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RESEARCH ARTICLE

Electroencephalographic correlates of target and outcome errors

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Abstract Different neural systems underlie the evaluation of different types of errors. Recent electroencephalographic evidence suggests that outcome errors—errors indicating the failure to achieve a movement goal—are evaluated within medial-frontal cortex (Krigolson and Holroyd 2006, 2007a, b). Conversely, evidence from a variety of manual aiming studies has demonstrated that target errors—discrepancies between the actual and desired motor command brought about by an unexpected change in the movement environment—are mediated within posterior parietal cortex (e.g., Desmurget et al. 1999, 2001; Diedrichsen et al. 2005). Here, event-related brain potentials (ERP) were recorded to assess medial-frontal and parietal ERP components associated with the evaluation of outcome and target errors during performance of a manual aiming task. In line with previous results (Krigolson and Holroyd 2007a), we found that target perturbations elicited an ERP component with a parietal scalp distribution, the P300. However, the timing of kinematic changes associated with accommodation of the target perturbations relative to the timing of the P300 suggests that the P300 component was not related to the online control of movement. Instead, we believe that the P300 evoked by target perturbations reflects the updating of an internal model of the movement environment. Our results also revealed that an error-related negativity, an ERP component typically associated with the evaluation of speeded response errors and error feedback, was elicited when participants missed the movement target. Importantly, this result suggests that a reinforcement learning system within medial-frontal cortex may play a role in improving subsequent motor output.

Keywords ERP · Manual aiming · Target perturbation · P300 · ERN

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Introduction

In order to achieve desired movement goals, the motor system needs to able to evaluate and correct many types of errors. For instance, the human sensorimotor system needs to be able to rapidly correct for *target errors*—errors in an ongoing motor command brought about by sudden, unexpected changes in the movement environment (cf., Diedrichsen et al. 2005). Behavioural studies of motor control have demonstrated just this, that when reaching to a target, the motor system is able to quickly modify the current motor command to correct for changes in target location—even if these changes occur after movement onset (Goodale et al. 1986; Krigolson and Heath 2006). For example, in a seminal study Goodale and colleagues found that participants making rapid aiming movements could modify their

movement trajectory to correct for an unexpected change in target location even though the target moved during saccadic suppression. In other words, in spite of being consciously unaware of the target error, participants were able to rapidly correct their movement trajectories to accommodate the new target locations. Recent neuroimaging and neuropsychological studies suggest that posterior parietal cortex (PPC) plays a key role in the evaluation of target errors and subsequent within movement adjustments to the motor command (Culham et al. 2003; Desmurget et al. 1999, 2001; Desmurget and Grafton 2000; Diedrichsen et al. 2005; Gréa et al. 2002; Pisella et al. 2000; Trillenberg et al. 2007).

In spite of the general effectiveness of the motor system to correct for target errors, sometimes the executed motor command fails to achieve the intended goal. Recent experimental evidence suggests that *outcome errors*—motor errors representing the failure to achieve the desired movement goal—are processed by a neural system within medial-frontal cortex involving anterior cingulated cortex (ACC) and the basal ganglia (Krigolson and Holroyd 2006, 2007a, b). Specifically, Krigolson and Holroyd conducted a series of recent experiments and examined the event-related brain potentials (ERPs) associated with successful and unsuccessful performance of continuous tracking and joystick aiming tasks. The results of these studies indicated that outcome errors modulated the amplitude of the error-related negativity (ERN), an ERP component that is sensitive to response errors (rERN; Falkenstein et al. 1991; Gehring et al. 1993) and error feedback (fERN; Miltner et al. 1997). Source localisation of the rERN and the fERN suggests a common source within medial-frontal cortex (specifically ACC; Holroyd et al. 2004; Miltner et al.; but see Nieuwenhuis et al. 2005c), and a current theory holds that these components are produced by a dopaminergic reinforcement learning signal sent from the basal ganglia to anterior cingulate cortex (Holroyd and Coles 2002).

To account for the roles of medial-frontal and posterior error systems in motor control, it has been proposed that error processing systems in the brain are hierarchically organised (Krigolson and Holroyd 2006, 2007a). Specifically, the hierarchical error processing hypothesis states that the motor system evaluates two levels of errors: "high-level" errors indicating a failure to achieve a movement goal (i.e., outcome errors), and "low-level" target errors indicating a discrepancy between the actual motor command and the appropriate motor command (i.e., target errors). Target errors are in principle correctable, but if they are not resolved during a movement then they result in an outcome error. For example, consider a goal-directed reaching task in which the target occasionally jumps to a new location following movement onset. The target error caused by the change in target location is a low-level error because it reflects a discrepancy between the actual motor command (which would be directed toward the previous target location) and the appropriate motor command (which should be directed toward the new target location). In a target perturbation paradigm, an online adjustment to the motor command is implemented to resolve the target error. However, if the motor system fails to correct the target error (because the change in target location occurred very late in the movement or the corrective movement was blocked) then an outcome error would occur. According to the hierarchical error processing hypothesis, target errors are mediated within PPC and/or the cerebellum (Desmurget et al. 1999, 2001; Desmurget and Grafton 2000; Gréa et al. 2002; Miall et al. 2001; Pisella et al. 2000) and outcome errors are evaluated by a medial-frontal reinforcement learning system involving anterior cingulate cortex and the basal ganglia (Holroyd and Coles 2002; Krigolson and Holroyd 2006, 2007a, b).

