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Surfing the attentional waves during visual curve tracing: Evidence from the sustained posterior contralateral negativity

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Abstract

Mental curve tracing is the process by which a contour is covertly followed between two landmarks. Completion time of this task increases as the distance between the landmarks does, even though the Euclidian distance is constant. This has been taken as evidence that attention does not cover a contour instantly, but rather moves from one point to another until the whole contour has been covered. This article provides an electrophysiological measurement of the time course of this spread of attention in humans using a sustained contralateral posterior negative (SPCN) event-related potential component. This component being elicited only when stimuli are presented laterally, the position of lateralization was varied to modulate the onset of this SPCN. Curves that became lateralized further from the central starting point yielded a later SPCN onset than curves that lateralized nearer. This provides converging evidence that attention moves along the curve.

Descriptors: Curve tracing, ERP, SPCN, selective attention

Boundaries and edges created by changes in luminance, texture, depth, or color as well as lines and curves in visual displays undoubtedly play a special role in a variety of fundamental visually guided behaviors ranging from pattern and object recognition to map reading (Marr, 1982). In this context, it has been argued that mechanisms designed to process boundaries, edges, and curves are likely to be particularly important. One such process is mental curve tracing, namely, the process of following a line or curve in a visual display, without moving the eyes, in order to individuate it from other stimuli or to compute relationships between stimuli in the display (e.g., between landmarks along the curve). Visual curve tracing was first studied empirically in humans by Jolicœur, Ullman, and Mackay (1986). They asked observers to determine if two landmarks in the display were on the same curve or not. When the landmarks were on the same curve the distance between them, along the curve, was varied. Displays were constructed such that the Euclidian distance between the landmarks was constant, and thus the absolute distance between the landmarks could not produce differences across different conditions of curve distance. Jolicœur et al. (1986) found that response times increased monotonically as the distance along the curve between the two landmarks increased, suggesting that visual curve tracing involved an underlying process that followed the curve at a finite and measurable speed.

The intertwined, complex curves used by Jolicœur et al. (1986) may have biased subjects to use this strategy because of the special characteristics of their task. However, Pringle and Egeth (1988) and Jolicœur, Ullman, and Mackay (1991) found similar patterns of results for very simple stimuli, suggesting that the results of Jolicœur et al. (1986) were not due to their particular task and were more likely the result of a basic visual routine (Ullman, 1984). This idea is further supported by Brown, Breitmeyer, Leighty, and Denney (2006), who found evidence of curve tracing in a spatial cueing task. They showed that the same-object effect was smaller when the distance between two points was longer, hence providing additional evidence for the existence and functional significance of curve tracing.

All this behavioral evidence also suggests that curve tracing is an attentive process and that it takes time for attention to travel along a curve. Attention appears not to be deployed over an entire curve in a single step, but rather attention appears to move or spread over the curve from one point to another such that longer curve distances lead to longer response times, as though points or segments along the curve are attended sequentially (Jolicœur, 1988; Jolicœur & Ingleton, 1991; Jolicœur et al., 1986, 1991; McCormick & Jolicœur, 1991; Pringle & Egeth, 1988).

A particularly interesting development in the study of curve tracing has been the use of brain measures of the underlying processes implementing curve tracing in animals. Roelfsema,

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Lamme, and Spekreijse (1998) found evidence of spreading activation along a curve in the primary visual cortex (area V1) of monkeys. They found that V1 neurons enhance their response if their receptive field falls on the traced curve relative to when it falls on a curve that is not traced, even if exactly the same stimulus is present in the neuron's receptive field. The data indicated that curve tracing is carried out in the visual cortex by the propagation of an enhanced response from neurons representing contour elements that are attended to neighboring neurons that represent adjacent contour elements until the entire curve is labeled by the enhanced response. The propagation of the response enhancement could occur in two manners: first, through lateral connections within area V1 that interconnect neurons tuned to contour elements in collinear configurations that usually belong to the same curve (Bosking, Zhang, Schofield, & Fitzpatrick, 1997; Schmidt, Goebel, Löwel, & Singer, 1997) and, second, with the help of feedback from higher visual areas (Roelfsema, 2006). In the studies by Roelfsema and colleagues, V1 neurons started to distinguish target from distractor curves about 130–180 ms following the onset of visual displays, and the modulation of the neuronal responses persisted for hundreds of milliseconds (Roelfsema et al., 1998; Roelfsema, Khayat, & Spekreijse, 2003).

