# Hierarchical Error Processing During Motor Control

by

Olave Edouard Krigolson
BEd., University of Victoria, 1997
MSc., Indiana University, 2003

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## ABSTRACT

The successful execution of goal-directed movement requires the evaluation of many levels of errors. On one hand, the motor system needs to be able to evaluate 'high-level' errors indicating the success or failure of a given movement. On the other hand, as a movement is executed the motor system also has to be able to correct for 'low-level' errors - an error in the initial motor command or change in the motor command necessary to compensate for an unexpected change in the movement environment. The goal of the present research was to provide electroencephalographic evidence that error processing during motor control is evaluated hierarchically. The present research demonstrated that high-level motor errors indicating the failure of a system goal elicited the error-related negativity, a component of the event-related brain potential (ERP) evoked by incorrect responses and error feedback. The present research also demonstrated that low-level motor errors are associated with parietally distributed ERP component related to the focusing of visuo-spatial attention and context-updating. Finally, the present research includes a viable neural model for hierarchical error processing during motor control.

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Carrie,

You are my sunshine

# General Introduction

How do we execute and control movement? In a seminal series of experiments Woodworth (1899) attempted to address this question by examining discrete and continuous goal-directed actions made with differing visual (eyes open versus eyes closed) and kinematic (movement time and movement velocity) restraints. From his results, Woodworth hypothesized a two component model of goal-directed action. The first component, termed the initial impulse, was thought to be programmed in advance of movement initiation and consisted of a ballistic limb movement to place the limb within the target vicinity. The second component, which Woodworth termed the current control phase, was thought to consist of online feedback based corrections to adjust for errors in the initial movement impulse. Woodworth's model also predicted that increasing the temporal constraints on a movement would result in a decrease in movement accuracy, as the feedback mechanisms attributed to the current control phase of the movement would not have sufficient time to process afferent information and make within movement modifications. Importantly, while the model suggested that some portion of the movement could be planned in advance (an idea similar to the later idea of a generalised motor program - see below, Schmidt, 1975), it also specified that afferent feedback could be used rapidly and efficiently in order to compensate for errors in motor output while a movement was being executed. Although Woodworth's model did not specifically address how these errors were being evaluated and corrected, his work implied that the system must make comparisons between the actual and the desired movement state in order to affect within movement modifications.

## *The Initial Motor Plan*

Woodworth's belief that all aiming movements contained a ballistic component is similar to the idea of a generalised motor program - a set of neural instructions that when executed results in a goal directed action. Motor programs allow movements to be planned in advance and executed entirely without the online use of afferent feedback (i.e., open-loop control). While this notion is contrary to Woodworth's original hypothesis, there is some evidence that people program movements in advance. For example, Henry and Rogers (1960) had participants perform a series of increasingly complex movement patterns and found that reaction time, the time from stimulus onset to movement onset, increased in relation to movement complexity. Henry and Rogers interpreted this result as evidence that participants were planning their actions in advance of movement onset; more complex actions took longer to plan and as a result reaction time is longer.

Other evidence supporting the existence of a generalised motor program stems from research examining the electromyogram (EMG) of arm muscles during rapid movements (Wadman, Denier van der Gon, Geuze, & Mol, 1979). Wadman and colleagues found that the EMG pattern for a normal arm movement was tri-phasic in nature, consisting of an initial impulse by the principal agonist followed by two subsequent bursts of activity (antagonist then agonist) to brake and stabilise the arm movement. During the course of the experiment the participants' arm movement was suddenly and unexpectedly blocked after movement onset. Interestingly, on blocked trials a tri-phasic EMG pattern similar to that for the unblocked movements was also observed. Wadman et al. interpreted these data as evidence that the arm movements were planned in advance of movement onset.

The kinematic characteristics of manual aiming movement also provide evidence for Woodworth's assertion that the initial phase of actions are ballistic and programmed in advance of the movement. For example, several studies have demonstrated the amount of time spent following peak velocity differs when participants make discrete reaching movements to targets that differ in size (Langolf, Chaffin, & Foulke, 1976; MacKenzie, Marteniuk, Dugas, & Liske, 1987; Soechting, 1984). It is important to note here that one of the more common measures of online movement control is the proportion of time spent after peak velocity, and thus increases or decreases in this measure are thought to be indicative of increased or decreased online movement control (Elliott, Helsen, & Chua, 2001). With this in mind, it is also important to note that the aforementioned studies did not find differences in the movement profiles before peak velocity, a result taken to suggest that up to this kinematic marker the movement was planned before movement onset.

Research examining the role of visual feedback in reaching accuracy also provides evidence for a ballistic movement phase. For instance, it has been demonstrated that memory-dependent reaches are less accurate and more variable than their full-vision counterparts (Heath, Westwood, & Binsted, 2004; Westwood & Goodale, 2003) Furthermore, Heath et al. analysed the proportion of endpoint variance explained by the limb position across the reaching trajectory ($R^2$) and found lower $R^2$ values for full-vision reaches at peak velocity (and peak deceleration) than for memory-guided reaches. The theory behind this analysis is that if reaches are specified in advance of movement onset then a greater proportion of the limb variability will be explained earlier in the movement than for reaches that rely to a greater extent on online control processes. Thus, Heath and colleagues concluded that memory-guided reaches are programmed in advance of movement onset, at least to a greater extent than their full-vision counterparts.

Complementing these results, Khan, Lawrence, Franks, and Elliott (2003) compared full-vision and memory-dependent reaches made with differing movement time restraints. Interestingly, their results indicated that for very brief movements (movement time ≈ 150 ms), there was no difference in accuracy between the visual conditions. Khan and colleagues suggested that this result indicates that for very rapid movements online control strategies are ineffective and thus the entire movement needs to be planned in advance. Furthermore, this result also implies that for slower movements only a portion of the movement is pre-programmed (i.e., Woodworth, 1899).

### *Programming the Movement: The Inverse Model*

A question of interest that stems from these findings is how the system determines the appropriate motor command to achieve a desired goal. Recently, it has been suggested that the motor system utilises an inverse model to generate the motor command (Desmurget & Grafton, 2000; Haruno, Wolpert, & Kawato, 1999, 2001; Haruno, Wolpert, & Kawato, 2003; Wolpert & Ghahramani, 2000; Wolpert, Ghahramani, & Gazzaniga, 2004). As input the inverse model receives information about the current state of the system (the initial position of the movement effector) and the desired state of the system (the position of the target), with this information being derived from afferent feedback, the efferent motor command, or a combination of the two (Jordan & Wolpert, 1999). From this information the inverse model determines the set of motor commands necessary to take the system from the current state to the desired state. Current theories propose that the inverse model specifies the parameters of the actual motor command by minimizing a cost function associated with the movement (Wolpert & Ghahramani, 2000). Consider an aiming movement to a stationary target. In this instance the inverse model would select an appropriate set of motor instructions that minimize the variability of the movement endpoint. Experimental evidence that demonstrates participants attempt to maximize the smoothness or minimize torques has been interpreted as support for this hypothesis (Flash & Hogan, 1985; Harris & Wolpert, 1998; Uno, Kawato, & Suzuki, 1989).

### *Online Motor Control*

In spite of the evidence outlined above, there is compelling evidence that Woodworth's initial assertion was correct and that goal directed actions also rely on online movement amendments to achieve endpoint accuracy. Indeed, if all movements were executed in an open-loop fashion then the motor system would be unable to accommodate changes in the movement environment or compensate for neuromotor noise. A frequently cited study by Goodale, Pelisson, and Prablanc (1986) provides strong evidence that online adjustments are made during goal-directed actions to accommodate changes within the movement environment. In their paradigm, Goodale and colleagues had participants make discrete reaching movements to a target that had just been perturbed to a new, more peripheral location. On a small percentage of the trials however, the target perturbed again to an even more peripheral location during the initial movement impulse. Thus, in order to achieve movement accuracy participants were required to make online modifications to their motor plan. Goodale et al.'s results indicated that participants were able to make these within movement modifications in spite of the temporal restraints of the task (participants were asked to reach "as quickly and accurately as possible"). Additionally, the results indicated no differences in movement time between the perturbed and non-perturbed trials negating the possibility that perturbed trial accuracy was due to a speed-accuracy trade-off. Furthermore, an examination of the velocity profiles for the perturbed trials revealed no discontinuities associated with a reprogramming of the movement. From these results Goodale and colleagues concluded that participants were able to make rapid and accurate adjustments to ongoing movements in order to accommodate the unexpected target perturbations.

Subsequent research by Chua and Elliott (1993) sought to further investigate the online control phase of goal directed movements by manipulating vision of the movement environment during a discrete aiming movement. Chua and Elliott had participants complete goal-directed aiming movements in one of four visual conditions. In the first, participants had vision at all times throughout the movement. In the second and third conditions, participants had vision of the movement environment during the first (prior to peak velocity) or second (after peak velocity) half of the movement respectively. In the fourth visual condition participants did not have vision of the aiming environment. The authors reasoned that if vision is important during the deceleration phase (after peak velocity) of an aiming movement to conduct online movement adjustments, then occluding vision during this phase should significantly reduce endpoint accuracy. Conversely, occluding vision prior to the peak velocity should have little impact on movement accuracy as this phase of the movement should have been planned in advance of movement onset. The results of this experiment reliably demonstrated that vision during the second half of the reaching movement facilitated movement accuracy. Presumably during this phase (i.e., the deceleration phase following the ballistic movement impulse) participants engaged in online control processes to nullify errors inherent to the initial movement trajectory.

More recently, Heath (2005) had participants complete reaches to targets in a variety of target and limb vision conditions. Specifically, following a preview phase of the movement environment participants had to make discrete aiming movements with or without vision of the target. Additionally, within each of the target vision conditions reaches were made with and without vision of the movement effector. The results of this study demonstrated that participants made highly effective and accurate adjustments to their reaching trajectories when vision of the target and / or limb was available. Conversely, when participants did not have vision of the target or their limb these within movement modifications were absent. Heath's results affirmed Woodworth's assertion that reaching movements are indeed comprised of ballistic and online control phases, and that the duration of these phases is mediated by the constraints of the movement environment. In other words, the degree of open-loop control in a movement increases in situations where the effectiveness of online control mechanisms are reduced or in situations where there is insufficient time to make within movement modifications (i.e., Elliott & Madalena, 1987).

Indeed, a variety of models and experimental data have affirmed Woodworth's initial two component model of goal-directed action(Chua & Elliott, 1993; Elliott, Helsen, & Chua, 2001; Glover & Dixon, 2001a, 2001b, 2002a, 2002b; Goodale et al., 1986; Heath, 2005; Heath, Hodges, Chua, & Elliott, 1998; Krigolson & Heath, 2004; Meyer, Abrams, Kornblum, Wright, & Smith, 1988; Milner & Goodale, 1993, 1995; Westwood & Goodale, 2003; Westwood, Roy, & Heath, 2003), albeit with some minor revisions to the initial hypothesis. However, all of the later models agree with Woodworth's initial hypothesis that, when possible, online feedback based corrections are critical to achieving accuracy during goal-directed actions. Recently patient, imagining, and transcranial magnetic stimulation studies have provided converging evidence the processes underlying online motor control may reside within posterior parietal cortex.

### *Posterior Parietal Cortex*

Desmurget et al. (1999) recently conducted a study in which right handed participants had to reach to targets that retained a stationary position or was perturbed during the initial saccadic eye movement. Replicating Goodale et al.'s (1986) original findings, Desmurget and colleagues found that during perturbed target trials participants accurately adjusted their reaching trajectory to accommodate the target movement, even though they were not consciously aware the target had moved. However, in another experimental condition transcranial magnetic stimulation (TMS) was applied to left posterior parietal cortex (PPC) for both unperturbed and perturbed target trials. Interestingly, TMS to left PPC negated the ability of participants to make online movement amendments during the perturbed target trials. In opposition to this finding, TMS to right PPC did not reduce the participants' ability to accommodate the target perturbations by making online adjustments to their aiming movement. Furthermore, TMS to left (or right) PPC did not disrupt the accuracy of stationary target trials suggesting that while this neural substrate was a part of the online control system, it does not play a crucial role in the programming of the initial movement impulse.

Two patient studies provide supportive evidence for Desmurget et al.'s (1999) findings. Using a similar paradigm, Grea et al. (2002) and Pisella et al. (2000) examined reaching movements to targets that perturbed after movement onset in normal participants and patients with lesions to PPC. In both instances, the experimental results revealed that patients with damage to PPC could plan motor movements as effectively as healthy participants – movements to targets that did not perturb after movement onset were equally accurate between the two experimental groups. However, when the movement target was perturbed after movement onset the results of both studies indicated that patients with damage to PPC had a reduced ability to make rapid and accurate adjustments to the initial motor plan. In conjunction with Desmurget et al.'s results, these data provide strong evidence that PPC is involved in the online control of visually guided actions.

Further confirmation of this hypothesis stems from another study by Desmurget et al. (2001) who utilised positron emission tomography (PET) to examine the neural substrate(s) underlying non-visual feedback control of movement (i.e., proprioceptive feedback). Again using a target perturbation paradigm, Desmurget and colleagues had participants reach to a target without vision of the movement effector. In one experimental condition the target remained stationary throughout the reaching movement whereas in the other experimental condition the target perturbed in location during the ocular saccade. As with previous studies (Desmurget et al., 1999; Goodale et al., 1986) the behavioural data indicated that participants were equivalently accurate between the stationary and perturbed target trials, a result indicating that the motor system was able to make rapid and accurate adjustments to accommodate the target perturbation. As with the

TMS and lesion studies, the PET analysis replicated previous findings and indicated that left PPC was activated during trials where a corrective sub-movement was necessary. Furthermore, activation of the right anterior intermediate cerebellum and left primary motor cortex were also observed. Desmurget and colleagues hypothesized that these neural substrates formed a network responsible for online motor control. Importantly, the results of this study also indicated that in addition to its role in visually based online motor control, PPC also mediated corrective mechanisms reliant upon proprioceptive feedback.

### *Online Motor Control: Forward Models*

While it seems clear that visual feedback is necessary to make online movement amendments, vision alone is not sufficient to achieve movement accuracy. Consider what occurs when an online movement adjustment is made. First and foremost, the system needs to make a comparison between the current state and the desired state of the limb at a point in time. The desired state of the limb is determined prior to movement onset and is available to the motor system throughout the movement. However, the determination of the current state of the limb is a more complex problem. If the system were to rely solely on visual and/or proprioceptive feedback then the online corrective movements would be subject to a sensory processing delay. Indeed, it appears that at least 80 to 100 ms is needed for an afferent signal to influence an ongoing movement (Jeannerod, 1986; Paillard, 1996). As a result, corrective sub-movements relying solely upon afferent feedback would not be effective for very fast movements.

To solve this problem, it has been proposed that the motor system takes advantage of a predictive forward model that utilises an efference copy of the motor command to estimate future movement states in advance of their occurrence. If one then places the forward model within an internal closed feedback loop, the system can act on the predictive information before it actually occurs, thus negating the sensory feedback delay (Desmurget & Grafton, 2000; Wolpert & Ghahramani, 2000). Further experimental and computational work suggests that the forward system also relies upon afferent feedback to improve its estimates of the current movement state (Wolpert, Ghahramani, & Jordan, 1995). Current theories suggest that the forward model either resides within PPC (Desmurget & Grafton, 2000) or the cerebellum (Blakemore, Frith, & Wolpert, 2001).

### *Frontal Systems and Error Processing*

While the previously outlined posterior error system appears to be dedicated to online motor control, another compelling line of research has identified a medial-frontal system responsible for evaluating rewards and punishments. The search for a reward mechanism in the brain dates back to the seminal work of Olds and Milner (1954). In their paradigm, an electrode was inserted into the hypothalamus of a rat and then connected to a lever within the rat's living environment. When the lever was depressed by the rat, an electrical current stimulated the hypothalamus. Observation of the rat's behaviour revealed repeated depression of the lever thus stimulating their hypothalamus. Rats were found to engage in this behaviour to the point of ignoring sleep, food, and the presence of other rats. Olds and Milner interpreted this finding as evidence that the thalamus contained a neural reward mechanism. Research extending Olds and Milner's findings has implicated the midbrain dopamine system as a likely candidate for a neural reward mechanism (Lindvall, Bjorklund, & Skagerberg, 1983). The midbrain dopamine system is comprised of the nuclei in the ventral tegmental area and the substantia nigra pars compacta with neurons from these areas projecting to multiple frontal cortical areas (Berger, Gaspar, & Verney, 1992).

Several studies have demonstrated a reward-dopamine relationship in monkeys by showing that dopamine neurons are more active when a reward is given (Ljungberg, Apicella, & Schultz, 1991, 1992; Mirenowicz & Schultz, 1994, 1996; Romo & Schultz, 1990). In all of these studies the activity of individual midbrain dopamine neurons was examined following reward presentation during a behavioural task. The results were similar in all cases, with greater activation of dopamine neurons observed following reward presentation. Interestingly, the activation of the dopamine neurons did not appear to differentiate between different types of rewards, but did distinguish between rewards and non-rewards. The primary conclusion arising from these early studies was that the midbrain dopamine system is a plausible candidate for the mediation of reinforcement learning (Robbins, Everitt, & Gazzaniga, 1995).

More recently, a systematic investigation of the relationship between stimuli, rewards, and dopamine activity has been undertaken by Shultz and colleagues (Schultz, 1997, 1998; Schultz, Dayan, & Montague, 1997). As previously discussed, in monkeys it has been demonstrated that presentation of an unpredicted reward results in a phasic increase in dopamine activity. After consistent stimulus-response training, however, the monkey learns that a stimulus predicts a given reward and an increase in dopamine activity is observed at the time of stimulus presentation. Additionally, in these situations where a learned stimulus elicits dopamine activity, no change in dopamine activity will be observed at the time of reward as the reward has already been predicted by the stimulus. Although there is no change in dopamine activity at the time of reward after a valid stimulus–response mapping is established, the system still monitors if the reward occurs as a decrease in dopamine activity is observed if a predicted reward is not presented after a conditioned stimulus. As such, the midbrain dopamine systems seems to act in a manner predicted by the method of temporal differences (c.f., Sutton & Barto, 1998) as the increases and decreases in dopamine activity seem to represent prediction errors as opposed to actual occurrences of stimuli or rewards. In other words, an increase in dopamine activity is observed when events are better than expected (an unpredicted reward occurs, a stimulus predicting a reward occurs) and a decrease in dopamine activity occurs when events are worse than expected (a predicted reward does not occur). When events are as expected, there is no change in dopamine activity (a predicted reward occurs).

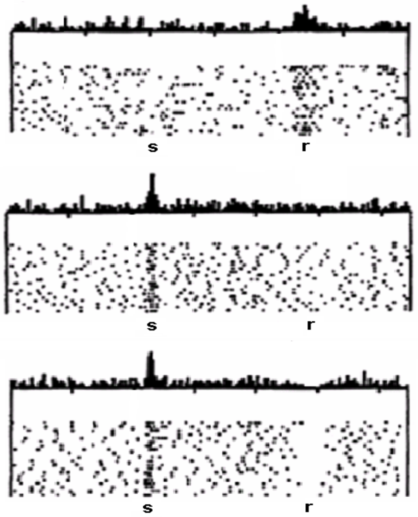

**Figure 1. The firing of a dopamine neuron in response to unpredicted and predicted rewards in monkey (adapted from Schultz, Dayan, & Montague, 1997). Early in learning, an unpredicted juice reward elicits an increase in phasic dopamine (top panel). After learning has occurred, a stimulus that consistently precedes the juice rewards elicits a phasic increase in dopamine, whereas the reward itself does not (middle panel). If a predicted reward does not occur, a phasic decrease in dopamine is observed at the time the reward should have occurred (bottom panel).**

### *A Mechanism for Dopamine Adaptations*

How do neurons in the midbrain dopamine system modify behaviour? Dopamine neurons in the ventral tegmental area and the substantia nigra pars compacta project to medial frontal cortex (Berger et al., 1992) and form synaptic triads with local neurons in these regions. At a regular synapse, synaptic transmission occurs between pre-synaptic and post-synaptic neurons when an action potential causes neurotransmitters to cross the synaptic cleft. However, in a synaptic triad the regular synapse contains a third neuron projecting onto the pre-synaptic neuron. It is hypothesized that this third neuron can modulate the excitability of the synapse by modulating the release of neurotransmitter (Dehaene, Changeux, & Nadal, 1987). Importantly, these synaptic triads are thought to mediate cortical function in the striatum by facilitating long term potentiation (LTP) and/or long term depression (LTD) at local synapses (Calabresi, Pisani, & Bernardi, 1996; Reynolds & Wickens, 2002; Wickens, Begg, & Arbuthnott, 1996; Wickens, Kotter, Houk, Davis, & Beiser, 1995) Therefore, the net result of dopamine release in the striatum via the synaptic triads is to enhance excitation of cortical neurons, thus reinforcing a particular pattern of neuronal firing. A selective focusing of target neurons via dopamine release ensures that the strongest firing neurons are facilitated and other cortical neurons are inhibited. As such, a specific pattern of neuronal firing is reinforced and strengthened, providing a viable mechanism for reinforcement learning (Brown & Arbuthnott, 1983; Schultz, 2002). Recently, it has also been suggested that the neuromodulatory effect of dopamine in pre-frontal cortex is to regulate the information held in working memory (Braver & Cohen, 2000; Seamans & Yang, 2004).

### *Electroencephalographic Evidence for Frontal Error Processing*

In humans the role of the medial-frontal system with regard to reward and error processing has been examined using event-related brain potentials (ERP). Initially, ERP studies paid little attention to error trials, however, Gehring, Goss, Coles, Meyer, and

Donchin (1993) sought to correct this deficit and their combined work resulted in the discovery of an ERP component sensitive to response errors. Gehring and colleagues had participants perform the Eriksen flanker task and recorded ERP data from both response errors and successful task performance. During the flanker task the participant classified a centrally presented letter (S or H) flanked by four distracter letters, two on either side. The distracter letters were either the same as the presented letter (i.e., S S S S S) or different (i.e., H H S H H). To classify the centrally positioned letter, the participants responded by squeezing one of two dynamometers. The participants completed the flanker task while focusing on either response speed, response accuracy, or a combination of both. To ensure this occurred, a monetary reward was given when responses were quicker than a cut-off time and a monetary penalty was assessed when errors occurred. Following the experiment, the ERP waveforms were analysed and a greater negativity was observed on error trials. This negative deflection in the ERP waveform was found to peak approximately 80 ms after a response error was maximal over front-central electrode sites. Interestingly, the results also revealed that the peak negativity in the condition where accuracy was the primary goal was greater than in the speeded response condition. Gehring and colleagues concluded the negative peak was representative of a neural error processing system and termed it the error-related negativity (ERN). Concurrent with this work, Falkenstein and colleagues (Falkenstein, Hohnsbein, & Blanke, 1991) reported a similar negative deflection in the ERP waveform following response errors which they termed the error negativity, or Ne.

Subsequent research by Bernstein, Scheffers, and Coles (1995) further investigated factors that would impact the magnitude of the ERN. Utilising a four choice reaction time paradigm where participants could respond with one of two fingers on either hand, Bernstein et al. created a situation where experimental errors varied in magnitude (i.e. wrong hand, wrong finger versus wrong hand, right finger versus right hand, wrong finger). An analysis of the results revealed that the ERN increased with the severity of the error, as the ERN amplitude was greatest for errors made with the wrong hand and the wrong finger relative to smaller ERN amplitudes associated with errors made by the right hand but the wrong finger. More recent research has also shown that an ERN is observed when an error is committed with the feet (Holroyd, Dien, & Coles, 1998) or the eyes (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). In conjunction with previous work, these later studies suggest that the ERN is generated by a generic error processing system independent of response modality.

Recently, Hajcak, Moser, Yeung, and Simons (2005) demonstrated in two separate experiments that the ERN is sensitive to the motivation of the participant. In the first experiment, participants performed a flanker task but were made aware before each trial whether an error would cost then a small or a large financial penalty. An analysis of the ERP data revealed that the ERN amplitude was greater for large monetary penalty errors as opposed to small monetary penalty errors. In the second experiment, two groups of participants performed the same flanker task in two different experimental conditions. In the first condition, participants were informed that their accuracy was being recorded and would be compared to other participants while in the second condition participants were simply told to do as well as they could. As with the first experiment, a larger ERN amplitude was found on trial were the participants were more motivated (i.e., when they were being evaluated). Together, the results of these two experiments suggest that ERN amplitude is impacted by the motivation of the participant.

While an ERN associated with a response error had been established, it was the work of Miltner, Braun, and Coles (1997) that found an ERN like waveform associated with negative feedback. In this particular experiment, on each trial participants heard an auditory stimulus and were asked to respond with a key press when they gauged a pre-specified time interval had past. Unlike previous studies investigating choice reaction time or speeded response tasks and the ERN, in this task participants were unaware of an error until they were presented with feedback. Feedback was presented to participants in either a visual, auditory, or somatosensory modality. An analysis of the ERP waveforms indicated that approximately 250 ms after receiving negative feedback there was a greater negativity over central electrode sites. Although the amplitude of the ERN was impacted by stimulus modality, in all of the experimental conditions the ERN had a greater negativity for feedback indicating an error had been committed. Termed a feedback ERN (fERN: as opposed to the previously identified response ERN: rERN), this ERP component was thought to be representative of the processing of error feedback. However, it is important to note that the fERN in this experiment was not found to be sensitive to the direction of the error (i.e., under or over estimation of the time period) and was also more broadly distributed over central electrode sites than previous research examining the rERN.

Recent research examining the fERN has demonstrated that its amplitude is modulated by reward/error expectancy in a manner similar to that of reward related phasic dopamine activity (i.e., Schultz, Dayan, & Montague, 1997). Holroyd and

Krigolson (in press) utilised a modified version of Miltner et al.'s time estimation task in which participants experienced "easy" and "difficult" blocks of trials (see above). Task performance was manipulated such that when guessing the duration of one second participants experienced easy blocks of trials in which they made few errors and difficult blocks of trials in which they made a lot of errors. As such, the paradigm allowed comparison of expected correct feedback with expected error feedback waveforms and unexpected correct feedback with unexpected error feedback waveforms. The results of the experiment indicated that the amplitude of the unexpected fERN difference waveform (unexpected error feedback – unexpected correct feedback) was significantly greater than the amplitude of the expected fERN difference waveform (expected error feedback – expected correct feedback). Furthermore, the experimental results revealed participants made larger behavioural modifications following unexpected error feedback. Importantly, these results are in line with reinforcement learning principles which suggest learning occurs following unexpected feedback (e.g., Rescorla-Wagner) and provide further evidence that the ERN reflects a prediction error (see below; see also Holroyd, Nieuwenhuis, Young, & Cohen, 2003).

