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Predictive information and error processing: The role of medial-frontal cortex during motor control

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Abstract

We have recently provided evidence that an error-related negativity (ERN), an ERP component generated within medial-frontal cortex, is elicited by errors made during the performance of a continuous tracking task (O.E. Krigolson & C.B. Holroyd, 2006). In the present study we conducted two experiments to investigate the ability of the medial-frontal error system to evaluate predictive error information. In two experiments participants used a joystick to perform a computer-based continuous tracking task in which some tracking errors were inevitable. In both experiments, half of these errors were preceded by a predictive cue. The results of both experiments indicated that an ERN-like waveform was elicited by tracking errors. Furthermore, in both experiments the predicted error waveforms had an earlier peak latency than the unpredicted error waveforms. These results demonstrate that the medial-frontal error system can evaluate predictive error information.

Descriptors: Error processing, ERN, ERP, Forward model, Reinforcement learning

Our ability to evaluate predictive information helps us to avoid making errors. For example, it is often necessary to correct for errors in motor execution while an action is in progress (i.e., trajectory adjustments; Chua & Elliott, 1993; Elliott, Helsen, & Chua, 2001; Goodale, Pelisson, & Prablanc, 1986; Khan, Lawrence, Franks, & Buckholz, 2004; Woodworth, 1899). The delays inherent in feedback processing (Desmurget & Grafton, 2000; Jeannerod, 1988; Paillard, 1996) suggest than such rapid online motor control mechanisms may operate in a predictive manner (i.e., a *forward model*; Desmurget & Grafton, 2000; Wolpert & Ghahramani, 2000). In these models, it is thought that the motor system utilizes sensory feedback and a copy of the current motor command to anticipate the future position of the limb. Thus, feedback processing delays are avoided by adjusting the movement according to the predicted position of the limb. Several lines of research suggest that the sensorimotor transformations necessary for this type of online motor control depend on posterior parietal cortex (PPC; Desmurget & Grafton, 2000; Desmurget et al., 1999, 2001; Grea et al., 2002) and the cerebellum (Blakemore, Frith, & Wolpert, 2001; Miall, Reckess, & Imamizu, 2001; Miall, Weir, & Stein, 1993).

In addition to this posterior error system, a second error processing system involving medial-frontal cortex appears to detect violations of "high level" system goals. Converging research has demonstrated that response errors (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993) and negative feedback (Holroyd & Coles, 2002; Miltner, Braun, & Coles, 1997; Nieuwenhuis, Holroyd, Mol, & Coles, 2004) elicit a negative deflection in the event-related brain potential (ERP). Termed the error-related negativity (ERN), this ERP component is thought to reflect the activity of a generic error-processing system (Holroyd & Coles, 2002; Holroyd, Yeung, Coles, & Cohen, 2005) involving anterior cingulate cortex (ACC; Brown & Braver, 2005; Holroyd & Coles, 2002; Holroyd, Nieuwenhuis, Mars, & Coles, 2004; but see Nieuwenhuis, Slagter, Alting von Geusau, Heslenfeld, & Holroyd, 2005). The reinforcement learning theory of the ERN (RL-ERN theory) proposes that the ERN amplitude is determined by the impact on ACC of an error signal carried by the midbrain dopamine system from the basal ganglia that indicates that ongoing events are worse than anticipated (Holroyd & Coles, 2002; for a review, see Holroyd et al., 2004).

We have recently provided evidence that the medial-frontal system is also sensitive to errors made during a continuous tracking task (Krigolson & Holroyd, 2006). Importantly, our results suggest that different levels of motor errors are processed by different brain systems. Specifically, we have proposed that "low-level" motor errors (i.e., trajectory discrepancies) are evaluated by posterior error systems whereas high-level motor errors (i.e., violations of a system goal) are evaluated by the medial-frontal system. In the present study we conducted two experiments to examine the predictive capabilities of the medial-frontal

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error system in a continuous motor task. Here, we show that, in addition to being sensitive to high-level motor errors, the medial-frontal system is also sensitive to internal and external information predicting a high-level error.

EXPERIMENT 1

The goal of Experiment 1 was to demonstrate that the medial-frontal system can process information predicting future performance during a continuous motor task. Specifically, we asked whether external information that explicitly predicted an upcoming error would elicit an ERN-like waveform in the context of a continuous tracking task. Participants performed a computer-based tracking task in which they used a joystick to maintain a cursor centered between two moving barriers that moved slowly through a base pattern (cf. Krigolson & Holroyd, 2006). As in our previous study, at random times during the base pattern the barriers moved unpredictably and rapidly to the left or right causing an unpredicted error. However, in the present experiment the unpredicted tracking errors were counterbalanced with an equivalent number of tracking errors that were predicted by the appearance of a visual cue. Based on our previous findings, we predicted that an ERN would be elicited by the unpredicted tracking errors. Furthermore, we predicted that an ERN with an earlier onset and peak latency would be elicited by the visual cue predicting an upcoming tracking error.

Methods

Participants

Fifteen right-handed undergraduate students (6 male, 9 female) with no known neurological impairments and with normal or corrected-to-normal vision participated in the experiment. All of the participants were volunteers who received extra credit in a first-year psychology course for their participation in addition to a monetary bonus associated with their performance of the experimental task (see below). 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

The participants were seated comfortably in front of a 17-in. flat screen computer monitor (1024 × 768 pixel display, 75 Hz refresh rate, LG L1732TX-S) in an electromagnetically shielded, soundproof booth and used a standard USB joystick to perform a computerized tracking task (cf. Krigolson & Holroyd, 2006) (written in MATLAB [Version 7.1, Mathworks, Natick, U.S.A.] using the Psychophysics Toolbox extension [Brainard, 1997]; the computer code can be downloaded from the Web site (http://web.uvic.ca/psyc/braincoglab/lab_research.html) of the Brain and Cognition Laboratory at the University of Victoria, Victoria, Canada). The display consisted of a red cursor (20 pixels × 20 pixels) initially positioned between two white barriers (60 pixels × 20 pixels) presented on a high contrast black background. The base tracking pattern consisted of back-and-forth barrier movement along a horizontal axis positioned two thirds of the way down the vertical axis of the monitor at a speed of 0.4 pixels/ms (henceforth p/ms). During the base tracking pattern the barriers paused for 500 ms when they passed through the middle of the screen and when they reversed direction as they approached the edges of the screen. Using their right hand, participants moved the cursor to the left and right with corresponding left and right displacements of the joystick in order to keep their cursor centered between the barriers. Joystick control in each direction was segmented such that four cursor speeds to the left and right were possible (0.0 p/ms, 0.4 p/ms, 0.8 p/ms, 1.2 p/ms), depending on the degree of joystick displacement in either direction.

As mentioned above, the participants' task entailed preventing the cursor from contacting the two barriers. An error was defined as contact between the cursor and either of the barriers, both of which turned red during the period of contact. During most of the experiment, participants navigated the base tracking pattern. In addition, participants also encountered rapid barrier movements that occurred during a randomly selected subset of the middle pauses of the base tracking pattern (see below). One half of the rapid barrier movements were unpredicted such that the barriers moved rapidly and randomly either to the right or to the left of the screen. The rapid barrier speed (2.0 p/ms), together with the unpredictable occurrence and direction of these movements, ensured that participants always contacted one of the barriers during these events. As such, these events constituted *unpredicted error trials*. For comparison purposes, participants also encountered an equivalent number of rapid barrier movements during which the computer program maintained the position of the cursor between the barriers for the duration of the event. These events thus constituted *unpredicted correct trials* (see Figures S1A and S1B). The duration of each unpredicted correct trial was identical to the duration of the last encountered unpredicted error trial. Note that if the first event encountered was randomly determined to be an unpredicted correct trial, then a duration of 200 ms was assigned (as derived from the average time to barrier contact from pilot research and a previous study utilizing this paradigm).