Lefebvre, Jolicœur, and Dell'Acqua (2010) provided electrophysiological evidence of curve tracing in humans. They also found evidence of the spread of attention on the curve to be traced using, however, a design that did not allow a verification of the temporal dynamics of the tracing process. To explore the temporal characteristics of visual curve tracing, in the present article we used the event-related approach to estimate the latency of the SPCN component elicited in a task engaging visual curve tracing subroutines. We show, for the first time, that brain activity increases at a later time for portions of a traced curve that is farther from the starting location of the attentional sweep along a target curve. The method and results are important because they allow us to bridge the gap between monkey neurophysiology and human electrophysiology and attention and to show that the fine temporal dynamics of activation in the visual system can be used to track the sweep of attention over a target object. This work was based on the event-related potential (ERP) method to analyze recordings of the electroencephalogram made while observers performed a task constructed to engage visual curve-tracing mechanisms. The particular ERP component of interest was the sustained posterior contralateral negativity (SPCN) and particularly differences in onset latency of the SPCN. This is explained below, following a brief introduction to the SPCN.

The Sustained Posterior Contralateral Negativity

As the name implies, the SPCN is a sustained electrophysiological response that is observed at posterior electrode sites and that is more negative over the hemisphere contralateral to an attended visual stimulus relative to the response observed at ipsilateral electrode sites. For example, if a target is situated in the right visual hemifield and a distractor is in the left hemifield, the activation recorded at electrode PO7 (located over the left hemisphere) will be more negative than the activation recorded at electrode P08 (at the symmetrically located position over the right hemisphere). Inversely, an attended target in the left hemifield will produce higher negativity at electrode site PO8 than at PO7. The onset of the SPCN is linked to the onset of the relevant, lateralized stimulus and to the speed with which processing of the relevant, selected stimulus can increase the activation of cells that respond to the stimulus in the visual system (Brisson & Jolicœur, 2007).

Jolicœur and colleagues argued that the SPCN reflects activity related to the maintenance of representations in visual short-term memory (e.g., Dell'Acqua, Sessa, Jolicœur, & Robitaille, 2006; Dell'Acqua, Sessa, Toffanin, Luria, & Jolicœur, 2010; Jolicœur, Brisson, & Robitaille, 2008; Jolicœur, Sessa, Dell'Acqua, & Robitaille, 2006a, 2006b; Perron et al., 2009). Vogel and colleagues, working on related problems, also found such activity (which they coined the contralateral delay activity, or CDA; Vogel & Machizawa, 2004), as did Klaver, Talsma, Wijers, Heinze, and Mulder (1999), who referred to the observed electrophysiological response as a contralateral negative slow wave (CNSW). It is clear, however, that an SPCN response can be observed in the presence of ongoing stimulation (as was, in fact, observed in the work of Klaver et al., 1999, during the encoding of a visual form). As such, the SPCN likely reflects activity of neural generators that can remain active during a retention interval of several seconds, in which case this activity supports visual short-term memory; but, it can also reflect activity driven from ongoing perceptual input (Drew & Vogel, 2008), as we show in the present article. Jolicœur et al. (2006b) argued that passage through the channel that implements visual short-term memory may be required for consciously controlled behavior from visual input. This view is consistent with single-cell recordings in monkeys demonstrating that neuronal activity in areas of the visual, parietal, and frontal cortex related to attention shifts is usually also modulated by working memory (Chelazzi, Miller, Duncan, & Desimone, 1993; Gnadt & Andersen, 1988; Rainer, Asaad, & Miller, 1998). In this view, it is expected that neural tissue that implements visual short-term memory would be active both during memory retention and also during ongoing active perception in the presence of a driving visual stimulus. We use this assumed property of the generators of the SPCN to study curve tracing.