### *Source Localization of the Error Related Negativity: Anterior Cingulate Cortex*

As reviewed above, the ERN seems to be representative of generic error processing system sensitive to whether an incorrect response has occurred or negative feedback has been received. Additionally, the ERN occurs regardless of response modality or stimulus presentation type. Finally, the amplitude of the ERN is sensitive to the motivation of the participant. A question that remains is the neural source of the ERN. Although there are problems with source localization using ERPs, several techniques have been introduced to deal with this particular problem (see Picton et al., 2000). The ERNs in the studies mentioned above all had maximal voltages over front–central regions of the scalp, but more recently further source information has been derived from ERN studies using the Brain Electrical Source Analysis (BESA) technique (for details see Scherg & Picton, 1991; Scherg, Vajsar, & Picton, 1989). Dehaene, Posner, and Tucker (1994) had participants perform two categorization tasks while recording ERP data. In the first, participants had to classify integers as being greater than or less than five using discrete key presses. In the second, participants had to classify whether a presented word belonged to a category previously specified by the experimenter. During both experiments, participants made errors given that a speeded-response was required. Analysis of the ERP data using the BESA technique suggested the source of the ERN was in anterior-cingulate cortex. Supporting this finding, more recent studies by Gehring, Himle, and Nisenson (2000) and Luu, Tucker, Derryberry, Reed, and Poulsen (2003) have also examined the ERN and used source localization techniques to also suggest anterior cingulate cortex as a neural generator of this ERP component.

Additional studies using magneto encephalogram (MEG) imaging and function magnetic resonance imaging (fMRI) also support the hypothesis that the ERN is generated in anterior cingulate cortex. Miltner et al. (2003) had participants perform a Go–NoGo task where they were instructed to respond to a high frequency tone but not a low frequency tone. On error trials a magnetic equivalent of the ERN was observed using MEG, and importantly source localization implied a generator in the region of anterior cingulate cortex. Complementing this result, Holroyd et al. (2004c) used fMRI to analyse errors made on a trial and error speeded response task. In one experimental condition, the stimulus-response mappings were fixed and thus after practice participants were aware when they had made a mistake (i.e., a response error). In the second experimental condition stimulus-response mappings were random forcing participants to wait for feedback to know if an error had been committed (i.e., a feedback error). In both cases, the fMRI data indicated that anterior cingulate cortex was more active on error trials than on correct trials, suggesting the involvement of this region in the processing of both response and feedback error information.

### *Linking Dopamine, the Error-Related Negativity, and Reinforcement Learning*

In a seminal paper, Holroyd and Coles (2002) sought to unify previous accounts linking phasic reward related dopamine activity in monkeys and reinforcement learning (i.e., Montague, Dayan, & Sejnowski, 1996; Schultz, Dayan, & Montague, 1997) with error processing in humans (the ERN). To accomplish this, Holroyd and Coles conducted an experiment where participants attempted to learn a series of stimulus-response mappings. Unbeknownst to participants, in one experimental condition the stimulus-response mappings were random and participants were rewarded (or punished) on 50% of the trials regardless of their response. In a second experimental condition however, the stimulus-response mappings were learnable (i.e., the relationship between the stimulus and a given response always stayed the same). The ERP data revealed that when participants were in a condition with random stimulus-response mappings a feedback ERN was observed. Conversely, when the stimulus–response mappings were fixed, a response ERN was observed.

To explain these results Holroyd and Coles (2002) developed a model based on previous temporal difference models of the dopamine system (e.g., Schultz, Dayan, &

Montague, 1997). In the Holroyd and Coles model the ERN is the observable scalp EEG following the impact of a dopaminergic reinforcement learning from the basal ganglia on anterior cingulate cortex. In this model, anterior cingulate cortex acts as a control filter, selecting the best motor controller to generate a specific response. Once selected, a response is generated and motor output occurs. To modify behaviour, an adaptive critic receives feedback about the response from sensory input and/or from an efference copy of the movement sent during the response output process. The adaptive critic then utilises this information to determine the consequences of the action and improve behaviour. If events are better or worse than expected, a reinforcement learning signal is sent via the midbrain dopamine system to the motor controllers and the control filter (anterior cingulate cortex) to modify future actions, and in a recursive manner to the adaptive critic to improve future predictions of reward.

In terms of reinforcement learning and the ERN, the Holroyd and Coles (2002) model predicts that early in learning a feedback ERN will be observed as the relationship between a given stimulus and response is not well established. The feedback ERN can be thought of as a prediction error occurring at the time of the feedback. Early in learning the system is not able to predict the outcome of actions and as such prediction errors are associated with feedback about the response outcome. The model explains the feedback ERN as the impact of the dopamine signal sent from the adaptive critic to anterior cingulate cortex when negative feedback is received. After practice a valid stimulus-response relationship is learned, and as a result a response ERN is observed when a response error is committed. In other words, the prediction error occurs at the time of the response as opposed to the time feedback is received; one could say that the system has developed a forward model of control able to predict the consequences of its actions. The Holroyd and Coles model explains the response ERN as the adaptive critic's evaluation that an error has been committed from the efference copy of the movement sent during the response output process. At this time the adaptive critic would send a reinforcement learning signal to anterior cingulate cortex via the dopamine system and a response ERN would be observed.

### *Summary*

There appears to be two distinct error processing systems in the human brain. One of these systems seems to be tasked with evaluating the 'low-level' motor errors as a movement unfolds. Specifically, this posterior system (typically associated with PPC), determines discrepancies between the current and desired motor command. This system is then able to implement modifications to the initial motor plan to allow for the achievement of movement accuracy. The posterior system is also able to correct for unexpected changes in the movement environment that occur as a movement unfolds (e.g., a change in target location). Conversely, there also appears to be a medial-frontal error system that is sensitive to response errors and negative feedback. This system seems to be tasked with evaluating 'high-level' errors that indicate the success or failure of a movement. To date, there are no studies examining the role of the medial-frontal system in motor control. Additionally, there are no electroencephalographic studies examining the activity of PPC during motor control.

The present research is comprised of four experiments that demonstrate that human error processing during motor control occurs hierarchically. The goal of Experiment One was to demonstrate that errors made during performance of a continuous tracking task elicited an ERN. The results of Experiment One did indicate this result, which suggests that the medial-frontal reinforcement learning system is sensitive to high-level motor errors. The goal of Experiment Two was to identify ERP components associated with the evaluation of low-level motor errors. Interestingly, the experimental results revealed that during performance of a joystick aiming task sudden changes in target location evoked an N140 and a P300 ERP component. Furthermore, a secondary goal of Experiment Two was to extend the findings of Experiment One by demonstrating that high-level motor errors made during performance of a joystick aiming task also elicited an ERN. Consistent with Experiment One, the results for Experiment Two revealed that high-level motor errors elicited an ERN.

A question remaining from Experiment Two was whether the N140 and P300 components reflected activation of a low-level error correction process for the online control of movement, or whether these components reflected another process such as the updating of a forward model for better control on subsequent trials. As with Experiment Two, the results for Experiment Three demonstrated that target perturbations elicited a P300 component, however, an N140 component was not observed. Comparison of the timing of the P300 with behavioural modifications associated with the online control of movement indicated that the P300 was too slow to be directly related to the online control of movement. Finally, the goal of Experiment Four was to further investigate whether there is an ERP component associated with the processing of low-level motor errors. To accomplish this, in Experiment Four participants made visually-guided and memory-guided aiming movements to a target location. The behavioural results from Experiment Four suggested that participants engaged online control processes to a greater extent in the visually-guided conditions, a result which was supported by differential parietal ERP activity between the visually-guided and memory-guided conditions.

# Experiment One[1]

## *Abstract*

Human goal-directed behaviour depends on multiple neural systems that monitor and correct for different types of errors. For example, tracking errors in continuous motor tasks appear to be processed by a system involving posterior parietal cortex, whereas errors in speeded response and trial-and-error learning tasks appear to be processed by a system involving frontal-medial cortex. To date, it is unknown whether there is a functional relationship between the posterior and frontal error systems. We recorded the event-related brain potential (ERP) from participants engaged in a tracking task to investigate the role of the frontal system in continuous motor control. Our results demonstrate that tracking errors elicit temporally distinct error-related ERPs over frontal and posterior regions of the scalp, suggesting an interaction between the subcomponents of a hierarchically organized system for error processing. Specifically, we propose that the frontal error system assesses high-level errors (i.e., goal attainment) whereas the posterior error system is responsible for evaluating low-level errors (i.e., trajectory deviations during motor control).

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[1] This experiment has been published: Krigolson, O. E., & Holroyd, C. B. (2006). Evidence for hierarchical error processing in the human brain. *Neuroscience, 137*(1), 13-17.

## *Introduction*

Errors differ in magnitude. A person driving a car, for example, is continually correcting small errors in the car's trajectory to accommodate the uneven surface of the road. However, a more serious error may occur when the person driving the car turn lefts at a street corner where they had intended to turn right. Studies in the cognitive neuroscience of motor control have indicated that such errors are processed by different neural systems (Kawato, 1999; Shadmehr & Wise, 2005; Wolpert & Ghahramani, 2000). Much of this work has focused on the role that posterior parietal cortex (PPC) plays in the online control of movement. This brain area is thought to estimate hand location in real-time and to compute motor errors (Desmurget et al., 2001) by predicting and evaluating peripheral feedback and/or an efference copy of the motor command (Desmurget & Grafton, 2000). In this manner the posterior error system can continuously modify motor output to adjust for "low-level" errors, such as updating a vehicle's trajectory to accommodate unexpected perturbations. Thus impairments to this system disrupt the ability to make online motor adjustments while a movement is in progress (Desmurget et al., 1999; 2001; Grea et al., 2002; Pisella et al., 2000). By contrast, frontal parts of the brain appear to detect and correct errors that violate "high-level" goals of the system, such as taking a wrong turn while driving. In particular, studies of the error-related negativity (ERN), a component of the event-related brain potential (ERP) sensitive to error commission, suggest that the anterior cingulate cortex (ACC) may comprise part of a generic error processing system for reinforcement learning (Brown & Braver, 2005; Holroyd & Coles, 2002; Holroyd, Larsen, & Cohen, 2004a; Holroyd, Nieuwenhuis, Mars, Coles, 2004b; Holroyd, Yeung, Coles, & Cohen, 2005). These studies have revealed that a “response ERN” is elicited when participants press the incorrect button in speeded response time tasks (Gehring et al., 1993; Holroyd & Coles, 2002; Holroyd et al., 2004b; Holroyd et al., 2005), and that a “feedback ERN” is elicited when people experience an outcome that is worse than anticipated (Holroyd & Coles, 2002; Holroyd et al., 2005; Miltner et al., 1997; Nieuwenhuis, Holroyd, Mol, & Coles, 2004). Both the response ERN and feedback ERN are thought to reflect error signals that indicate a violation of a “high-level” goal and that are utilised for the adaptive modification of behaviour.

An important unresolved question concerns whether these different neural systems can process errors in parallel and, if so, how the systems interact. Here we demonstrate that the frontal system contributes to continuous motor control by showing that tracking errors elicit an ERN. Further, we show that these errors also elicit a subsequent negative deflection in the ERP over posterior parietal cortex. These results suggest a hierarchical interaction between the frontal and posterior elements of a general system for error processing.

## *Methods*

Fifteen undergraduate participants (6 male, 9 female) performed a computer tracking task by manipulating a joystick to keep a cursor centred between two moving barriers. The barriers moved in unison according to a predictable sequence of alternating left and right movements separated by brief stationary periods in the middle of the screen (*straightaway sections*). A *tracking error* was defined as a contact between the cursor and one of the barriers. Successful performance consisted of the participant maintaining the cursor in a central location between the two barriers (*on target*). In addition, on a randomly-selected subset of the straightaway sections (20%) participants encountered a *difficult corner*. At these times the barriers moved rapidly and unpredictably to the left or to the right, with an equal probability of moving in either direction. Further, on half of the difficult corners the participant maintained full control of the cursor (*unlocked difficult corners*). The speed of these barrier movements ensured that participants always made an error whenever the unlocked difficult corners occurred. By contrast, on the other half of the difficult corners the computer program moved the cursor with the barriers (*locked difficult corners*) so a tracking error did not occur. For the locked corners the period of time the computer controlled the participant's cursor was matched on a trial to trial basis with the duration to barrier contact associated with the preceding unlocked difficult corner. The locked and unlocked difficult corners were identical in all other respects. Importantly, these conditions allowed a comparison of the ERPs associated with correct trials and error trials while controlling for a general effect of surprise induced by the sudden barrier movement (Holroyd, 2004). Electroencephalogram data were recorded from 38 electrodes using a 10-20 layout and were analysed using standard techniques (see online supplementary material for more detail). For the error trials, the ERP data were averaged according to the time of the barrier contact. For the correct trials, the ERP data were averaged according to times that were matched with the barrier contact times on the error trials. Additionally, ERP data were averaged for the occasional tracking errors that occurred during the regular performance of the task, independent of the unlocked difficult corners (*regular tracking errors)*.

## *Results*

Participants on average experienced 79 unlocked difficult corners, 80 locked difficult corners, and made 108 regular tracking errors throughout the course of the experiment. The ERP associated with the unlocked trials was more negative than the ERP associated with the locked trials from 26 ms before to 150 ms after the tracking error occurred. This difference was maximal at channel FCz, a finding that is consistent with previous observations of the ERN (Gehring et al., 1993; Holroyd & Coles, 2002; Holroyd et al., 2004a, 2004b; Holroyd et al., 2005; Miltner et al., 1997)(Figure 2a). A peak analysis of the unlocked-locked difference wave at channel FCz (Figure 2c) demonstrated that tracking errors resulted in a significantly greater negativity than on-target performance [$t(14) = -6.51$, $p < 0.001$, -3.61 uV difference 73 ms after the barrier contact]. Furthermore, an onset analysis (Rodriguez-Fornells, Kurzbuch, & Munte, 2002) conducted on the difference wave indicated that this negative deflection began approximately 26 ms before the barrier contact. These findings were confirmed by the results of a spatiotemporal principal component analysis (PCA)(Dien, 2002; Dien, Spencer, & Donchin, 2003) of the ERP data, which yielded 11 spatial factors that accounted for 95.6% of the total variance. The first of these spatial factors exhibited loadings with a frontal-central scalp distribution (Figure 3a; 0.93, 0.93, 0.95 loadings over channels FC1, FCz, and FC2, respectively). A temporal PCA on the scores associated with the first spatial factor yielded a temporal factor (accounting for 34.4 % of the total variance) with maximal loadings (> 0.9) from 70 to 122 ms after the barrier contact. This epoch corresponded to the time of the negative peak difference recorded at channel FCz. Finally, a comparison was made between the tracking errors made during the normal tracking pattern and the locked and unlocked corners. This analysis revealed that the negativity elicited by the regular tracking errors was about the same amplitude as that of the unlocked corners [$t(14) = 0.34$, $p > 0.05$], but was significantly larger than that

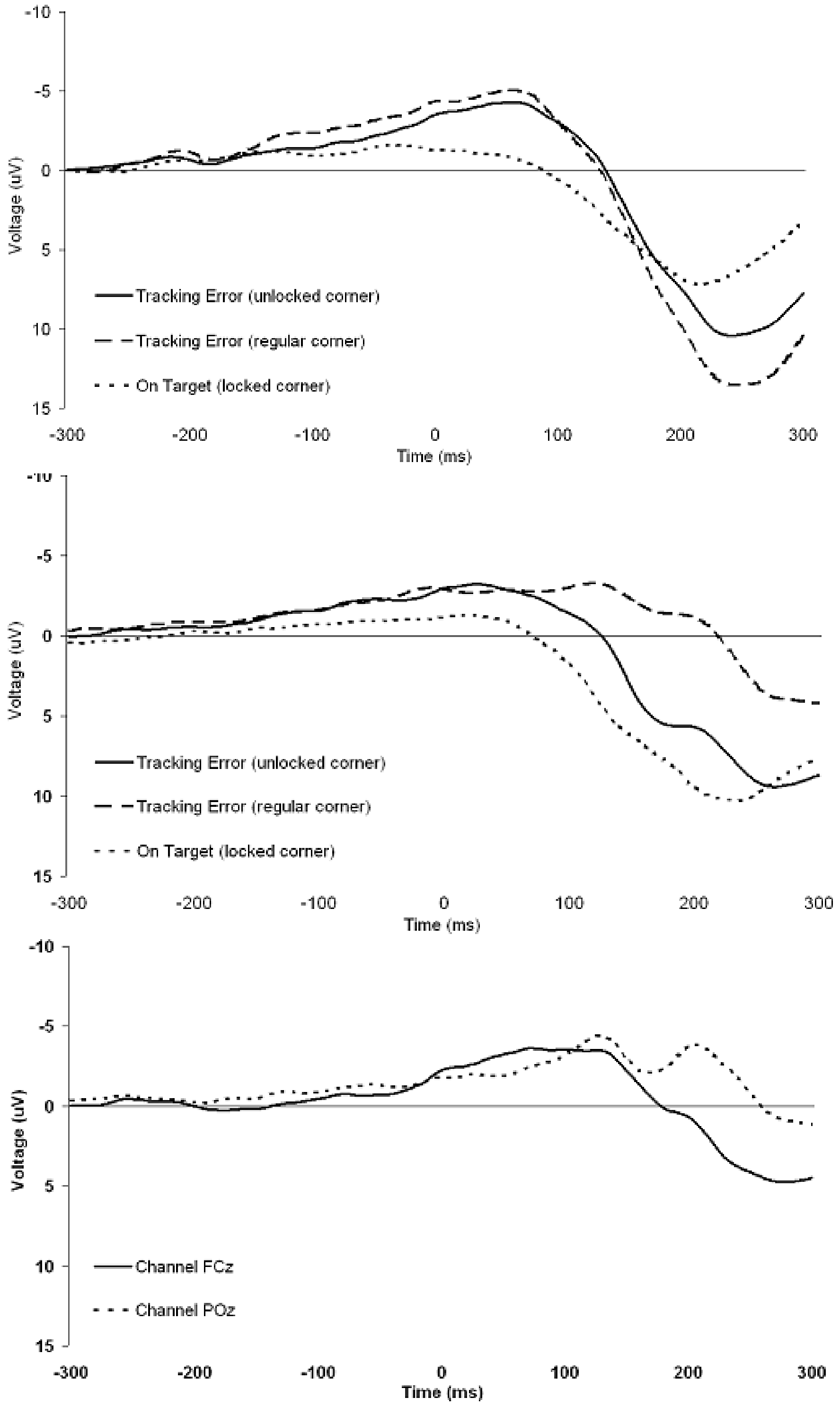

**Figure 2. (a) Averaged ERP waveforms recorded at channel FCz for unlocked tracking errors, regular tracking errors, and locked on-target events. (b) Averaged ERP waveforms recorded at channel POz for both unlocked tracking errors, regular tracking errors, and locked on-target events. (c) Averaged ERP difference waves associated with channels FCz and POz. Zero ms 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.**

of the locked corners [$t(14) = 3.85$, $p < 0.001$] (see Figure 2a). The spatiotemporal PCA also revealed a second spatial factor with loadings that were maximal over posterior areas of the scalp (Figure 3b; 0.96 and 0.89 loadings over channels POz and Oz, respectively). A temporal PCA on the scores associated with the second spatial factor yielded a temporal factor (accounting for 35.0 % of the total variance) with maximal loadings (>0.9) from 146 to 166 ms after the barrier contact. To explore this finding further, we conducted a peak analysis of the locked-unlocked difference wave associated with channel POz. This analysis revealed a negativity after tracking errors that peaked 82 ms later than the frontal negativity recorded at channel FCz [$t(14) = -4.29$, $p < 0.001$, -4.40 uV difference] (see Figure 3b, 3c for more detail).

## *Discussion*

The ERP component observed immediately following tracking errors in the present study is consistent with previous observations of the response-ERN (Gehring et al., 1993; Holroyd & Coles, 2002; Holroyd et al., 2005) and the feedback-ERN (Holroyd & Coles; Holroyd et al., 2004a; Miltner et al., 1997). Specifically, we observed during sudden barrier movements a negativity that peaked 73 ms following tracking errors but that was reduced or absent when participants remained on target. The frontal-central spatial distribution of this component is consistent with previous reports that the ERN is generated in frontal-medial cortex, probably in the ACC (Holroyd & Coles; Holroyd et al., 2004c; Miltner et al., 1997). Nevertheless, the timing of this negativity is different from that of the response ERN and the feedback ERN, presumably because the error information associated with tracking errors becomes available to the system at a different time. Note that the onset analysis of the ERN waveform

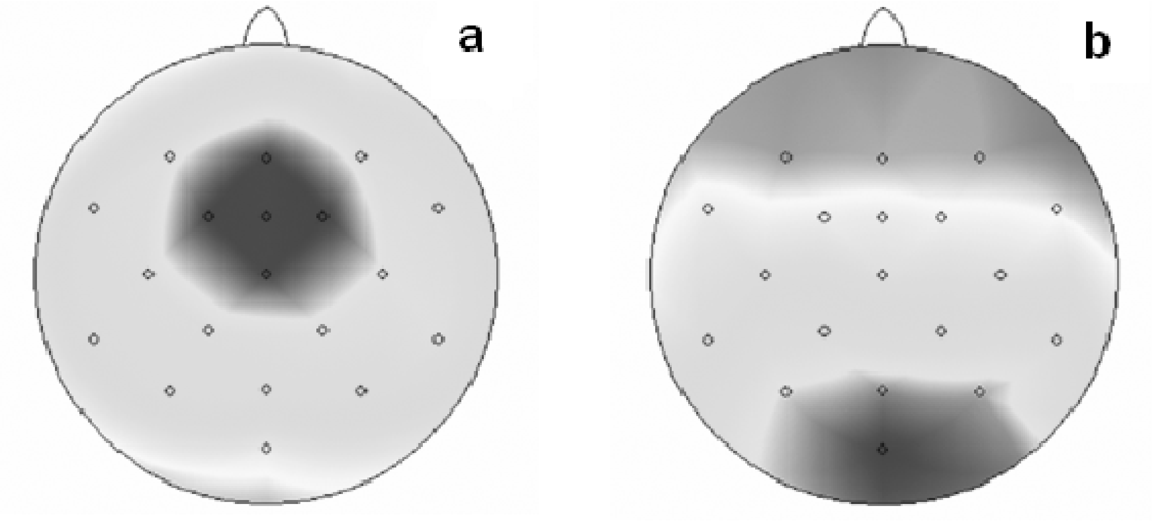

**Figure 3. Spatial PCA factor loadings projected onto the surface of the human head for the first (a, frontal-central, accounting for 42.7% of the total variance) and second (b, posterior, accounting for 32.0% of the total variance ) factors. The top of each map points toward the nose.**

revealed that the frontal-medial system began to detect the error in advance of the actual barrier contact, rather than subsequent to the barrier contact, in which case it would have been expected to elicit a classic feedback ERN peaking about 250 ms following the error. This negativity also does not appear to depend on an efference copy of the motor command, as is the case with the response ERN (Allain, Hasbroucq, Burle, Grapperon, & Vidal, 2004). Rather, it appears that the frontal system can detect these errors by adopting a predictive mode of control (Desmurget & Grafton, 2000; Holroyd & Coles, 2002; Holroyd et al., 2005). Although commonly thought to be processed by posterior parts of the brain (Desmurget et al., 1999; Desmurget & Grafton; Desmurget et al., 2001; Grea et al., 2002; Kawato, 1999; Pisella et al., 2000), these results suggest that the frontal system is sensitive to “high-level” tracking errors (i.e., barrier crossings) in a continuous motor task.

If the tracking errors in the present study are indeed evaluated by the frontal system, what then is the role of the posterior system? Previous research has demonstrated that “low-level” motor errors (i.e., trajectory modifications) are evaluated by PPC (Desmurget et al., 1999; Desmurget & Grafton, 2000; Desmurget et al., 2001; Grea et al., 2002; Kawato, 1999; Pisella et al., 2000). It has been suggested that the posterior error system residing in PPC either operates using a forward model of control (Desmurget & Grafton, 2000; Wolpert & Ghahramani, 2000) or in an online manner using visual feedback (Chua & Elliott, 1993; Elliott et al., 2001; Goodale et al., 1986; Goodale, Westwood, & Milner, 2004). Interestingly, the results of the present study revealed a negative deflection in the ERP that was distributed over occipital-parietal regions of the scalp and that peaked about 82 ms after the ERN. To our knowledge these data comprise the first ERP evidence of a posterior error system.

One may ask why posterior activity was not revealed in the ERP during the period before the tracking error occurred. In the present study the errors elicited by the unlocked difficult corners occurred very rapidly (on average about 218 ms following the onset of the corner) and unpredictably. Although the posterior system may have attempted to prevent a tracking error from occurring, the speed and the unpredictability of the unlocked difficult corners may have been beyond its capacity to correct. This inference is in line with models that suggest the posterior motor control system depends of visual feedback during a movement (i.e., Goodale et al., 2004) and is supported by the results of goal directed reaching experiments that have demonstrated that participants are not able to adjust movement trajectories during very rapid movements (Carlton, 1981; Desmurget et al., 1999). Furthermore, the unpredictable nature of the unlocked difficult corners may have negated the ability of a predictive error system to utilize a forward model of control. Instead, in the present study the frontal-medial system appears to have determined that these tracking errors violated a high-level goal of the system, namely, to avoid crossing the barriers. It seems likely that an optimal movement control strategy would most likely involve both frontal and posterior systems operating in both feedback and feedforward manners (Desmurget & Grafton, 2000; Holroyd & Coles, 2002; Seidler, Noll, & Thiers, 2004). As such, one possible explanation for timing of the frontal-central and posterior ERP components in the present study may be that the high-level error information, once evaluated by the frontal system, was then communicated to the posterior system for the adaptive modification of behaviour.

In summary, we have observed for the first time that tracking errors in a continuous movement task elicit both an ERN and a subsequent ERP component that is distributed over posterior regions of the scalp. These results indicate that the frontal-medial system is sensitive to errors in a computational domain normally associated with posterior parts of the brain, and suggest an interaction and posterior elements of a hierarchically organised system for error processing.

# Experiment Two[2]

## *Abstract*

Error processing during motor control involves the evaluation of “high-level” errors (i.e., failures to meet a system goal) by a frontal system involving anterior cingulate cortex and the evaluation of “low-level” errors (i.e., discrepancies between actual and desired motor commands) by a posterior system involving posterior parietal cortex. We have recently demonstrated that high-level errors committed within the context of a continuous tracking task elicited an error-related negativity (ERN) – a component of the event-related brain potential (ERP) generated within medial-frontal cortex that is sensitive to error commission. The purpose of the present study was to demonstrate that low-level motor errors do not elicit an ERN, but may instead evoke other ERP components associated with visual processing and online motor control. Participants performed a computer aiming task in which they manipulated a joystick to move a cursor from a start to a target position. On a random subset of trials the target jumped to a new position at movement onset, requiring the participants to modify their current motor command. Further, on one half of these "target perturbation" trials the cursor did not respond to corrective movements of the joystick. Consistent with our previous findings, we found that the uncorrectable errors elicited an ERN. We also found that the target perturbations on both correctable and uncorrectable trials did not elicit an ERN, but rather evoked two other ERP components, the N100 and P300. These results suggest that medial-frontal cortex is insensitive to low-level motor errors, and are in line

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[2] This experiment has been published: Krigolson, O. E., & Holroyd, C. B. (2007b). Hierarchical error processing: different errors, different systems. Brain Research, 1155, 70-80.

with a recent theory that holds that the P300 reflects stimulus-response optimization by the impact of locus coeruleus activity on posterior cortex.