Although the ERN appears to be the product of the high-level error processing system (c.f. Krigolson and Holroyd 2006, 2007a, b), to our knowledge an ERP signature of low-level error processing has yet to be reliably identified. With that said, in a previous study, it was found that target errors which occurred during performance of a joystick aiming task elicited a P300 component (Krigolson and Holroyd 2007a). The P300 is a parietally distributed positive deflection in the ERP that has been associated with the updating of an internal model of the environment (Donchin and Coles 1988) and more recently with decision-related phasic activity of the locus-coeruleus norepinephrine (LC-NE) system (Nieuwenhuis et al. 2005a, b). Interestingly, in the aforementioned study (Krigolson and Holroyd 2007a) the timing of the P300 immediately preceded corrective movements that were implemented to accommodate target errors—a result which suggested that the component might reflect parietal activity associated with the online control of movement. Somewhat problematically, Krigolson and Holroyd's results could not completely resolve whether or not the P300 reflected evaluation of target errors within PPC. Specifically, the "online control" required to accommodate the change in target location consisted of a single, discrete manipulation of a computer joystick during performance of a relatively slow (movement times $\approx$ 1,500 ms) aiming task—a movement that is physically and temporally quite different from the limb trajectory adjustments required to accommodate target perturbations during rapid goal directed reaching movements. Furthermore, in the Krigolson and Holroyd study task constraints made it advantageous for participants to make corrective movements during the later stages of the joystick aiming movement. Specifically, participants might have implemented a corrective movement that was based on a diagonal path to the target from the initial movement trajectory—a strategy which

would have resulted in participants deliberately not adjusting for the target perturbation as soon as it occurred (the perturbation occurred at movement onset). Thus, from Krigolson and Holroyd's results it is not clear whether or not the P300 reflects visual processing of the target jump, the online control of movement, or some other process. In the present study we sought to further evaluate the role of the P300 in the mediation of target errors by forcing participants to make rapid online adjustments to an ongoing motor command during performance of a manual aiming task and then making a comparison between these corrections and the timing of the P300. In other words, we sought to further investigate whether parietal parts of the brain produce an ERP component related to low-level error processing that is analogous to the ERN.

In the present study we recorded the electroencephalogram (EEG) while participants made rapid, two-dimensional aiming movements to a visually defined target. To evoke online corrective limb adjustments, on some trials the location of the movement target changed unexpectedly following movement onset. In line with previous findings (Krigolson and Holroyd 2007a), we predicted that target errors would elicit a P300. However, to clarify the relationship between the P300 and the evaluation of target errors, in the present study we had participants make rapid aiming movements (movement times between 400 and 700 ms) and compared the timing of the P300 with the timing of kinematic changes associated with accommodation of target errors by the motor system (specifically, changes in vertical acceleration). We predicted that if the P300 was elicited directly by the error correction process itself, then its onset would occur before any changes in movement kinematics following target errors. Conversely, if P300 onset occurred after participants' corrective movements, then that would indicate that the P300 was not directly associated with the online evaluation of target errors.

A second goal of the present experiment was to evaluate the role of the medial-frontal cortex in the evaluation of endpoint error. Previous work has examined the ERP components associated with tracking errors (Krigolson and Holroyd 2006) and blocked corrective movements (Krigolson and Holroyd 2007a), but to date there has not been an analysis of the ERP components associated with the evaluation of endpoint error. Within the hierarchical framework, missing a movement target constitutes an outcome error and thus should elicit the ERN. Importantly, we hypothesized that an ERN elicited by off-target trials would suggest that the involvement of the medial-frontal reinforcement learning system in improving subsequent motor output. Finally, in order to further investigate the hierarchical hypothesis, we also sought to replicate previous findings by preventing participants from making corrective movements on one half of the target perturbation trials. In line with previous findings (Krigolson and Holroyd 2007a), we anticipated that an ERN would be elicited by precluding the implementation of an online corrective movement.

Methods

Participants

Fifteen right-handed undergraduate students (18–26 years of age; 8 males, 7 females) with no known neurological impairments and with normal or corrected-to-normal vision participated in the experiment. The participants provided informed consent approved by the Office of the Vice-President, Research, University of Victoria, and the study was conducted in accordance with the ethical standards prescribed in the 1964 Declaration of Helsinki.

Apparatus and procedure

Participants were seated comfortably in front of an aiming apparatus similar to that employed by Held and Gottlieb (1958). The apparatus consisted of a two-sided box (74 cm high, 96 cm wide, 60 cm deep) divided in half by a fully silvered mirror inclined at 20°. A 17 in. computer monitor (LG 1750 SQ: 8 ms response rate) was placed upside down on the superior surface of the apparatus in order to project stimuli onto the surface of the mirror. A graphics tablet (WACOM Intuos 2, 30 cm × 45 cm, sampling rate: 125 Hz) was placed directly below the mirror such that movements made on the surface of the graphics tablet corresponded to movements of a cursor (a 0.5 cm × 0.5 cm red square) on the surface of the mirror (see Electronic supplementary material Fig. 1). The distance between the eyes and the mirror was approximately 45 cm. Participants held a stylus in the reaching hand (i.e., their right hand) and completed pointing movements to a visually defined target. Participants started each trial by placing the stylus on a common home position (i.e., a 1.5 cm × 1.5 cm white square located on the left hand edge of the display) and then initiated a left to right aiming movement to a target (i.e., 1.5 cm × 1.5 cm white square) in response to an auditory start tone. Note that if the participant moved the stylus before movement onset, the trial did not commence and the participant had to reset the cursor to the start position following an error message.