The SPCN, a lateralized ERP, is observed when one actively maintains, or processes, a lateralized visual stimulus. However, it is not the mere presence of a lateralized stimulus that is needed, but rather that the stimulus be attended and transferred to appropriate processing mechanisms in the brain. We reasoned that tracing a visual curve would engage these mechanisms and lead to a measurable electrophysiological response (the SPCN) if the curve that was actively traced was in the left or right visual field. Evidence in support of this assumption was provided by Lefebvre et al. (2010). In the present work, we extend their work significantly by showing that the onset of the SPCN can be earlier or later, depending on when the expected leading edge of activation along a mentally traced curve enters into either the left or right visual hemifield. We achieved this by creating visual displays that encouraged observers to begin tracing on the vertical midline and by drawing curves that had a portion of curve of different lengths along the midline before branching out to the left or right to a lateralized terminal landmark. This construction is illustrated in Figure 1. Each display had a colored disk at fixation and several disks at the terminal end of lateralized line segments. Participants had to determine if the colored disk at fixation had the same color as the disk to which it was connected by a continuous path. The path could branch left or right from the starting point (fixation) or at a point on the vertical midline some distance from the starting point.

The key predictions for the outcomes of the experiment hinged on the underlying temporal dynamics of visual curve tracing.

Suppose that curve tracing is achieved, in the human brain, by means of a sequential activation of the target curve that is associated with an increase in neural activity of the cells whose receptive fields contain portions of the curve. In this case, we expected to observe an earlier onset of lateralized electrical activity when the curve passed into the left or right visual field sooner (relative to the starting point) than when the curve deviated from the midline at a later point along the tracing path. The curves were designed to produce expected early lateralization or expected late lateralization by very small changes in the configuration of small oblique branches providing bridges from the curve along the vertical midline to one of the lateralized lines (thus creating a continuous path to one of the colored disks at the bottom of the display). If, instead, tracing does not involve a sequential dynamic scan of the curve, but rather a simultaneous activation of the curve, or the task is performed by some other unknown mechanisms, then there is no reason to expect a delayed onset of the SPCN for late-lateralization curves relative to early-lateralization curves. Indeed, if observers could, somehow, activate the terminal position of the target curve using mechanisms other than sequential curve tracing and deploy his or her attention at this location (in order to determine whether the color matched the color at fixation), then we would expect equivalent lateralized responses (latency and amplitude) regardless of the details of the path of the curve leading to this point (particularly given that the length of the paths for "early" lateralization and "late" lateralization were the same, as were all other aspects of the displays).

Given that early lateralization curves had a longer lateralized portion of to-be-traced curve than in late lateralization curves, we also expected the amplitude of the SPCN to differ across conditions. Although we expected about the same number of cells in the visual system to be actively involved in the curve tracing process for both types of curves, more of them would be lateralized for early lateralization curves. This greater number of cells responding to a lateralized stimulus would produce a stronger lateralized ERP. It is important to note that the displays were always closely left–right balanced, overall, and that any differences in lateralized ERP response would have to reflect how the display was processed (i.e., mentally traced) rather than variations of the physical properties of the stimuli. Also, the attended end points of the target curves were also exactly the same for early and late lateralization curves. Thus, any differences across conditions could not arise from differences in lateralized physical characteristics of the displays or in differences in end points of the curves.

Method

Participants

Thirty-six students from l'Université de Montréal took part in the experiment. Thirteen of them were male, and six were left-handed. Their mean age was 22.7 years ($SD = 3.2$). All reported normal or corrected-to-normal vision and no history of neurological disorder. They received a monetary compensation of $20 CAN and gave informed consent prior to their participation.

Apparatus and Stimuli

Participants were seated 57 cm from a computer screen (17-in. CRT color monitor, 640 × 480 pixels at 60 Hz) in a dimly lit, electrically shielded room. Their head position was controlled by a chin rest. Stimulus presentation and behavioral data recording was controlled via E-Prime software. Participants entered their responses using two adjacent keys on a standard computer keyboard.

Stimuli consisted of seven white, straight, vertical lines (CIE $x = .275$, $y = .306$, $Y = 37$ cd/m^2), displayed on a black (CIE $x = .428$, $y = .489$, $Y = .14$ cd/m^2) background (Figure 1). All lines started on the horizontal midline, and ended 5° of visual angle below the midline. One line descended from the center of the screen, whereas three were in the left visual field and three were in the right visual field. A colored disk was at the end of each of the six lateral lines. The lines were separated horizontally by 1°. The two outermost lines were therefore 3° from the vertical midline. The five interior lines were broken by a 0.8° gap in one or two places each. Straight oblique branches also stemmed from the four lines situated to the left and right of the center line. The branches had an orientation of 45° and sloped away from the vertical midline, as illustrated in Figure 1. On each trial, some of the gaps were filled, and one or two branches extended so as to link adjacent vertical lines. These small changes to the display created a single uninterrupted path from the fixation disk to the end disk of one and only one of the two inner lateralized lines in the left or right visual field. The two outermost lines were distractors on all trials. Unlike the five other lines, they were unbroken and did not have branches stemming from them.