## *Introduction*

Human error processing appears to be hierarchically organised such that different neural systems are tasked with different types of error evaluation (Krigolson & Holroyd, 2006; but see also (Doya, 2000; Doya, Kimura, & Kawato, 2001). On one hand, the motor system continuously corrects for "low-level" motor errors as movements unfold. Within a hierarchical framework, low-level errors are defined as discrepancies between the actual and appropriate motor command precipitated by neuromotor noise or by unexpected changes in the movement environment. For example, as one reaches to pick up a glass, the motor system continually adjusts the reaching trajectory so that the hand accurately finds the target. Importantly, these "low-level" errors are correctable—in the sense that such minor discrepancies can be easily overcome—and appear to be evaluated and corrected by error systems associated with posterior parts of the brain (see below). On the other hand, the motor system must also recognize "high-level" errors that indicate that a movement goal can not be achieved. Within the context of the above example, the motor system also determines whether or not the glass has been successfully attained so that it can plan subsequent motor commands accordingly. We have recently provided evidence that high-level errors in continuous motor tasks are processed within medial-frontal cortex (Krigolson & Holroyd). Note that within this hierarchical framework, low-level errors become high-level errors if left uncorrected. Thus, if for some reason the posterior system is not able to correct a discrepancy between the actual motor command and the appropriate motor command, then a high-level error will ensue.

Seminal research by Woodworth (1899) indicated that movements can be corrected as they unfold, a hypothesis confirmed by research demonstrating that ongoing motor plans can be rapidly adjusted during goal-directed reaching (Goodale et al., 1986). A large body of evidence suggests that the neural substrates underpinning real-time low-level error evaluation include posterior parietal cortex (PPC) and the cerebellum (Blakemore et al., 2001; Desmurget et al., 1999; Desmurget & Grafton, 2000; Desmurget et al., 2001; Grea et al., 2002; Miall, Malkmus, & Robertson, 1996; Miall, Reckess, & Imamizu, 2001; Pisella et al., 2000). Whether these online adjustments depend on a predictive forward model of control (Desmurget & Grafton, 2000; Wolpert & Ghahramani, 2000) or on a feedback-based control mechanism (Chua & Elliott, 1993; Goodale et al., 1986; Goodale et al., 2004; Grea et al., 2002; Heath, 2005; Heath et al., 1998; Khan et al., 2006; Khan & Lawrence, 2005; Khan, Lawrence, Franks, & Buckolz, 2004; Khan et al., 2003; Krigolson & Heath, 2004) remains unclear. Regardless of which position is correct, it is evident that low-level error information can be used to modify ongoing motor behaviour while an action is in progress.

Recent electroencephalographic studies have identified a component of the event-related brain potential (ERP) associated with high-level error processing. Seminal work by two independent research groups found that "slips" made during a speeded response task elicited a negative deflection in the ERP peaking about 100 ms after error commission (the response error-related negativity: rERN)(Falkenstein et al., 1991; Gehring et al., 1993). Subsequent research by Miltner, Braun, and Coles (1997) demonstrated that error feedback in trial-and-error learning tasks elicited a similar negative deflection in the ERP that reaches maximum amplitude about 250 ms following feedback onset (the feedback error-related negativity: fERN). Source localisation studies of the rERN and the fERN have suggested a common source for both ERP components within anterior cingulate cortex (ACC: Holroyd et al., 2004c; Miltner et al., 1997; but see also Nieuwenhuis, Heslenfeld, von Geusau, Mars, Holroyd, & Yeung, 2005b). Together, the rERN and fERN are proposed to be elicited by a generic error processing system evaluating, respectively, an efference copy of the motor command and external error information. Specifically, the Reinforcement Learning theory of the error-related negativity (ERN)(RL-ERN theory, Holroyd & Coles, 2002) holds that the amplitude of the ERN is determined by the impact on the ACC of a reinforcement learning signal carried by the midbrain dopamine system from the basal ganglia. Further, the theory holds that the error signal is generated by the earliest indication that events are worse than expected: an rERN is elicited when the efference copy of the motor command provides the first indication that an error has occurred, and a fERN is elicited when external information provides the first information that an error has occurred (for a review see Holroyd et al., 2004b).

The ERN also appears to be elicited by errors made during the performance of continuous motor tasks. In a recent study participants performed a continuous computer tracking task in which they attempted to keep a cursor centered between two moving barriers (Krigolson & Holroyd, 2006). In this task an error was defined as contact between the cursor and either of the barriers. We found that high-level tracking errors (cursor - barrier contact) elicited a negative deflection in the ERP, with a latency and scalp distribution consistent with the ERN. These results indicate that medial-frontal cortex is sensitive to high-level errors in continuous motor tasks. Further, we have recently replicated this finding and demonstrated that medial-frontal cortex is also sensitive to internal and external information predicting upcoming tracking errors (Krigolson & Holroyd, 2007a). Specifically, we found that ERNs were elicited by visual cues and by losses of joystick control when these events predicted subsequent tracking errors.

Our primary goal in the present experiment was to identify ERP correlates evoked by the evaluation of low-level motor errors. Specifically, we hoped to demonstrate that low-level motor errors do not elicit an ERN, but instead elicit other posterior ERP components. In addition, we also hoped to provide further evidence for the hierarchical error processing hypothesis by demonstrating that high-level errors committed in a manual aiming task also elicit an ERN. We had participants perform a computer based manual aiming task with three experimental conditions randomly intermixed across trials. In the first experimental condition (control), participants manipulated a joystick to move a cursor horizontally from a start position to a target square. In the second experimental condition (correctable), the target square jumped to a new vertical location immediately following movement onset, forcing participants to adjust for the target perturbation with a further manipulation of the joystick. In the third experimental condition (uncorrectable), the target jumped as in the correctable condition, but the participants' cursor failed to respond to corrective joystick movements. By creating a discrepancy between the actual motor command (joystick movement towards the original target location) and the appropriate motor command (joystick movement towards the new target location), the target perturbations in the correctable and uncorrectable conditions both resulted in low-level motor errors. Given that these errors were correctable in principle - and thus constituted low-level errors by definition - we predicted that the target perturbations would elicit posterior ERP components but not an ERN. By contrast, the failure of the joystick in the uncorrectable condition constituted a high-level error as it made the goal of reaching the target unattainable. In these instances we predicted that the high-level errors would elicit an ERN.

## *Methods*

### *Participants*

Fifteen right-handed college aged participants (9 male, 6 female) with no known neurological impairments and with normal or corrected-to-normal vision took part in the experiment. All of the participants were volunteers who received extra-credit in a first or second year psychology course for their participation. 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 a computer monitor in an electromagnetically shielded, soundproof booth and used a standard USB joystick to perform a computerized aiming task (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 website of the Brain and Cognition Laboratory at the University of Victoria, Victoria, Canada). To perform the task, participants used a joystick to move a cursor (a 0.5 cm by 0.5 cm red square) 24 cm from a start position square (a 1.5 cm by 1.5 cm white square) on the left hand side of a computer display to target position (a 1.5 cm by 1.5 cm white square) on the right hand side of a computer display. Pressure applied to the joystick in the desired direction moved the cursor in the corresponding direction. The cursor moved at a constant velocity, irrespective of the amount of pressure applied. Participants could direct the cursor only in the up/down and rightward directions; leftward movements (away from the target) were not possible. Note that this control structure allowed for diagonal forward movements (i.e., to the right and up, to the right and down). Once a movement started, the cursor's trajectory could be modified along the vertical axis but not along horizontal axis; the forward movement could only be stopped, not reversed. To stop the cursor, participants returned the joystick to its resting position.

The experiment consisted of 300 trials that were divided between three randomly intermixed aiming conditions (100 trials in each condition). Each trial began with a two second visual preview period during which the participant could see the start position, the target position, and the cursor. At the end of the preview period the cursor changed colour from white to green, indicating to the participants that the movement could begin. If the participant applied pressure to the joystick before the cursor changed colour, then the trial was immediately cancelled, an error message was presented, and another trial (selected at random) began.

In the first aiming condition (control), participants were required to move the cursor horizontally from the start position to the target position. In the second (correctable) and third (uncorrectable) aiming conditions, the target simultaneously disappeared and re-appeared at a new location immediately following movement onset (within one refresh cycle), either up or down 8 cm from its initial position (equal probability for each direction). As such, trials in these conditions required participants to correct their initial movement by shifting pressure on the joystick from a horizontal to a diagonal direction. However, in the uncorrectable condition the cursor was unresponsive to diagonal displacements of the joystick. Thus, it was possible to correct for the target perturbations in the correctable condition but not in the uncorrectable condition. Note that participants could not determine at the start of each trial which condition would occur, as the target perturbations always followed movement onset. Furthermore, following a perturbation, participants could differentiate the uncorrectable from the correctable conditions only by attempting a corrective movement[3]. Participants were instructed to be as accurate as possible and to stop the cursor so that it made contact with the target.

Participants performed five blocks of 60 trials with 20 trials from each of the three conditions randomly intermixed within each block. Between blocks participants relaxed during self-paced rest periods. The base aiming task (the control condition) was explained to participants before the experiment. Participants were also told that on some trials (the correctable and uncorrectable conditions), the target would jump to a new location, and that on some of these trials the joystick would not work and as such they would be unable to reach the movement target. Participants then completed five practice trials in each of the aiming conditions before the experiment began.

### *Behavioural Analysis*

The aiming program recorded reaction time (the time from when the cursor changed colour to movement onset in ms), movement time (the time from movement

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[3] Note that we were concerned that predictive attempts to correct for a target perturbation (i.e., a diagonal movement of the joystick) before a target perturbation occurred would bias our results. As such, if diagonal pressure was applied to the joystick before a target perturbation occurred then we removed that trial from further analysis (less than 1% of experimental trials were discarded because of this criterion).

onset to movement offset in ms), and the time of the corrective movement in the correctable and uncorrectable conditions (in ms). Accuracy was recorded as a binary value (whether the cursor ended the trial on-target or off-target). Participants also completed a short questionnaire upon completion of the experiment. Behavioural data were analysed using a univariate ANOVA with post-hoc comparisons done by simple effects analysis. An alpha level of 0.05 was assumed for all statistical tests.

### *Data Acquisition*

The electroencephalogram (EEG) was recorded from 41 electrode locations using BrainVision Recorder software (Version 1.3, Brain Products, GmbH, Munich, Germany). The electrodes were mounted in a fitted cap with a standard 10-20 layout and were referenced to the average. The vertical and horizontal electro-oculograms 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 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).

### *Data Analysis*

The EEG data were filtered through a (0.1 Hz – 20 Hz passband) phase shift free Butterworth filter and re-referenced to linked mastoids. Ocular artefacts were removed using the algorithm described by Gratton, Coles and Donchin (1983). Trials in which the change in voltage at any channel exceeded 35 uVs per sampling point were also discarded. In total, less than 1% of the data were discarded. All waveform segments (regardless of marker position) were baseline corrected to a 200 ms epoch from 500 to 300 ms before the movement initiation cue.

To evaluate the effect of the target perturbation on the ERP, 1000 ms epochs of data (from 200 ms before movement onset to 800 ms after movement onset) were extracted from the continuous EEG for each trial, channel, and participant for each of the three experimental conditions. ERPs were then created by averaging the EEG data by condition for each electrode, channel, and participant. These data were submitted to a spatiotemporal principal component analysis (STPCA: see below) to identify electroencephalographic activity that co-varied across electrodes and time. Subsequent to this analysis two separate difference waves were created by subtracting the control waveform from the correctable and uncorrectable waveforms for each channel and participant. A peak detection analysis was conducted on each of the difference waves for channels of interest identified by the STPCA. The ERN was identified as the maximum negative deflection in the difference wave from 100 to 400 ms and the P300 (see below) was identified as the maximum positive deflection in the difference wave from 200 to 500 ms.

To analyse the corrective movements associated with target perturbations, a 1000 ms window of data was extracted and averaged with respect to the time of the first diagonal (corrective) movement of the joystick in the correctable and uncorrectable conditions (400 ms before the corrective movement, 600 ms after). These data were submitted to a STPCA. Following the STPCA difference waves were created by subtracting the correctable ERPs from the uncorrectable ERPs for each channel and participant. A peak analysis was conducted on this difference waveform using the criteria outlined above.

The amplitudes of the difference waves were tested against zero with a one sample t-test. Amplitudes and latencies of the difference waves were compared directly with paired samples t-tests. For all statistical tests an alpha level of 0.05 was assumed.

### *Spatiotemporal Principal Component Analysis*

As noted above, the ERP data were submitted to STPCA (analysed using the MATLAB PCA toolbox; Dien, 2002; Dien et al., 2003; Krigolson & Holroyd , 2006). First, spatial factor loadings were obtained by submitting to a PCA the observations (for the target perturbation analysis: 750 ERP samples, 250 time points x 3 conditional waveforms; for the movement correctable analysis: 600 ERP samples, 200 time points x 3 conditional waveforms) for each participant and electrode (Varimax rotation, no Kaiser correction). Next, the spatial factor scores for each spatial factor were submitted separately to a temporal PCA. 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

### *Behavioural Data*

Reaction time did not differ between the three experimental conditions, $F(2,28) = 2.22$, $p > 0.05$ (see Table1 for all behavioural results). The analysis of movement time yielded a main effect for experimental condition, $F(2,28) = 7.76$, $p < 0.01$, indicating that movements in the uncorrectable condition were longer than in the control ($t(14) = 3.01$, $p < 0.01$) and the correctable ($t(14) = 2.44$, $p < 0.05$) conditions. The time at which participants attempted to correct for the target perturbation did not differ between the correctable and uncorrectable conditions, $t(14) = 0.25$, $p > 0.05$. Participants were significantly more accurate in the control condition than in the correctable condition, t(14) = 3.60, $p < 0.01$ (note that accuracy associated with the uncorrectable condition is not meaningful as by design participants always erred on these trials). Unfortunately, an electroencephalographic analysis of endpoint error could not be conducted due to an insufficient number of off-target trials in the control and correctable conditions.

Table One
Behavioural data as a function of experimental condition.

| Condition | Reaction Time (ms) | Movement Time (ms) | Correction Time (ms) | Accuracy (%) |
|---|---|---|---|---|
| Control | 519 +/- 12 | 1377 +/- 15 | n/a | 93.2 |
| Correctable | 534 +/- 14 | 1391 +/- 21 | 471 +/- 122 | 84.1 |
| Uncorrectable | 530 +/- 14 | 1441 +/- 129 | 470 +/- 107 | 0 |

### *Electroencephalographic Data: The Target Perturbation*

A spatial PCA conducted on the three ERPs averaged to the time of the target perturbation yielded a primary spatial factor that accounted for 40.5% of the spatial variance, and that exhibited loadings that were maximal at channel Pz (see Figure 4a). The factor scores associated with this factor were submitted to a temporal PCA, which yielded an epoch from 324 to 500 ms that accounted for 43.9% of the temporal variance. These results are consistent with the spatial distribution and latency of the P300 (Dien et al., 2003; Dien, Spencer, & Donchin, 2004; Donchin & Coles, 1988; Spencer, Dien, & Donchin, 2001). Given these results, we examined the P300 directly by conducting peak analyses on the difference waves constructed by subtracting the control ERPs from the correctable and uncorrectable ERPs recorded at channel Pz (see above; Figures 4b). Both the correctable (16.5 uV: t(14) = 8.18, $p < 0.001$) and uncorrectable (16.6 uV: t(14) = 8.19., $p < 0.001$) difference waves were characterized by a large positive deflection in this time range. The amplitude (t(14) = 0.72, $p > 0.05$) and latency (t(14) = 0.65, $p > 0.05$;

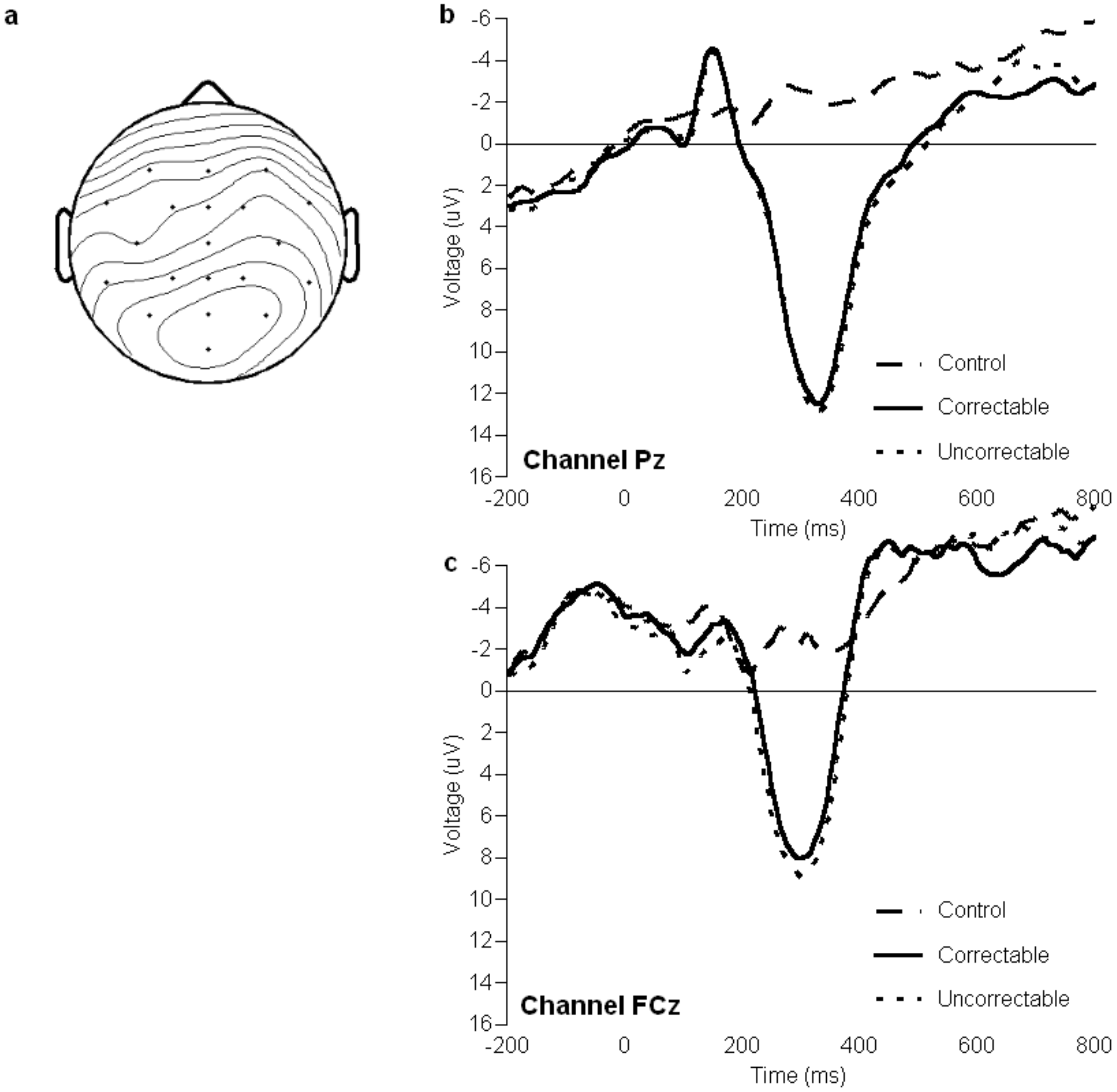

**Figure 4. Target Perturbation: (a) Spatial PCA factor loadings projected onto the surface of the human head for the posterior factor. (b) Averaged ERP waveforms recorded at channel Pz for the control, correctable, and uncorrectable conditions. (c) Averaged ERP waveforms recorded at channel FCz for the control, correctable, and uncorrectable conditions. Note that zero ms corresponds to movement onset. Negatives voltages are plotted up by convention.**

328 vs 324 ms) of the peaks of the difference waves did not differ from one another. Note that the spatial PCA did not yield a front-central PCA factor consistent with the ERN. Further, a peak analyses on the difference waves associated with channel FCz did not yield a statistically significant negative deflection within the time window of the ERN (100 – 400 ms; see Figure 4c). Post-hoc visual inspection of the data revealed an N100 component in the correctable and uncorrectable difference waves that peaked 140 ms following the target perturbation. The peak amplitude of the N100 was lateralised over left visual cortex, being maximal at channel PO7 (Figure 5), where it was statistically different from zero for both the correctable (-8.29 uV: $t(14) = 8.34$, $p < 0.001$) and uncorrectable (-8.19uV: $t(14) = 8.30$, $p < 0.001$) difference waves[4]. In sum, these results suggest that the target perturbation elicited an N100 and a P300 component, but not an ERN.

*Electroencephalographic Data: The Corrective Movement*

To evaluate the impact of uncorrectable errors, we conducted a STPCA on the ERP data averaged to the time of the movement correction[5]. This analysis yielded two spatial factors of interest: a posterior factor with maximal loadings at channels Pz (0.93) and POz (0.96) accounting for 51.2% of the spatial variance, and a frontal-central factor (Figure 6a) with maximal loadings at channel FCz accounting for 18.0% of the spatial variance. As the posterior factor appeared to be associated with the P300 that preceded the corrective movement, we focused our analysis on the frontal-central factor. The

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[4] Note that Figure 3 shows the waveforms locked to the target perturbation plotted for channel Pz. However, the statistical analysis of the N100 component was done on channel PO7 where it was maximal.

[5] For comparison purposes we also examined the ERP data for the control condition. As there was no corrective movement in this condition, we averaged the ERP data for the control condition on a trial-by-trial basis according to the time of the corrective movement on each preceding correctable or uncorrectable trial.

temporal PCA on the spatial factor scores associated with the frontal-central factor yielded a temporal factor with maximal loadings from 196 to 256 ms following the attempted correction (accounting for 22.7% of the temporal variance), an epoch consistent with the latency of the fERN. Given these results, we conducted a peak analysis on the difference waves constructed by subtracting the correctable ERPs from the uncorrectable ERPs (Figures 6b and 6c) for channel FCz. This analysis revealed a significant negative deflection (t(14) = 8.9, p < 0.001: -5.3 uV) with a latency of 248 ms and a frontal-central distribution, a result consistent with the spatial distribution and timing of the fERN (Ruchsow, Grothe, Spitzer, & Kiefer, 2002; Krigolson & Holroyd, 2006; Miltner et al., 1997). Note however that the movement times were longer in the uncorrectable condition relative to the correctable condition, so this difference between the ERPs could reflect differential motor activity rather than error processing *per se*. To show that this front-central negativity was not in fact a motor potential, we created difference waves for each participant based on subsets of trials in the correctable and

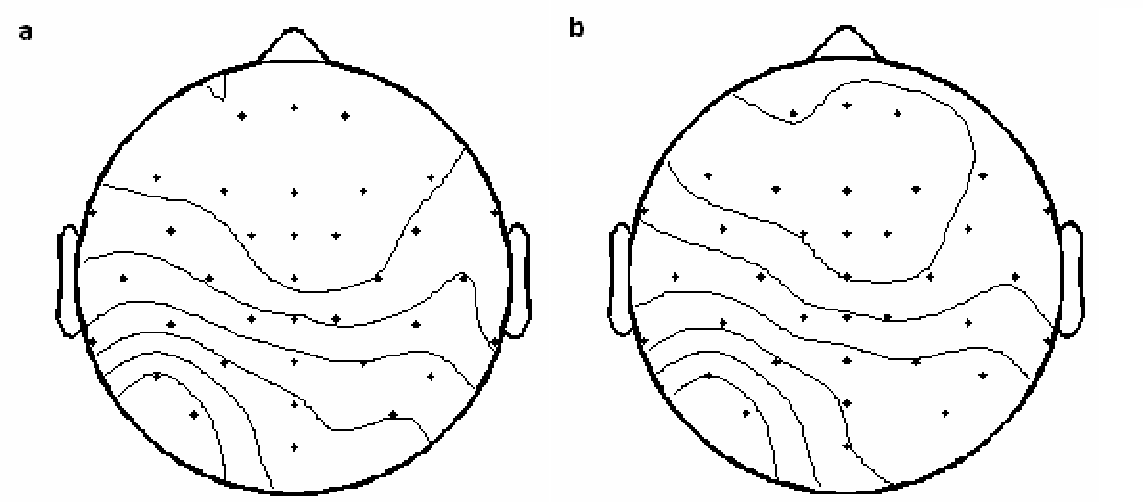

**Figure 5. Target Perturbation: (a) Scalp distribution of the N100 peak associated with the target perturbation for the correctable difference wave. (b) Scalp distribution of the N100 peak associated with the target perturbation for the uncorrectable difference wave.**

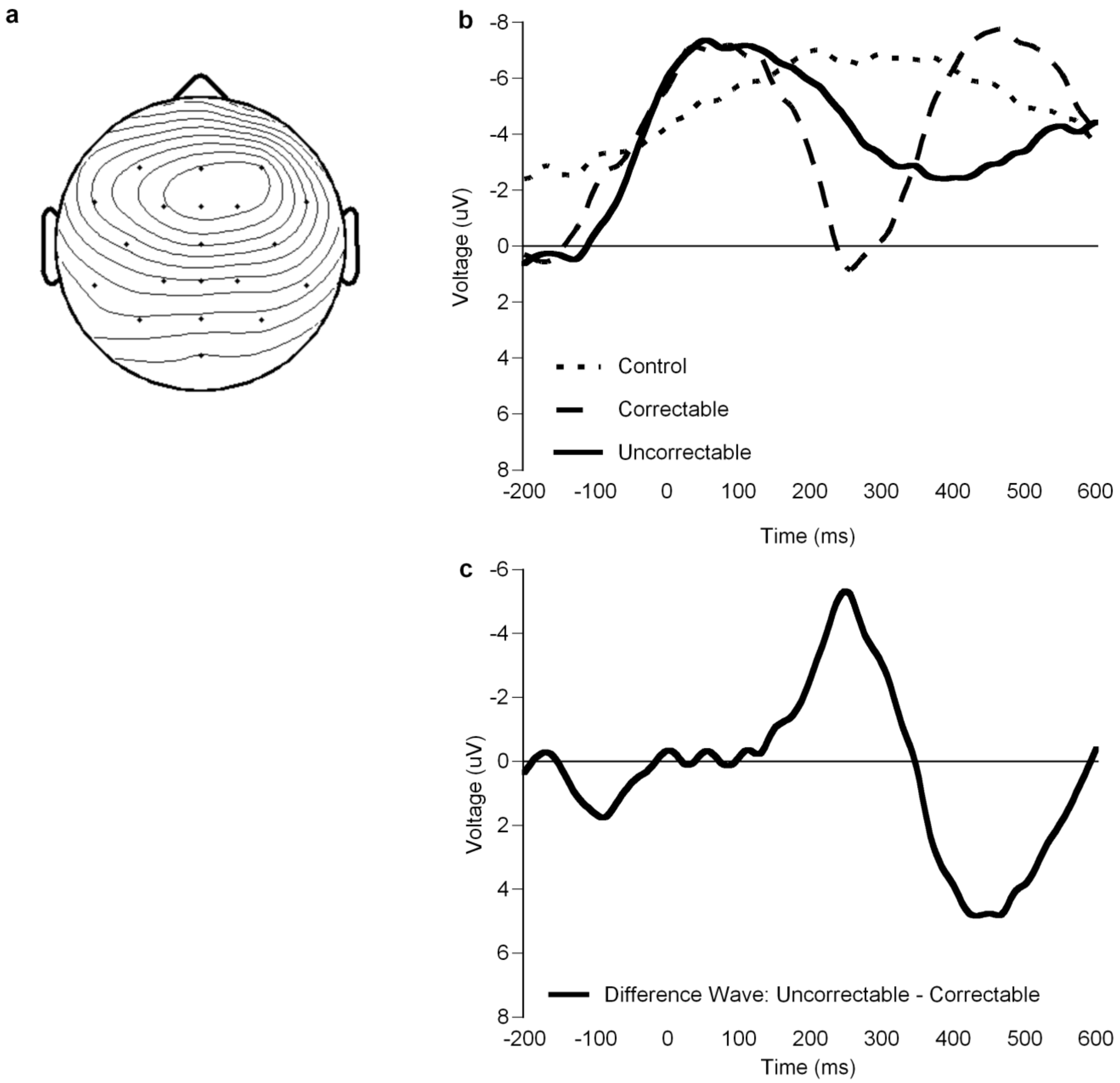

**Figure 6. Movement Correction: (a) Spatial PCA factor loadings projected onto the surface of the human head for the front-central factor. (b) Averaged ERP waveforms recorded at channel FCz for the control, correctable, and uncorrectable conditions. (c) Difference wave (uncorrectable – correctable) at channel FCz. Note that zero ms corresponds to movement onset. Negatives voltages are plotted up by convention.**

uncorrectable conditions that were matched according to their movement times. In line with the previous analysis, we found a significant negative deflection ($t(14) = 3.9$, $p < 0.01$: - 9.00 uV) with a latency of 260 ms and a front-central scalp distribution. These results suggest that the uncorrectable errors in fact elicited a fERN.