Participants completed aiming movements in three experimental conditions. In the first condition (i.e., the *control* condition) the target presented to participants in advance of movement onset also served as the target during movement execution; that is, the target location was not perturbed. In the second condition (i.e., the *correction* condition) participants made aiming movements to the target

location as in the control trials; however, immediately following movement onset the target jumped to a new location (either up or down 8 cm, equal probability for each direction). As such, correction trials required an online modification of the initial aiming movement. The third condition (i.e., the *blocked* condition) was identical to the correction condition with the exception that the participants' cursor was locked to the horizontal axis following the perturbation. As such, on blocked trials participants always missed the target location as they were unable to alter the vertical cursor position to accommodate the target perturbation. In other words, in the blocked condition participants were only able to modify their initial motor plan in terms of movement along the horizontal access.

Participants were instructed that their goal was to move the cursor onto the target square as "fast and accurately" as possible. In total, participants performed five blocks of 60 trials with 20 trials from each of the three conditions randomly interleaved within each block. To ensure that participants did not hesitate following the start of the movement to determine whether a target perturbation would occur, and whether or not they would be able to correct for it, they were required to complete the movement within 400–700 ms. Participants were instructed that if their movement time (the time from movement onset to movement end) occurred outside of this time window then a replacement trial (selected from the three conditions at random) would occur. Between blocks participants relaxed during self-paced rest periods. The aiming task was explained to participants before the task began and they completed five trials in each of the aiming conditions during a practice block.

Behavioural analysis

Displacement data for the participants' cursor were recorded for each trial. Following data collection we filtered displacement data via a second-order dual-pass Butterworth filter using a low-pass cut-off frequency of 10 Hz. The displacement data were then differentiated using a three-point central finite difference algorithm to obtain instantaneous velocities in the vertical movement axis. The same algorithm was used to differentiate the velocity values to obtain instantaneous acceleration values for each time point. Dependent variables used in subsequent analyses were reaction time (ms), movement time (ms), constant error (pixels) and variable error (pixels) in the horizontal (CE_h, VE_h) and vertical (CE_v, VE_v) movement axes, and instantaneous acceleration in the vertical movement axis (pixels/ms^2). To get a temporal estimate of when participants began to correct for a target perturbation, we utilised a stepwise procedure in which a 40 ms window of instantaneous acceleration values (i.e., ±20 ms) were averaged for each time point for each experimental condition (control, correction, blocked) and participant across the aiming trajectory. The resulting acceleration values for the correction and blocked conditions were then tested against the control acceleration values with a series of two-tailed t-tests against zero (see Rodriguez-Fornells et al. 2002). Thus the onset of the corrective movement, as determined by a statistical difference in the acceleration profile between the control and correction/blocked conditions was defined as the first time point at which five consecutive t-tests showed a significant difference ($P < 0.05$).[1] Note that we inverted the signed acceleration values for downwards target perturbations to allow us to collapse across perturbation direction (up, down) and focus our analysis on conditional differences.

Electrophysiological analysis

The EEG was recorded from 41 electrode locations using Brain Vision Recorder software (Version 1.3, Brain Products, Munich, Germany). The electrodes were mounted in a fitted cap and were referenced to a common ground: Fp1, Fpz, Fp2, F7, F3, Fz, F4, F8, FT9, FC5, FC1, FCz, FC2, FC6, FT10, T7, C3, Cz, C4, T8, TP9, CP5, CP1, CPz, CP2, CP6, TP10, P7, P3, Pz, P4, P8, PO7, POz, PO8, Oz, M1, M2, LHEOG, RHEOG, VEOG. The vertical and horizontal electro-oculograms were recorded from electrodes placed above and below the right eye (FP2, VEOG) and on the outer canthi of the left and right eyes (LHEOG, RHEOG), respectively. Electrode impedances were kept below 10 kΩ. The EEG data were sampled at 250 Hz, amplified (Quick Amp, Brain Products, GmbH, Munich, Germany) and filtered through a passband of 0.017 Hz–67.5 Hz (90 dB octave roll off). Following data collection the EEG data were filtered through a 0.1–20 Hz passband phase shift free Butterworth filter and re-referenced to linked mastoids. Ocular artefacts were removed using the algorithm described by Gratton et al. (1983). Trials in which the change in voltage at any channel exceeded 35 μVs per sampling point were also discarded. In total, less than 5% of the data were discarded. All waveform segments irrespective of the point of analysis were baseline corrected to a 200 ms epoch from −500 to −300 ms before the movement initiation cue.

To analyse the target perturbations, epochs of data spanning from 400 ms before movement onset to 600 ms after movement onset were extracted from the continuous EEG for each experimental condition (control, correction,

[1] Onset procedures such as the one we employed here are not typically used on acceleration data in this manner. However, we felt that this was a viable technique for identifying when participants began to correct for the target perturbation. Furthermore, this technique allowed for a direct comparison with the ERP data.

blocked). In a similar manner, to examine the corrective movements epochs of data spanning from 200 ms before the corrective movement to 400 ms after the corrective movement were extracted from the continuous EEG for the correction and blocked conditions. As there was no target perturbation during the control trials, we did not compare the waveforms for this condition the waveforms for the other two conditions. Finally, to analyse the waveforms associated with on-target (the cursor landed on the target square) and off-target (the cursor landed off the target square) trials, epochs of data spanning from 200 ms before movement end to 600 ms after movement end were extracted from the continuous EEG. To ensure a sufficient number of trials for this analysis, the on-target and off-target epochs were pooled across the control and correction conditions. Given that participants were prevented from reaching the target in the blocked condition these endpoint errors were not analysed. ERPs were created for each segment by averaging the EEG data by condition for each electrode, channel, and participant for each of the events of interest: movement onset, the corrective movement, and movement end.