Procedure

Eight participants completed one session of testing including 32 practice trials and 480 experimental trials. The rest (28) completed 64 practice trials and 640 experimental trials. The target disk was on the left of fixation on half the trials and on the right on the other half. It was never at the end of the central line, which had no colored disk at the lower end. In half the trials, the uninterrupted path deviated from the vertical midline at fixation, whereas in the other half, it deviated laterally 2.6° below fixation. Each of the four possible terminal disks at the bottom of the display was tested in 25% of the trials. The order of presentation of all types of trial was random.

Trials started with a feedback cross made of five plus or minus signs, depending on the response accuracy on the preceding trial. The first trial in a block started with a cross composed of five plus signs. Participants initiated a trial by pressing the space bar when ready. The feedback cross was immediately replaced by a single fixation cross displayed at the center of the screen. Participants were asked to maintain fixation on this location and to refrain from blinking during the entire trial. The fixation cross remained alone on the screen for an average of 600 ms (± 150 ms jitter). The test display then appeared, and the fixation cross was replaced by a colored disk. The task was to indicate if the color at fixation was the same (half of the trials) or different (half of the trials) as the color at the end of the continuous curve that started at fixation. Half the participants responded by pressing the "c" or "v" key with their left hand. The other half responded by pressing the "b" or "n" with their right hand. The display remained on the screen for 2.5 s. Participants were instructed to respond as accurately as possible, without speed pressure. There was an equal number of early lateralization trials and late lateralization trials terminating at each of the four possible terminal disks, requiring each of the possible responses.