## *Discussion*

### *Low-Level Errors: The N100 and the P300*

In this study we sought to demonstrate that low-level motor errors do not elicit an ERN, but rather elicit other posterior ERP components associated with the processing of visual target information. Furthermore, we sought to demonstrate that high-level errors within the same trial do elicit an ERN. Participants manipulated a joystick to move a cursor from a start position on the left of a computer screen to a target position on the right. Complicating this, on some trials following movement onset the target disappeared and jumped to a new location, requiring participants to execute a corrective motor command. Further, on one half of these target perturbation trials the joystick did not respond to the corrections. At the start of each trial participants could not predict whether or not a target perturbation would occur, and at the time of the target perturbation they could not differentiate the correctable from the uncorrectable trials until they attempted the movement correction. Thus, although the perturbation invalidated the current motor command, the goal of the task (moving the cursor to the target) was in principle still attainable at the time of the perturbation. For this reason, the medial-frontal system should not have evaluated the perturbation as a high-level error, and indeed we found that the perturbation did not elicit an ERN. By contrast, the perturbation constituted a low-level motor error because it resulted in a discrepancy between the actual and appropriate motor command. Thus the posterior error system should have evaluated the perturbation as a low-level error, and in fact we found that the perturbation elicited two distinct posteriorly distributed ERP components, the N100 and the P300 (Figure 7).

The N100 was elicited shortly following the target perturbation (140 ms) and was maximal over left visual cortex. This result is consistent with previous reports that target stimuli appearing in the left or right visual field elicit a posterior N100 lateralized over the contralateral hemisphere (Kasai, Morotomi, & Katayama, 2003; Pazo-Alvarez, Amenedo, & Lorenzo-Lopez, 2004; Spironelli, Tagliabue, & Angrilli, 2006). Because all of the target perturbations in the present study occurred in the right visual field, we believe that the left lateralised N100 component reflects the detection of the target perturbation by visual cortex. Interestingly, the timing of the N100 in the present study (140 ms) is similar to the minimum time believed to be required for feedback-reliant control processes (Desmurget & Grafton, 2000; Jeannerod, 1988; Paillard, 1996).

Although it may not be surprising that the target perturbations in the present study elicited an ERP component normally evoked by changes in the visual environment, our results may be considered in terms of the functional consequences of the underlying neural process. The timing of the N100 in the present study (≈ 140ms) is too slow to simply reflect the arrival of visual information in primary visual cortex (which typically is observed as a C1 waveform and begins 40-60 ms after stimulus onset; Clark, Fan, & Hillyard, 1995). As such, it seems likely that the N100 observed in the present study may reflect neural activity associated with a more advanced stage of visual processing, such as the detection of an unexpected environmental change, the magnitude of the change, the location of the change, and so on (c.f. Milner & Goodale, 1993). Importantly, these

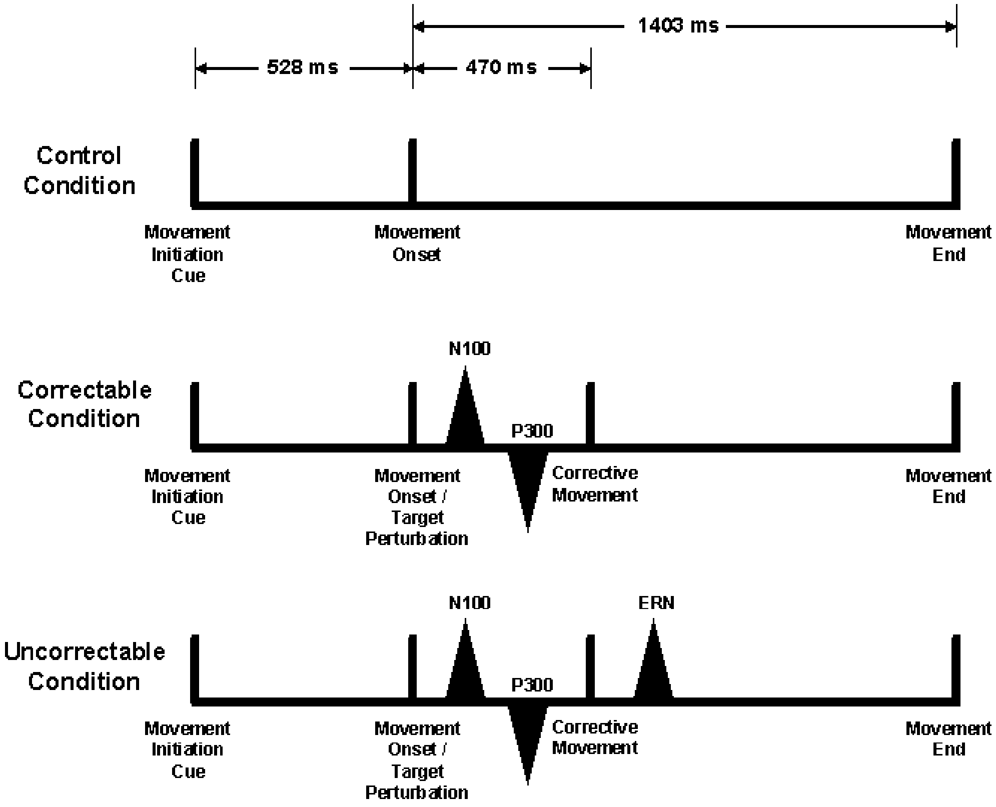

**Figure 7. Experimental Timeline: A summary of the experimental events and the evoked ERP components in the control, correctable, and uncorrectable conditions. Note that the times reported here are averages of the values reported in Table 1.**

functions are necessary for the detection and correction of low level errors by the posterior system. Although the N100 in the present study may be representative of low-level error evaluation by posterior parietal cortex, a neural region associated with the online control movement (c.f. Desmurget et al., 1999; 2001; Grea et al., 2002; Pisella et al., 2000), the scalp distribution of the N100 is inconsistent with this hypothesis. Alternatively, the scalp distribution of the N100 may be more consistent with the "ventral visual stream", and recent evidence suggests that information from the ventral stream can also be utilised for the online control of movement (i.e., Krigolson & Heath, 2004; Krigolson, Clark, Heath, & Binsted, 2007).

We also found that the target perturbation elicited an ERP component characterized by a timing, polarity, and scalp distribution that is consistent with the P300 (Donchin & Coles, 1988; Nieuwenhuis et al., 2005a). The P300 is a large, positive-going, parietally-distributed deflection in the ERP that peaks 300 ms or later following stimulus onset (Sutton, Braren, Zubin, & John, 1965), and is typically elicited by surprising events. For example, in the "oddball task" participants respond to (or silently count) infrequently occurring target stimuli, which elicit a large P300 (Bekker, Kenemans, & Verbaten, 2005; Donchin & Coles, 1988; Duncan-Johnson & Donchin, 1977; Jackson, Jackson, & Roberts, 1999). Although the location(s) of the generator(s) that produce the P300 is not exactly clear, research suggests a P3 source near the temporal-parietal junction (Calhoun, Adali, Pearlson, & Kiehl, 2006; Halgren et al., 1995; Kiss, Dashieff, & Lordeon, 1989; Knight, Scabini, Woods, & Clayworth, 1989; Menon, Ford, Lim, Glover, & Pfefferbaum, 1997; Molnar, 1994; Smith et al., 1990; Verleger, Heide, Butt, & Kompf, 1994; Yamaguchi & Knight, 1991, 1992).

As with the N100, it is unsurprising that the target perturbations elicited a P300, which is normally elicited by unexpected task-relevant events. Nevertheless, our results may provide insight into the cognitive process that generates the P300. A prominent theory of the P300 holds that this ERP component indexes “context updating” (Donchin & Coles, 1988), such that it elicited by the active updating of an internal model of the environment upon receipt of new information. It is interesting to note that an important aspect of rapid motor control involves comparing the consequences of the current motor command to the movement’s desired end-state (Desmurget & Grafton, 2000). To do so, the system must continuously predict the outcome of the current motor command while simultaneously monitoring the external environment for changes that would negate its effectiveness. This process entails the development and execution of an internal forward model of the environment that must be updated in response to unpredicted events. Given that the context-updating hypothesis proposes that the P300 is elicited by just such an occurrence (Donchin & Coles, 1988), and given the proximal location of the temporal-parietal junction to PPC, we speculate that the P300 in this task may index the revision of an internal forward model by posterior cortex and thus may reflect part of the low-level error evaluation process.

The results of the present study are also compatible with a more recent theory that holds that the P300 is evoked by the impact of phasic activity of the locus-coeruleus norepinephrine (LC-NE) system on posterior cortex (Nieuwenhuis et al., 2005a; Nieuwenhuis, Gilzenrat, Holmes, & Cohen, 2005b). Phasic activity of the LC-NE system is associated with the presentation of task relevant stimuli (Aston-Jones & Bloom, 1981; Aston-Jones, Rajkowski, Kubiak, & Alexinsky, 1994; Foote, Aston-Jones, & Bloom, 1980; Nieuwenhuis et al., 2005a) and appears to be time-locked to the behavioural response initiated by the stimulus (Bouret & Sara, 2004; Clayton, Rajkowski, Cohen, & Aston-Jones, 2004; Rajkowski, Majczynski, Clayton, & Aston-Jones, 2004). In this context, the LC-NE system appears to increase the gain of target neurons in cortex, the thalamus, and the cerebellum to optimize the decision making process (Servan-Schreiber, Printz, & Cohen, 1990).

In the present experiment the target perturbation created a low-level motor error which forced participants to rapidly change their current motor command. Given that phasic activity of the LC-NE system appears to facilitate rapid decision making, it seems reasonable to assume that the target perturbation would activate this system. The LC-NE system projects to parietal cortex (amongst other brain regions; Aston-Jones, Foote, & Bloom, 1984; Berridge & Waterhouse, 2003) and thus may have a direct impact on PPC, a neural region associated with the online control of movement (Desmurget et al., 1999, 2001; Grea et al., 2002; Pisella et al., 2000). If the LC-NE theory of P300 generation is correct (Nieuwenhuis et al., 2005a, 2005b), then this process would also elicit a P300. Consistent with this inference, it appears that the P300 in this task may reflect a rapid adjustment by the posterior system to low-level motor errors.

Of equal importance is the result that the target perturbations did not elicit an ERN. Within a hierarchical framework, errors are only evaluated as "high-level" by the medial-frontal system if they indicate that the movement goal is no longer attainable. Within the context of the present study, at the time of the target perturbations participants did not know whether or not they would lose vertical control of the cursor. As such, the target perturbations in the present study construed low-level motor errors as they resulted in discrepancies between the actual and the desired motor commands. However, the target perturbations did not indicate that the goal of the task was unattainable, and thus an ERN was not elicited as the medial-frontal system was not activated by these errors.

### *High-Level Errors and the Error Related Negativity*

The second goal of this study was to demonstrate that high-level errors committed during the performance of a discrete manual aiming task elicited an ERN. In the uncorrectable condition of the present experiment corrective joystick movements were prevented. As such, the low-level errors brought about by the target perturbations in this condition became high-level errors when joystick control was reduced. Interestingly, we found that the blocking of corrective joystick movements elicited a deflection in the ERP that exhibited a latency and scalp distribution consistent with the fERN (Holroyd et al., 2004a; Miltner et al., 1997) which is thought to be produced in medial-frontal cortex (Holroyd & Coles, 2002; but see Nieuwenhuis et al., 2005c).

It may be asked why such errors would elicit a fERN as opposed to a rERN (e.g., Gehring et al., 1993). Recall that the RL-ERN theory holds that the ERN is elicited by the first indication that ongoing events are worse than expected. According to the theory, the rERN is elicited by an efference copy of the motor command, and as such occurs relatively quickly following the error response (~ 100 ms), whereas the fERN is elicited by an external feedback stimulus and thus occurs following feedback presentation (~ 250 ms). Importantly, participants in this task attempted to correct the low-level errors on both correctable and uncorrectable trials, so in both conditions the efference copy would have indicated that the system was producing the appropriate response. Instead, participants relied on visual feedback from the cursor to determine whether or not their error correction was successful. When the joystick failed, this external visual feedback indicated that the system goal would be violated and elicited a fERN. These results are consistent with a previous finding that high-level errors made during the performance of a continuous tracking task also elicited an ERN (Krigolson & Holroyd, 2006, 2007a).

An alternative explanation for the frontal-central negativity elicited by the blocked joystick movement is that it is a motor potential. There is some evidence to suggest that enhanced motor potentials are associated with increased force production or movement duration (i.e., Slobounov & Ray, 1998). In the present experiment it is possible that motor-related differences between correctable and uncorrectable trials gave rise to the frontal-central negativity observed in the difference wave. However, the frontal-central negativity was not lateralised over left motor cortex, suggesting that it was not directly elicited by the motor correction. Furthermore, the ERPs associated with the correctable and uncorrectable conditions began to differ from one another approximately 120 ms following the attempted movement correction (Figure 6). Considering the long delay before participants responded to the target perturbation (≈ 470 ms), 120 ms would seem be too little time for the system to both detect the error and initiate the correction. On the other hand, the scalp distribution and latency of the negativity is exactly consistent with the fERN. For these reasons we believe that the front-central negativity in the present experiment is in fact a fERN

### *A Theory of Hierarchical Error Processing*

The results of the present study, together with our previous work examining the ERN in the context of a continuous tracking task (Krigolson & Holroyd, 2006, 2007a), suggest that the error processing system is organized hierarchically: high-level errors are evaluated by a neural system involving medial-frontal cortex, and low-level errors are evaluated by a neural system involving posterior and occipital cortex. The present study complements the previous work by demonstrating that low-level errors elicit an N100 and a P300, but not an ERN. Although it seems likely that the N100 and P300 do not directly reflect the sensorimotor transformations implemented by PPC for the online control of movement, they may instead reflect processes that support PPC function (such as context-updating, or LC-NE optimization of response execution). Conversely, our results indicate that only high-level errors elicit an ERN, suggesting that the medial-frontal system is concerned with only whether or not the motor command will be ultimately successful.

How do the posterior and frontal error systems work together? Within a motor learning context, high-level error information can be utilized to improve the accuracy of subsequent motor commands. For instance, it has been proposed that once a motor command is put into execution, the posterior system monitors and corrects low-level errors in real-time via a forward model of control (i.e., Desmurget & Grafton, 2000; Wolpert & Ghahramani, 2000), feedback-based control (Chua & Elliott, 1993; Goodale et al., 1986; Grea et al., 2002; Heath, 2005; Khan & Lawrence, 2005; Khan et al., 2003, 2004; Krigolson & Heath, 2004), or both. Conversely, a growing body of evidence suggests that the medial-frontal system (including the basal ganglia) plays an important role in error evaluation (i.e., Holroyd & Coles, 2002). While this frontal system may receive error information during a movement, it does not appear to act on this information unless a high-level goal is not achieved. When this occurs, the frontal system elicits the ERN. Converging evidence from monkey studies have demonstrated that the midbrain dopamine system carries a temporal difference prediction error from the basal ganglia to frontal cortex (Schultz et al., 1997; Schultz & Dickinson, 2000), including the ACC, where the impact of this signal may generate the ERN (Holroyd & Coles, 2002). Interestingly, it has recently been demonstrated that a temporal difference prediction error system can be used to train forward models (Branning, Watz, Aisa, & O'Reilly, 2005). Thus, one potential function of the high-level error signal generated in medial-frontal cortex could be to train the forward models implemented by the posterior error system.

Although the neural generators that support the type of feed-forward control outlined above remain unclear, they likely involve the supplementary motor area (Hikosaka, Nakamura, Sakai, & Nakahara, 2002; Matsumura et al., 2004)**,** the cerebellum (Floyer-Lea & Matthews, 2004; Wolpert, Miall, & Kawato, 1998), and/or the basal ganglia (Doyon & Benali, 2005; Hikosaka et al., 2002). The results of the present study, along with our previous research (Krigolson & Holroyd, 2006, 2007a), provide evidence that these feed-forward error signals are also sent to ACC in order to modify subsequent motor output (c.f. Holroyd & Coles, 2002).

### *Conclusion*

We have proposed that errors come in two kinds: high-level errors that occur when a task goal has been violated and low-level errors that reflect a currently invalid but correctable course of action. Our results indicate that low-level errors associated with a target perturbation elicit two posteriorly distributed ERP components, an N100 and a P300, but do not elicit an ERN. We suggest that the ERP components evoked by the target perturbation reflect processes that support the online control of movement. Specifically, we propose that the N100 reflects detection by the visual system of changes in the target location. We also suggest that the P300 reflects the updating of an internal model of the visual environment, which in turn facilitates an updating of the PPC's current forward model of control. Furthermore, we speculate that the P300 may also reflect the impact of phasic LC-NE activity on PPC to optimize the corrective movement. The scalp distribution and proposed neural generators of the N100 and P300 are consistent with these suggestions. In addition, our results also indicate that high-level errors elicit the ERN. Given that the ERN appears to be generated in ACC, these data support the hypothesis that the medial-frontal system, which includes the basal ganglia and the ACC, plays a role in the detection and evaluation of high-level errors in continuous motor tasks. Taken together with our previous findings, these results support our hypothesis that that human error processing is hierarchically organised.

# Experiment Three[6]

## *Abstract*

Recently, we have demonstrated that “high-level” motor errors, which indicate the failure to obtain a movement goal, elicit an error-related negativity (ERN), a component of the event-related brain potential (ERP) sensitive to response errors and error feedback (Gehring et al., 1993; Krigolson & Holroyd, 2006; Miltner et al., 1997). The human sensorimotor system also rapidly evaluates and corrects errors that occur during ongoing movements. These “low-level” motor errors, which reflect a discrepancy between the desired and the actual motor command, are thought to be evaluated within posterior parietal cortex (PPC) (Desmurget et al., 1999). Here, we recorded electroencephalographic data from participants engaged in a manual aiming task to determine whether or not PPC activity elicits an ERP correlate evoked by online motor control processes. Specifically, participants executed manual aiming movements over a graphics tablet to a target location while viewing a virtual display. On some trials, the target jumped to a new location following movement onset, requiring participants to adjust their current motor command online. We found that the target perturbations elicited a parietally-distributed ERP component, the P300, which we propose reflects the updating of a forward model for online motor control. Further, we also found that in line with our previous work, an ERN was elicited when participants ended a trial off target. To the best of our knowledge this experiment represents the first investigation using electroencephalography to examine neural activity associated with manual aiming.

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[6] This experiment has been submitted for publication.

## *Introduction*

The human sensorimotor system can rapidly accommodate changes in the movement environment. For instance, when reaching to a target, the motor system can quickly adjust the current motor command to correct for changes in target location – even if these changes occur after movement onset (Goodale et al., 1986). Interestingly, Goodale and colleagues found that participants making rapid aiming movements could change their movement trajectories to correct for a change in target location, even if the target moved during an ocular saccade. In other words, participants rapidly corrected for target “jumps” even if they were not aware that the target had moved. In conjunction with findings from other studies, Goodale et al.’s results form the basis of the action – perception model, a framework in which visual information is thought to be processed by a parallel system involving the dorsal and ventral visual streams (Milner & Goodale, 1995). Visual input relevant to action is processed within the dorsal visual stream. Recent neuroimaging and neuropsychological studies suggest that posterior parietal cortex (PPC) is one of the neural substrates within the dorsal visual stream associated with the online control of movement (Desmurget et al., 1999; Desmurget & Grafton, 2000; Desmurget et al., 2001; Grea et al., 2002; Pisella et al., 2000). While the ventral visual stream is typically associated with the processing of perceptual visual input, recent research suggests that information from the ventral stream processes is also used for movement planning and control (Krigolson & Heath 2004, Krigolson et al., 2006; Obhi & Goodale, 2005).

Despite the online control process implemented by the PPC and other brain areas, sometimes actions fail to achieve the intended goal. Such motor errors are processed by a neural system involving frontal parts of the brain, in particular the basal ganglia and anterior cingulate cortex (ACC). We recently conducted a series of studies in which we examined the event-related brain potentials (ERPs) associated with successful and unsuccessful performance of continuous tracking and joystick aiming tasks (Krigolson & Holroyd, 2006, 2007a, 2007b). Our results indicate that failure to achieve a movement goal modulates 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 the medial-frontal cortex (specifically ACC; Holroyd et al., 2004c; Miltner et al.; but see Nieuwenhuis et al., 2005c), and a current theory holds that these components are produced by the impact of the activity of a dopaminergic system for reinforcement learning on anterior cingulate cortex (Holroyd & Coles, 2002).

To account for these data, we have suggested that error processing systems in the brain are hierarchically organised (Krigolson & Holroyd, 2006, 2007a, 2007b). Specifically, we have proposed that the motor system evaluates two levels of errors: "high-level" errors indicate a failure to achieve a movement goal, whereas "low-level" errors indicate a discrepancy between the actual motor command and the appropriate motor command. Low-level errors are in principle correctable, but if they are not corrected then they lead to the occurrence of high-level errors. For example, consider a goal-directed reaching task in which the target occasionally jumps to a new location following movement onset. The target perturbation creates a low-level error because it brings about 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; c.f., Goodale et al., 1986). So long as the current motor command is corrected and the target attained, a high-level error does not occur. However, if the target perturbation cannot be corrected (for example, because it occurred very late in the movement or because the motor system failed to respond to the target jump), then a high-level error would occur. According to the hierarchical error processing hypothesis, low-level errors are mediated within PPC and/or the cerebellum (Desmurget et al., 1999, 2001; Desmurget & Grafton, 2000; Grea et al., 2002; Miall et al., 2001; Pisella et al., 2000) and high-level errors are evaluated by a system involving anterior cingulate cortex and the basal ganglia (Krigolson & Holroyd, 2006, 2007a, 2007b).

Although the ERN appears to be the product of a high-level error processing system, to our knowledge an ERP signature of low-level error processing has yet to be identified. Here we investigated whether posterior parts of the brain produce an ERP component related to low-level error processing that is analogous to the ERN. In a previous aiming study, we found that a change in target location that required a corrective joystick movement elicited an ERP component called the P300 (Krigolson & Holroyd, 2007b). The P300 is a parietally-distributed positive deflection in the ERP associated with the updating of an internal model of the environment (Donchin & Coles, 1988) and with decision-related phasic activity of the locus-coeruleus norepinephrine (LC-NE) system (Nieuwenhuis et al., 2005a, 2005b). Somewhat problematically, our previous results could not decide which theory of the P300 best accounted for our data. Furthermore, in our previous study the “online control” required to accommodate the change in target location consisted of a single, discrete manipulation of a computer joystick. From the results of our previous study it was not clear whether or not the P300 we observed following the target perturbation reflected visual processing associated with the online control of movement. As such, in the present study we sought to further evaluate the role of the P300 in error correction by providing a finer measure of the timing of the P300 relative to the onset of corrective sub-movements.

Here we recorded the electroencephalogram (EEG) from participants making rapid, two dimensional aiming movements to a target location by moving a stylus across a graphics tablet. To evoke online control processes, on some trials the target jumped to a new location following movement onset. In line with our previous findings, we predicted that the target perturbations would elicit the P300. To clarify the relationship between the P300 and the online control of movement, we compared the timing of the P300 with the timing of the low-level error corrections (specifically, changes in vertical acceleration) brought about by the target perturbations. We predicted that if the P300 is elicited directly by the error correction process itself, then its onset should occur before any observable changes in behaviour following the target perturbation. Conversely, if P300 onset follows the corrective movement, then that would indicate that the P300 is not directly associated with the online control of movement.

A second goal of the present experiment was to examine the evaluation of high-level motor errors by medial-frontal cortex within the context of a more ecologically valid manual aiming task. To accomplish this, we “blocked” one half of the participants’ corrective movements on perturbation trials so that it was not possible to correct for the target jump. We anticipated that, as with our previous findings, an ERN would be elicited by blocking the corrective movements. To further gauge the role of medial-frontal error systems, we also examined the ERPs associated with movement end for on-target and off-target trials. Given that missing the target also constitutes a high-level error, we predicted that an ERN would be elicited when participants ended a trial off-target.

## *Methods*

### *Participants*

Fifteen right-handed undergraduate students (8 male, 7 female) 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 rectangular box (74 cm high, 96 cm wide, 60 cm deep) divided in half by a mirror inclined at 20 degrees. A 17 inch 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 x 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 by 0.5 cm red square) on the surface of the mirror. The distance between the eyes and the mirror was approximately 45 cm. The participants' task was to use the stylus to move a cursor from a start position (a 1.5 cm by 1.5 cm white square) on the left hand side of the display to a target location (another 1.5 cm by 1.5 cm white square) on the right hand side of the display. At the start of each trial participants were required to align the cursor with the start location and to then wait for an auditory movement initiation cue.

Participants completed aiming movements in three experimental conditions. In the first condition (*control*) participants made aiming movements to the target location. In the second condition (*correction*) 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 (*blocked*) was identical to the correction condition except 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 correct their initial motor plan.