Given the novelty of this paradigm for an EEG experiment, we submitted the mean ERP waveforms for each event of interest (timepoints × conditions × participant) to a spatial principal component analysis (PCA) to identify channels for further analysis (using the MATLAB PCA toolbox; Dien 2006; Dien et al. 2003; see also Krigolson and Holroyd 2006). For the waveforms locked to the target perturbations, there were 11,250 observations (250 timepoints × 3 conditions × 15 participants) for each electrode (41), for the waveforms locked to the corrective movement there were 4,500 observations (150 timepoints × 2 conditions × 15 participants) for each electrode (41), and for the waveforms locked to movement end there were 6,000 observations (200 timepoints × 2 conditions × 15 participants) for each electrode (41) in each spatial PCA. Each of the spatial PCAs was done with a Varimax rotation. For display purposes, the spatial factor loadings were plotted using custom Matlab scripts built on the open source EEGLAB toolbox (Delorme and Makeig 1994; http://sccn.ucsd.edu/eeglab).

To analyze the P300 evoked by the target perturbations, we applied a peak detection algorithm to the conditional ERPs locked to movement onset for the posterior channel identified by the spatial PCA (i.e., the posterior channel with maximal spatial loadings). For each condition and participant, P300 amplitude was identified by finding the maximum positive value at this channel between 200 and 500 ms following movement onset; P300 latency corresponded to the time of maximum amplitude. The onset of the P300 was obtained using a stepwise procedure in which a 40 ms window of data was averaged for each sample (i.e., ±20 ms) for each condition. The resulting values for the correction and blocked conditions were then tested against the control condition with a series of two tailed t-tests against zero (Rodriguez-Fornells et al. 2002). The onset of the difference in the ERPs between the correction and blocked conditions was defined as the first time point at which five consecutive t-tests showed a significant difference ($P < 0.05$).[2]

To analyze the corrective movements and movement end, the ERN was associated with the channel at which the loadings for the frontal-central spatial factor were maximal. For the blocked corrective movements, we created difference waves by subtracting the ERPs for correction trials from the ERPs for blocked trials. Likewise, for the endpoint errors, we created difference waves by subtracting the ERPs for on-target trials from the ERPs for off-target trials. This difference-wave approach isolates the ERN by removing other ERP components that are equivalent across conditions (Holroyd and Krigolson 2007; Holroyd et al. 2004; Krigolson and Holroyd 2006, 2007a, b). For both comparisons, the ERN was identified as the maximum negative peak in the difference waves from 0 to 400 ms post-event (blocked correction, movement end), for each condition and participant.

Behavioural and ERP data were submitted to repeated-measures ANOVA and paired samples *t* tests. An alpha level of 0.05 was used to interpret all omnibus tests.

Results

Behavioural data

Neither reaction time, $F(2,28) = 0.18$, $P > 0.05$, nor movement time, $F(2,28) = 1.84$, $P > 0.05$, were found to differ between the experimental conditions. An analysis of CE_h and CE_v indicated main effects for experimental condition (CE_h: $F(2,28) = 19.95$, $P < 0.001$, CE_v: $F(2,28) = 9.41$, $P < 0.001$).[3] Post-hoc examination of the main effect in the horizontal axis (CE_h) revealed that participants were more accurate on control compared to correction trials ($t(14) = 4.99$, $P < 0.001$) and blocked trials ($t(14) = 4.93$,

[2] Note that due to the inherent difficulties in single trial ERP analysis we were unable to get an onset value of the P300 for each trial, or for that matter even for individual subjects, and so could not compare this quantity with single-trial acceleration values.

[3] The constant and variable error values reported here for the blocked condition reflect the final endpoint of the participants' stylus. As the cursor was locked to the horizontal axis in this condition, the vertical error associated with the final cursor location was simply the distance from the midline to the target. Additionally, as there was no variation in the vertical cursor position the variable error of the cursor was always zero.

$P < 0.001$) (the latter two conditions were found not to differ: CE_h ($t(14) = -0.74$, $P > 0.05$). Decomposition of the main effect for CE_y revealed that participants overshot the target in the vertical axis (CE_v) on correction trials ($t(14) = -2.24$, $P < 0.05$) and blocked trials ($t(14) = -3.23$, $P < 0.01$). There was no effect of variable error in the horizontal direction (VE_h), $F(2,28) = 2.02$, $P > 0.05$. In contrast, a main effect of variable error in the vertical direction (VE_v), $F(2,28) = 32.59$, $P < 0.001$, indicated that control trials were less variable than correction ($t(14) = -6.42$, $P < 0.001$) and blocked ($t(14) = -5.38$, $P < 0.001$) trials, which in turn did not differ from each other ($t(14) = 1.25$, $P < 0.05$) (see Table 1 for mean and standard error values).

An onset analysis was conducted to compare the vertical acceleration profiles of the control, correction, and blocked experimental conditions. The results of this analysis indicated that the vertical acceleration values for the correction condition differed from the control condition 220 ms following the target perturbation, and that the vertical acceleration values for the blocked condition also differed from the control condition 220 ms following the target perturbation (P's < 0.05; see Fig. 1).

