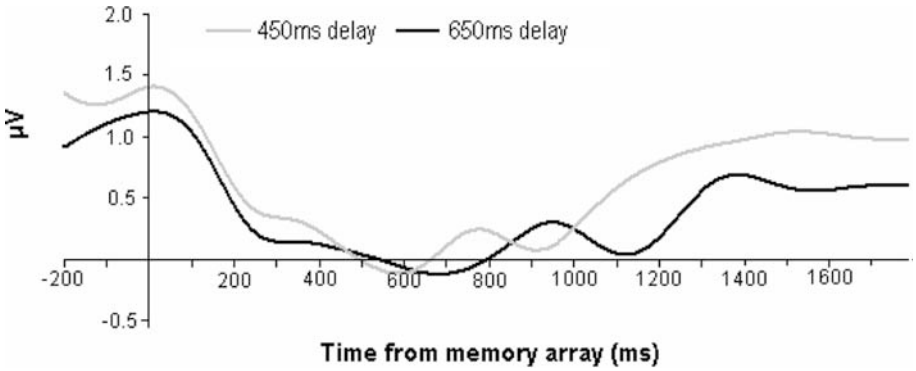

Fig. 4 Grand average SPCN waveforms for different retention intervals, baseline corrected relative to the 200 ms period prior to the onset of the memory test array (in the short-retention interval condition, that is, 400–600 ms from onset of the memory array; note that the same baseline period was used for the two retention interval conditions). Note that the data is shown with the filter applied during the Jackknife procedure

The interaction between memory load and visual field was not significant, $F(1, 12) = 2.68$, MSE = 0.24, $p > 0.127$.

SPCN

As expected from the overall interaction, the pattern of results for the SPCN differed from that of the N2pc. Contrary to the N2pc, there was an interaction between memory load and visual field. Decomposition of this interaction revealed that the memory load effect was significant in the lower visual field, $F(1, 12) = 8.72$, MSE = 0.60, $p < 0.013$, a memory load of three yielding alarger SPCN (−1.93 μV) than a memory load of 2 (−1.03 μV), but not for the upper visual field, a memory load of two yielding a slightly larger SPCN (−0.81 μV) than a memory load of 3 (−0.69 μV), $F(1, 12) = 0.48$, MSE = 0.20, $p > 0.503$. Note that this interaction could not be due to the absence of an SPCN in the load 2 condition: a *t* test revealed that the mean amplitude during the 450–600 ms interval was significantly different than 0, $t(12) = -2.95$, $p < 0.013$.

Retention interval (450 vs. 650 ms)

We examined the effects of the duration of the retention interval by segmenting the EEG into longer segments (−200 to 1,800 ms relative to the onset of the memory array) than for the previous analyses. As explained earlier, rather than exclude trials with ocular artefacts we applied a correction algorithm. The corrected EEG was then averaged separately for each retention interval. These curves were baseline corrected based on the mean amplitude in a window of 400–600 ms (200 ms prior to the presentation of the test array, at the shortest retention interval). We chose this new baseline to equate the curves as much as possible just prior to when they might start to deviate from each other, given that, in principle, all conditions were identical up to that point. The resulting curves, given that they start from a time during which the SPCN was in full force, should deviate toward the positive when the SPCN returns to baseline.