To provide participants with predictive information about the upcoming events, half of the rapid barrier movements were preceded by a color-coded stimulus. Specifically, in these instances the rapid barrier movements were preceded for 500 ms by a visual stimulus that indicated the occurrence and type of the subsequent barrier movement. Because of this manipulation, the subsequent events constituted *predicted error trials* and *predicted correct trials*. Each predictive stimulus consisted of a blue or green square (50 × 50 pixels) centered horizontally and presented above the barriers on the computer monitor (150 pixels above the cursor movement axis: 6° of angle; see Figures S1A and S1B). The predictive stimulus colors associated with a particular condition were randomly counterbalanced between participants. In all other respects the unpredicted and predicted trials were identical. The durations of the predicted correct trials were time matched in duration to the predicted error trials in the same manner outlined above for the unpredicted error and correct trials.

In total, participants experienced four types of rapid barrier movements, two of which were predicted (predicted error and predicted correct trials) and two of which were unpredicted (unpredicted error and unpredicted correct trials). The frequency of the rapid barrier movements (40% of the middle pauses) was such that participants encountered six rapid barrier movements per minute on average (1.5 unpredicted errors, 1.5 unpredicted corrects, 1.5 predicted errors, 1.5 predicted corrects). In total, participants encountered approximately 360 events during the experiment (90 events per condition on average). Importantly,

participants were not instructed as to the meaning of the predictive cues. Instead, there were told that at some points in the tracking pattern they would see signs appear above the barriers, and that these signs might provide some useful information to help with tracking performance. Thus, to utilize the predictive information they had first to learn the color–event relationship during the course of the experiment. At the end of the experiment participants were tested to ensure that they had learned the mappings of the signs.

The experimental phase consisted of 15 four-minute blocks, between which participants relaxed during self-paced rest periods. The tracking task was explained to participants before the experiment began and participants were given 1 four-minute practice block that did not contain any rapid barrier movements to practice the base tracking task. As motivation, participants began the experiment with a $15.00 CAN performance bonus and were instructed that each time they committed a tracking error they would lose $0.10. During each rest block participants were told of the current balance of their performance bonus and at the end of the experiment were given the amount of money owed to them.

Behavioral Analysis

The tracking program recorded the start time of each of the rapid barrier movements and the time of the barrier contact for error trials. Participants completed a short questionnaire upon completion of the experiment.

Data Acquisition

The electroencephalogram (EEG) was recorded from 37 electrode locations using BrainVision Recorder software (Version 1.3, Brainproducts, GmbH, Munich, Germany). The electrodes were mounted in a fitted cap with a standard 10-20 layout and were referenced to a common ground. The vertical and horizontal electrooculograms were recorded from electrodes placed above and below the right eye and on the outer canthi of the left and right eyes, respectively. Electrode impedances were kept below 20 kΩ. The EEG data were sampled at 250 Hz in addition to being amplified (Quick Amp, Brainproducts, GmbH, Munich, Germany) and filtered through a passband of 0.017–67.5 Hz (90 dB octave roll off).

Data Analysis

The EEG data were filtered through a (0.1–20 Hz passband) phase-shift-free Butterworth filter and re-referenced to linked mastoids. Ocular artifacts were removed using the algorithm described by Gratton, Coles, and Donchin (1983). Trials in which the change in voltage at any channel exceeded 50 μV per sampling point were also discarded. In total, less than 5% of the data were discarded.

For each rapid barrier movement (both unpredicted and predicted), a 1000-ms epoch of data was extracted from the continuous EEG, extending from 700 ms before barrier contact on error trials, and from the equivalent matched point in time on correct trials, to 300 ms after the event. The data were baseline corrected by subtracting from each sample the average activity recorded at that electrode during a -700 to -500-ms window at the start of the epoch. ERPs were created by averaging the EEG data by condition (unpredicted errors, unpredicted corrects, predicted errors, predicted corrects) for each electrode and participant. Difference waves were then created by subtracting the correct trial ERPs from the corresponding error trial ERPs for both the unpredicted and predicted conditions for each electrode and participant. The amplitude and latency of each difference wave was obtained by identifying its maximum deflection within a 600-ms window ($\pm$ 300 ms around barrier contact). The peak positive and negative amplitudes of this deflection were statistically tested against zero using a one-sample t test and an alpha of .05. The onset of the difference waves was obtained using a stepwise procedure in which a 40-ms window of data was averaged for each sample (i.e., $\pm$ 20 ms), with the resulting values subjected to a series of one-tailed t tests against zero (Rodriguez-Fornells, Kurzbach, & Munte, 2002). The onset latency of the difference wave was defined as the first time point at which five consecutive t tests showed a significant difference ($p < .05$). Note that the rationale behind creating difference waves was to remove any effects induced purely by the sudden barrier movements and/or the appearance of the predictive cues.

Spatiotemporal Principal Component Analysis

The ERP data were submitted to a spatiotemporal principal component analysis (PCA) to identify electrophysiological activity that covaried across electrodes and time (analyzed using the MATLAB PCA toolbox; Dien, 2002; Dien, Spencer, & Donchin, 2003; cf. Krigolson & Holroyd, 2006). First, spatial factor loadings were obtained by submitting to a PCA the observations (500 ERP samples: 250 time points $\times$ 2 difference waves) for each participant and electrode (Varimax rotation, no Kaiser correction), for both the predicted and unpredicted conditions. Next, the spatial factor scores associated with the resulting first and second spatial factors were submitted separately to temporal PCAs. For display purposes, the spatial factor loadings were plotted using custom Matlab scripts built on the open source EEGLAB toolbox (Delorme & Makeig, 2004; http://sccn.ucsd.edu/eeglab).

Results

Behavioral Results

The unpredicted error and correct trials had similar mean durations, 179 ms and 180 ms, respectively. The mean durations of the predicted error and correct trials were also similar (677 ms, 678 ms). Note that the durations of the predicted trials were greater than that of the unpredicted trials because they included the 500 ms associated with the appearance of the predictive cue.

Electrophysiological Results

First, we conducted a spatial PCA on the ERP data (see Figure 1A,B) to identify patterns of data that covaried spatially. The first factor yielded by this analysis exhibited loadings that were maximal over frontal-central regions of the scalp (for channels FCz, FC1, FC2, all loadings were over .96) and accounted for 54.3% of the total variance (see Figure 1C). Importantly, this distribution is consistent with that of the ERN (Gehring et al., 1993; Holroyd et al., 2004; Miltner et al., 1997; cf. Krigolson & Holroyd, 2006). To investigate the dynamics of this factor we conducted a temporal PCA on the associated factor scores. This analysis yielded two primary epochs, the first occurring from 60 ms to 152 ms following barrier contact (accounting for 49.8% of the variance) and the second occurring from -140 ms to -44 ms preceding barrier contact (accounting for 20.2% of the variance).