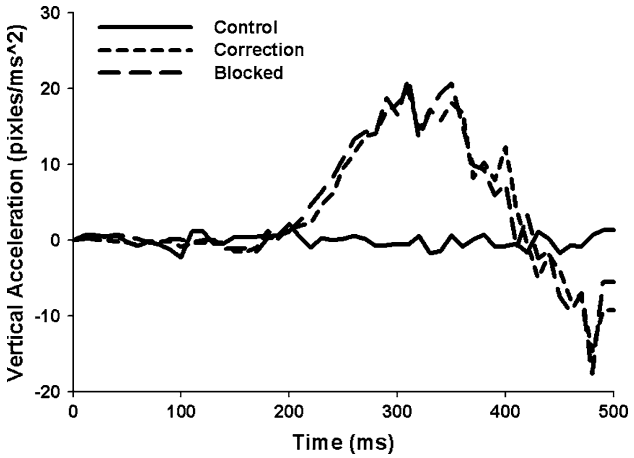

Fig. 1 Instantaneous acceleration in the *vertical axis* for the first 500 ms of the reaching trajectory

Electrophysiological data

The target perturbation. To examine the effect of the target perturbation on behaviour, a spatial PCA was conducted on the ERP waveforms averaged relative to movement onset. The spatial PCA yielded a primary spatial factor that loaded maximally at channel Pz and that accounted for 26.3% of the spatial variance (Fig. 2a). These results are consistent with the spatial distribution of the P300 (Dien et al. 2003, 2004; Donchin and Coles 1988; Spencer et al. 2001). To examine this component more directly, we conducted a peak analysis on the data recorded at channel Pz for each condition. The peak analysis yielded a main effect for condition, $F(2,28) = 18.89$, $P < 0.001$, indicating a larger P300 amplitude for trials with target perturbations (correction: $t(14) = -4.81$, $P < 0.001$; blocked: $t(14) = -4.27$, $P < 0.001$) than for the control trials (Fig. 2b). P300 amplitude for correction and blocked trials did not differ ($t(14) = 0.265$, $P > 0.05$). The peak analysis also indicated that the latencies of the P300 for control condition (417 ms), the correction condition (362 ms) and the blocked condition (341 ms). The onset analysis of the P300 revealed that the correction and blocked waveforms began to differ from the control waveforms at 256 and 240 ms, respectively (both P's < 0.05).

The corrective movement. The results of a previous study (Krigolson and Holroyd 2007a) found that blocking participants' corrective movements during a joystick aiming task elicited an ERN, a result we predicted we would replicate in the present experiment. To test this prediction we submitted the two ERPs averaged to the time of onset of the corrective movement to a spatial PCA. As above, the spatial PCA yielded a factor with loadings that were consistent with the scalp distribution of the P300 (maximal spatial loadings for channel Pz), but did not yield a factor with loadings that were consistent with the scalp distribution of the ERN (maximal spatial loadings at or near channel FCz). Furthermore, we investigated the presence of the ERN directly by applying a peak detection analysis to the difference waves (recorded at channel FCz) derived by subtracting the ERP on correction trials from the ERP on blocked

Table 1 Reaction time, movement time, constant error (horizontal and vertical axes), and variable error (horizontal and vertical axes) for control, correction, and blocked aiming movements

Condition	Reaction time (ms)	Movement time (ms)	Horizontal axis		Vertical axis	
			Constant error (pixels)	Variable error (pixels)	Constant error (pixels)	Variable error (pixels)
Control	234 ± 18	540 ± 8	4 ± 4	49 ± 2	−7 ± 2	34 ± 2
Correction	236 ± 19	539 ± 7	−12 ± 5	49 ± 3	4 ± 6	80 ± 8
Blocked	236 ± 19	543 ± 8	−10 ± 4	52 ± 3	14 ± 8	77 ± 9

Also reported is the standard error of the mean for each score

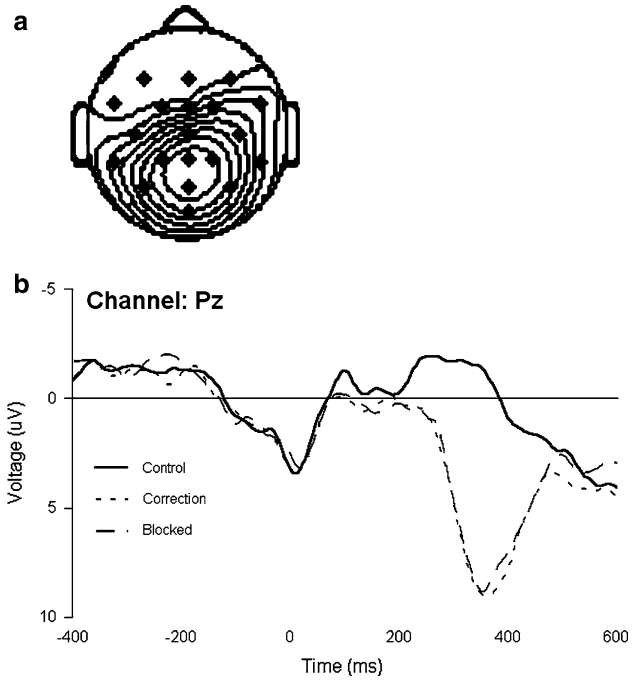

Fig. 2 Movement onset: **a** Spatial PCA factor loadings projected onto the surface of the human head for the posterior factor. **b** Averaged ERPs recorded at channel Pz for the control, correction, and blocked conditions. Note that zero ms corresponds to movement onset. Negatives voltages are plotted up by convention

trials (Fig. 3). The result of this analysis was not significant ($P > 0.05$). Together, these results indicate that, contrary to our prediction, blocking the corrective movement did not elicit an ERN at the time of the correction.