The grand average ERPs resulting from these procedures are shown in Fig. 4. The reader will note that the data were filtered in a lowpass, 3 Hz, 48 dB/o, in accordance with the Jackknife procedure. The waveform for the short-retention interval condition deviates toward the positive about 200 ms earlier than the waveforms for the long-retention interval. We used a jackknife approach to determine whether the waveforms returned to baseline at different latencies for the two retention intervals. In the jackknife approach each of the *n* subjects was removed once from *n* grand average based on $n - 1$ subjects (see Kiesel, Miller, Jolicœur, & Brisson, 2008; Miller, Patterson, & Ulrich, 1998; Ulrich & Miller, 2001). We performed a jackknife analysis in which we estimated, for each jackknifed waveform, the latency at which the waveform crossed a voltage of 0.6 μV separately for the short and long-retention interval waveforms, and subjected these estimates to an ANOVA that considered retention interval as a within-subjects factor. Given that the measurements are taken on a curve that includes virtually the same subjects, the variance of the resulting set of measurements is smaller than the variance of the measurements that would have been obtained from each individual subject curves. The results of the ANOVA were therefore corrected to take into account the smaller variability of the jackknifed estimates (see Miller, Patterson, & Ulrich, 1998; Ulrich & Miller, 2001). The analysis confirmed that the SPCN returned to baseline earlier in the shorter retention interval condition (mean = 1,092 ms) than in the longer retention interval condition (mean = 1,296 ms), $F(1, 12) = 11.73$, MSE = 159.78, $p < 0.006$.

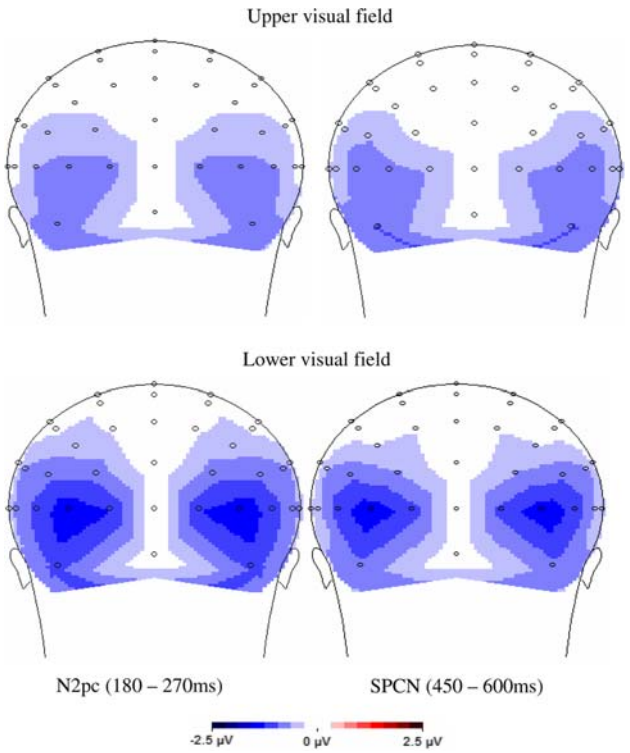

Fig. 5 Distribution of mean voltage during the N2pc (170–280 ms from onset of the memory array), *left column*, and the SPCN (450–600 ms) *right column*, for upper visual field memory arrays, *top row*, and for lower visual field memory arrays, *bottom row*. Since ipsilateral data from the left and the right, as well as contralateral data from both sides are averaged in the subtraction procedure used to create these maps, we end with data for one side of the scalp. We added a mirror reflection of these data for aesthetic purposes

In Fig. 5 we show maps of the distributions of mean voltage during the N2pc (170–280 ms) and SPCN (450–600 ms). The voltage maps were quite similar across the N2pc and SPCN, suggesting that the neural generators of the two components are both in posterior visual areas.

Discussion

In this study we used a paradigm that combined a simple visual spatial task with a VSTM task. Stimuli to be encoded in VSTM first had to be selected from a more complex display on the basis of a pop-out colour (e.g., green) from one side of a left-right bilateral display in which an equal amount of distractor information (e.g., in red) on the other side was to be ignored. The purpose of this manipulation was to induce a clear and distinct N2pc component on the basis of the deployment of visual spatial attention to the to-be-memorised items, which could, in principle, be isolated from the later expected SPCN component associated with maintenance in VSTM (e.g., Jolicœur et al., 2006a, b; Vogel & Machizawa, 2004). In previous work (e.g., Klaver et al., 1999; Vogel & Machizawa, 2004) focusing on VSTM, the relevant information was cued well ahead of the onset of the memory array, making the isolation of N2pc from SPCN more difficult. As can be seen in Fig. 2, our procedure was successful in eliciting distinct N2pc and SPCN waves, enabling us to target analyses on each component designed to evaluate the impact of our experimental manipulations.