Given that the factor loadings for the first spatial factor were maximal at channel FCz, we focused our analysis on the

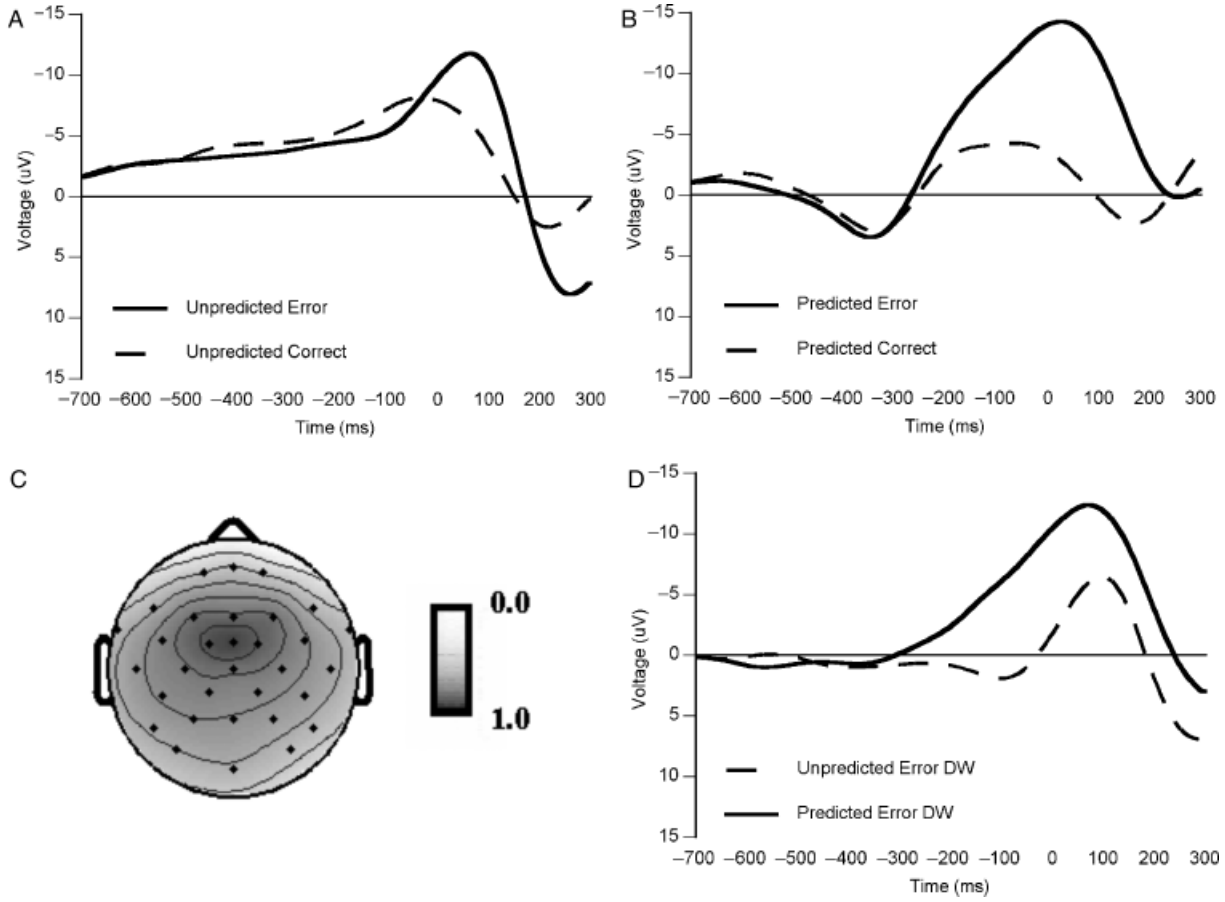

Figure 1. Experiment 1. A: Averaged ERP waveforms recorded at channel FCz for unpredicted error and correct trials. B: Averaged ERP waveforms recorded at channel FCz for predicted error and correct trials. C: Spatial PCA factor loadings projected onto the surface of the human head for the frontal-central factor, accounting for 54.3% of the total variance. D: Averaged ERP difference waves associated with channel FCz for the unpredicted and predicted errors. Zero milliseconds corresponds to the time of barrier contact on error trials and to a matched point in time on correct trials. Note that negative voltages are plotted up by convention.

difference waveforms (see above) associated with unpredicted and predicted tracking errors (Figure 1D). For the unpredicted trials, a negative deflection in the difference-wave reached maximum amplitude 100 ms following barrier contact, $t(14) = -7.68$, $p < .001$. For the predicted trials, a negative deflection in the difference wave reached maximum amplitude 66 ms following barrier contact, $t(14) = -9.04$, $p < .001$. A comparison of the latencies of these deflections indicated that the peak difference associated with the predicted trials occurred prior to that of the unpredicted trials (66 ms vs. 100 ms), $t(14) = 3.32$, $p < .01$. Unexpectedly, the deflection associated with the predicted trials was significantly larger than that of the unpredicted trials, – 14.1 μV versus – 8.7 μV, $t(14) = 4.93$, $p < .001$ (Figure 1D). In addition, we conducted an onset analysis on the difference waves. This analysis revealed that the onset of the deflection associated with the unpredicted trials occurred 12 ms prior to barrier contact, whereas the onset of the deflection associated with the predicted trials occurred 192 ms preceding barrier contact. Importantly, the latencies and onsets of the predicted and unpredicted deflections coincide with the epochs identified by the temporal PCA for the first spatial factor. Specifically, the first epoch (occurring from 60 ms to 152 ms following barrier contact) includes the peaks of both the unpredicted and predicted difference waves whereas the second epoch (occurring from – 140 ms to – 44 ms before barrier contact) appears to be associated with the onset of the predicted difference wave.

Recall that participants were not told the meaning of the predictive visual cues. As such, we anticipated that as participants inferred the meaning of the cues during the experiment, the latency of the predicted difference wave would occur sooner on trials later in the experiment than on trials earlier in the experiment, whereas the latency of the difference wave on unpredicted trials would not change. To test this prediction we created 30 running averages (20 trials per average) for both the unpredicted and predicted difference waves (cf. Holroyd & Coles, 2002). Visual inspection of the data (Figure 2, top panel) suggests backward propagation of the latency of the difference wave over the course of the experiment for predicted trials but not for unpredicted trials. In line with this observation, a linear trend analysis conducted on the peak latencies of the predicted difference wave confirmed that the peak latencies back propagated in time during the experiment, $F(1,14) = 11.05$, $p < .01$. Conversely, there was no significant trend in the peak latencies for the unpredicted difference wave, $F(1,14) = 2.19$, $p > .05$.

The spatiotemporal PCA also yielded a second spatial factor with maximal loadings distributed over channels PO7, PO8, and Oz (loadings of .90, .93, and .93, respectively) that accounted for 21.7% of the spatial variance. A temporal PCA on the factor

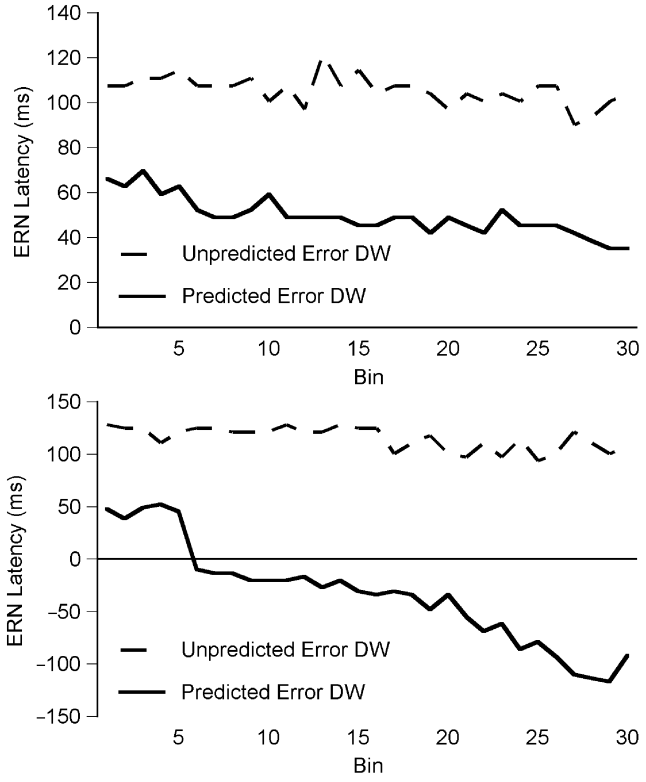

Figure 2. Top panel: Running averages for both the unpredicted and predicted ERN difference wave latencies in Experiment 1. Bottom panel: Running averages for both the unpredicted and predicted ERN difference wave latencies in Experiment 2. Running average bin size = 20 trials.