Participants were instructed that their goal was to stop the cursor on the target square. In total, participants performed five blocks of 60 trials with 20 trials from each of the three conditions randomly intermixed within each block. To ensure that participants did not hesitate following the start of the movement to determine whether a target perturbation would occur, they were required to complete the movement within 400 to 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 5 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 the displacement data were filtered with 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 both the horizontal and vertical movement axes. The same algorithm was utilised again to differentiate the velocity values to obtain instantaneous accelerations. Note that we simplified data analysis by rectifying the displacement data in the vertical axis. As such, we focused our analysis on the amplitude of the target displacements and the participants' corrective movements but not their direction. Dependent variables used in subsequent analyses were reaction time (ms), movement time (ms), and constant error (pixels) and variable error (pixels) in the horizontal ($CE_h$, $VE_h$) and vertical ($CE_v$, $VE_v$) movement axes. Furthermore, the instantaneous acceleration values in the horizontal and vertical axes were computed at 10%, 30%, 50%, 70% and 90% of the reaching trajectory. Finally, to get a temporal estimate of when participants began to correct for a target perturbation, we utilised a stepwise procedure in which a 30 ms window of data was averaged for each instantaneous acceleration data point (i.e., +/- 15 ms) for each condition 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 (Rodriguez-Fornells et al., 2002). Thus the onset of the corrective movement, as determined by a statistical difference in the acceleration profile, was defined as the first time point at which five consecutive t-tests showed a significant difference ($p < 0.05$)[7].

*Electroencephalographic Analysis*

The EEG was recorded from 41 electrode locations using BrainVision Recorder software (Version 1.3, Brain Products, GmbH, 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 Hz – 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 uVs 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.

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[7] 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.

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, 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 corrective movement during the control trials, no comparisons were made between this condition and 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. Note that 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 ERP data for each event of interest 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 & Holroyd, 2006). 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).

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 ($p < 0.05$)[8].

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 & Krigolson, in press; Holroyd, 2004; Krigolson & Holroyd 2006, 2007a, 2007b). For both comparisons, the ERN was identified as the maximum negative peak in the difference waves from 0 to

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[8] 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.

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. Bonferroni corrections were applied where appropriate.An alpha level of 0.05 was used to interpret all omnibus tests and only significant effects are reported.

## *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 ($CE_h$: $F(2,28) = 19.95$, $p < 0.001$, $CE_v$: $F(2,28) = 9.41$, $p < 0.001$)[9]. 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 to blocked trials ($t(14) = 4.93$, $p < 0.001$). Correction and blocked trials did not differ in terms of $CE_h$ ($t(14) = -0.74$, $p > 0.05$). 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,

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[9] 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.

which in turn did not differ from each other ($t(14) = 1.25$, $p < 0.05$) (see Table 2 for mean and standard error values).

Table Two
Reaction Time, Movement Time, Constant Error (horizontal and vertical axes), and Variable Error (horizontal and vertical axes) for control, correction, and blocked aiming movements. Also reported is the standard error of the mean for each score.

| Condition | Reaction Time (ms) | Movement Time (ms) | Constant Error (pixels) | Variable Error (pixels) | Constant Error (pixels) | Variable Error (pixels) |
|---|---|---|---|---|---|---|
| | | | Horizontal Axis | | Vertical Axis | |
| Control | 235 | 540 | 4 | 49 | -7 | 34 |
| | +/- 18 | +/- 8 | +/- 4 | +/- 2 | +/- 2 | +/- 2 |
| Correction | 236 | 539 | -12 | 49 | 4 | 80 |
| | +/- 19 | +/- 7 | +/- 5 | +/- 3 | +/- 6 | +/- 8 |
| Blocked | 236 | 543 | -10 | 52 | 14 | 77 |
| | +/- 19 | +/- 8 | +/- 4 | +/- 3 | +/- 8 | +/- 9 |

We analysed acceleration at select points of the reaching trajectory using a 5 (marker: 10%, 30%, 50%, 70%, 90%) by 3 (condition: control, correction, blocked) repeated measures ANOVA. An analysis of horizontal acceleration yielded main effects for marker, $F(4,56) = 67.61$, $p < 0.001$, and condition, $F(2,28) = 5.19$, $p < 0.01$, and a marker by condition interaction, $F(8,112) = 3.27$, $p < 0.01$ (Figure 8a). Post-hoc analysis of this interaction indicated that movements on control trials had larger accelerations at 50% of the trajectory than did movements on correction ($t(14) = 3.62$, $p < 0.01$) and blocked ($t(14) = 2.70$, $p = 0.01$) trials, which in turn did not differ from one another ($t(14) = -1.12$, $p > 0.05$). An analysis of vertical acceleration also indicated main effects for marker, $F(4,56) = 38.99$, $p < 0.001$, and condition, $F(2,28) = 372.87$, $p < 0.001$, and a marker by condition interaction, $F(8,112) = 58.73$, $p < 0.001$ (Figure 8b). Further analysis of the interaction revealed that vertical acceleration was impacted by experimental

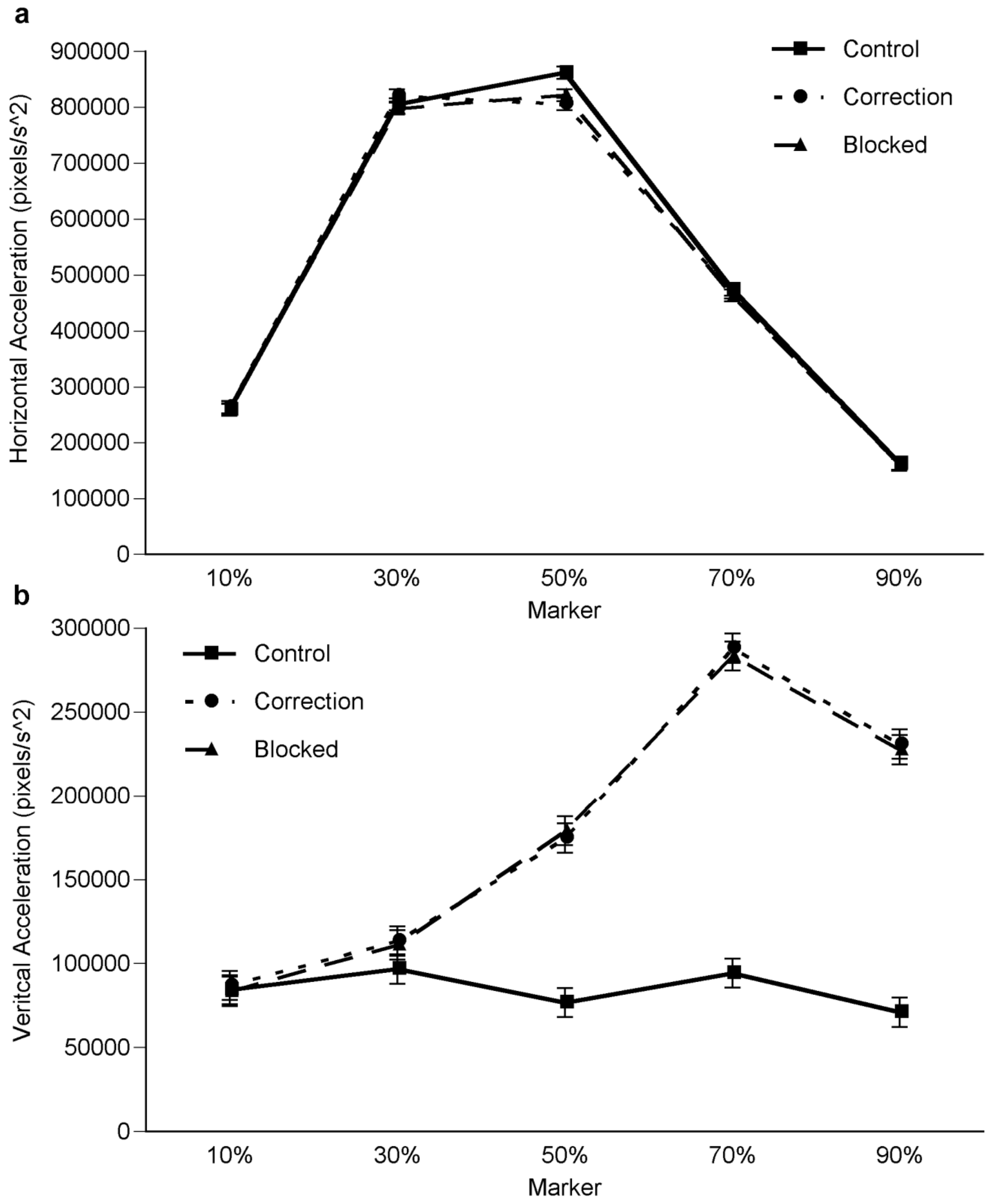

**Figure 8. Instantaneous acceleration in the (a) horizontal and (b) vertical axes at 10%, 30%, 50%, 70%, and 90% of the reaching trajectory. Note that absolute acceleration values are reported to remove directional effects. Error bars represent the standard error of the mean.**

condition at 30% ($F(2,28) = 12.52$, $p < 0.001$), 50% ($F(2,28) = 52.13$, $p < 0.001$), 70% ($F(2,28) = 153.77$, $p < 0.001$), and 90% ($F(2,28) = 172.25$, $p < 0.001$) of the aiming trajectory, but not at 10% ($F(2,28) = 1.86$, $p > 0.05$) of the aiming trajectory. Specifically, correction (30%: $t(14) = -4.67$, $p < 0.001$; 50%: $t(14) = -7.96$, $p < 0.001$; 70%: $t(14) = -12.01$, $p < 0.001$; 90%: $t(14) = -14.66$, $p < 0.001$) and blocked (30%: $t(14) = -3.86$, $p = 0.001$; 50%: $t(14) = -7.10$, $p < 0.001$; 70%: $t(14) = -13.81$, $p < 0.001$; 90%: $t(14) = -13.17$, $p < 0.001$) trials exhibited greater vertical accelerations than did control trials. Furthermore, at each of these points in the reaching trajectory correction and blocked trials did not differ in terms of vertical acceleration (30%: $t(14) = 0.65$, $p > 0.05$; 50%: $t(14) = -0.82$, $p > 0.05$; 70%: $t(14) = 0.87$, $p > 0.05$; 90%: $t(14) = 0.60$, $p > 0.05$). To get a temporal estimate of when the acceleration profiles began to differ, we also conducted an onset analysis comparing the vertical acceleration values for the correction and blocked conditions with the control condition. The results of this analysis indicated for both the correction and blocked conditions, vertical acceleration values were statistically different from the control condition at 230 ms following the target perturbation ($p$'s $< 0.05$).

### *Electroencephalographic 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 (Figure 9a). These results are consistent with the spatial distribution of the P300 (Dien et al., 2003, 2004; Donchin & 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

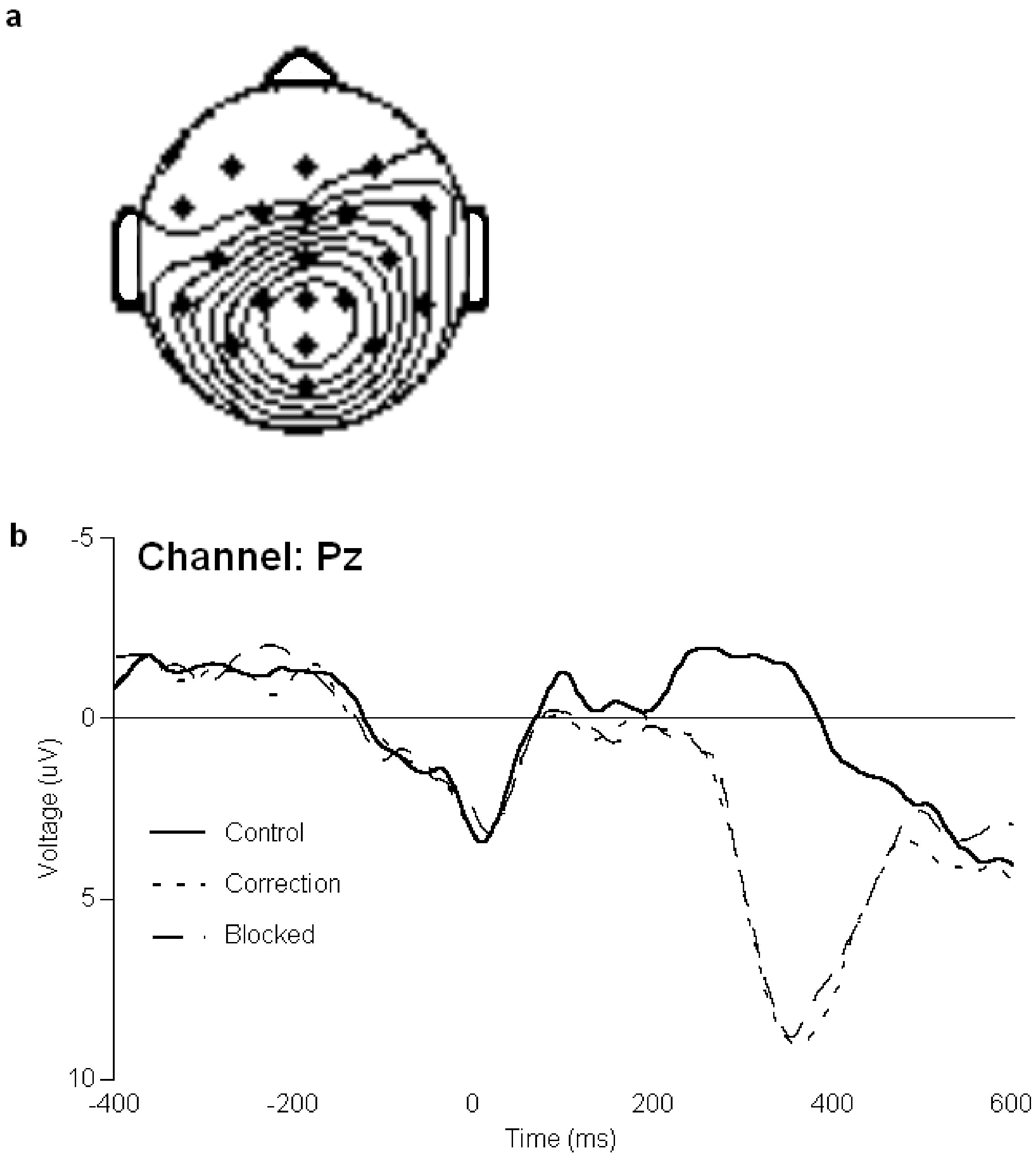

**Figure 9. Movement Onset: (a) Spatial PCA factor loadings projected onto the surface of the human head for the posterior factor. (b). Averaged ERPs recorded at channel Pz for the control, correction, and blocked conditions. Note that zero ms corresponds to movement onset. Negatives voltages are plotted up by convention.**

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 (Figure 9b). 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 ms and 240 ms, respectively (both p's $< 0.05$).

*The Corrective Movement.* Based on our previous research (Krigolson & Holroyd, 2007b), we predicted the corrective movement on blocked trials would elicit an ERN relative to the corrective movement on correction trials. 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 trials (Figure 10). The result of this analysis was not significant ($p > .05$). Together, these results indicate that, contrary to our prediction, blocking the corrective movement did not elicit an ERN at the time of the correction.

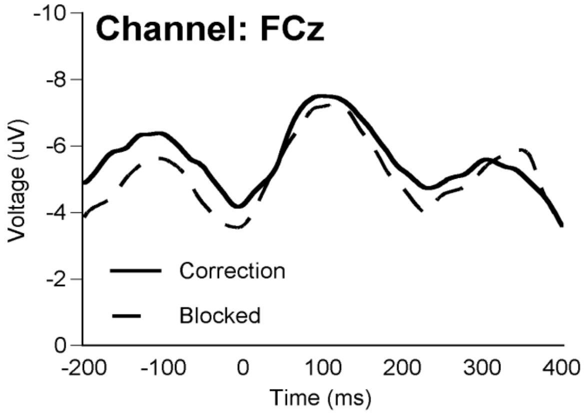

**Figure 10. Movement Correction: Averaged ERPs for correction and blocked trials recorded at channel FCz. Zero ms indicates the average point in time at which a corrective movement was attempted. Negatives voltages are plotted up by convention.**

*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; Figure 11a). A peak analysis of the difference waves (off-target minus on-target) associated with channel FCz revealed the presence of an ERN, $t(14) = 5.54$, $p < 0.001$, at a latency of 268 ms (Figure 11c). The spatial PCA also yielded a second factor with loadings that were maximal at channel Pz (Figure 11b). 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, $t(14) = -7.90$, $p < 0.001$ (Figure 11d). 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 uV versus -6.9 uV, $t(14) = 0.42$, $p > 0.05$).

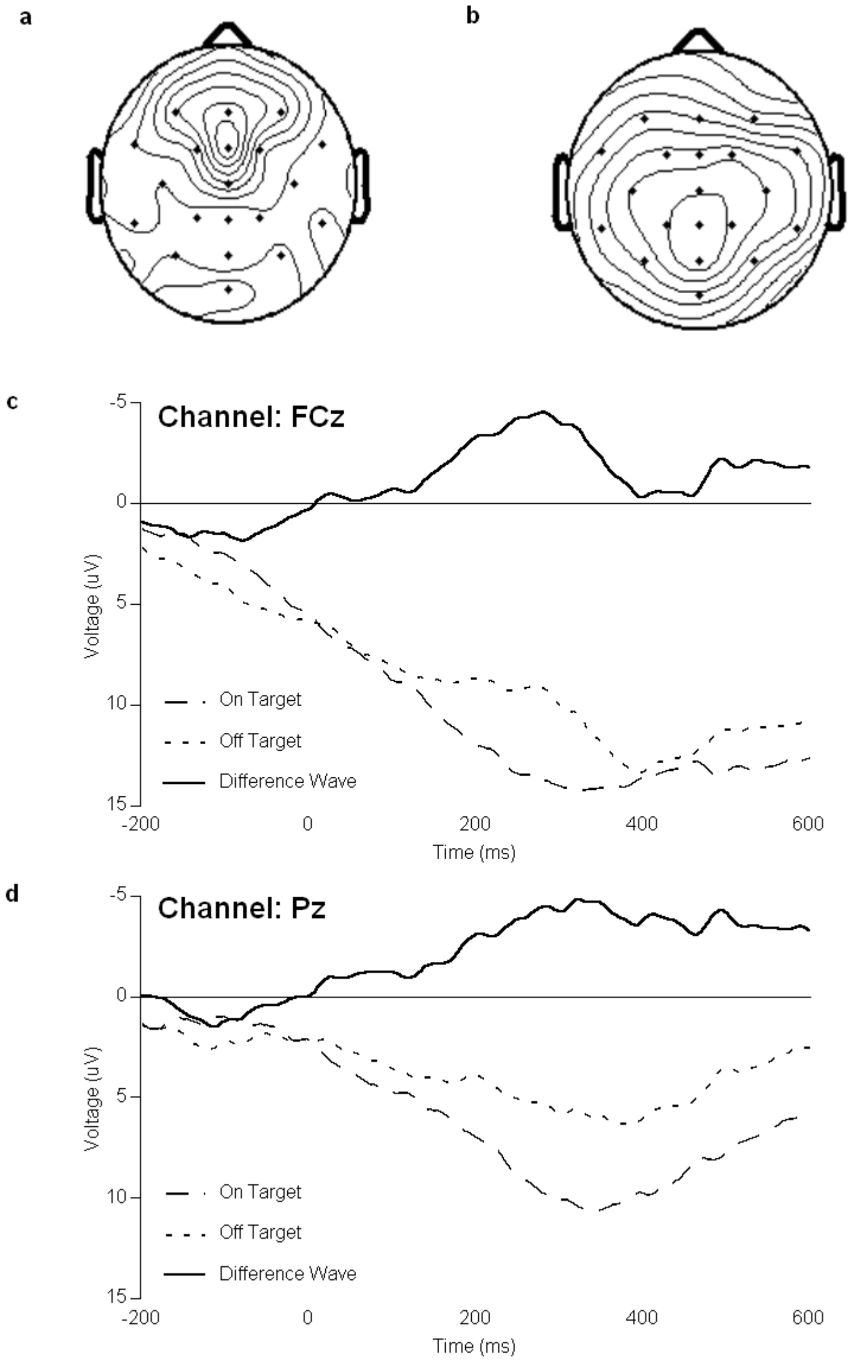

**Figure 11. Movement End: (a) Spatial PCA factor loadings projected onto the surface of the human head for the frontal-central factor. (b) Spatial PCA factor loadings projected onto the surface of the human head for the posterior factor. (c) Averaged ERPs recorded at channel FCz for on target and off 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 ms corresponds to movement end. Negatives voltages are plotted up by convention.**

## *Discussion*

In the present experiment we examined error-related ERP components evoked during performance of a manual aiming task. Specifically, we sought to examine the ERP components elicited by a change in target location, by blocking the corrective movement associated with the target perturbation, and by missing the target. In line with the hierarchical error processing hypothesis, we predicted that low-level errors resulting from a target perturbation would elicit an ERP component distributed over posterior areas of the scalp, and that high level errors due to the blocking of a corrective movement or due to missing the movement target would elicit the ERN. Further, we hoped to clarify the relationship between the posterior ERP components and online control processes by comparing the timing of the P300 with the timing of the corrective movements associated with the target perturbation.

### *Low-Level Errors*

Low-level motor errors brought about by a sudden change in the movement environment appear to be corrected by PPC (Desmurget et al., 1999, 2001; Grea et al., 2002; Pisella et al., 2000). Previously, we demonstrated that low-level errors in a joystick manual aiming task elicited a P300, and we suggested that in this context the P300 may be reflect an online control process (Krigolson & Holroyd, 2007b). In the present study we sought to clarify the relationship between the P300 and online control by comparing the timing of this ERP component relative to the moment of the error correction. We predicted that if the P300 reflects an online control process (or a process that supports online control, namely dorsal stream visual processing), then its onset should occur before kinematic changes indicative of online control (i.e., changes in acceleration).

Conversely, if the onset of the P300 comes after behavioural changes associated with the online control of movement then it cannot have played a role on implementing those modifications – however, it may still reflect the updating of a forward model of control. As in our previous study, in the present experiment we found that target perturbations indeed elicited a P300 (Donchin & Coles, 1988; Nieuwenhuis et al., 2005a, 2005b). Importantly, the onset of the P300 occurred 256 ms and 240 ms following the target perturbation on correction and blocked trials, respectively, whereas differences in the vertical acceleration profiles for these conditions began to occur at about 230 ms (Figure 12). 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. Although these values may appear to be similar, it is worth noting that the behavioural modifications that began at 230 ms occurred well in advance of the latency of the peak P300 amplitude (correction condition: 362 ms, blocked condition 341 ms). Thus, the major portion of the process that gives rise to the P300 appears to have occurred following the change in behaviour.

The P300 is characterized by a parietal scalp distribution and appears to be generated in 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 online motor control (Desmurget et al., 1999; 2001, Desmurget & Grafton, 2000; Grea et al., 2002; Pisella et al., 2000). 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).

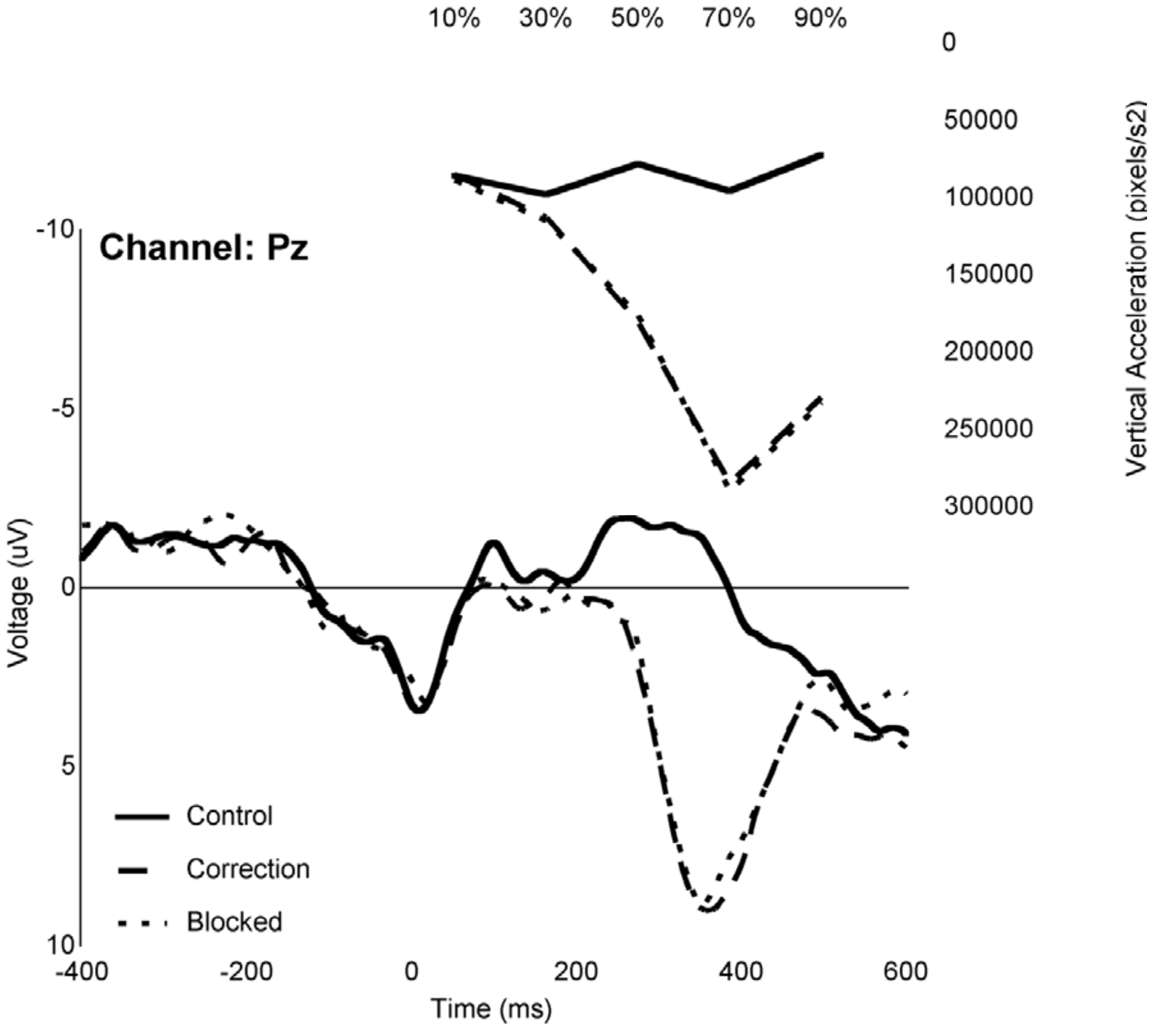

Figure 12. A comparison of the instantaneous vertical acceleration kinematic profiles and the conditional ERPs associated with the target perturbation. Note that any amplitude scaling similarities are coincidental.

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, Cohen, Servan-Schreiber, Rajkowski, & Aston-Jones, 1999). We previously speculated that phasic LC-NE activity should precede and facilitate the rapid changes in motor behaviour brought about by the target perturbation (Krigolson & Holroyd, 2007b). 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 correction. However, the timing of the P300 relative to the corrective movement in the present experiment seems to rule out this possibility.