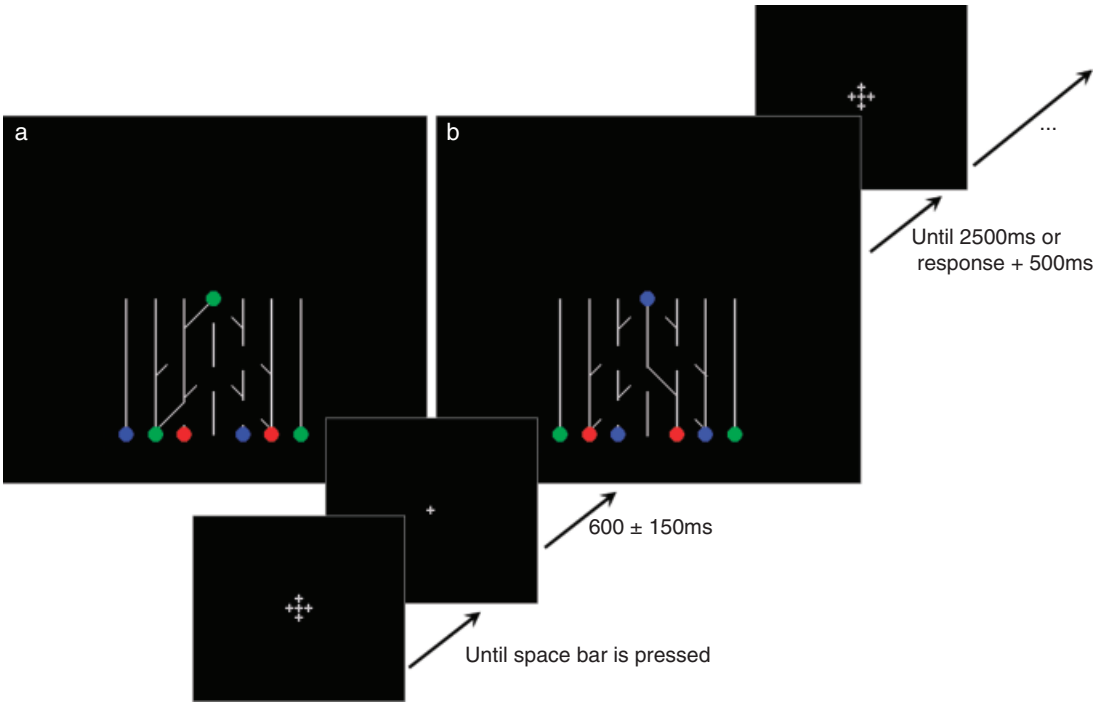

Figure 1. Illustration of the stimuli and trial sequence. Participants pressed a button to have the feedback from the previous trial replaced by a centered fixation cross. This fixation cross was replaced by the test display (see text for details) 600 ± 150 ms later, which remained on the screen for 2500 ms or until response. The test display consisted of a central colored dot and seven bars descending downward from an imaginary line separating the screen in two. The six lateral lines ended with a colored dot. On each trial, only one path connected the center fixation dot to a colored dot at the bottom of the display. The task was to decide whether the color of the dot at fixation was the same of different from the color of the dot at the end of the connecting path. Two examples of test displays are shown in the figure. Example a shows a leftward early lateralization trial associated with a "same" correct response. Example b shows a rightward late lateralization trial associated with a "different" correct response. Feedback was shown 500 ms after participants entered their response.

Electrophysiological Recordings and Analysis

A BioSemi Active Two system and an elastic head cap with 64 Ag/AgCl active electrodes at standard 10–10 system positions plus five external electrodes were used to record brain electrical activity. External electrodes were applied to the left (HEOGl) and right (HEOGr) of the outer canthi and below (VEOGd) the left eye. An electrode was also applied to each mastoid. HEOG and VEOG waveforms were obtained by subtracting left HEOG from right HEOG and Fp1 from VEOGd, respectively. Recording was continuous, at a sampling rate of 256 Hz (low-pass filtered at 67 Hz). The signal was re-referenced off-line to the averaged mastoids.

VEOG and HEOG channels were filtered with a 5 Hz, 48 dB/octave low-pass filter to facilitate ocular artifact rejection. Trials with HEOG activity exceeding 35 μV in a 200-ms sliding window or VEOG activity varying by more than 50 μV over a 150-ms period were removed from analyses, as were trials with EEG activity varying by more than 100 μV over a 50-ms period or exceeding ± 60 μV from baseline during a single segment at electrodes PO7/O8. For all other electrodes, signal was removed, for that particular electrode only, if voltage varied by more than 100 μV in a 50-ms period. Trials with an incorrect response were also removed from analysis. If, after artifact and error removal, less than 50% of the trials remained, data from that participant were removed from analysis, resulting in the loss of data from nine participants. Data from six other participants were removed from analysis because the difference between the residual HEOG signal, averaged separately for left-target curve trials and for right-target curve trials, was higher than 3 μV (equivalent to a horizontal eye movement of about 0.19° of visual angle or greater in the direction of the target curve). The residual HEOG signal for the remaining participants was − 0.06 μV for right-sided targets and − 1.03 μV for left-sided targets, when averaged from 250 to 850 ms after stimulus onset.

To calculate the SPCN, we computed a mean contralateral waveform by averaging the waveform at PO7 for right-target curve trials with the waveform at PO8 for left-target curve trials, separately for early and late lateralization trials. We then computed, separately for each lateralization condition, the mean ipsilateral waveform by averaging the waveform at PO7 for left-target curve trials with the waveform at PO8 for right-target curve trials. Finally, we subtracted the mean ipsilateral waveform from the mean contralateral waveform in each condition, producing one mean SPCN difference wave for early lateralization trials and another for late lateralization trials ($[(PO7_{right}+PO8_{left})-(PO7_{left}+PO8_{right})]/2$).

Results

Behavioral Data

Mean accuracy was 95%. We averaged accuracy for all trials of each of the 21 retained participants (see the section "Electrophysiological Recordings and Analysis") separately for left and right and early and late lateralization displays and then compared them in an ANOVA with side (left vs. right) and later-

alization (early vs. late) as within-subjects factors. The ANOVA did not yield any main or interaction effect, all *p*s > .44.