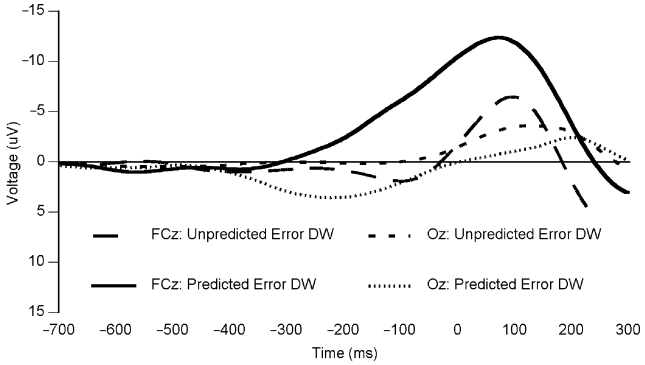

Figure 3. Experiment 1. Averaged ERP difference waves associated with channels FCz and Oz for unpredicted and predicted errors. Zero milliseconds corresponds to the time of barrier contact on error trials and to a matched point in time on correct trials. Note that negative voltages are plotted up by convention.

scores associated with this factor yielded three temporal factors, one from 24 ms to 144 ms following barrier contact (accounting for 36.0% of the total variance), one from 188 ms to 296 ms following barrier contact (accounting for 30.3% of the total variance), and one from -320 to -156 ms preceding barrier contact (accounting for 12% of the total variance). To investigate this finding further, the unpredicted and predicted difference waves associated with channel Oz were submitted to a peak analysis (Figure 3). The results of this analysis indicated that both the unpredicted (-6.2 μV) and predicted (-6.2 μV) negative peaks and their latencies (130 ms, 166 ms) were similar (peak analysis: $t[14] = 0.01$, $p > .05$, latency analysis: $t[14] = 1.54$, $p > .05$). In a previous study we found that the ERN associated with unpredicted tracking errors was followed by a posterior negativity (cf. Krigolson & Holroyd, 2006). For this reason, in the present study we compared the latencies of the posterior negativities for the unpredicted and predicted difficult corners with their respective frontal counterparts. This analysis indicated that in both cases, the frontal negativity occurred earlier than the posterior negativity (unpredicted: $t[14] = 2.44$, $p < .05$, 100 ms vs. 130 ms; predicted: $t[14] = 4.9$, $p < .001$, 66 ms vs. 166 ms; Figure 3).

EXPERIMENT 2

As in Experiment 1, the goal of Experiment 2 was to show that the medial-frontal system can evaluate predictive information within the context of a continuous tracking task. However, whereas in Experiment 1 the predictive information was explicit in nature (a visual cue), in Experiment 2 the predictive information resulted from a comparison between an internal motor command and its behavioral consequence. In Experiment 2, participants performed a continuous tracking task in which the base tracking pattern and the unpredicted error and correct trials were identical to Experiment 1. However, in contrast to Experiment 1, the predicted errors in Experiment 2 consisted of trials of the regular tracking pattern during which the control of the joystick was greatly reduced. In these instances, participants were unable to maintain the cursor positioned between the barriers and inevitably an error occurred. For comparison purposes, we sampled equivalent durations of the regular tracking pattern when participants did not make an error. Given the results of Experiment 1 and our previous work, we again predicted that the unpredicted tracking errors would elicit an ERN. Additionally, we predicted that the reductions in joystick control would elicit an ERN with an earlier latency and onset than that of the unpredicted errors.

Methods

Participants

Fifteen right-handed undergraduate students (4 male, 11 female) with no known neurological impairments and with normal or corrected-to-normal vision participated in Experiment 2. All of the participants were volunteers who received extra credit in a first-year psychology course for their participation in addition to a monetary bonus associated with their performance of the experimental task (see below). 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

The same apparatus and base tracking pattern that was used in Experiment 1 was used in Experiment 2. As in Experiment 1, on 40% of the middle pauses during the base tracking pattern participants encountered either an unpredicted error, unpredicted correct, predicted error, or predicted correct trial (see below) with half of these trials consisting of rapid barrier movements (unpredicted error and unpredicted correct trials; see above, equal

probability of each) and the other half of the trials split equally between predicted error trials and predicted correct trials. In Experiment 2, predicted error trials occurred during the base tracking pattern when the responsiveness of the joystick was suddenly reduced by 90%, such that the cursor always drifted slowly into one of the barriers. Thus, the speed of the cursor and the duration of these reduced control trials ensured that these events always resulted in predicted errors. For comparison purposes, an equivalent number of trials of the regular tracking pattern (matched in duration to the predicted error trials) were sampled. Given that participants made a minimal number of errors during the base tracking pattern, these trials constituted *predicted correct* trials (see Figure S2). Segments were only included as predicted correct trials if the participant did not commit a tracking error in the preceding period before the segment (1000 ms), during the segment, or in the preceding period after the segment (1000 ms). Note that the participants were not explicitly told that reduced control of the joystick would lead to a tracking error. Instead, participants were simply told that for brief random durations during the experiment they would find that the cursor did not respond to the joystick as it normally did. It was left to participants to realize that an error always occurred when joystick control was reduced. Participants encountered approximately 360 events during Experiment 2 (90 unpredicted errors, 90 unpredicted corrects, 90 predicted errors, 90 predicted corrects: 6 events per minute on average). In all other aspects Experiment 2 was identical to Experiment 1.

Behavioral Analysis

As in Experiment 1, the tracking program recorded the start time and the time of barrier contact for each of the unpredicted and predicted error trials. For unpredicted and predicted correct trials the start time of the trial was recorded. Participants completed a short questionnaire upon completion of the experiment.

Data Acquisition and Analysis

Data acquisition and analysis were carried out in the same manner as in Experiment 1.

Spatiotemporal Principal Component Analysis

A spatiotemporal PCA was conducted on the ERP data in the same manner as in Experiment 1.

Results

Behavioral Results

The mean durations of the unpredicted error and correct trials were similar (177 ms, 179 ms) as were the durations of the predicted error and correct trials (680 ms, 681 ms). By design, the cursor speed associated with reduced control was set such that the average duration of predicted error trials was equivalent to the duration of these events in Experiment 1. For this reason, the durations of the predicted trials were greater than those of the unpredicted trials in Experiment 2 as they were in Experiment 1.

Electrophysiological Results

The analyses conducted in Experiment 2 paralleled those in Experiment 1. First, we conducted a spatial PCA on the conditional ERP waveforms (Figure 4A,B) to identify patterns of data that covaried spatially. The first factor yielded by this analysis exhibited loadings that were maximal over front-central regions of the scalp (for channels FCz, FC1, and Cz all loadings were over .95), accounting for 47.2% of the total variance (Figure 4C). This result is consistent with the scalp distribution of the ERN (Holroyd et al., 2004; Krigolson & Holroyd, 2006). To examine the timing of this spatial factor, a temporal PCA was conducted on the factor scores associated with the first spatial factor. The temporal PCA identified two epochs, one from -264 to -16 ms (31.0% of the total variance) and one from 56 to 152 ms (26.3% of the total variance).