Movement end. Finally, to analyse movement end we submitted the on-target and off-target ERPs to a spatial PCA. The spatial PCA yielded a primary factor with loadings that were maximal at channel FCz (accounting for 41% of the spatial variance; Fig. 4a). A peak analysis of the difference waves (off-target minus on-target) associated with channel FCz revealed the presence of an ERN-like ERP component, $t(14) = 5.54$, $P < 0.001$, at a latency of 268 ms (Fig. 4c). The spatial PCA also yielded a second factor with loadings that were maximal at channel Pz (Fig. 4b). A peak analysis of the difference waves (off-target minus on-target) associated with channel Pz revealed a negative-going deflection in the ERP that peaked at 362 ms, $t(14) = -7.90$, $P < 0.001$ (Fig. 4d). A direct comparison of these two peaks revealed that the negative peak recorded at channel Pz occurred significantly later than the negative peak recorded at channel FCz, $t(14) = -3.12$. The amplitudes of both peaks were similar (−6.5 vs. −6.9 μV, $t(14) = 0.42$, $P > 0.05$).

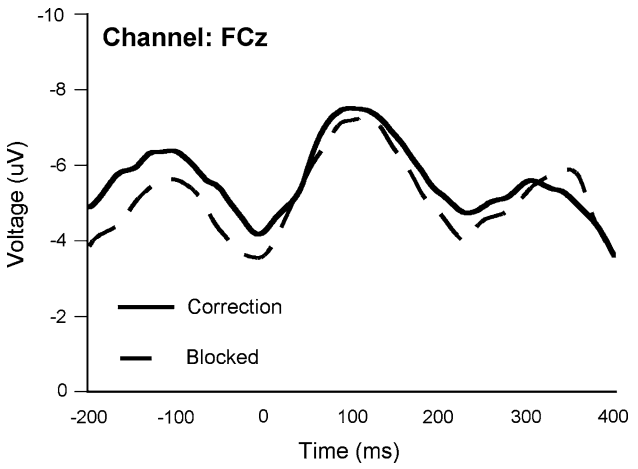

Fig. 3 Movement correction: Averaged ERPs for correction and blocked trials recorded at channel FCz. Zero millisecond indicates the average point in time at which a corrective movement was attempted. Negatives voltages are plotted up by convention

Discussion

In the present experiment we examined medial-frontal and parietal error-related ERP components during performance of a manual aiming task. Specifically, we sought to examine the ERP components elicited by target and outcome errors. In line with the hierarchical error processing hypothesis, we predicted that target errors would elicit an ERP component distributed over parietal areas of the scalp (i.e., a P300). In an important extension of earlier work, we sought to compare the timing of the P300 with the timing of corrective movements associated with the target errors in order to clarify the relationship between the P300 and online control processes. In the present study we also predicted that outcome errors brought about by blocking the corrective movement and/or by missing the movement target would elicit an ERN.

Target errors

Target errors brought about by a sudden change in the movement environment appear to be mediated within PPC (Desmurget et al. 1999, 2001; Diedrichsen et al. 2005; Gréa et al. 2002; Pisella et al. 2000). Previously, it was demonstrated that target errors in a joystick aiming task elicited a P300: a finding that was interpreted to reflect an underlying role of the P300 in the online control process (Krigolson and Holroyd 2007a). In the present study we sought to clarify the relationship of the P300 relative to online control processes by comparing the timing of this ERP component relative to the timing of kinematic changes associated with mediation of target errors. We predicted that if the P300 reflected the evaluation and correction of target errors, then it should occur before kinematic changes indicative of online control (i.e., changes in vertical acceleration).

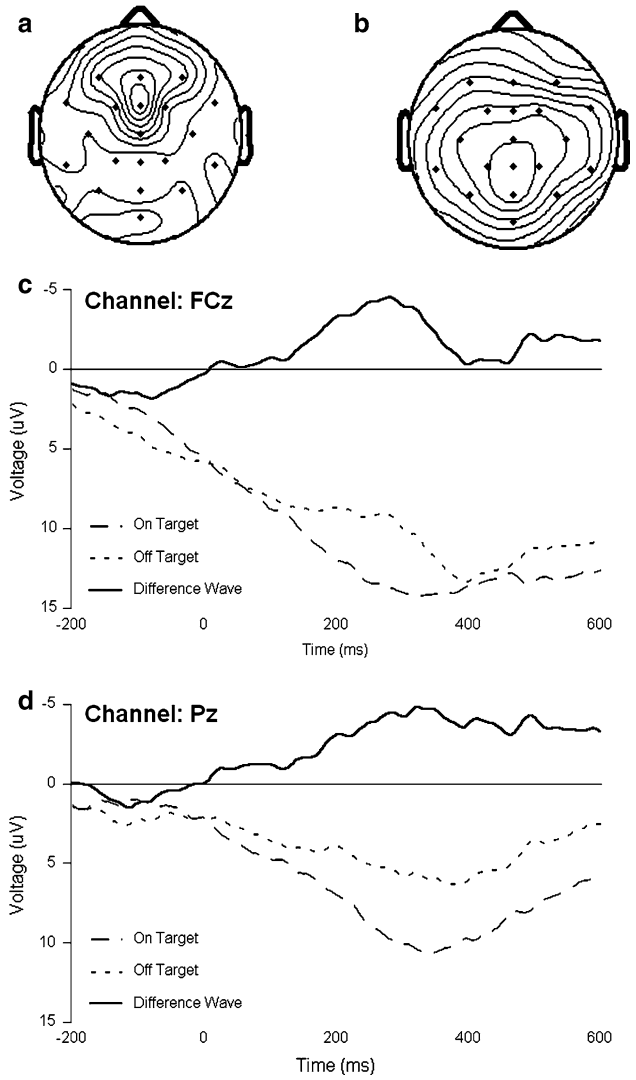

Fig. 4 Movement end: **a** Spatial PCA factor loadings projected onto the surface of the human head for the frontal-central factor. **b** Spatial PCA factor loadings projected onto the surface of the human head for the posterior factor. **c** Averaged ERPs recorded at channel FCz for on target and off target performance and the associated difference wave. **d** Averaged ERPs recorded at channel Pz for on target and off target performance and the associated difference wave. Note that zero millisecond corresponds to movement end. Negatives voltages are plotted up by convention