By contrast, 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 & Coles, 1988). In this regard, it is relevant that PPC has access to a representation of the current movement environment (Desmurget et al., 1999; 2001; Grea et al., 2002; Pisella et al., 2000), and that this representation is updated upon receipt of new information indicating that the environment has changed. Further, the PPC is thought to implement online control by relying on forward models that predict future internal and external events (i.e., Desmurget & Grafton, 2000; Wolpert & Ghahramani, 2000). Unpredicted events such as target perturbations are, by definition, failures of the forward model to predict the event, and thus constitute errors that can be used by the forward model to improve future control. We suggest that the P300 in the present study may reflect changes to the PPC’s forward model of control, in an attempt to improve upon its predictions of the future environment. 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 & Coles, 1988).

Importantly, although the target perturbations in the present study required participants to amend their ongoing motor plans, the participants could not know at that time whether or not their motor corrections would be successful. As such, the target perturbations constituted low-level motor errors. Supporting this conclusion, we found that the target perturbations elicited a P300 but not an ERN, which suggests that medial-frontal cortex did not process these events as high-level errors. This finding is consistent with our hypothesis that error processing occurs in a hierarchical fashion.

### *High-Level Errors*

On the basis of the results of a previous study (Krigolson & Holroyd, 2007b), we predicted that blocked trials relative to correction trials would elicit an ERN shortly after the failed corrective movement. By our definition, the blocked corrective movements constituted high-level errors because the system goal of reaching the target was no longer attainable. However, contrary to this prediction, in the present experiment blocked trials did not elicit an ERN. One explanation for this results stems from the similarity between the correction and blocked trials in terms of movement time, endpoint error, variable error, and acceleration profiles (Figure 8 and Table 2). These kinematic data indicate that nearly all physical aspects of the movement were identical on the 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 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 error processing within the basal ganglia – ACC is inhibited when conflicting feedback is received. Alternatively, the motor correction may be implemented ballistically. Previous research suggests that at least 100 ms is required to implement feedback based amendments to ongoing motor output (Desmurget & Grafton, 2000; Jeannerod, 1988; Paillard, 1996). As such, it may be that while participants were able to adjust their initial motor command following the target perturbation, due to time constraints they released the corrective movement ballistically, and thus were unable to evaluate the success (or failure) of the movement on-line. In other words, once the correction was made participants did not have sufficient time to evaluate the success of the correction before movement end[10].

Assuming that a participants' movement is not blocked, then the earliest indicator as to whether or not the movement goal has been achieved is the evaluation of endpoint error. Thus, in line with the reinforcement learning theory of the ERN we predicted that for the control and correction conditions missing the target would constitute a high-level 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 & Coles, 2002; Holroyd et al., 2004a; Miltner et al., 1997). This result provides further support for the hypothesis that high-level motor errors are evaluated within the basal ganglia – ACC system, and that this information is used for the purpose of

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[10] 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 & 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 jitter in fact occurred.

reinforcement learning (Krigolson & Holroyd, 2006, 2007a, 2007b; see also Holroyd & Coles, 2002). Consistent with this hypothesis, we observed a negative-going ERP component distributed over posterior areas of the scalp 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 & Holroyd, 2006, 2007a, 2007b).

### *Conclusion*

To the best of our knowledge, the present study is the first to use ERPs to analyse the neural basis of motor control in a discrete manual aiming task. We found that target perturbations evoke a P300, an ERP component with a parietal scalp distribution that has been associated both with context updating (Donchin & Coles, 1988) and activity of the LC-NE system (Nieuwenhuis et al., 2005a, 2005b). Importantly, 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 implement the online control of movement (i.e., PPC), it does not appear that the P300 is directly related to such on-line control. Rather, we propose the P300 may reflect the updating of a forward model of motor control. 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 high level errors (as evidenced by target misses, which elicited the ERN) but not by low-level errors (as evidenced by the target perturbations, which did not elicit the ERN). Contrary to a previous result (Krigolson & Holroyd, 2007b), the results of the present study indicated an ERN was not elicited when a corrective movement was blocked. Interestingly, this result suggests that during performance of a rapid aiming task corrective sub-movements may be implemented ballistically and as such, the success (or failure) of the corrective movement is not evaluated until movement end. Taken together, these results support our hypothesis that motor control is hierarchical in nature (Krigolson & Holroyd, 2006, 2007a, 2007b).

# Experiment Four

## *Abstract*

The successful execution of goal-directed movement requires contributions from both movement planning and online control processes. In the present experiment we assessed the relative contributions of these processes to goal-directed reaching by recording event-related brain potential (ERP) data while participants made visually-guided and memory-guided aiming movements to two target locations. Consistent with previous results, we found that visually-guided aiming movements were more accurate and less variable than memory-guided reaches. Further, we found that visually-guided and memory-guided reaches elicited similar pre-movement cortical motor potentials. By contrast, our results indicated that visually-guided reaches were associated with both larger cortical motor potentials during the movement and smaller constant error upon movement completion than memory-guided reaches were. Interestingly, we also observed an enhanced positive deflection in the ERP over parietal cortex during the later stages of the visually-guided aiming movements, a result which may reflect the contribution of posterior parietal cortex to the online control of movement. Finally, we found that trials that ended off-target in the visually guided condition elicited an error-related negativity – an ERP component associated with the evaluation of response errors and error feedback. To the best of our knowledge, the results of the present study represent the first attempt to contrast visually-guided and memory-guided aiming movements with electroencephalographic data. Importantly, our results demonstrated differences in cortical motor potentials and parietal ERP activity that mirrored the behavioural differences we observed between these conditions.

## *Introduction*

In a seminal paper, Woodworth (1899) proposed that goal-directed aiming movements rely on a combination of movement planning and online control processes. He hypothesized that goal-directed actions could be subdivided into two distinct phases: an initial ballistic movement phase resulting from a movement plan generated before movement onset and a “current control” phase characterized by feedback-based adjustments to the initial movement plan. Since Woodworth’s seminal manuscript, a multitude of behavioural studies have supported his contention that goal-directed actions consist of these two distinct phases, now more frequently termed planning and control (Abahnini & Proteau, 1999; Abbs, Gracco, & Cole, 1984; Bullock & Grossberg, 1988; Elliott et al., 2001; Glover, 2004; Meyer et al., 1988; Schmidt, 1975).

In recent years, neuroimaging studies have indicated that movement planning and control processes are implemented by different brain regions. Studies utilising functional magnetic resonance imaging and electroencephalographic recordings suggest that supplementary motor area (SMA) is associated with movement planning and that primary motor area (M1) is related to movement execution (Evarts & Fromm, 1980; Fromm & Evarts, 1981; Kornhuber & Deecke, 1965; Lee, Chang, & Roh, 1999; Wise, 1985; Wise & Evarts, 1981). In a similar vein, a series of converging studies have demonstrated that posterior parietal cortex (PPC) plays a key role in the online control of movement (Desmurget et al., 1999, 2001; Grea et al., 2002; Pisella et al., 2000). Despite these efforts, to date there has not yet been a comprehensive neuroimaging study demonstrating the relative timing of the neural regions associated with movement planning (SMA, M1) and online control (PPC, M1).

### *Movement Planning Processes*

The planning phase of goal-directed actions is thought to result in the generation (or recall) of a motor program, a set of neural instructions specifying the motor commands necessary to achieve the desired movement goal (Schmidt, 1975), which when executed results in a ballistic movement. Several sources of behavioural evidence support the existence of movement planning processes implemented by motor programs. For example, studies which report few, if any, changes in the trajectory of a movement effector during the initial stages of a reaching movement have been interpreted as evidence for an initial ballistic movement impulse (Khan et al., 2006). Studies which manipulate the availability of visual information during movement provide further evidence for movement pre-programming. In general, these studies have found that withdrawing vision of the movement environment during the first half of a reaching movement does not reduce the movement's accuracy, but withdrawing vision during the second half of the movement does (Carlton, 1981; Chua & Elliott, 1993). These results have been interpreted as evidence that at least the first half of the movement plan was specified prior to movement onset.

Neuroimaging studies provide further evidence that an initial motor plan is specified in advance of movement onset. For example, in a study employing functional magnetic resonance imaging, Lee, Chang, and Roh (2006) examined the hemodynamic response evoked by the preparation and execution of voluntary finger movements. In line with previous research (i.e., Evarts & Fromm, 1980; Wise, 1985; Wise & Evarts, 1981) they identified activity within premotor area (PMA) associated with movement preparation and activity within primary motor area (M1) associated with movement execution. Lee et al. were also able to temporally separate the activity within supplementary motor area (SMA); they identified early activity within anterior SMA associated with motor program selection and movement preparation and late activity within posterior SMA associated with movement execution.

There is also electroencephalographic evidence for preparatory neural activity prior to movement onset. The *bereitschaftspotenital* (BP) and the *reafferente Potentiale* (RP) are cortical motor potentials associated with movement preparation and execution. Typically, the BP is observed in experimental tasks where participants perform simple movements, such as depressing a series of buttons (e.g., Kornhuber & Deecke, 1964). After considerable research, it appears that the BP is actually comprised of two primary ERP subcomponents, the early BP which reflects pre-movement activity within SMA (Cui & Deecke, 1999a, 1999b; Praamstra, Stegeman, Horstink, & Cools, 1996) and the late BP which reflects pre-movement activity within lateral pre-motor area (Cui & Deecke, 1999a, 1999b; Shibasaki, Barrett, Halliday, & Halliday, 1980; Shibasaki & Hallett, 2006; Yazawa et al., 2000) and M1 (Gerloff et al., 1998; Shibasaki et al.; Shibasaki & Hallett)(Figure 13). As with the BP, the RP is also comprised of ERP subcomponents, called the N+50, P+90, N+160 and P+300 (Figure 13; Cui & Deecke, 1999a, 1999b; Shibasaki & Hallett; Shibasaki et al.). Although less in known about these post-movement ERP components, the N+50 is thought to reflect the recruitment of motor neurons within area M1 whereas the later components are thought to reflect the processing of kinaesthetic feedback (Shibasaki et al.) and/or reafferent activity from motor areas (Cui & Deecke, 1999a, 1999b).

### *Online Control Processes*

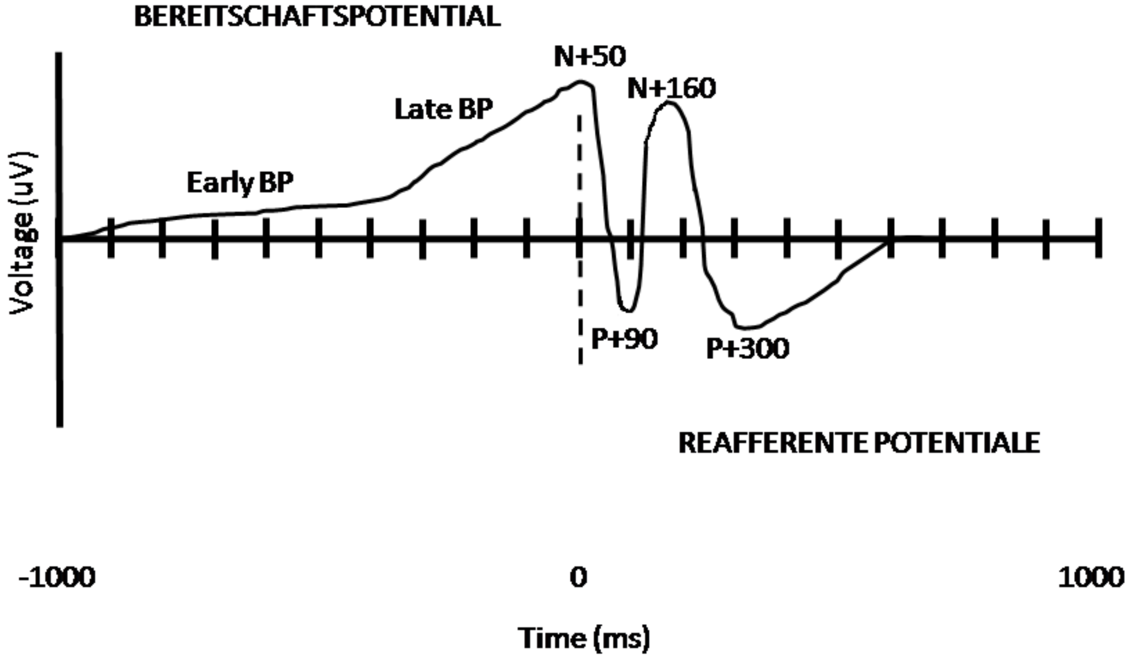

**Figure 13. Diagram of the Berietshaftspotential and the reafferente Potentiale.**

In line with Woodworth's (1899) assertion that goal-directed aiming movements have two distinct phases, experimental evidence suggests that online, feedback-based adjustments to the initial motor command also plays a crucial role in movement accuracy. For instance, Goodale and colleagues (1986) had participants make rapid, discrete reaching movements to a target that jumped to a more peripheral location following movement onset. Although the target perturbations occurred during the ocular saccade such that the participants were unaware of them, the participants nevertheless modified the reaching trajectory to accommodate the change in target location. Goodale et al.'s results also indicated that the target jumps did not result in an increase in movement time, ruling out the possibility of a speed-accuracy trade-off (Fitts, 1954), 1954). From these results, Goodale and colleagues concluded that it is possible to make rapid and accurate visually based adjustments to an ongoing movement to accommodate unexpected changes in the environment. This ability to execute fast, feedback-based adjustments to an ongoing movement has been confirmed by a large number of behavioural experiments (Chua & Elliott, 1993; Elliott, Chua, Pollock, & Lyons, 1995a; Elliott et al., 1999; Elliott, Lyons, Chua, Goodman, & Carson, 1995b; Elliott & Madalena, 1987; Goodale et al., 1986; Heath et al., 1998; Heath et al., 2004; Jeannerod, 1986, 1988; Keele & Posner, 1968; Khan & Lawrence, 2005; Khan et al., 2003, 2004, 2006; Meyer et al., 1988; Pratt & Abrams, 1996).

A series of transcranial magnetic stimulation (Desmurget et al., 1999; Tunik, Frey, & Grafton, 2005), positron emission tomography (Desmurget et al., 2001), functional magnetic resonance imaging (Frey, Vinton, Norlund, & Grafton, 2005), and brain lesion (Grea et a., 2001; Pisella et al., 2000) studies have indicated that posterior parietal cortex implements – at least in part – the sensorimotor transformation underlying online control. For example, Desmurget et al. (1999) conducted a study in which participants reached to targets that either remained stationary or jumped to a new position during the initial saccadic eye movement. Replicating Goodale et al.'s (1986) original findings, Desmurget and colleagues found that participants accurately adjusted their reaching trajectory to accommodate the target perturbation, even though they were not consciously aware that the target had moved. In a key experimental manipulation, Desmurget et al. also found that transcranial magnetic stimulation applied to left PPC during the reaching movements negated the participants' ability to correct for the target jumps, whereas transcranial magnetic stimulation to right PPC did not. Furthermore, they found that neither transcranial magnetic stimulation applied to left or right PPC impaired accuracy when the target remained stationary, suggesting that while left PPC played an important role in making online adjustments to an ongoing motor command, it did not seem to play a role in the programming of the initial movement impulse.

In a subsequent study Desmurget and colleagues (2001) utilised positron emission tomography to examine the neural substrates underlying online motor control in a target perturbation paradigm in which participants could not see their movement effector. As with their previous work and that of others (e.g., Goodale et al., 1986), Desmurget et al. found that participants were about as accurate on stationary target trials as on perturbed target trials, and that left PPC was activated to a greater extent than other neural regions on the perturbed target trials. Interestingly, their results also revealed activation of the right anterior intermediate cerebellum and left primary motor area. From their results, Desmurget and colleagues hypothesized that these neural substrates formed a network responsible for online motor control. Given that participants only received kinaesthetic feedback from the movement effector in this study, Desmurget et al.'s results also suggested that PPC activation during online motor control was not simply related to the processing of visual feedback, but instead was related to the processing of the sensorimotor transformations necessary for the modification of an ongoing motor command.

In the present study we utilised event-related brain potentials (ERPs) to examine the relative timing of movement planning and online control processes during the performance of visually-guided and memory-guided aiming movements. Comparisons of visually-guided and memory-guided reaches provide an excellent context for studying movement planning and online control processes. In a typical experiment, participants begin each trial with a visual preview period in which they can see the start location, the movement effector and the movement target. On some trials, participants reach to the target with full vision of the effector and target, allowing for movement planning and control to benefit from real-time egocentric visual information. By contrast, in memory-guided conditions vision of the movement environment is occluded for some period of time beginning prior to movement onset and ending after movement termination. As such, memory-guided aiming movements are thought to be planned in their entirety before movement onset from a stored sensory representation of the environment that is necessarily less accurate than the real-time visual information available during visually-guided conditions (Elliott, Calvert, Jaeger, & Jones, 1990; Glover, 2004; Heath, 2005; Westwood, Heath, & Roy, 2001; 2003). Furthermore, the absence of real-time visual information in memory-guided conditions reduces the ability to correct the motor command during the movement itself (Heath, 2005; Westwood & Goodale, 2003). Given these constraints, it is not surprising that memory-guided reaches are typically found to undershoot the movement target and exhibit greater endpoint variability than their visually-guided counterparts (Adamovich, Berkinblit, Fookson, & Poizner, 1998; Darling & Miller, 1993; Elliott & Madalena, 1987; Glover, 2004; Goodale et al., 2004; Heath, 2005; Heath & Westwood, 2003; Heath et al., 2004; Westwood, Heath, & Roy, 2000, 2001, 2003).

In the present study we hypothesized that differences in the planning of visually-guided and memory-guided reaches would be reflected as differences in the cortical motor potentials associated with these conditions. Given that memory-guided reaches typically undershoot the desired target location, we predicted that memory-guided reaches would have reduced cortical motor potentials prior to (BP) and following (RP) movement onset, reflecting a reduction in movement planning and/or online control processes relative to visually-guided reaches. To date, no ERP component has been attributed to the online control of movement implemented by PPC. However, given that visually-guided reaches are also thought to rely on online control processes to a greater extent than memory-guided reaches, we also predicted that there would be conditional differences in the posterior ERP waveforms during the later stages of aiming movements. Finally, in line with previous research (Experiment Three), we compared the ERPs associated with movement end for on-target and off-target trials in the visually-guided condition, with the prediction that off-target performance would elicit an ERN.

## *Methods*

### *Participants*

Fifteen right-handed undergraduate students (18 to 22 years of age; 7 male, 8 female) 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 rectangular box (74 cm high, 96 cm wide, 60 cm deep) divided in half by a mirror inclined at 20 degrees. A 17 inch 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. On each trial the projected aiming environment consisted of a start position (a 1.5 cm by 1.5 cm white square) located to 15.5 cm to the left of center and one of two targets (also 1.5 cm by 1.5 cm white squares) positioned horizontally to the right of the start position (proximal target: 31 cm, distal target: 32 cm). A graphics tablet (WACOM Intuos 2, 30 cm x 45 cm, sampling rate: 125 Hz) was placed directly below the mirror, with the stylus of the tablet controlling the movements of a cursor (a 0.5 cm by 0.5 cm red square) which was also projected onto the surface of the mirror. Movements of the projected cursor were directly scaled to movements of the stylus on the surface of the graphics tablet. Participants were seated such that the distance between their eyes and the midline of the mirror was approximately 45.

Participants were instructed to complete ballistic aiming movements "as quickly and accurately as possible" to a presented target in three experimental conditions: full-vision (FV), delay-one (D1), and delay-three (D3). At the start of each trial in all three conditions the participant was required to move the cursor onto the start location. Immediately after, a two second target preview phase began. At the start of the preview period the target appeared (either the proximal or distal target, equal probability for each). On FV trials participants heard an auditory tone immediately after the preview phase which cued them to make an aiming movement to the presented target. On D1 trials the start location, cursor, and target disappeared immediately following the preview period, and remained occluded for the duration of the trial. One second after the aiming environment was occluded participants heard an auditory cue signalling movement initiation. Thus, on D1 trials participants made an aiming movement to the remembered target location. D3 trials were identical to D1 trials in all regards except that participants had to wait three seconds following occlusion of the aiming environment before they heard the movement initiation tone. In all of the experimental conditions the position of the participants' heads relative to the mirrored surface obstructed vision of the participants' movement effector (their limb and the stylus).

On each trial, regardless of experimental condition, participants had to complete their aiming movement with a movement time between 400 and 700 ms. Participants were instructed that if their movement time was outside of this time window then they would have to repeat the trial. Participants were given practice trials at the start of the experiment to learn the movement time requirements (see below). Note that at the end of FV trials participants were able to see the position of the cursor relative to the target location. This was not the case in the D1 and D3 conditions; for trials in the D1 and D3 conditions the aiming environment remained invisible following each trial to avoid giving participants feedback about their movement accuracy. On these trials vision of the cursor and start position did not return until participants returned their cursor to within 5 cm of the start location.

Participants completed 300 aiming trials divided into six blocks of 50 trial each, with two blocks for each condition (FV, D1, D3). All trials within a block were associated with the same condition. Within each block participants completed 25 aiming movements to the two target locations (proximal, distal) each. The order of the blocks was randomised between participants, and trial displacement was randomised within each block. Between blocks participants relaxed during self-paced rest periods. The aiming task was explained to participants before the task began and they completed 10 trials in each of the aiming conditions during a practice block. If more than three of the participants trials during the practice block were outside the movement time constraints, participants completed another practice block.

### *Behavioural Analysis*

Displacement data for the cursor were recorded for each trial. Following data collection the displacement data were filtered with a second order dual-pass Butterworth filter using a low-pass cut-off frequency of 10Hz. The displacement data were then differentiated using a three-point central finite difference algorithm to obtain instantaneous velocities in both the horizontal and vertical movement axes. The same algorithm was utilised again to differentiate the velocity values to obtain instantaneous accelerations. Dependent variables used in subsequent analyses were reaction time (ms), movement time (ms), time after peak velocity (the amount of time in ms spent after the maximal velocity during the movement), constant error (the number of pixels the participant undershot or overshot the centre of the target) and variable error (the standard deviation of each participant's constant error scores in pixels). Instantaneous position and acceleration values in the horizontal axis were also computed at 10% increments of the aiming trajectory. Note that the absolute value of the instantaneous acceleration values was utilised here, thus movement deceleration appears as a positive deflection in subsequent figures.

### *Electroencephalographic Analysis*

The electroencephalogram (EEG) was recorded from 41 electrode locations (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) using Brain Vision Recorder software (Version 1.3, Brain

Products, GmbH, Munich, Germany). The electrodes were mounted in a fitted cap and were referenced to a common ground. 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 Hz – 20 Hz passband phase shift free Butterworth filter and re-referenced to linked mastoids. Ocular artefacts were removed using the algorithm described by Gratton, Coles and Donchin (1983). Trials in which the change in voltage at any channel exceeded 35 uVs per sampling point were also discarded. In total, less than 5% of the data were discarded.

For each of the event of interest (movement onset, movement end), ERP waveforms were created for each condition by averaging the EEG data for each electrode channel and participant. To examine the cortical motor potentials before and after movement initiation ERPs for each condition (FV, D1, D3) were averaged with respect to movement onset (300 ms before to 700 ms after). Recall that cortical motor potentials have components associated with movement planning and execution that are maximal over frontal-central electrode sites and components associated with the recruitment of motor neurons that are maximal over contralateral electrode sites. As such, to directly examine these components we defined the frontal-central BP as the mean voltage across channels FC1, FCz, and FC2 and the contralateral BP as the mean voltage for channels C3 and C4 from 300 ms before movement onset to movement onset. To examine the within movement cortical motor potentials (WMCMP; i.e., the reafferente Potenitale), we defined the frontal-central WMCMP as the mean voltage across channels FC1, FCz, and FC2 and the contralateral WMCMP as the mean voltage for channels C3 and C4 from movement onset to 500 ms. Furthermore, given that we were also interested to see whether or not there was an electroencephalographic marker associated with the online control of movement, we calculated the mean voltage during the later stages of the aiming movements (from 300 to 500 ms) for an average of two parietal electrodes (Pz, POz)[11].

Given our hypothesis that an ERN would be elicited when participants finished a trial off-target, we also created ERP waveforms for FV trials averaged with respect to movement end (200 ms before movement end to 600 ms after) for on-target and off-target trials. Subsequent to this, we created difference waves for each participant by subtracting the on-target ERPs from the off-target ERPs for each electrode channel. The ERN for each participant was identified as the maximal negative deflection of the difference wave between 0 and 400 ms following movement end. All ERPs (irrespective of marker position) were baseline corrected using a 200 ms epoch from -500 to -300 ms before movement onset.

Each behavioural and electroencephalographic dependent measure was submitted to a 3 (experimental condition: FV, D1, D3) repeated-measures ANOVA[12]. Furthermore, to specifically compare lateralised components (e.g., the contralateral BP) we utilised a 2

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[11] We specifically chose this time window as online motor control is typically thought to occur following peak velocity during the later stages of the aiming movement. Furthermore, we chose these electrode sites as they correspond roughly to the location of PPC.

[12] Note that while we found typical effects of target location (proximal, distal) for the behavioural data (i.e., increased movement time with increased target displacement, greater acceleration with greater target displacement, etc.), we did not find any statistically significant differences between these conditions for the electrophysiological data. For this reason the following ERP analyses have been collapsed across target location.

(channel: C3, C4) by 3 (experimental condition: FV, D1, D3) repeated measures ANOVA. Post-hoc decomposition of main effects was carried out via paired samples t-tests as appropriate. A one-sample t-test against zero was also utilised to test the difference waves associated with movement end. For all statistical tests an alpha level of 0.05 was assumed. Bonferroni corrections were applied where appropriate.

## *Results*

### *Behavioural Results*

Examination of reaction time data revealed a main effect of experimental condition F(2,28) = 7.72, p = 0.002. Post-hoc decomposition of this effect indicated that FV reaches (334 ms) had shorter reaction times than D1 (353 ms: t(14) = 2.17, p = 0.048) and D3 (367 ms: t(14) = 3.83, p = 0.002) reaches. D1 and D3 reaches did not differ in terms of reaction time (t(14) = 1.77, p = 0.098). The analysis of movement times did not indicate an effect for experimental condition, F(2,28) = 1.89, p = 0.169 (Figure 14a). Nevertheless, a subsequent analysis of time after peak velocity revealed a main effect for experimental condition, F(2,28) = 13.13, p =< 0.001. Post-hoc analysis indicated that FV reaches took longer to complete following peak velocity than did D1 (t(14) = 3.48, p = 0.003) and D3 (t(14) = 4.04, p = 0.001) reaches, which did not significantly differ from each other (t(14) = 1.34, p = 0.201); Figure 14b).

Analysis of constant error revealed a main effect for experimental condition, F(2,28) = 7.61, p = 0.002. Post-hoc analysis indicated that FV reaches were more accurate than D1 (t(14) = 2.43, p = 0.02) and D3 (t(14) = 3.31, p = 0.005) reaches, whereas D1 and D3 reaches did not significantly differ from each other (t(14) = 1.16, p = 0.264; Figure 14c). Likewise, the examination of variable error also demonstrated an effect for experimental condition, F(2,28) = 16.42, $p < 0.001$; post-hoc analysis indicated that reaches in the FV condition were less variable than D1 (t(14) = 4.12, p = 0.001) and D3 (t(14) = 5.88, $p < 0.001$) reaches, which did not significantly differ from each other(t(14) = 0.80, p = 0.439)(see Figure 14d).