We also examined the difference waves associated with unpredicted and predicted tracking errors at channel FCz (Figure 4D), where the first spatial factor loadings were maximal. A peak analysis indicated that the predicted errors associated with reduced joystick control resulted in a negative deflection with an earlier latency (-32 ms) than that of the unpredicted errors (103 ms), $t(14) = 5.08$, $p < .001$. These latencies are consistent with the first two factors yielded by the temporal PCA of the factor scores associated with the first spatial factor. The peak analysis of the difference waveforms also indicated that the negative deflection of the predicted error difference wave (-7.7 μV) was larger than that of the unpredicted error difference wave (-5.5 μV), $t(14) = -7.30$, $p < .001$ (Figure 4D). Finally, an onset analysis conducted on the two difference waves indicated that the onset of the negative deflection for predicted errors was earlier than that of the negative deflection for unpredicted errors (-436 ms vs. 16 ms).

As in Experiment 1, participants were not instructed that the predictive information inevitably led to a tracking error. As such, participants had to learn this for themselves, and we predicted that the latency of the difference wave associated with the predictive errors would occur earlier as the experiment progressed. As in Experiment 1, we created 30 running averages (20 trials per average) for both the unpredicted and predicted difference waves (cf. Holroyd & Coles, 2002). Visual inspection of these running averages suggests that the latency of the negativity occurred earlier at the end of the experiment compared to the beginning of the experiment for the predicted, but not for the unpredicted, trials (Figure 2, bottom panel). This impression was confirmed by a linear trend analysis on the latencies of the predicted difference wave, which indicated that the peak latency of the predicted difference wave propagated backward in time during the experiment, $F(1,14) = 12.75$, $p < .01$. As with the first experiment, a trend analysis on the peak latencies for the unpredicted difference waves indicated that they did not change throughout the experiment, $F(1,14) = 4.17$, $p > .05$.

The second spatial factor identified by the spatial PCA accounted for 22.8% of the total variance and was distributed over posterior regions of the scalp (maximal loadings greater than .90 over channels P8, PO7, PO8, and Oz). A temporal PCA conducted on the factor scores associated with this spatial factor yielded epochs from 220 to 300 ms (28.6% of the total variance), 28 to 136 ms (26.8% of the total variance) and -348 to -88 ms (18.2% of the total variance). To investigate these results further, we conducted a peak analysis on the difference waves (both unpredicted and predicted) associated with channel Oz. This analysis indicated that the negative deflection associated with channel Oz was significantly larger in amplitude, $t(14) = 3.15$, $p < .01$, -7.4 μV versus -4.6 μV, and later in latency, $t(14) = 4.86$, $p < .001$, 159 ms versus -118 ms, for unpredicted errors relative to predicted errors. As in Experiment 1, we compared the latencies of the frontal and posterior negativities for the unpredicted and predicted errors. For the unpredicted errors the posterior negativity occurred significantly later than the frontal

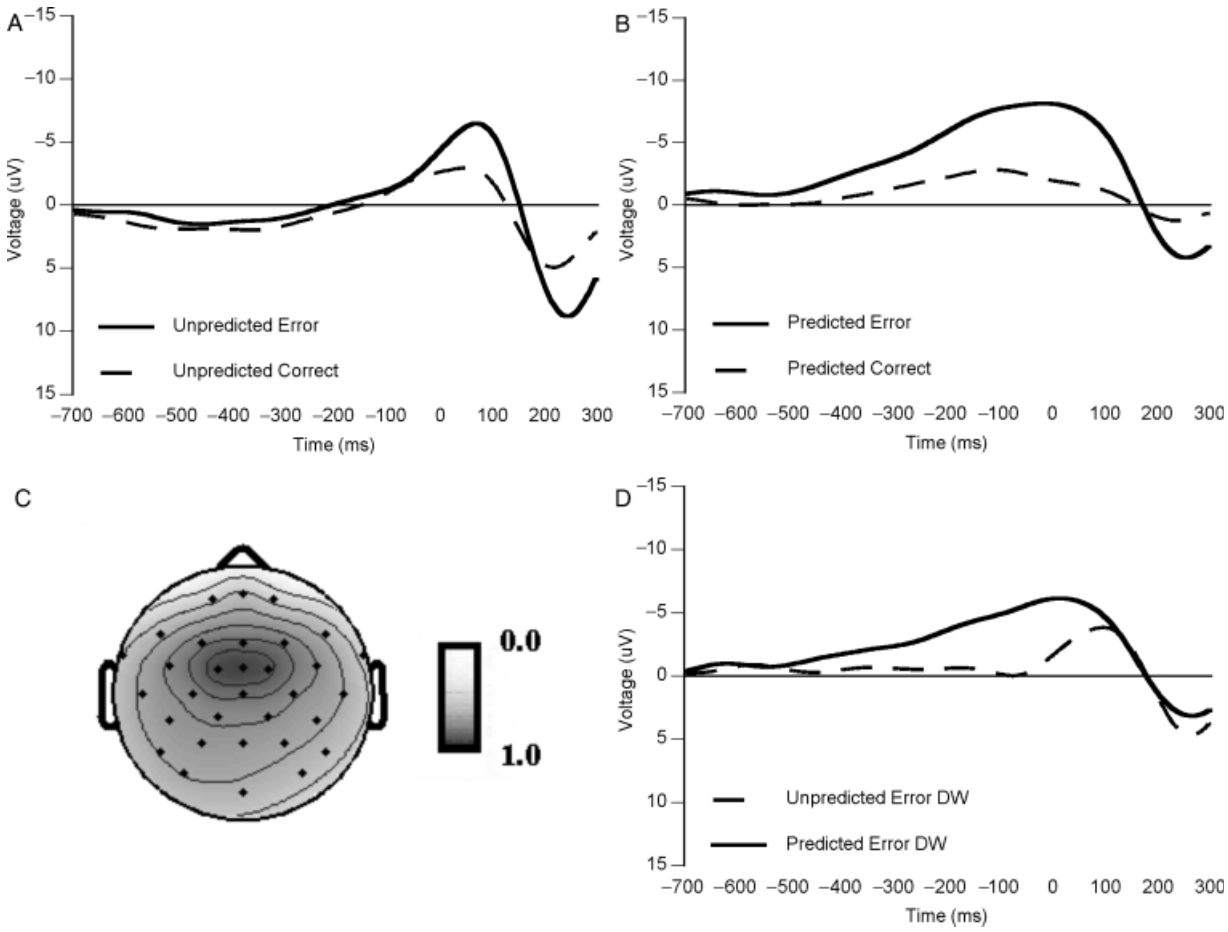

Figure 4. Experiment 2: A: Averaged ERP waveforms recorded at channel FCz for unpredicted error and correct trials. B: Averaged ERP waveforms recorded at channel FCz for predicted error and correct trials. C: Spatial PCA factor loadings projected onto the surface of the human head for the frontal-central factor, accounting for 54.3% of the total variance. D: Averaged ERP difference waves associated with channel FCz for the unpredicted and predicted errors. Zero milliseconds corresponds to the time of barrier contact on error trials and to a matched point in time on correct trials. Note that negative voltages are plotted up by convention.

negativity, $t(14) = 3.72$, $p < .001$, 103 ms versus 159 ms. Interestingly, the reverse was true for the predicted errors, with the posterior negativity occurring before the frontal negativity, $t(14) = 5.48$, $p < .001$, – 347 ms versus – 32 ms (Figure 5).