Electrophysiological Data

Figure 2 shows the SPCN waveforms for early lateralization (black line) and late lateralization (gray line) trials. The onset of the SPCN in the early lateralization trials was earlier (latency at -1 μV = 134 ms) than the onset of the SPCN for the late lateralization condition (latency at -1 μV = 238 ms). The two conditions seemed to vary not only in terms of latency but also in their maximum amplitude. In fact, the mean amplitude of the SPCN in the early lateralization condition was 2.5 times that of the mean amplitude of the SPCN in the late lateralization trials (mean calculated from 250 ms to 850 ms after the onset of the stimuli). We evaluated this difference statistically by computing the mean amplitude of each waveform for each subject and submitting the mean amplitudes to a one-way ANOVA. This ANOVA confirmed that the mean SPCN amplitude in the early lateralization condition (-2.20 μV) was larger than in the late lateralization condition (-0.8 μV), $F(1,20) = 24.85$, $MS_e = 0.75$, $p < .001$, partial $\eta^2 = .55$.

Given that two components, with the same onset, can appear to have different latencies when one curve has a larger amplitude than the other (Roelfsema et al., 2003), we could not compare the latencies in the two conditions based on a fixed amplitude criterion. We used a jackknife procedure (Kiesel, Miller, Jolicœur, & Brisson, 2008; Miller, Patterson, & Ulrich, 1998; Ulrich & Miller, 2001) based on the percentage of amplitude instead of a fixed value to evaluate whether the component onset latency difference was statistically significant. The latency at which the amplitude reached 25% of its maximum was used as a criterion. A one-way ANOVA showed that the latency in the early lateralization condition yielded an earlier SPCN (128 ms) than in the late lateralization condition (mean: 166 ms), $F'(1,20) = 9.50$, $MS_e = 4.11$, $p < .01$, partial $\eta^2 = .84$, $CI_{95} = 12.46, 64.64$. We did not expect that these ERP differences would be associated with differences in reaction times, because the total length of curve that had to be traced was similar for the early and late lateralization conditions. Indeed, despite an earlier SPCN onset, early lateralization trials were not completed significantly faster, on average, than late lateralization trials (823 vs. 813 ms) $F(1,20) = 4.09$, $MS_e = 250.81$, $p > .05$, partial $\eta^2 = .17$. In Figure 3 we display the scalp distribution of the mean amplitude of SPCN (in a window of 250–850 ms) for the early lateralization and late lateralization conditions. In both cases the distribution had a clear lateralized peak and resembled what has been found in earlier work with the SPCN (e.g., Jolicœur et al., 2008; Perron et al., 2009).

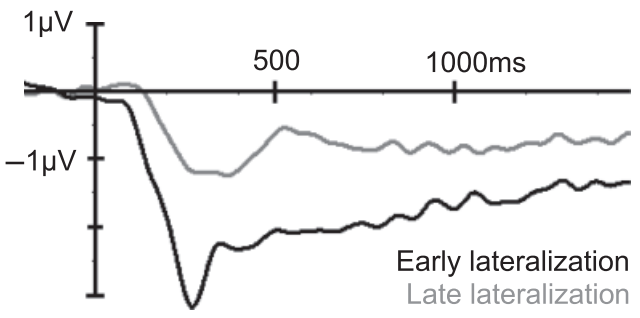

Figure 2. Grand average SPCN waveforms at electrodes PO7/PO8 for early (black) and late lateralization (gray) trials. The waveforms shown were lowpass filtered (8 Hz, 24 dB/octave) for aesthetic purposes. All analyses reported in the article were computed on unfiltered data.