To further examine the impact of experimental condition on the aiming movements, we calculated limb displacement across the reaching trajectory and submitted these values to a 3 (experimental condition: FV, D1, D3) by 11 (trajectory position: 0%, 10%, … 100%) repeated measure ANOVA. The analysis revealed main effects for experimental condition, F(2,28) = 19.28, $p < 0.001$, and trajectory position, F(10,140) = 1834.47, $p < 0.001$, and an interaction between experimental condition and trajectory position, F(20,280) = 12.68, $p < 0.001$. Decomposition of the interaction between experimental condition and trajectory position revealed that FV reaches had a greater displacement from the start position at each point in the reaching trajectory than D1 and D3 reaches(with the exception of the 0% maker; see Table 3 for all post-hoc statistical tests and Figure 15a). Furthermore, the post-hoc analyses indicated that D1 and D3 reaches had the same displacement at each marker.

Table Three

Limb position across the reaching trajectory. T-scores of the post-hoc comparisons for the interaction between experimental condition and marker.

| | Marker | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0% | 10% | 20% | 30% | 40% | 50% | 60% | 70% | 80% | 90% | 100% |
| FV vs D1 | n/a | 5.1*** | 5.5*** | 5.8*** | 6.0*** | 5.6*** | 4.9*** | 4.1*** | 3.3** | 2.7* | 2.4* |
| FV vs D3 | n/a | 5.7*** | 6.1*** | 6.2*** | 6.5*** | 6.1*** | 5.6*** | 4.8*** | 4.1*** | 3.6** | 3.3** |
| D1 vs D3 | n/a | 0.3 | 0.7 | 1.0 | 1.4 | 1.1 | 1.0 | 1.0 | 1.0 | 1.3 | 1.3 |

Note that the degrees of freedom for all t-tests are 14

Significance is indicated as follows: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

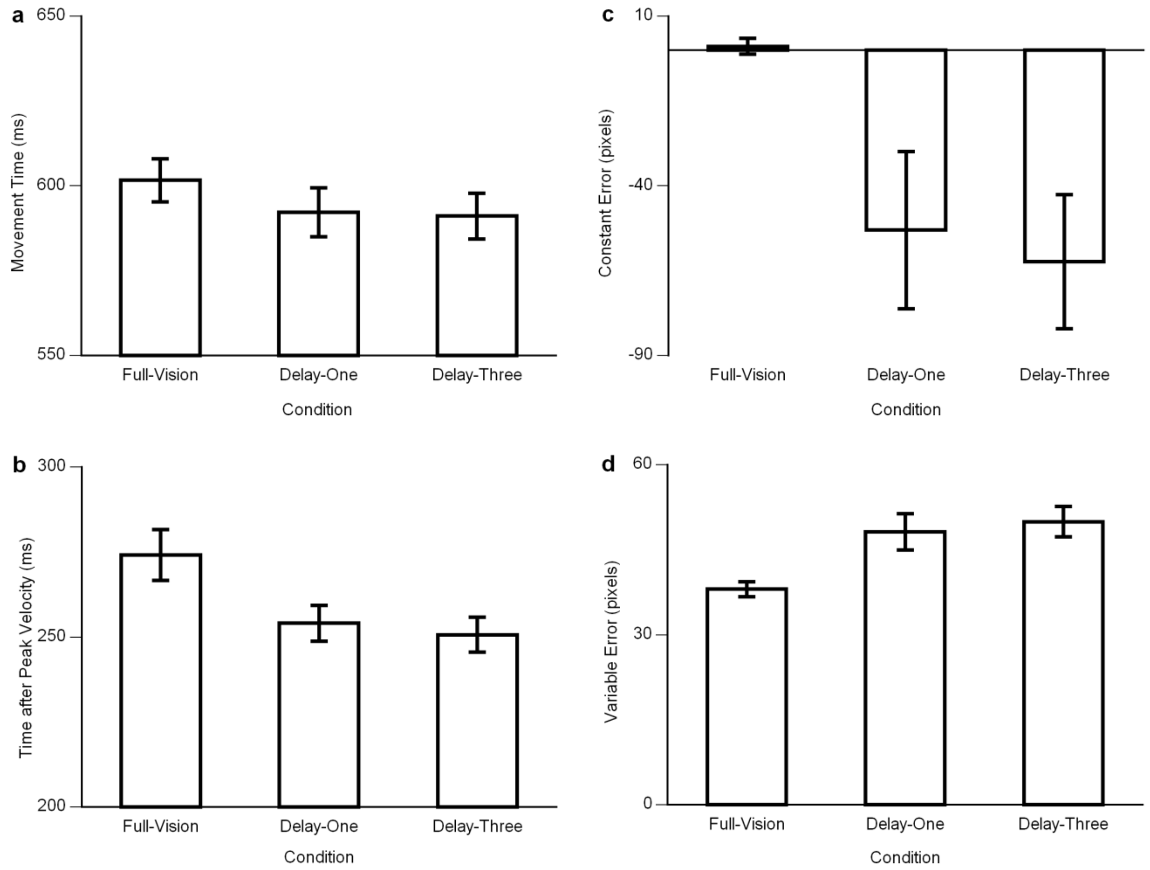

**Figure 14. Behavioural Results: (a) Movement time (ms) as a function of experimental condition. (b) Time after peak velocity (ms) as a function of experimental condition. (c) Constant error (pixels) as a function of experimental condition. (d) Variable error (pixels) as a function of experimental condition. Error bars represent the standard error of the mean.**

Additionally, we also calculated instantaneous acceleration across the reaching trajectory and submitted these values to a 3 (experimental condition: FV, D1, D3) by 9 (limb position: 10%, 20%,… 90%) repeated measures ANOVA. The analysis yielded a main effect for trajectory position, $F(8,112) = 26.72$, $p < 0.001$, and an interaction between experimental condition and trajectory position, $F(16,224) = 5.98$, $p < 0.001$. Post-hoc examination of the experimental condition by trajectory position interaction indicated that acceleration for FV reaches was greater than D1 and D3 reaches at 20% and 60% of the reaching trajectory, and was less than D1 and D3 reaches at 90% of the reaching trajectory. Acceleration for FV reaches was also greater than that of D1 reaches at 10% of the reaching trajectory. In sum, these data suggest that peak acceleration occurred earlier for FV reaches relative to D1 and D3 reaches (20% versus 30%) as did peak deceleration (70% versus 80%)(70% of the reaching trajectory; see Table 4 and Figure 15b for more detail).

Table Four
Instantaneous acceleration across the reaching trajectory. T-scores of the experimental condition and marker.

| | Marker | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 10% | 20% | 30% | 40% | 50% | 60% | 70% | 80% | 90% |
| FV vs D1 | 2.68* | 2.45* | 2.04 | 0.28 | 1.72 | 3.62** | 2.15 | 0.96 | 6.04*** |
| FV vs D3 | 1.6 | 2.24* | 1.01 | 1.06 | 0.78 | 2.86* | 1.15 | 1.78 | 6.74*** |
| D1 vs D3 | 0.84 | 0.3 | 1.16 | 1.34 | 1.56 | 1.08 | 1.4 | 1.94 | 0.81 |

Note that the degrees of freedom for all t-tests are 14
Significance is indicated as follows: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

### *Electroencephalographic Results*

*Prior to Movement Initiation.* Visual inspection of the ERPs locked to movement onset revealed an initial negative deflection (the BP: Cui & Deecke, 1999a, 1999b; Shibasaki & Hallet, 2006; Shibasaki et al., 1980) that was maximal at channel FCz (mean voltage: FV= -1.7uV; D1 = -1.83 uV; D3 = -2.45 uV; Figure 16). Statistical analysis of

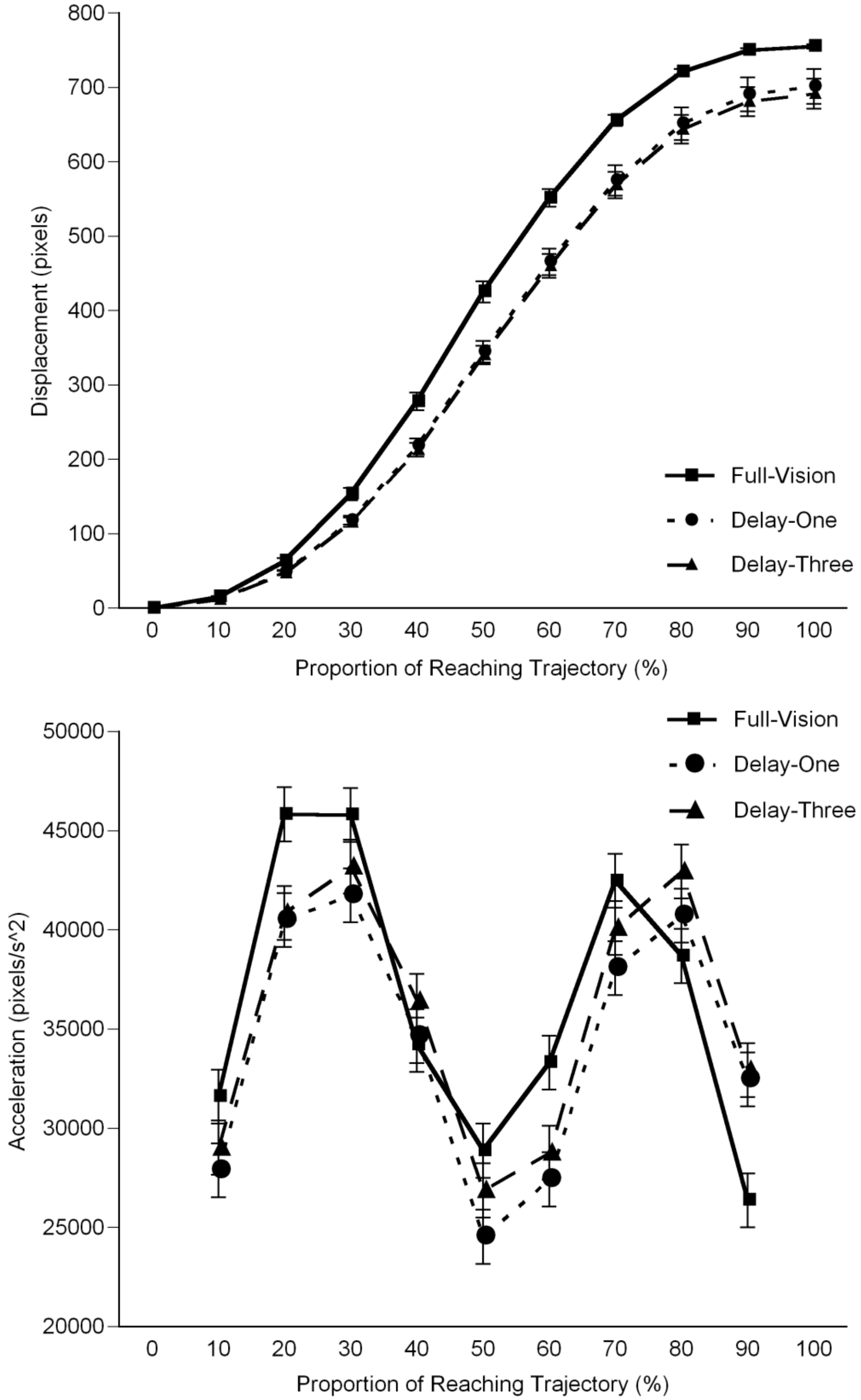

**Figure 15. Behavioural Results: (a) Displacement (pixels) as at 10% increments of the reaching trajectory for each experimental condition. (b) Instantaneous acceleration (pixels/ms2) at 10% increments of the reaching trajectory for each experimental condition. Error bars represent the standard error of the mean.**

the frontal-central BP (see above) revealed that it did not differ in amplitude between experimental conditions, F(2,28) = 0.77, p = 0.473. Analysis of the contralateral BP revealed an effect for channel, F(1,14) = 11.85, p = 0.004, that indicated the BP measured at channel C3 had a greater mean negative voltage than the BP measured at channel C4.

*During the Movement.* Visual inspection of WMCMPs following movement onset revealed an initial negative peak at 50 ms that was maximal at channel FCz in all three conditions (Figure 16; mean amplitude from 0 to 100 ms: FV = -9.67 uV; D1 = -7.53 uV; D3 = -8.95 uV). The timing and scalp distribution of this peak are similar to previous accounts of the N+50 motor potential (Cui & Deecke, 1999a, 1999b; Tarkka & Hallett, 1991; Shibasaki & Hallett, 2006; Figure 16). Following this initial peak, we also observed a positive-going deflection at 180 ms which was followed by a negative-going deflection at 300 ms at the frontal-central electrode sites. Given that the type of behaviour executed in the present experiment differed greatly from the type executed in previous motor potential experiments (e.g., button presses, discrete finger movements), it is difficult to compare these peaks with the other within-movement cortical motor potentials previously described in the literature (e.g., P+90, N+160, P+300). For this reason, we did not examine specific components (e.g., the N+50), but instead examined the mean voltage for frontal-central WMCMPs and contralateral WMCMPs.

The analysis of the frontal-central WMCMPs indicated an effect for experimental condition, F(2,28) = 8.66, p < 0.001. Post-hoc decomposition of this main effect demonstrated that FV reaches had larger negative mean voltages during the aiming movement than D3 reaches (t(14) = 2.48, p = 0.026), which in turn had larger negative mean voltages than D1 reaches (t(14) = 2.40, p = 0.031). Given the conditional effect on

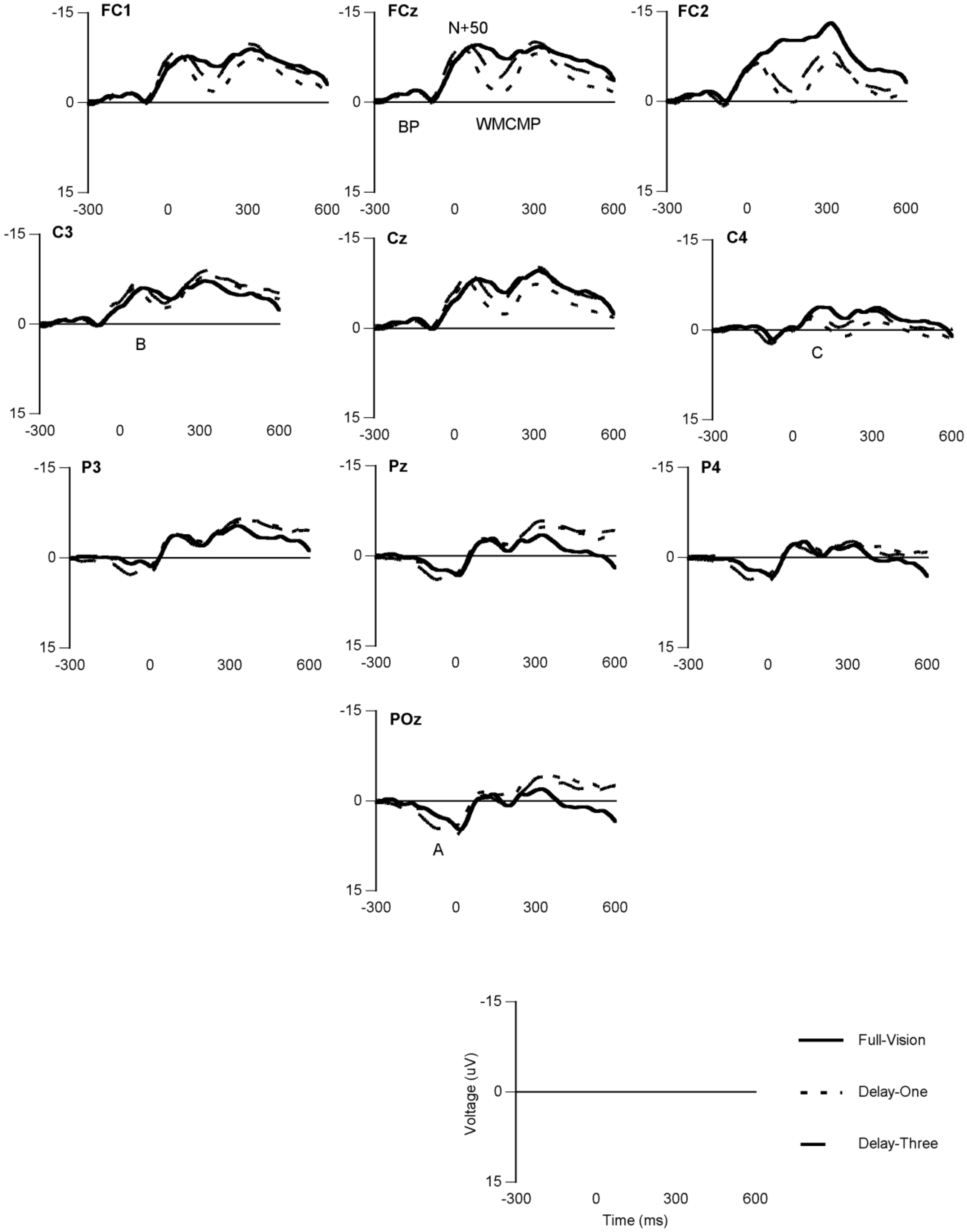

**Figure 16. Averaged ERP waveforms for the full-vision, delay-one, and delay-three conditions. Zero ms corresponds to movement onset. Negative voltages are plotted up by convention.**

the WMCMPs, we also decided to investigate the relationship between the amplitude of the WMCMPs and constant error. Interestingly, we found that the amplitudes of the frontal-central WMCMPs were negatively correlated with the constant error values (Pearson $r = -0.323$, $p < 0.05$). In other words, larger WMCMPs were associated with greater movement accuracy. The analysis of the contralateral WMCMPs demonstrated a main effect for channel, $F(1,14) = 47.62$, $p < 0.001$, which indicated a greater mean negative voltage at channel C3 relative to channel C4 during the aiming movements. Recall that all aiming movement were made with the right hand. As such, this contralateral negativity is in line with lateralised cortical motor potentials associated with movement execution (Cui & Deecke, 1999a, 1999b; Shibasaki et al., 1980; Shibasaki & Hallet, 2006)(see Figure 17).

*Online Control.* Recall that activity within posterior parietal cortex is thought to play an important role in the online control of movement (see introduction). Interestingly, visual inspection of the waveforms associated with the aiming movement revealed a positive-going deflection starting at approximately 350 ms that was maximal over posterior electrode sites (specifically, channel Pz: Figure 16) and that was not present over frontal-central sites. We statistically examined this deflection by submitting the mean voltage from 400 to 500 ms at channel Pz to a 3 experimental condition (FV, D1, D3) repeated measures ANOVA. This analysis indicated an effect for experimental condition, $F(2,28) = 4.32$, $p = 0.023$. Post-hoc analysis indicated that FV reaches had a greater positive mean voltage than D1 ($t(14) = 2.57$, $p = 0.022$) and D3 ($t(14) = 2.71$, $p = 0.017$) reaches during this time window. The mean positive voltage between 400

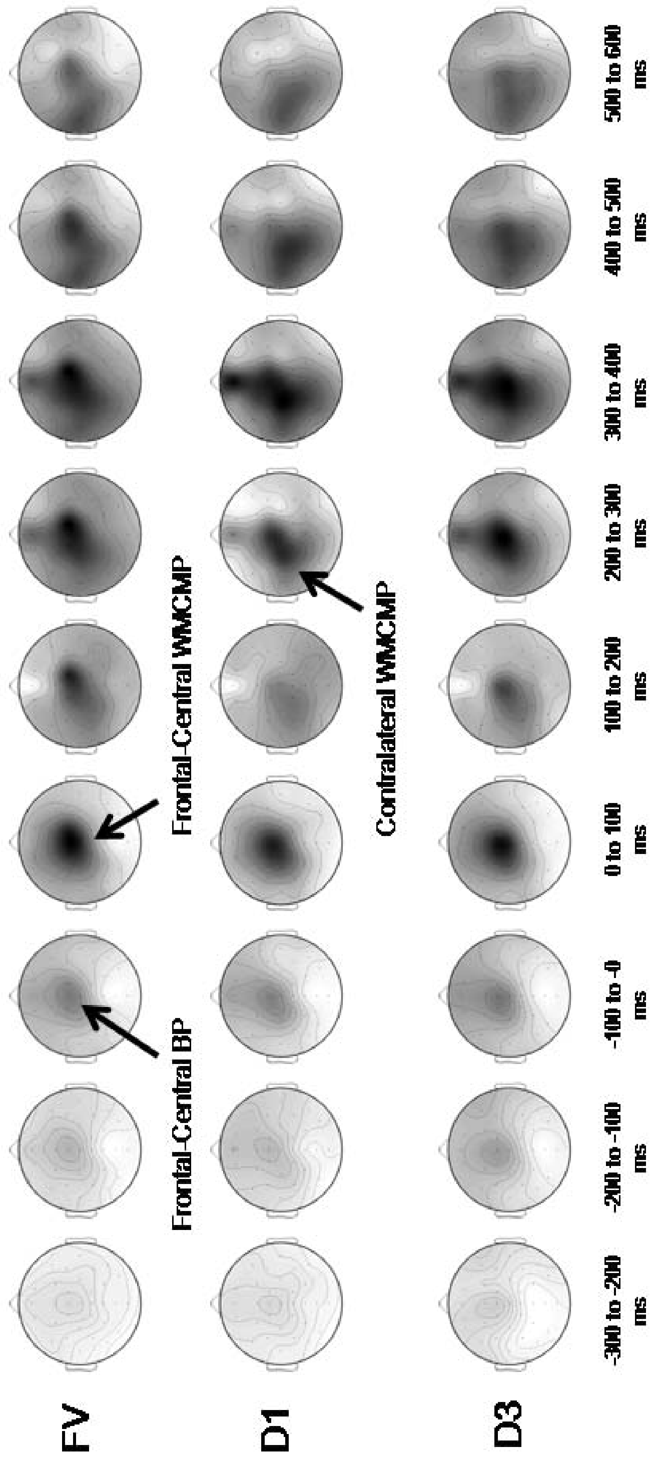

**Figure 17. Standardized mean scalp topographies (100 ms windows) from 300 ms prior to onset to 500 ms after movement onset for each experimental condition.**

and 500 ms did not differ between D1 and D3 reaches (t(14) = 0.04, p = 0.966).

*Movement End.* Finally, given previous research examining the ERN within the context of motor tasks, we analysed the difference waves created by subtracting the on-target from the off-target ERPs for FV reaches (Figure 18a). This analysis revealed a negative-going deflection (-6.29 uV, latency 235 ms) that was maximal at channel FCz (Figure 18b) and that was significantly greater than zero (t(14) = 6.33, p < 0.001). The scalp distribution and timing of this ERP component is consistent with previous work examining ERNs evoked during the performance of motor tasks (Krigolson & Holroyd, 2006, 2007a, 2007b) and other accounts of the fERN (Holroyd & Coles, 2002; Holroyd & Krigolson, in press; Miltner et al., 1997).

## *Discussion*

### *Cortical Motor Potentials and Movement Planning*

When one reaches to a target in the absence of visual information, real-time egocentric information is not available for movement planning. Instead, memory-guided reaches are planned from a stored representation of the environment, and as a result are less accurate than visually-guided reaches. The behavioural results of the present study support this contention. Specifically, we found that FV reaches were more accurate and less variable than D1 and D3 reaches. Of particular interest in the present study was whether or not these differences in movement accuracy could be explained in terms of differences in the cortical motor potentials associated with reach planning.

Prior to movement onset in all of the experimental condition (FV, D1, D3), we observed cortical motor potentials that shared similarities with those observed during the

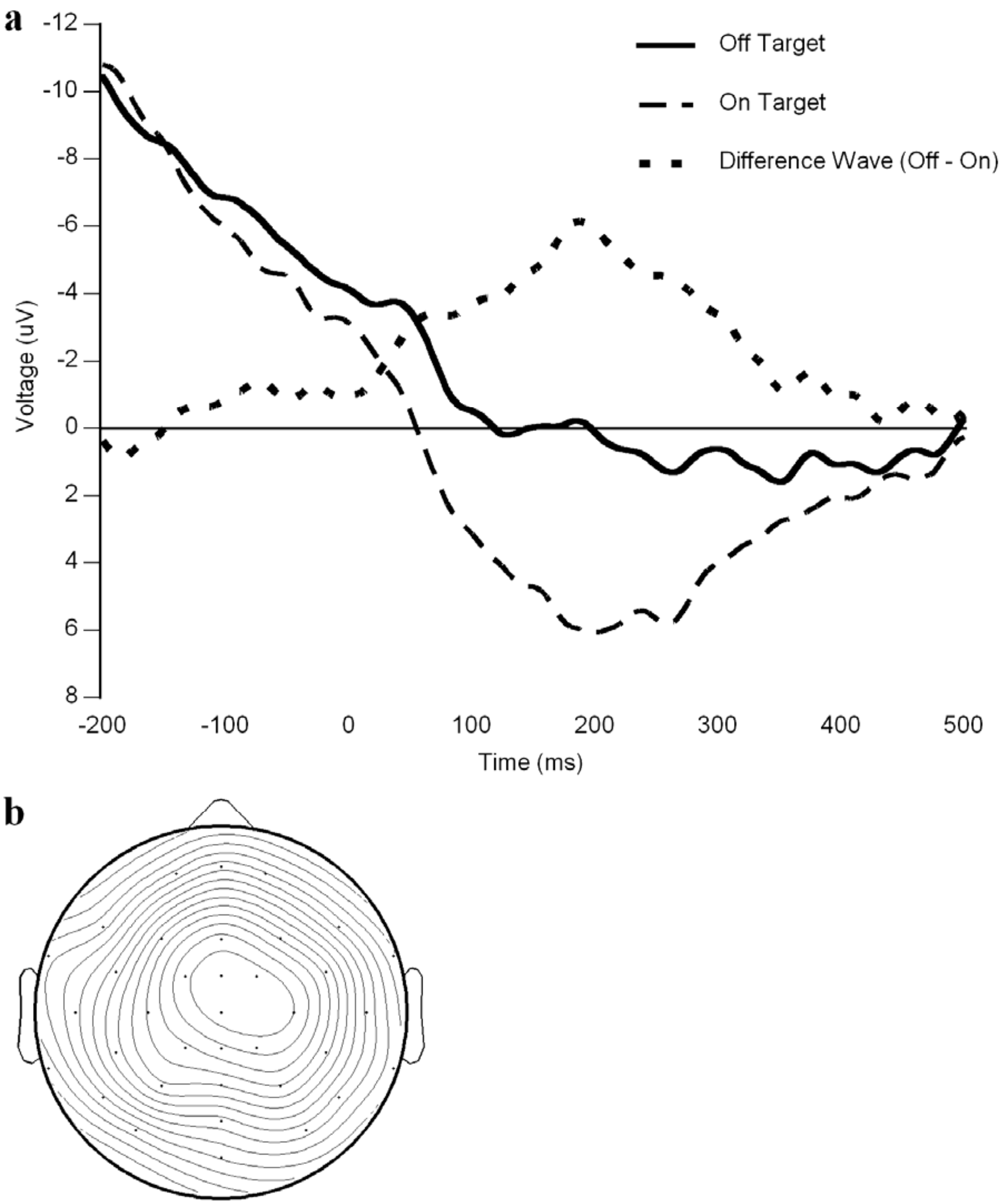

**Figure 18. (a) Averaged ERP waveforms and the difference waveform locked to movement end for on-target and off-target trials in the full-vision condition. Zero ms corresponds to movement end. Negative voltages are plotted up by convention. (b) Scalp distribution of the ERN for the difference waveform at 188 ms.**

performance of simple finger movements. Specifically, we found a negative ERP component with a frontal-central scalp distribution consistent with accounts of the early BP (Shibasaki et al., 1980; Shibasaki & Hallett, 2006). Recall that the early BP has been localised to SMA and is thought to be related to movement preparation and planning (and termed here the frontal-central BP). While the duration of the frontal-central BP in the present experiment may seem quite brief (less than 300 ms), this finding is not inconsistent with previous accounts that have found that the duration of the BP is related to the speed of the movement, such that rapid movements tend to have short BPs (Jankelowitz & Colebatch, 2002; Masaki, Takasawa, & Yamazaki, 1998. As we found no differences in the amplitude of the frontal-central BP across experimental conditions, we conclude that there are no observable differences in the ERP between visually-guided and memory-guided reaches associated with this early preparatory SMA activity. Recall that the late BP is associated with recruitment of motor neurons in area M1 (Gerloff et al., 1998; Shibasaki & Hallett, 2006) and as such it is typically maximal over a central region of the scalp contralateral to the hand utilised to perform the experimental task (e.g., channel C3 for right handed movements). The results of the present experiment demonstrated that the contralateral BP was greater at channel C3 relative to channel C4, a not surprising result given that all participants were right handed. However, the as with the frontal-central BP, the contralateral BP was not impacted by experimental condition.