VSTM was tested by presenting a pattern at fixation (Fig. 1). This aspect of the experimental design was intended to require a deployment of visual spatial attention in the region surrounding the fixation point, at the time of the memory test. If the SPCN reflected ongoing deployment of visual spatial attention at the location of previously encoded visual objects—in other words a prolonged N2pc—rather than a distinct component reflecting activity in VSTM, then the SPCN should have disappeared 600 ms after the onset of the memory array, because this was the time at which the test array was presented in half of the trials (short-retention interval condition). As can be seen in Fig. 4, the SPCN waves began to attenuate significantly (indicated by a positive-going shift in Fig. 4) long after the presentation of the test array, for both retention interval conditions. If the SPCN was just a long N2pc, presenting the test array at fixation would cause a disappearance of the SPCN about 200 ms following the onset of the test array (because the test array was at fixation, and neither in left nor right visual field, attention to this stimulus cannot generate an N2pc). Thus, the fact that the positive-going shifts in Fig. 4 begin long after an expected cancellation of the N2pc, associated with a shift of attention towards fixation, provides further evidence for the distinctive nature of the SPCN. This finding suggests that the SPCN reflects activity specifically related to VSTM, rather than ongoing visual spatial attention.

We gathered more evidence of the association between the SPCN and VSTM through our manipulation of the retention interval. We used two different retention intervals to test whether the SPCN would return to baseline later with a longer retention interval, which it did. Moreover, the difference in return to baseline was about the same (204 ms) as the difference between intervals (200 ms). If we assume that stimuli are maintained in VSTM only as long as necessary, then this pattern of return to baseline is consistent with a link between the SPCN and VSTM.

The SPCN was also influenced by the number of items in the memory array, the larger memory load yielding a greater SPCN, whereas the N2pc was not influenced by memory load, as shown in the decomposition of the three-way interaction. This difference constitutes a clear functional dissociation between the two components, as well as evidence in favour of a link between VSTM and the SPCN.

However, the interaction of memory load with visual field, for the SPCN, makes the portrait a bit more complex, and will be discussed below. As can be seen in Fig. 2, the field of presentation of the memory array (upper vs. lower visual field) had a major impact both on the amplitude of the N2pc and of the SPCN. The lower N2pc amplitude for upper visual field stimuli was expected on the basis of results of Luck et al. (1997). Here we show for the first time that the amplitude of the SPCN is also affected in a very similar way by presentation in the upper versus lower visual field. This finding is important because it shows that the anatomical, and/or attentional, field effects that influence the N2pc also have a commensurate impact on the SPCN. Also, the effect of the field of presentation (upper vs. lower) was similar across the components, suggesting that the neural generators of the N2pc and of the SPCN are structured in the same orientation relative to the recording electrodes, with receptive fields in upper versus lower visual fields projecting to more ventral and dorsal portions of visual cortex, respectively. Perhaps the very same generators are involved but they perform different functions at different times. The N2pc might reflect an initial deployment of attention to the general region of interest (and hence not influenced by the number of items in that region), whereas a later differentiation of items would take place as information is transferred to VSTM, leading to effects on SPCN.