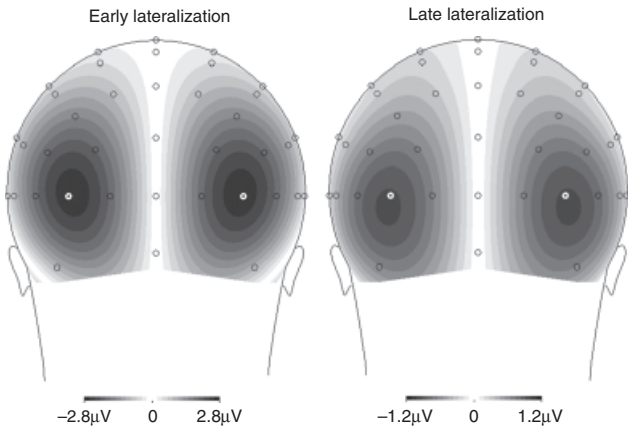

Figure 3. Distribution of mean voltage of the SPCN (250–850 ms from onset of the test display). The positions of electrodes PO7 and PO8 are highlighted in white. Because the SPCN is computed by averaging the contralateral response (e.g., the signal at PO7 for a right-sided curve with the signal at PO8 for a left-sided curve) and subtracting from it the averaged ipsilateral response (e.g., $[(PO7_{right}+PO8_{left}) - (PO7_{left}+PO8_{right})]/2$), the resulting distributions are necessarily symmetric by virtue of the method used to compute the SPCN. We mirror reflected these data for viewing purposes.

The similarity of the shape of the distributions across the two conditions suggests that the same underlying functional component was elicited in the two conditions, and any small differences in distribution probably reflects the differences in details of the retinotopy of the cells activated by early lateralization curves versus late lateralization curves.[1]

1. In a recent study, Van Dijk, Van Der Werf, Mazaheri, Medendorp, and Jensen (2010) suggested that SPCN-like waveforms might be caused by a decrease in the amplitude of alpha oscillations contralateral to the relevant stimuli. They found a significant correlation between the amplitude of alpha-band power and the amplitude of an SPCN-like response in their task. We note that this study is possibly compromised because their displays were unbalanced: No distracting stimuli were presented in the opposite field to make sure the contralateral differences observed were not simply due to physical differences in the display. Furthermore, their experiment did not include a manipulation of memory load, making the functional link to the SPCN less clear. On the other hand, another study, by Grimault et al. (2009), did use a balanced display in their visual short-term memory task. They found that alpha oscillations, as measured by a magnetoencephalography system, decreased at posterior sites contralateral to the stimulation. Although alpha-band amplitude decreased in amplitude contralateral to the attended hemifield, alpha-band amplitude increased with increasing memory load. This load-related increase in alpha oscillations was, however, bilateral, with no evidence of contralateral/ipsilateral differences. The modulations in alpha-band power caused by changes in memory load were not significantly correlated with changes in SPCN amplitude on a subject-by-subject basis. This led the authors to suggest the two modulations could be independent and reflect different processes.

To verify if the SPCN observed during curve tracing might be linked with brain activity in the alpha band, we performed a number of new analyses. If the SPCN is caused by modulations of alpha-band oscillations, then we should observe similar effects as for the SPCN, namely, a difference in amplitude between the early and late lateralization conditions. Also, there should be a correlation between the SPCN amplitude values and alpha-band amplitude values at the individual subject level. To see if our data could provide support for this hypothesis, we first extracted the alpha signal from our data. We performed a fast Fourier transform

Discussion

Covert visual curve tracing of a lateralized curve was associated with a clear pattern of lateralized electrical brain activity that was larger over the hemisphere contralateral relative to the traced curve (Lefebvre et al., 2010). Most importantly, this SPCN was affected by the position of the curve in a manner that was consistent with the expected temporal dynamics of the deployment of visual attention during the tracing process. We used the onset latency of the SPCN as a way to measure when attention reached the position where the target curve departed from the vertical midline toward a lateralized path. It is critical to remember that attending to a location on the vertical midline will produce ERPs that are equivalent at left-sided and right-sided electrodes. Thus, as long as the tracing path remains on the vertical midline we should observe no net lateralization of the ERPs. Assuming that activation spread along the target curve at a finite speed, we would thus expect that the onset of the SPCN would occur at a later time if the curve deviated from the midline further along the postulated tracing path. The results provide a strong confirmation of this prediction of the attentional-shift and spreading-activation models of curve tracing (Jolicœur et al., 1986, 1991; Roelfsema, 2006; Roelfsema, Lamme, & Spekreijse, 2000). We note that large effects on the SPCN were observed with nearly identical physical displays. These displays differed in subtle details of the positions of a few gaps and oblique branches, which, in and of themselves, were extremely unlikely to have produced the observed effects on the basis of lateralized sensory differences. The observed differences between early and late lateralization displays were expected, however, on the basis of how observers would deploy visual spatial attention, on the assumption that curves were traced sequentially from the fixation point to the connected terminal colored disk.