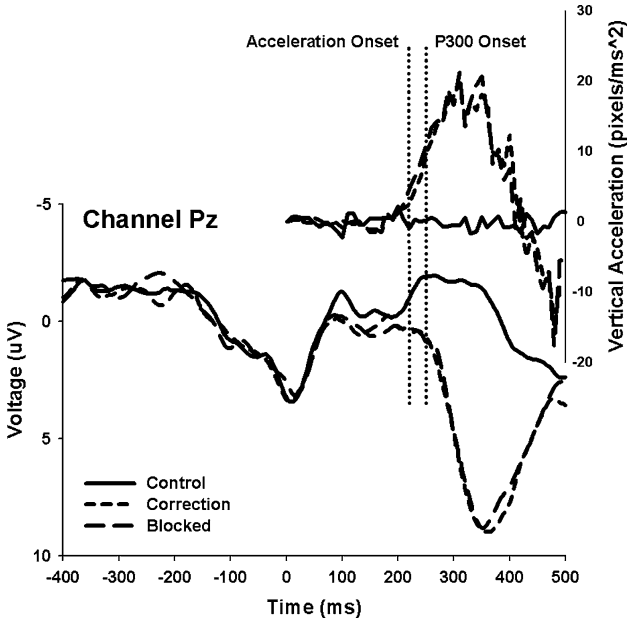

Fig. 5 A comparison of the instantaneous vertical acceleration profiles and the conditional ERPs associated with the target perturbation. Note that any amplitude scaling similarities are coincidental

Conversely, if the P300 came after behavioural changes associated with the online control of movement then it cannot have played a role in the evaluation of target errors. In line with previous results (Krigolson and Holroyd), in the present study we found that target errors elicited a P300. Importantly, the onset of the P300 in the correction and blocked conditions occurred following the onset of changes in vertical acceleration associated with the online control of movement (onset of P300 $\approx$ 250 ms, onset of changes in vertical acceleration 220 ms following the target perturbations; see Fig. 5). In other words, the onset of the P300 occurred after the point in time when participants began to adjust their motor output to accommodate the target perturbation. It is also worth noting that the behavioural modifications that began approximately 220 ms occurred well in advance of the peak latency of the P300 in the correction (362 ms) and blocked (341 ms) conditions.

The P300 is characterized by a parietal scalp distribution and appears to be generated at or near the temporal-parietal junction (Calhoun et al. 2006; Halgren et al. 1995; Kiss et al. 1989). This brain area lies adjacent to PPC, and thus seems consistent with a role for this area in the evaluation and correction of target errors (Desmurget et al. 1999, 2001; Desmurget and Grafton 2000; Diedrichsen et al. 2005; Gréa et al. 2002; Pisella et al. 2000—but see also Rizzolatti and Matelli 2003). Further, a recent theory holds that the P300 is produced by the impact of phasic activity of the LC-NE system on posterior cortex (Nieuwenhuis et al. 2005b). Importantly, phasic release of norepinephrine by the LC-NE system is thought to facilitate rapid decision making by increasing the gain of cortical neurons (Nieuwenhuis et al. 2005b; Usher et al. 1999). We previously speculated that phasic LC-NE activity should precede and facilitate the rapid changes in motor behaviour brought about by target errors (Krigolson and Holroyd 2007a). If so, and if the impact of the LC-NE system on cortex produces the P300, then the onset of the P300 should occur before the movement corrections associated with the accommodation of target errors. The timing of the P300 relative to the corrective movement in the present experiment suggests the P300 elicited here does not reflect decision related activity of the LC-NE system. With that said, assuming the LC-NE

system is responsible for generation of the P300 component (Nieuwenhuis et al. 2005b), the result of the present study are compatible with LC-NE theories which suggest that phasic activity of this system plays a role in learning. Specifically, it has been recently suggested that phasic increases in NE are elicited by unexpected task relevant changes in the environment, and that this signal is used to optimize the learning process (i.e., improve the quality of subsequent motor output) (Dayan and Yu 2006; Yu and Dayan 2005).

In contrast to the LC-NE theory of the P300, the "context updating" hypothesis holds that the P300 is elicited by the updating of an internal model of the environment in response to new, task-relevant information (Donchin and Coles 1988). In terms of motor control, it is important that movement planning and control processes have access to an accurate representation of the movement environment in order to facilitate movement accuracy. For instance, during performance of a target perturbation aiming task participants can use information relating the frequency and location of target jumps to minimize endpoint error. In a similar vein, recent computational accounts of motor control posit that predictive forward models are used to optimize movement accuracy (i.e., Desmurget and Grafton 2000; Wolpert and Ghahramani 2000). However, for a predictive motor control system function optimally, it is essential that the system have access to an accurate internal model of the movement environment in addition to an efference copy of the current motor command. We suggest that the P300 in the present study reflects an updating of an internal model of the movement environment. In other words, the P300 reflects the updating of an internal model of the frequency and/or location of target jumps. Importantly, this information can be used in a forward manner to improve the accuracy of subsequent motor output. This view seems consistent with some of the original motivation for the context updating hypothesis, which is that because the P300 can occur following the response, the neural process which generates it cannot be directly involved in the application of stimulus-response mappings (Donchin and Coles 1988).