Discussion

The results of the present experiments demonstrate that external predictive visual information and predictive error comparisons between an internal motor command and related behavior elicit an ERN-like waveform. Specifically, in both experiments we found that predicted errors elicited an ERN-like waveform with an earlier latency and onset than unpredicted errors. These findings demonstrate that the medial-frontal system can process external and internal predictive cues related to subsequent motor performance. Our results also replicate our previous observation that unpredicted errors made during a continuous tracking task elicit an ERN-like waveform (cf. Krigolson & Holroyd, 2006). The results of the present experiment are consistent with the RL-ERN theory (Holroyd & Coles, 2002) and also support our hypothesis that motor errors are processed by a hierarchical system.

Previous research has demonstrated that the ERN is elicited by information indicating an upcoming undesired outcome in a trial-and-error learning task (Holroyd & Coles, 2002). Holroyd and Coles found that when stimulus–response mappings could be learned, response errors elicited the ERN. Conversely, when stimulus–response mappings could not be learned (because the feedback stimuli occurred at random), error feedback elicited the ERN. In other words, on trials with fixed stimulus–response mappings the system could predict the outcome of the trial at the time of error commission, whereas on the trials with random stimulus–response mappings the system relied on feedback stimuli to determine the outcome of the trial. In the present study we also found that predictive information indicating that an error was about to occur—derived from an external "sign" in Experiment 1 and from a comparison between an internal motor command and its consequence in Experiment 2—elicited an ERN-like waveform with an earlier latency and onset than unpredicted errors. However, in contrast to the trial-and-error learning task—in which the response ERN peaked within 100 ms following the error—in the present study the ERN-like waveform peaked much later ($\sim$500 ms) following the occurrence of the predictive information.

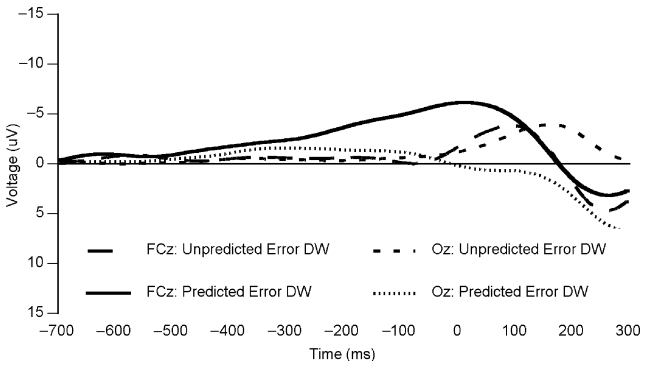

Figure 5. Experiment 2. Averaged ERP difference waves associated with channels FCz and Oz for unpredicted and predicted errors. Zero milliseconds corresponds to the time of barrier contact on error trials and to a matched point in time on correct trials. Note that negative voltages are plotted up by convention.

Because we did not tell participants the meaning of the predictive cues until after the experiment was completed, one possible explanation for this result was that the learning process was incomplete at the time the experiment ended. Thus, in Experiment 1 participants were required to learn that one of the two "signs" predicted an upcoming (and inevitable) error, and in Experiment 2 they were required to learn that joystick failure inevitably led to an error. If they failed to learn these relationships completely by the end of the experiment, then the error signal underlying the ERN-like waveform would not have fully "propagated back in time" from the time of barrier contact to the time of the predictive event. In support of this argument, we found that the peak ERN-like waveform latency in both experiments propagated backward in time during the course of the experiment (Figures 2 and S3). Also consistent with this position, a recent experiment demonstrated that predictive cues can elicit a feedback ERN when the meanings of the cues are explicitly told to the participants at the start of the task (Baker & Holroyd, 2006). Furthermore, the onset analyses in the present study revealed that the onset of the ERN-like waveform preceded the trial events, indicating that the medial-frontal system began to process the predictive information well in advance of the tracking errors.

It might be suggested that the frontal-central ERP component elicited by predictive error information is a contingent negative variation (CNV) rather than an ERN. The CNV is a slow negative-going ERP component thought to reflect a combination of motor preparation and stimulus expectancy (Bennett, Golob, & Starr, 2004; Brunia, 1999). However, we believe that the ERP component observed in the present experiment is not a CNV for several reasons. First, for the predicted events in both experiments, the peak of this ERP component varied highly in latency across individual subjects (Figure S4), perhaps because participants learned the meaning of the predictive information at different rates. When averaged across subjects, this latency jitter resulted in the ERP component's apparently slow development in the grand average. Second, the running average analysis conducted in the present experiment suggests that the ERN-like waveform to predictive information occurred earlier as the experiment progressed, which is inconsistent with a CNV that peaks immediately before the event. Rather, the slow propagation of the predicted ERN-like negativity seems to have resulted in a waveform that only appears like a CNV in the grand average (see also Figure S3). Third, by creating difference waves we sought to remove any anticipatory effects that were independent of the valence of the predictive information, a manipulation that theoretically would remove the CNV from the data; although there is some evidence that a CNV can be elicited by predictive cues (e.g., Brunia, 1999; Falkenstein, Hoormann, Hohnsbein, & Kleinsorge, 2003; Van Boxtel & Brunia, 1994), it is not clear why the CNV would be larger for cues that predict upcoming errors compared to cues that predict upcoming correct responses. With these considerations in mind, we believe that the ERN-like waveform elicited by the predictive cues in the present experiments is an ERN. Furthermore, even if the waveforms were a CNV and not an ERN, the results nevertheless demonstrate that the frontal system is sensitive to information that predicts upcoming errors.

One may also ask why the amplitudes of the predicted difference waveforms were greater than that of the unpredicted difference waveforms. In both cases, information indicative of upcoming error feedback elicited a larger ERN-like waveform than did the error feedback itself. This result is consistent with previous studies that showed that, all other things being equal, the amplitude of the response ERN is bigger than the amplitude of the feedback ERN (Holroyd & Coles, 2002; Nieuwenhuis et al., 2002). One possibility is that the ERN is larger when the motor action is still in progress, compared to when the action terminates. It is also possible that the present ERN-like waveforms reflect the summation of a phasic ERN with a slower CNV that is larger for predicted errors than for predicted corrects. Further research is needed to clarify this issue.

The Role of the Medial-Frontal System in Motor Control

A large body of literature suggests that motor errors are processed exclusively by posterior parts of the brain, especially by PPC (Desmurget & Grafton, 2000; Desmurget et al., 1999, 2001; Grea et al., 2002; Milner & Goodale, 1993) and the cerebellum (Blakemore et al., 2001; Miall et al., 2001). These regions are thought to adjust for motor errors "on the fly" by integrating sensory feedback with internal sensorimotor information to generate corrective movements (Andersen, Snyder, Bradley, & Xing, 1997; Desmurget et al., 1999, 2001; Grafton, Mazziotta, Woods, & Phelps, 1992; McKay, 1992; Pisella et al., 2000; Rushworth, Johansen-Berg, & Young, 1998). In contrast to this position, we have previously suggested that motor errors are processed by a hierarchical system: High-level errors (that indicate a system goal has been violated) are processed by the medial-frontal system, whereas low-level errors (that can be corrected without violating the system goal) are processed by PPC (Krigolson & Holroyd, 2006). Our proposal was motivated by the observation that errors in a continuous tracking task generate an ERN-like waveform, an electrophysiological signal associated with error commission. According to the RL-ERN theory (Holroyd & Coles, 2002), the ERN is elicited by the first indication that ongoing events are worse than expected by the frontal monitoring system.