The situation becomes more complex when we take into account the interaction between memory load and visual field observed for the SPCN mentioned earlier. Recall that the SPCN had a larger amplitude for three bars than for two bars, but only for memory arrays presented in the lower visual field. There was no increase in SPCN amplitude as the number of items was increased for memory arrays presented in the upper visual field (Fig. 2). These are perhaps the most intriguing results of the present work. Note that there were no differences in overall accuracy or response time as a function of field (upper vs. lower) of presentation. To remain consistent with previous research, we suggest that representations of stimuli encoded from the lower visual field may have a greater tendency to be encoded as distinct elements, perhaps because of a greater attentional resolution (He et al., 1996). Items shown in the upper visual field may tend to be encoded more holistically, as a single pattern, perhaps as a result of a greater difficulty to individuate elements due to lower attentional resolution. The difference between two- and three-bar patterns would be attenuated for memory arrays shown in the upper visual field because the additional bar would be less likely to be encoded as a distinct item. Of course, this interpretation is highly speculative, but it is broadly consistent with earlier work showing differences in attentional resolution across the upper and lower visual fields (He et al., 1996). The other, anatomical account would be that the orientation of the cells in the primary visual cortex, which could explain the smaller effect observed in the upper visual field, would also make the distinction between the two memory loads harder to observe, because of a floor effect. Basically, if the orientation of the cells makes the SPCN difficult to detect, than it would also make variations in the SPCN difficult to detect too.

In conclusion, the SPCN is strongly affected by whether stimuli are encoded from the upper or lower visual field, much in the same way as the N2pc, suggesting a strong neuroanatomical connection between these two components. Additional evidence for this connection comes from several experiments, including the present one (Fig. 5), in which the distribution of voltages on the scalp was very similar across the two components (e.g., Robitaille & Jolicœur, 2006; Brisson & Jolicœur, 2007b). Nonetheless, other evidence (such as the differential effect of the number of items on the two components) suggests strongly that N2pc and SPCN reflect distinct aspects of visual processing, with N2pc reflecting the deployment of visual spatial attention and the SPCN reflecting the maintenance of information in VSTM. The present work contributes to our growing understanding of the functional and neuroanatomical underpinnings of these two important cognitive functions.

Acknowledgments This research was supported by research grants from the Natural Sciences and Engineering Research Council of Canada, The Canadian Institutes of Health Research, the Canadian Research Chair program, and by an infrastructure grant from the Fonds de Recherche en Santé du Québec to the last author.

References

Brisson, B., & Jolicœur, P. (2007a). Electrophysiological evidence of central interference in the control of visuospatial attention. *Psychonomic Bulletin & Review, 14*, 126–132.

Brisson, B., & Jolicœur, P. (2007b). A psychological refractory period in access to visual short-term memory and the deployment of visual-spatial attention: Multitasking processing deficits revealed by event-related potentials. *Psychophysiology, 44*, 323–333.

Dell'Acqua, R., Sessa, P., Jolicœur, P., & Robitaille, N. (2006). Spatial attention freezes during the attention blink. *Psychophysiology, 43*, 394–400.

Eimer, M. (1996). The N2pc component as an indicator of attentional selectivity. *Electroencephalography and Clinical Neurophysiology, 99*, 225–234.

Gratton, G., Coles, M. G. H., & Donchin, E. (1983). A new method for off-line removal of ocular artifacts. *Electroencephalography and Clinical Neurophysiology, 55*, 468–484.

He, S., Cavanagh, P., & Intriligator, J. (1996). Attentional resolution and the locus of visual awareness. *Nature, 383*, 334–337.

Hillyard, S. A., & Galambos, R. (1970). Eye movement artifact in the CNV. *Electroencephalography and Clinical Neurophysiology, 28*, 173–182.

Hickey, C., McDonald, J., & Theeuwes, J. (2006). Electrophysiological evidence of the capture of visual attention. *Journal of Cognitive Neuroscience, 18*, 604–613.

Jolicœur, P., Brisson, B., & Robitaille, N. (2008). Dissociation of the N2pc and sustained posterior contralateral negativity in a choice response task. *Brain Research*, *1215*, 160–172.

Jolicœur, P., Sessa, P., Dell'acqua, R., & Robitaille, N. (2006a). Attentional control and capture in the attentional blink paradigm: Evidence from human electrophysiology. *Psychological Research, 19*, 560–578.

Jolicœur, P., Sessa, P., Dell'Acqua, R., & Robitaille, N. (2006b). On the control of visual spatial attention: Evidence from human electrophysiology. *Psychological Research, 70*, 414–424.

Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (2000). *Principles of neural science* (4th ed.). New York: McGraw-Hill.

Kiesel, A., Miller, J., Jolicœur, P., & Brisson, B. (2008). Measurement of ERP latency differences: A comparison of single-participant and jackknife-based scoring methods. *Psychophysiology*, *45*, 250–274.

Kiss, M., Jolicœur, P., Dell'Acqua, R., & Eimer, M. (2008). Attentional capture by visual singletons is mediated by top-down task set: New evidence from the N2pc component. *Psychophysiology*, *45*, 1013–1024.

Klaver, P., Talsma, D., Wijers, A. A., Heinze, H.-J., & Mulder, G. (1999). An event-related brain potential correlate of visual short-term memory. *NeuroReport, 10*, 2001–2005.

Leblanc, É., Prime, D., & Jolicœur, P. (2008). Tracking the location of visuospatial attention in a contingent capture paradigm. *Journal of Cognitive Neuroscience*, *20*, 657–671.

Lins, O. G., Picton, T. W., Berg, P., & Scherg, M. (1993). Ocular artifacts in EEG and event-related potentials 1: Scalp topography. *Brain Topography, 6*, 51–63.

Luck, S. J. (2005). *An introduction to the event-related potential technique*. Cambridge, MA: The MIT Press.

Luck, S. J., Girelli, M., McDermott, M. T., & Ford, M. A. (1997). Bridging the gap between monkey neurophysiology and human perception: An ambiguity resolution theory of visual selective attention. *Cognitive Psychology, 33*, 64–87.

Luck, S. J., & Hillyard, S. A. (1994a). Electrophysiological correlates of feature analysis during visual search. *Psychophysiology, 31*, 291–308.

Luck, S. J., & Hillyard, S. A. (1994b). Spatial filtering during visual search: Evidence from human electrophysiology. *Journal of Experimental Psychology: Human Perception and Performance, 20*, 1000–1014.

Maunsell, J. H. R., & Newsome, W. T. (1987). Visual processing in monkey extrastriate cortex. *Annual Reviews of Neuroscience, 10*, 363–401.

McCollough, A. W., Machizawa, M. G., & Vogel, E. K. (2007). Electrophysiological measures of maintaining representations in visual working memory. *Cortex, 43*, 77–94.

Miller, J., Paterson, T., & Ulrich, R. (1998). Jackknife-based method for measuring LRP onset latency differences. *Psychophysiology, 35*, 99–115.

Robitaille, N., & Jolicœur, P. (2006). Fundamental properties of the N2pc as an index of spatial attention: Effects of masking. *Canadian Journal of Experimental Psychology, 60*, 101–111.

Robitaille, N., Jolicœur, P., Dell'Acqua, R., & Sessa, P. (2007). Short-term consolidation of visual patterns interferes with visuo-spatial attention: Converging evidence from human electrophysiology. *Brain Research, 1185*, 158–169.

Sereno, M. I., Dale, A. M., Reppas, J. B., Kwong, K. K., Belliveau, J. W., Brady, T. J., et al. (1995). Borders of multiple visual areas in humans revealed by functional magnetic resonance imaging. *Science, 268*, 889–893.

Ulrich, R., & Miller, J. (2001). Using the jackknife-based scoring method for measuring LRP onset effects in factorial designs. *Psychophysiology, 38*, 816–827.

Vogel, E. K., & Machizawa, M. G. (2004). Neural activity predicts individual differences in visual working memory capacity. *Nature, 428*, 748–751.

Woodman, G. F., & Luck, S. J. (2003). Serial deployment of attention during visual search. *Journal of Experimental Psychology: Human Perception & Performance, 29*, 121–138.

Wyszecki, G., & Stiles, W. S. (1982). *Color Science: Concepts and methods, quantitative data and formulae* (2nd ed.). New York: Wiley.