The paradigm we developed allowed us to measure the rate of attentional deployment along the traced curve using a noninvasive measure of brain activity, in the absence of instructions to respond quickly (as is required in response time studies). Based on the estimated latency difference of 81 ms in the onset of the SPCN across conditions and the fact that the late lateralization curves required tracing 2.6° along the vertical midline prior to branching left or right, whereas early lateralization paths deviated from the midline at the starting location of the postulated tracing process, the results suggest a speed of attentional spread of 30°/s (at this speed, it takes 31 ms to travel 1°). It is difficult to compare the rate estimated from our procedure with earlier estimates because previous work has shown that the rate of curve tracing depends on a number of factors, including the proximity of other curves and the curvature of the target curve (Jolicœur et al., 1991; see also Jolicœur & Ingleton, 1991). Nonetheless, the estimated speed of displacement of the attentional wavefront appears entirely reasonable and is broadly consistent with earlier results. For example, Jolicœur et al. (1991) found tracing rates of approximately 15°–50°/s with similar displays, whereas Jolicœur et al. (1986) obtained 40°/s. With their very simple display and in the absence of distractor curves, Pringle and Egeth (1988) unsurprisingly obtained faster tracing speeds ranging from 116°/s to 231°/s.

We note that our methods allowed us to eliminate contributions of eye movements to the results (indeed, moving the eyes to the target curve would eliminate differential lateralization of the signal on the basis of attentional activation). Thus, the results reflect an entirely covert deployment of visual attention, guided by a visual curve, which is presumably used to guide eye movements when more complex displays (e.g., maps, circuit diagrams) are processed (e.g., to plan a trip).

The presence of a clear SPCN in response to curve tracing suggests that, as in the monkey visual cortex, cells in the human brain that respond to visual curves increase their firing rate when the cells respond to the curve of interest. The present work suggests that it is now possible to use latency differences in the onset of the SPCN when this enhanced response is observed to estimate when the attentional enhancement effect reaches a particular point on the object of interest. Although, in other conditions, it may be possible for attention to jump from one location to another, complex visual scenes sometimes require the integration of distant points linked by a common curve. In the presence of distractor curves, attention cannot jump from one curve to another without some way to ensure that the portion of curve between these points is, indeed, one and the same curve. Under these conditions, it appears that attention cannot jump very far and instead appears to move along the curve smoothly from one location to another and to activate points along the curve between the start and end locations of the larger attentional shift. We have devised a noninvasive electrophysiological method to track this attentional sweep in the human brain and to quantify the speed of attentional movement through visual space along a target curve.

(FFT) of the unfiltered, artifact-free data at electrodes PO7 and PO8 in a time window from 250 to 850 ms after stimulus presentation separately for left and right and early and late lateralization trials. FFTs were performed on each trial in order to estimate the induced oscillatory power, and we then averaged the estimates for each condition and side to obtain mean induced power. Power was averaged in a frequency interval from 9 to 12 Hz separately for each subject and condition, and an SPCN-like calculation (contralateral minus ipsilateral power) was performed using these values. Interestingly, alpha-band power was lower over the contralateral hemisphere relative to the traced curve (mean = 0.023 μV^2) $F(1,20) = 6.93$, $p < .02$. However, there were no other significant findings. The contralateral decrease in alpha-band power was not significantly different across the late lateralization condition (– 0.03 μV^2) and the early lateralization condition (– 0.016 μV^2), $F(1,20) = 1.75$, $p > .20$. To check further, we also correlated the amplitude SPCN and the amplitude of the alpha-band contralateral-ipsilateral difference separately for the two conditions. Neither correlation was significant, late: $r = .32$, $t(19) = -1.46$, $p > .16$, early: $r = .31$, $t(19) = 1.41$, $p > .17$. In short, there was a decrease of alpha-band oscillatory power at posterior sites contralateral to the traced curve, even with balanced displays, as observed by Grimault et al. (2009) and others. However, there was no significant difference in alpha-band amplitude between the late and early lateralization conditions, and, more importantly, there was no significant correlation between SPCN voltage amplitude and contralateral–ipsilateral differences for alpha waves. Although it is possible that these relationships could be stronger in other situations, neither the present results nor those of Grimault et al. (2009) provided support for the hypothesis put forth by Van Dijk et al. (2010) that the SPCN is the reflection of a modulation of alpha-band oscillations.

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