The role of the medial-frontal system with regard to the modification of motor behavior remains unclear. For instance, if the error-related process reflected by the ERN is used to modify subsequent behavior, it has yet to be demonstrated how these changes are implemented in the brain. A clue to this question can be found in research involving people with Parkinson's disease. People with Parkinson's disease are impaired at generating internally driven ballistic actions (Flowers, 1976, 1978a, 1978b;

Sheridan, Flowers, & Hurrell, 1987) and at utilizing efference copy to detect their errors (Demirci, Grill, McShane, & Hallett, 1997; Klockgether & Dichgans, 1994; Moore, 1987; Rickards & Cody, 1997; Stern, Mayeux, Rosen, & Ilson, 1983; see also Holroyd, Praamstra, Plat, Coles, 2002), but are relatively unimpaired at responding to external cues. One possibility is that damage to the midbrain dopamine system impairs the ability to learn new internally driven ballistic movements. Given that the midbrain dopamine system appears to carry a predictive error signal for reinforcement learning (Schultz, 1998; Schultz, Dayan, & Montague, 1997) and that the ERN has been suggested to be elicited by the impact of this signal on anterior-cingulate cortex (Holroyd & Coles, 2002), it seems plausible that the ERN may reflect a training signal. In principle, high-level error signals that indicate that a goal has been violated can be used to train internal models for motor control (Wolpert & Ghahramani, 2000). Interestingly, a recent computational model has demonstrated how a dopamine-like reinforcement learning signal could be used to train an internal model implemented by PPC (Branning, Watz, Aisa, & O'Reilly, 2005). In light of these observations, we speculate that not only is the medial-frontal system responsible for evaluating predictive information during motor control, it may also be responsible for developing the internal models utilized by PPC.

The Posterior Error System

In both experiments the ERN-like waveform associated with unpredicted errors was followed by a negative deflection of the difference wave distributed over posterior regions of the scalp. This finding is consistent with the results of a previous study, in which we speculated that the activity reflected the adaptive modification of a posterior error-processing system (Krigolson & Holroyd, 2006; but see also Hill & Raab, 2005), perhaps located within PPC (Desmurget et al., 1999, 2001). Further, in Experiment 1 the ERN-like waveform associated with predicted errors was also followed by a small posterior negativity. Although one might speculate that in this case the posterior system would have been active before the medial-frontal system in an attempt to avoid the upcoming error, the predictive stimulus did not actually provide information about the direction of the barrier movement. As such the posterior system would not have been able to use the predictive information to prevent the error. In contrast, in Experiment 2 there was a very small negative posterior waveform that peaked before the ERN-like waveform did. Here participants had enough time (because the predicted tracking errors occurred slowly) and information (because the participants could see the direction of the barrier movements) to avoid the error, but the reduced control of the joystick negated the usefulness of the predictive information (i.e., the loss of joystick control). In spite of this, the posterior system may have still attempted to avoid the upcoming error, eliciting the posterior activity seen in the ERP. Note that the posterior components associated with both of the predictive waveforms were very small, and we are unsure about their meaning. Further research is needed to investigate this posterior ERP component.

In conclusion, the results of the two experiments presented here suggest that, during the performance of a continuous motor task, predictive information about high-level errors is processed and evaluated within medial-frontal cortex and provide further support for the argument that human error processing is hierarchical in nature (Krigolson & Holroyd, 2006). The findings are also consistent with the RL-ERN theory, which predicts that the ERN will be elicited by the first indication that ongoing events are worse than expected.

REFERENCES

Andersen, R. A., Snyder, L. H., Bradley, D. C., & Xing, J. (1997). Multimodal representation of space in the posterior parietal cortex and its use in planning movements. *Annual Review of Neuroscience*, *20*, 303–330.

Baker, T. E., & Holroyd, C. B. (2006). Which way do I go? Neural activation in response to feedback processing and decision making in a virtual T-maze task. *Psychophysiology*, *43*, S22 [abstract].

Bennett, I. J., Golob, E. J., & Starr, A. (2004). Age-related differences in auditory event-related potentials during a cued attention task. *Clinical Neurophysiology*, *115*, 2602–2615.

Blakemore, S.J, Frith, C. D., & Wolpert, D. M. (2001). The cerebellum is involved in predicting the sensory consequences of action. *NeuroReport*, *12*, 1879–1884.

Brainard, D. H. (1997). The psychophysics toolbox. *Spatial Vision*, *10*, 433.

Branning, P., Watz, B., Aisa, B., & O'Reilly, R. (2005). Simulated robot arm coupled with posterior parietal cortex model performs block stacking task. Poster session presented at the Computational Cognitive Neuroscience Conference, Washington, DC.

Brown, J. W., & Braver, T. S. (2005). Learned predicitions of error likelihood in the anterior cingulate cortex. *Science*, *307*, 1118–1121.

Brunia, C. H. M. (1999). Neural aspects of anticipatory behaviour. *Acta Psychologia*, *101*, 213–242.

Chua, R., & Elliott, D. (1993). Visual regulation of manual aiming. *Human Movement Science*, *12*, 365–401.

Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics. *Journal of Neuroscience Methods*, *134*, 9–21.

Demirci, M., Grill, S., McShane, L., & Hallett, M. (1997). A mismatch between kinesthetic and visual perception in Parkinson's disease. *Annual Review of Neurology*, *41*, 781–788.

Desmurget, M., Epstein, C. M., Turner, R. S., Prablanc, C., Alexander, G. E., & Grafton, S. T. (1999). Role of posterior parietal cortex in updating reaching movements to a visual target. *Nature Neuroscience*, *2*, 563–567.

Desmurget, M., & Grafton, S. T. (2000). Forward modeling allows feedback control for fast reaching movements. *Trends in Cognitive Science*, *4*, 423–431.

Desmurget, M., Grea, H., Grethe, J. S., Prablanc, C., Alexander, G. E., & Grafton, S. T. (2001). The functional anatomy of non-visual feedback loops during reaching: A positron emission tomography study. *Journal of Neuroscience*, *21*, 2919–2928.

Dien, J. (2002). Principal components analysis of ERP data. In T. C. Handy (Ed.), *Event-related potentials* (1st ed, pp. 189–208). Cambridge, MA: MIT Press.

Dien, J., Spencer, K. M., & Donchin, E. (2003). Localization of the event-related potential novelty response as defined by principal component analysis. *Cognitive Brain Research*, *17*, 637–642.

Elliott, D., Helsen, W. F., & Chua, R. (2001). A century later: Woodworth's (1899) two-component model of goal-directed aiming. *Psychological Bulletin*, *127*, 342–357.

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. *Clinical Neurophysiology*, *78*, 447–455.

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Kleinsorge, T. (2003). Short-term mobilization of processing resources is revealed in the event-related potential. *Psychophysiology*, *40*, 914–923.

Flowers, K. A. (1976). Visual "closed-loop" and "open-loop" characteristics of voluntary movement in patients with Parkinsonism and intention tremor. *Brain*, *99*, 269–310.

Flowers, K. A. (1978a). Lack of prediction in the motor behaviour of Parkinsonism. *Brain*, *101*, 35–52.