The results of present study are also in line with the hierarchical error processing hypothesis. Although the target errors that occurred in the present study required participants to amend their ongoing motor plans, the participants could not know in advance of movement execution whether or not an explicit movement correction would be required and whether that corrective control would be successful. As such, the target errors constituted low-level motor errors. Supporting this conclusion, we found that although the target errors elicited a P300 component, they did not elicit an ERN. This suggests that medial-frontal cortex did not process target errors as high-level errors.

Outcome errors

On the basis of the results of a previous study (Krigolson and Holroyd 2007a), here we predicted that the blocking of corrective movements would elicit an ERN. In the present experiment blocking participants' corrective movements caused an outcome error to occur as the goal of reaching the target was made unattainable. However, and contrary to the above prediction, in the present experiment we found that blocked trials did not elicit an ERN. One explanation for this result stems from the similarity between the correction and blocked trials in terms of movement time, endpoint error, variable error, and acceleration profiles (see Fig. 1; Table 1). These kinematic data indicate that nearly all physical aspects of the movement were identical on correction and blocked trials. Indeed, the only apparent difference between these conditions was that on the blocked trials the participants' cursor did not respond to their corrective movements. Thus, the medial-frontal error processing system received conflicting input: visual feedback indicating a failure of a system goal, and kinaesthetic feedback indicating correct performance. This result suggests that either error processing within the basal ganglia—ACC system is inhibited when conflicting feedback is received, or alternatively that this system places a priority on kinaesthetic feedback to evaluate limb position during goal directed reaching.

Another explanation for these data may be that motor corrections in the present task were implemented ballistically (i.e., the stochastic optimized sub-movement model: Meyer et al. 1988). Previous research suggests that at least 100 ms is required to implement feedback based amendments to ongoing motor output (Desmurget and Grafton 2000; Jeannerod 1988; Paillard 1996). As such, it may be that while participants were able to adjust their initial motor command following target errors, due to the temporal constraints of the task participants were not able to evaluate the success (or failure) of the corrective movements on-line (or at least this process was delayed). In other words, once the correction was made participants did not have sufficient time to evaluate the success of the correction before movement end.[4] A further and perhaps more simple explanation

[4] It is worth noting that a much slower ERN may have been elicited following the corrective movements as participants may have been slow to detect the error in the presence of conflicting feedback. Indeed, the reinforcement learning theory of the ERN would predict this—an ERN should be elicited at the earliest indicator that events are worse than expected (Holroyd and Coles 2002). If participants were not able to evaluate the response itself, then feedback (whenever it is determined) would elicit an ERN. However, in this case participants may have detected the error at different times during the movement trajectory. Given the methodology of the present experiment it is not possible to ascertain whether such temporal instability in fact occurred.

of our results is an extension of this idea—perhaps the medial-frontal system does not evaluate the success or failure of rapid aiming movements until the movement is completed. As such, the blocked corrective movement was not evaluated as an outcome error and did not elicit an ERN as the movement was not completed at that point in time.

Assuming that a participants' movement is not blocked, then the earliest indicator of whether or not an outcome error has occurred is the evaluation of endpoint error. Thus, in line with the hierarchical error processing theory we predicted that in the control and correction conditions missing the target reflected an outcome error and should elicit an ERN. Confirming this hypothesis, we observed a negative deflection in the ERP with a scalp topography and latency consistent with the fERN (Holroyd and Coles 2002; Holroyd et al. 2005; Miltner et al. 1997). This result provides further support for the hypothesis that high-level motor errors are evaluated by a reinforcement learning system comprised of the basal ganglia and ACC, and that this information is used for the purpose of improving subsequent motor output (Krigolson and Holroyd 2006, 2007a, b; see also Holroyd and Coles 2002). Interestingly, we also observed a negative-going ERP component distributed over posterior areas of the scalp which temporally lagged the ERN following off-target trials. We have previously speculated that this ERP component may reflect a training signal sent from the frontal system to the posterior system to modify future motor behaviour (Krigolson and Holroyd 2006, 2007a, b).

Conclusion

To the best of our knowledge, the present study is the first to use ERPs to analyse the neural basis of error evaluation in a discrete manual aiming task. We found that target errors evoked a P300, an ERP component with a parietal scalp distribution that has been associated both with context updating (Donchin and Coles 1988) and decision related activity of the LC-NE system (Nieuwenhuis et al. 2005a, b). The results of the present study suggest that although the scalp distribution and proposed neural generators of the P300 are consistent with neural regions believed to evaluate and correct target errors (i.e., PPC), it does not appear that the P300 is directly related to the online control of movement. Rather, we propose the P300 reflects the updating of an internal model of the movement environment that is used to help plan and execute future motor output. In addition, our results provide further support for the hypothesis that the basal ganglia—ACC system is sensitive to high-level motor errors. Specifically, we found that the basal ganglia—ACC system was sensitive to outcome errors (as evidenced by target misses, which elicited the ERN) but not by target errors (as evidenced by the target perturbations, which did not elicit the ERN). Taken together, the results of the present experiment support the hypothesis that error processing during motor control is hierarchical in nature (Krigolson and Holroyd 2006, 2007a, b).

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