Flowers, K. A. (1978b). Some frequency response characteristics of Parkinsonism on pursuit tracking. *Brain*, *101*, 19–34.
Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science*, *4*, 385–390.
Goodale, M. A., Pelisson, D., & Prablanc, C. (1986). Large adjustments in visually guided reaching do not depend on vision of the hand or perception of target displacement. *Nature*, *320*, 748–750.
Grafton, S. T., Mazziotta, J. C., Woods, R. P., & Phelps, M. E. (1992). Human functional anatomy of visually guided finger movements. *Brain*, *115*, 565–587.
Gratton, G., Coles, M. G. H., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology*, *55*, 468–484.
Grea, H., Pisella, L., Rossetti, Y., Desmurget, M., Tilikete, C., Grafton, S., et al. (2002). A lesion in posterior parietal cortex disrupts on-line adjustments during aiming movements. *Neuropsychologia*, *40*, 2471–2480.
Hill, H., & Raab, M. (2005). Analyzing a complex visuomotor tracking task with brain-electrical event related potentials. *Human Movement Science*, *24*, 1–30.
Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, *109*, 679–709.
Holroyd, C. B., Nieuwenhuis, S., Mars, R., & Coles, M. G. H. (2004). Anterior cingulate cortex, selection for action, and error processing. In M. Posner (Ed.), *Cognitive neuroscience of attention* (pp. 219–231). New York: Guilford Publishing, Inc.
Holroyd, C. B., Praamstra, P., Plat, E., & Coles, M. G. H. (2002). Spared error potentials in mild to moderate Parkinson's disease. *Neuropsychologia*, *40*, 2116–2124.
Holroyd, C. B., Yeung, N., Coles, M. G. H., & Cohen, J. D. (2005). A mechanism for error detection in speeded response time tasks. *Journal of Experimental Psychology General*, *134*, 163–191.
Jeannerod, M. (1988). *The neural and behavioral organization of goal-directed movements*. Oxford: Clarendon Press.
Khan, M. A., Lawrence, G. P., Franks, I. M., & Buckolz, E. (2004). The utilization of visual feedback from peripheral and central vision in the control of direction. *Experimental Brain Research*, *158*, 241–251.
Klockgether, T., & Dichgans, J. (1994). Visual control of arm movement in Parkinson's disease. *Movement Disorders*, *9*, 48–56.
Krigolson, O. E., & Holroyd, C. B. (2006). Evidence for hierarchical error processing in the human brain. *Neuroscience*, *137*, 13–17.
McKay, W. A. (1992). Properties of reach-related neuronal activity in cortical area 7a. *Journal of Neurophysiology*, *67*, 1331–1345.
Miall, R. C., Reckess, G. Z., & Imamizu, H. (2001). The cerebellum coordinates eye and hand tracking movements. *Nature Neuroscience*, *4*, 638–644.
Miall, R. C., Weir, D. J., & Stein, J. F. (1993). Intermittency in human manual tracking tasks. *Journal of Motor Behaviour*, *25*, 53–63.
Milner, A. D., & Goodale, M. A. (1993). Visual pathways to perception and action. In T. P. Hicks, S. Molotchnikoff, & T. Ono (Eds.), *Progress in brain research* (Vol. 95, pp. 317–337). Amsterdam: Elsevier.
Miltner, W. H. R., Braun, C. H., & Coles, N. G. H. (1997). Event-related potentials following incorrect feedback in a time-estimation task: Evidence for a ''generic'' neural system for error detection. *Journal of Cognitive Neuroscience*, *9*, 788–798.
Moore, A. P. (1987). Impaired sensorimotor integration in Parkinsonism and dyskinesia: A role for corollary discharges? *Journal of Neurology, Neurosurgery, and Psychiatry*, *50*, 544–552.
Nieuwenhuis, S., Holroyd, C. B., Mol, N., & Coles, M. G. H. (2004). Reinforcement-related brain potentials from medial-frontal cortex: Origins and functional significance. *Neuroscience and Biobehavioural Review*, *28*, 441–448.
Nieuwenhuis, S., Ridderinkhof, K. R., Talsma, D., Coles, M. G. H., Holroyd, C. B., Kok, A., & Van der Molen, M. W. (2002). A computational account of altered error processing in older age: Dopamine and the error-related negativity. *Cognitive, Affective & Behavioral Neuroscience*, *2*, 19–36.
Nieuwenhuis, S., Slagter, H. A., Alting von Geusau, N. J., Heslenfeld, D. J., & Holroyd, C. B. (2005). Knowing good from bad: Differential activation of human cortical areas by positive and negative outcomes. *European Journal of Neuroscience*, *21*, 3161–3168.
Paillard, J. (1996). Fast and slow feedback loops for the visual correction of spatial errors in a pointing task: A re-appraisal. *Canadian Journal of Physiology and Pharmacology*, *74*, 401–417.
Pisella, L., Grea, H., Tilikete, C., Vighetto, A., Desmurget, M., Rode, G., et al. (2000). An 'automatic pilot' for the hand in human posterior parietal cortex: Toward reinterpreting optic ataxia. *Nature Neuroscience*, *3*, 729–736.
Rickards, C., & Cody, F. W. J. (1997). Proprioceptive control of wrist movements in Parkinson's disease. *Brain*, *120*, 977–990.
Rodriguez-Fornells, A., Kurzbach, A. R., & Munte, T. F. (2002). Time course of error detection and correction in humans: Neurophysiological evidence. *Journal of Neuroscience*, *22*, 9990–9996.
Rushworth, M. F. S., Johansen-Berg, H., & Young, S. A. (1998). Parietal cortex and spatial-postural transformation during arm movements. *Journal of Neurophysiology*, *79*, 478–482.
Schultz, W. (1998). Predictive reward signal of dopamine neurons. *Journal of Neurophysiology*, *80*, 1–27.
Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science*, *275*, 1593–1599.
Sheridan, M. R., Flowers, K. A., & Hurrell, J. (1987). Programming and execution of movement in Parkinson's disease. *Brain*, *110*, 1247–1271.
Stern, Y., Mayeux, R., Rosen, J., & Ilson, J. (1983). Perceptual motor dysfunction in Parkinson's disease: A deficit in sequential and predictive voluntary movement. *Journal of Neurology, Neurosurgery, and Psychiatry*, *46*, 145–151.
Van Boxtel, G. J. M., & Brunia, C. H. M. (1994). Motor and non-motor components of the contingent negative variation. *International Journal of Psychophysiology*, *17*, 269–279.
Wolpert, D. M., & Ghahramani, Z. (2000). Computational principles of movement neuroscience. *Nature Neuroscience*, *3*, 1212–1217.
Woodworth, R. S. (1899). The accuracy of voluntary movement. *Psychological Review*, *3*, 1–119.

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SUPPLEMENTARY MATERIAL

The following supplementary material is available for this article (all Figures provided in JPG format):

Figure S1A. Experiment One Diagram of the experimental procedure. Note that the yellow arrow did not appear on the participants screen during rapid barrier movements.

Figure S1B. Experiment Two Diagram of the experimental procedure. Note that the yellow arrow did not appear on the participants screen during rapid barrier movements.

Figure S2. Top Panel: Difference waveforms for unpredicted and predicted errors for the first and last running average bins in Experiment One. Bottom Panel: Difference waveforms for unpredicted and predicted errors for the first and last running average bins in Experiment Two.

Figure S3. Top Panel: Individual participant difference waveforms for predicted errors in Experiment One. Bottom Panel: Individual participant difference waveforms for predicted errors in Experiment Two.

Figure S4. Top Panel: Averaged ERP waveforms recorded at channel FCz for unpredicted (left) and predicted (right) tracking errors locked to the onset of the rapid barrier movement or the predictive cue for Experiment One. Bottom Panel: Averaged ERP waveforms recorded at channel FCz for unpredicted (left) and predicted (right) tracking errors locked to the onset of the rapid barrier movement or the predictive cue for Experiment Two.

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