Subsequent to the front-central BP, we also observed a positive-going deflection in the ERP over parietal cortex prior to movement onset. Although this result differs from the results of most other studies which indicate maximal BP amplitude over frontal-central or contralateral central areas of the scalp (e.g., Shibasaki et al., 1980), a recent study by Wheaton, Yakota, and Hallett (2005) yielded results that are comparable to our own. These investigators compared BP components evoked when participants performed complex movements (such as making gestures and pantomiming tool use) with BP components associated with simple finger movements. Similar to the results of the present study, Wheaton and colleagues found that the BP associated with complex movements was more negative over parietal electrode sites, in particular left parietal cortex. Wheaton et al. proposed that the posterior BP was related to the role of parietal cortex in the planning and execution of complex movements, such as grasps, gestures, and manual aiming movements (Andersen, Snyder, Bradley, & Xing, 1997; Desmurget et al., 1999, 2001; Fogassi & Luppino, 2005; Frey et al., 2005; Fridman et al., 2006; Grea et al., 2001; Haaland, Harrington, & Knight, 2000; Kimura & Archibald, 1974; Pisella et al., 2000; Tunik et al., 2005; Tunik, Rice, Hamilton, & Grafton, 2007). Although the polarity of this effect differs in the present experiment, the parietal deflection observed here may reflect the contribution of the PPC to the planning and execution of manual aiming movements.

Although we observed no conditional differences between the cortical motor potentials prior to movement onset, this was not the case for the frontal-central WMCMPs following movement onset. Our results indicated that the frontal-central WMCMPs associated with FV reaches exhibited greater mean negative amplitudes across the entire reaching trajectory than the WMCMPs associated with D1 and D3 reaches (which did not differ from each other; specifically see channel FC2 in Figure 16). Importantly, this pattern of results mirrors our results for limb displacement which we also found was also reduced for D1 and D3 reaches relative to FV reaches across the aiming trajectory. Furthermore, our results for constant error indicated that D1 and D3 reaches were undershot the target to a greater extent than FV reaches (Figures 14a, 15a). Finally, we found a significant negative correlation between the frontal-central WMCMPs and constant error – a result further suggesting a relationship between these measures. As with the pre-movement contralateral BP, the results of the present experiment revealed that the contralateral WMCMPs were greater over channel C3 than channel C4 – however, there were no conditional differences between the contralateral WMCMPs.

Previous research has associated the cortical motor potentials following the onset of brief discrete movements with the initial recruitment of motor neurons in primary motor area (N+50), kinaesthetic feedback, and/or reafferent activity from motor areas (P+90, N+160, P+300; Cui & Deecke, 1999a, 1999b; Shibasaki et al., 1980; Shibasaki & Hallett, 2006). We believe the WMCMPs associated with the aiming trajectories in the present study reflect multiple N+50 peaks - in other words, ERP deflections reflecting activity in frontal motor structures such as SMA, LPA, and/or M1. Indeed, given the greater movement duration in the present study relative to that of previous research, it stands to reason that continued activity within the aforementioned motor areas would be required. Furthermore, consistent with the execution of right-handed aiming movements, in all conditions we observed greater activity over left M1 (i.e., the contralateral WMCMP; see Figure 16, 17). Given these findings, we suggest that the reduced WMCMPs associated with D1 and D3 reaches resulted from relative inactivity of SMA, LPA, and/or M1 neurons in these conditions, which in turn led to reduced displacement being traversed across the trajectory and thus undershooting of the movement target.

We also observed an interesting relationship between the WMCMPs and the acceleration profiles for FV, D1, and D3 reaches. Visual inspection of instantaneous acceleration values plotted across the reaching trajectory (Figure 15b) and the WMCMPs at the frontal-central electrode sites (Figure 16, channels FC1, FCz and FC2) highlights the similarities of these two measures. Note that visual inspection of the frontal-central WMCMPs reveals two negative peaks, one at approximately 50 ms (N+50) and one at approximately 320 ms, time points which correspond roughly to 20% and 60% of the reaching trajectory[13]. In a similar manner, our analysis of acceleration across the reaching trajectory revealed that at 20% and 60% of the reaching trajectory, FV reaches had greater instantaneous acceleration values than D1 and D3 reaches (Figure 15b). Indeed, this relationship results suggests that the N+50 and the N+320 WMCMPs reflect activity within SMA, LPA, and/or M1 that results in changes in force production associated with an ongoing movement and thus reduced acceleration (and therefore displacement – see above). In sum, we propose that the reduced WMCMPs in the D1 and D3 conditions resulted in reduced force being generated in these conditions (and thus reduced accelerations) which resulted in reduced limb displacement across the aiming trajectory in the D1 and D3 conditions which led to greater target undershooting in these conditions – a hypothesis supported by the significant negative correlation between the WMCMPs and constant error.

Although the results of the present study suggest a relationship between cortical motor potentials, force, and reach accuracy it remains unclear why the cortical motor

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[13] As we solely wanted to make a broad comparison here, we determined ERP latency values by visual inspection of the grand average waveforms. In a similar fashion, we determined the movement time values associated with 20% and 60% of the aiming trajectory by multiplying these percentages with the mean movement time.

potentials for memory-guided reaches were reduced across the movement trajectory. Theories derived from behavioural evidence hold that the visuo-motor system operates in real-time – movements are planned just prior to movement onset to take advantage of accurate up to date egocentric visual information (Glover, 2004; Goodale & Milner, 2002; Westwood & Goodale, 2003; Westwood, Heath, & Roy, 2001, 2003). When reaches are executed in memory-guided conditions, information for movement planning is derived from a stored sensory representation of the movement environment which degrades with time, making it not as metrically precise as real-time egocentric visual information (Elliott & Madalena, 1987, Glover, 2004; Heath, 2005; Krigolson & Heath, 2004; Westwood, Heath, & Roy, 2000, 2001, 2003). Thus, the reduction in accuracy observed in memory-guided conditions can be attributed to a more proximal estimate of the target location (Binsted, Rolheiser, & Chua, 2006; Glover, 2004; Rolheiser, Binsted, & Brownell, 2006; Westwood, Heath, & Roy, 2001, 2003). With this in mind, one possible explanation why the cortical motor potentials were reduced for D1 and D3 reaches in the present experiment is that reaches in these conditions were planned with a stored sensory representation that indicated the movement target was more proximally located than it actually was.

### *Online Control*

To date, we are not aware of an ERP component associated with the sensorimotor transformations necessary for the online control of movement. However, in a series of studies Krigolson & Holroyd (2006, 2007a, 2007b) have found posterior ERP components that may be linked to this process. In the first of these studies participants performed a continuous tracking task in which they manipulated a joystick to keep a cursor centred between two moving barriers. Krigolson and Holroyd (2006) found that when a tracking error occurred (the participant's cursor contacted one of the barriers) an ERN - an ERP component associated with the evaluation of response errors and error feedback - was elicited. However, following the ERN an ERP component with a parietal scalp distribution was observed which Krigolson and Holroyd speculated may represent the updating of online control systems following a tracking error (see also Krigolson & Holroyd, 2007a). In another study, Krigolson and Holroyd (2007b) had participants complete aiming movements on a computer monitor by manipulating a joystick. The results of this study indicated that if the target jumped to a new location during the movement, two posterior ERP components were elicited: an early lateralised negative component (latency ≈ 140 ms) with a parietal-occipital scalp distribution and a later positive component (latency ≈ 350 ms) with a central parietal scalp distribution. Although the exact nature of these components remains unclear, Krigolson and Holroyd (2007b) speculated that the early component may reflect processing of the target jump and that the late component (the P300) may reflect either context updating of the movement environment (Donchin & Coles, 1988) and/or decision related phasic activity of the locus coeruleus norepinephrine system (Nieuwenhuis et al., 2005b).

When aiming movements are made with vision, the motor system takes advantage of this and implements highly effective online adjustments to the current motor command to ensure that the movement is accurate. In line with this, we found that in the FV condition a greater proportion of movement time was spent following peak velocity than in the D1 and D3 conditions, a result typically associated with greater engagement of online control processes (Elliott et al., 1999; Heath et al., 1998; Jeannerod, 1986). Recall that converging evidence from imaging and patient studies suggests that the sensorimotor transformations necessary for online adjustments to an ongoing motor command occur within PPC (Desmurget et al., 1999; 2001; Grea et al., 2001; Pisella et al., 2000). Interestingly, in the present study we found that the waveforms associated with the FV condition exhibited a greater mean positive voltage over parietal electrode sites during the second half of the aiming movement – an effect possibility indicative of PPC activation related to the online control of movement. To date, there is no known ERP component associated with PPC and online motor control processes, however, parietal ERP components have been observed following a sudden change in target location during the performance of a joystick aiming task (c.f. Krigolson & Holroyd, 2006). Thus, it may be that the greater parietal cortical motor potentials associated with FV reaches in the present study are reflective of PPC and online control processes. With that said, given the brief duration required to implement visually based online corrections (≈ 100 to 120 ms: Desmurget & Grafton, 2000; Jeannerod, 1988; Paillard, 1996), or even the proposed "instantaneous" corrections possible in a system reliant upon forward models (c.f. Desmurget & Grafton, 2000; Wolpert and Ghahramani, 2000), it is difficult to infer whether the changes in cortical motor potentials we observed in the present study reflect a motor plan generated in advance of movement onset or online adjustments to the current motor command. Further research is needed to clarify this issue.

### Movement End

Previous research utilising electroencephalographic means to examine error processing and motor control has demonstrated that certain motor errors elicit an ERN (Krigolson & Holroyd, 2006, 2007a, 2007b), an ERP component associated with the evaluation of response errors (Gehring et al., 1993; Falkenstein et al., 1991) and the processing of error feedback (Miltner et al., 1997). The ERN is thought to reflect the activity of a reinforcement learning system involving anterior cingulate cortex and the basal ganglia (Holroyd & Coles, 2002). A recent theory has incorporated this reinforcement learning theory of the ERN into a hierarchical framework where different levels of motor errors are processed by different brain systems (Krigolson & Holroyd, 2006, 2007a, 2007b). Specifically, the hierarchical theory proposes that "low-level" motor errors (e.g., deviations between the desired and actual motor command) are evaluated by posterior error systems (i.e., PPC) whereas high-level motor errors (i.e., violations of a system goal) are evaluated by the anterior cingulate – basal ganglia system. In line with the reinforcement learning theory of the ERN, the hierarchical theory of error processing suggests that high-level errors are utilised to modify future motor actions. In the FV condition in the present study we found that an ERN was elicited when participants finished a trial off-target. This result fits the hierarchical error processing hypothesis, as finishing off-target violated the goal of the task and thus an ERN was elicited. Furthermore, on off-target trials a reinforcement learning signal could be utilised to modify future motor output in order to improve the accuracy of subsequent aiming movements.

### *Conclusion*

To the best of our knowledge the results of the present study represent the first attempt to utilise electroencephalographic recordings to analyse the differences between visually-guided and memory-guided aiming movements. Here, we report three important findings. One, our results demonstrate that the amplitude of cortical motor potentials differ when vision is, and is not, available during aiming movements. Importantly, our results suggest that the reduced accuracy typically associated with memory-guided aiming movements can be attributed to reduced cortical motor potentials during the aiming movements. Two, the results of the present study also revealed greater posterior ERP activity during FV trials, a result that may be representative of PPC activity related to the online control of movement. Three, our results support the hierarchical error processing theory (c.f., Krigolson & Holroyd, 2006, 2007b) as we found that an error-related negativity was elicited when participants finished a trial off-target in the FV condition.

# General Discussion

The planning and control of goal-directed movement requires the evaluation of different types of errors. First, the motor system needs to be able to evaluate and correct for errors that occur during an ongoing movement. Termed "low-level" errors, these errors reflect a discrepancy between an actual and a desired motor command. Second, the motor system also needs to be able to evaluate whether or not the movement goal has been achieved. Importantly, these "high-level" errors indicate the failure of a system goal and therefore indicate the need to modify subsequent motor behaviour. The primary goal of the present research was to identify the electroencephalographic correlates of low-level and high-level motor errors. Additionally, the present research sought to provide a theoretically and biologically plausible framework for the interaction of the neural systems responsible for the evaluation of low-level and high-level motor errors.

## *Electroencephalographic Correlates of Low-Level Error Processing*

### *The N140*

Recall that in Experiment Two participants performed a joystick aiming task which required them to move a cursor on a computer monitor from a start location to a target location by manipulating a computer joystick. On some trials, the target moved to a new location immediately following movement onset thus changing the desired motor command. As such, on target perturbation trials the change in target location constituted a low-level motor error as it created a discrepancy between the actual and the new desired motor command. The results of Experiment Two indicated that two components were elicited by the target perturbation, a N140 component and a P300 component (see below). The N140 component exhibited a lateralized scalp distribution that was maximal at electrode PO7. Given that the C1 waveform (40-60 ms) is representative of processing in primary visual cortex (Clark et al., 1995), the timing of the N140 along with its scalp distribution suggest that it represented a more advanced stage of visual processing. Note that in Experiment Two participants were required to fixate in the middle of the screen at all times, placing the target (and thus the target perturbations) in the right visual field. The morphology of the N140 component observed is similar to that of the N1, an ERP component sensitive to target location within the visual field that is thought to reflect the orienting of visuo-spatial attention (Clark & Hillyard, 1996; Kremlacek, Kuba, Chulbnova, & Kubova, 2004; Shoji & Ozaki, 2006; Van Velzen & Eimer, 2003). It is also worth noting that the latency of the N140 observed in Experiment Two is too early to be the N2PC, a posterior contralateral ERP component which is thought to reflect the focusing of attention onto a target item (Praamstra, 2006; Praamstra & Oostenveld, 2003).

In Experiment Three participants performed an aiming task similar to the one utilised in Experiment Two. However, there were three important differences between the paradigms worth noting. One, in Experiment Three participants viewed a virtual display and moved the target from the start location to the target location by making two dimensional aiming movements on a graphics tablet. Two, in Experiment Three the mean movement time was much less than in Experiment Two (550 ms versus 1500 ms). Three, in Experiment Three participants fixated on the target location placing the target in central vision as opposed to fixating on the middle of the display as in Experiment Two. The results for Experiment Three indicated that a small N140 component was elicited by the target perturbations, however this component was not statistically different from the control condition. In Experiment Three the reduction in N140 amplitude may be attributed to the fact that participants fixated on the target location as opposed to the middle of the computer display. As such, the results of Experiment Three are in line with those of Experiment Two in that they support the hypothesis that the N140 reflects information processing sensitive to visual field (e.g., the orienting of visuo-spatial attention: Clark & Hillyard, 1996). However, the results of Experiment Three also suggest that the processes underlying the N140 are not involved in the online control of movement.

In sum, the results of Experiments Two and Three suggest that the N140 most likely represents the processing of visual information sensitive to visual field. Supporting this, the scalp distribution for the N140 in Experiment Two was maximal over left occipital cortex, and not posterior parietal cortex – a neural region that is thought to play a key role in the online control of movement (Desmurget et al., 1999; Desmurget et al., 2001; Grea et al., 2002; Pisella et al., 2000). Furthermore, the relationship between N140 amplitude and the location of the target relative to the participants' fixation point also suggests that this component is not directly related to the online control of movement. Indeed, the N140 observed in the present research may be the N1, an ERP component sensitive to visual field that is associated with the focusing of visuo-spatial attention on a target location (Clark & Hillyard, 1996). Further research is needed to clarify the exact relationship between the N140 component and online motor control processes.

### *P300*

In Experiment Two it was also found that the target perturbations evoked a P300 component. The P300 is a large positive ERP component with a parietal scalp distribution that increases in amplitude with decreasing target frequency (Donchin & Coles, 1988). There are several reasons to hypothesize that the P300 may be representative of processes underlying online motor control. One, the P300 has a source near the temporal-parietal junction placing it near PPC (Calhoun et al., 2006; Halgren et al., 1995; Kiss et al., 1989). Two, the P300 has recently been linked to decision related phasic activity of the LC-NE system (Nieuwenhuis et al., 2005b). Phasic increases of norepinephrine are thought to facilitate rapid decision making by increasing the gain of targeted neurons. Within the context of Experiment Two, when the target unexpectedly changes location the motor system needs to make rapid decisions to change the current motor command. Thus, while the P300 may not directly be representative of PPC activity and the online control of movement, it may be representative of processes that facilitate this process. Interestingly, in Experiment Two participants adjusted their motor output to accommodate the target perturbation after the onset and peak of the P300, a result in line with the LC-NE motor control hypothesis.

Unfortunately, the results of Experiment Three cast doubt on the possibility that the P300 is representative of processes supporting the online control of movement. Specifically, the results of Experiment Three paralleled those of Experiment Two - target perturbations elicited a P300 waveform. However, in Experiment Three it was possible to directly compare the onset of the P300 with the onset of changes in vertical acceleration of the corrective movements. Importantly, this comparison indicated that the onset of the P300 waveform occurred after the change in vertical acceleration, a result suggesting that the P300 is not representative of online control processes within PPC or phasic activity of the LC-NE system (Nieuwenhuis et al., 2005a, 2005b).

While the aforementioned results seem to rule out the possibility that the P300 associated with the online control of movement, the P300 may be indicative of feed-forward motor control processes. In particular, the results of the Experiments Two and Three are in line with research that suggests the P300 is representative of context-updating (Donchin & Coles, 1988). The P300 context-updating hypothesis posits that a P300 is elicited when an internal model of the environment is updated due to the receipt of new information. Within the context of this hypothesis, it seems reasonable to assume that a change in target location would result in an updating of the internal model of the environment. Interestingly, recent computational theories propose that the motor system utilises forward predictive models during the planning and execution of goal-directed actions (Desmurget & Grafton, 2000; Wolpert & Ghahramani, 2000). Essentially, forward models utilise efference copy, afferent feedback, and information about the movement environment to predict the consequences of motor actions. I propose that the P300 evoked by the target perturbations in Experiments Two and Three may reflect an updating of an internal model of the environment which is utilised to improve the effectiveness of forward predictive models of control. As with the N140, further research is needed to clarify this contention.

### *Slow Parietal Waveforms*

When reaches are made with vision, the motor system is thought to take advantage of the availability of real-time egocentric information from the dorsal visual stream to implement error nullifying corrections to the current motor command (Goodale & Milner, 1992; Goodale et al., 2004; Milner & Goodale, 1993). Recall that in Experiment Four, participants made reaches to a stationary target in both visually-guided and memory-guided conditions. The behavioural results indicated that during full-vision reaches participants engaged online control processes to a greater extent than during memory-guided reaches. Specifically, while no statistical differences between visually-guided and memory-guided reaches were observed in terms of movement time, visually-guided reaches spent a greater proportion of movement time following peak velocity. Although it is difficult to gauge the extent to which online movement modifications occur during an aiming movement strictly from behavioural data, it is generally accepted that increased time after peak velocity reflects greater online control during a reaching movement (Elliott et al., 1999; Heath et al., 1998; Jeannerod, 1986).

Interestingly, in Experiment Four a comparison of the visually-guided and memory-guided waveforms during the second half of the aiming movement (i.e., after peak velocity) revealed that visually-guided reaches exhibited a positive deflection that was maximal over parietal cortex (channel Pz) relative to memory-guided reaches during this time range. While this scalp area is consistent with evidence localising the online control of movement to posterior parietal cortex (Desmurget et al., 1999; Desmurget et al., 2001; Grea et al., 2002; Pisella et al., 2000), it is difficult to say whether or not the positivity observed for visually-guided reaches during this time frame reflects the online control of movement. Furthermore, visual observation of the parietal positivity revealed that its amplitude continued to increase following movement end, a result that is difficult to reconcile with online motor control processes that would have occurred during the actual movement. With that said, it is possible that ERP component overlap is an issue here and the parietal ERP component associated with online motor control could be superimposed on a parietal ERP component such as the P300.

## *Electroencephalographic Correlates of High-Level Error Processing*

### *The ERN*

As outlined in the introduction, the ERN is a component of the event-related brain potential that is sensitive to response errors (the response ERN: rERN) and error feedback (the feedback ERN: fERN) (Falkenstein et al., 1991; Gehring et al., 1993; Miltner et al., 1997). Recently, it has been proposed that the ERN represents a temporal difference prediction error conveyed by the midbrain dopamine system from the basal ganglia to anterior cingulate cortex. The prediction error signal represented by the ERN is thought to occur at the earliest indicator that events are worse than expected, and as such, the timing of the ERN represents the evaluation of motor efference copy (rERN) or error feedback (fERN; Holroyd & Coles, 2002). Importantly, this reinforcement learning signal is thought to play an important role in helping to train motor control units to select appropriate behaviours for a given context. Within the context of motor control, the basal ganglia - anterior cingulate system could be utilised to improve future motor output.

In Experiment One participants performed a continuous tracking task in which their sole goal was to keep a cursor from coming into contact with two moving barriers. The experimental results revealed that when participants committed a tracking error, whether it was a result of an experimental manipulation or whether it was due to an internal error, an ERN was elicited. In other words, an ERN was elicited in Experiment One whenever participants failed to achieve the desired system goal of not coming into contact with one of the two barriers. Also of interest was the timing of the ERN, which was neither consistent with the rERN or the fERN but may have reflected the propagation of the error signal with learning. In other words, as participants learned that certain barrier movements inevitably led to a tracking error the ERN propagated back in time from the time of barrier contact to the onset of these events. However, it is also possible that the unique timing of the ERN in Experiment One was due to the novelty of the task. Importantly, these results provide the first evidence that the basal ganglia - anterior cingulate reinforcement learning system is sensitive to errors made during the performance of a continuous motor task.

As outlined above, in Experiment Two participants performed a computer aiming task in which they manipulated a joystick to move a cursor from a start position to a target location. Recall that on some trials, the target jumped to a new location forcing participants to make a corrective movement to the current motor output. Furthermore, on half of these trials the participants' cursor did not respond to corrective movements of the joystick. Interestingly, the analysis of the ERP waveforms locked to movement onset revealed that an ERN was not elicited by the target perturbations. On all target perturbation trials, at the time of the target jump, participants did not know whether or not they could successfully correct for the change in target location. When the target perturbations occurred, to the best of their knowledge, participants still thought the goal of the task was achievable. Within the context of a hierarchical error processing hypothesis, an ERN should only be elicited when a system goal is violated. As such, and as predicted by the hypothesis, the target perturbations in Experiment Two did not elicit an ERN as the target perturbations did not reflect the failure of a system goal.

Also of interest in Experiment Two were the blocked corrective movements. A comparison of the waveforms for the unblocked and blocked corrective movements revealed than an ERN was elicited by the blocked corrective movements. Again, this result is in line with the hierarchical hypothesis as a blocked corrective movement indicated that the goal of the task – to reach the target – was not attainable. The timing of the ERN evoked by the blocked corrective movement was consistent with the fERN (Miltner et al., 1997). While at first one might think that a rERN would be elicited by a blocked corrective movement, participants were not actually able to evaluate efference copy to evaluate the success (or failure) of a corrective joystick movement. Instead, to evaluate the success of the motor command participants had to make a comparison between the trajectory of the cursor and their motor command – a process more akin to the processing of external feedback. As such, the blocked corrective movements elicited a fERN.

In Experiment Three participants performed a manual aiming task similar to the joystick aiming task utilised in Experiment Two. However, in Experiment Three participants manipulated a computer cursor by making actual aiming movements with a stylus instead of by manipulating a joystick. As with Experiment Two, the analysis of the waveforms locked to movement onset revealed that an ERN was not elicited by the target perturbations. However, opposite to the findings of Experiment Two, the results for Experiment Three indicated that blocking the corrective movement did not elicit an ERN. While at first glance this result appears to be contradictory to the hierarchical hypothesis, there are two possible reasons why an ERN was not elicited by the blocked corrective movements in Experiment Three. Following the blocked corrective movements in Experiment Three the medial-frontal system received conflicting feedback from the visual and proprioceptive systems, and as a result the evaluation of whether or not an error had occurred was not possible. More likely however, it could be that given the restricted time constraints of this task the corrective movements were implemented ballistically, and as a result the evaluation of the movement was left until movement end.

The evaluation of endpoint error in a manual aiming task also reflects the evaluation of a system goal. Indeed, if a participant finishes a trial off-target then this would indicate a failure of the system goal as opposed to finishing a trial on-target (which would indicate that the system goal had been achieved). In Experiments Three and Four participants made two dimensional manual aiming movements in visually-guided and memory-guided conditions to a target location. In both experiments, examination of the visually-guided conditions revealed that an ERN was elicited when participants finished a trial off-target. The timing of the ERN in both experiments was consistent with previous accounts of the fERN (Miltner et al., 1997), a result that makes sense given that to evaluate endpoint error participants had to make a visual comparison between the final position of the cursor and the target location.

In sum, the results of all four experiments suggest that an ERN is elicited as soon as the medial-frontal system detects that a high-level motor error has occurred. Given the hypothesized role of the medial-frontal system in reinforcement learning, these data suggest that motor learning at the neural level may be driven to some extent by reinforcement learning principles. In other words, when a motor command is successfully executed the medial-frontal system releases a dopaminergic signal that helps reinforce the motor command that was responsible for the movement. Conversely, when a movement is executed incorrectly the medial-frontal system may utilise the midbrain dopamine system to signal that an alternative motor command should be selected.

### *Post Movement Parietal Components*

The ERN appears to be elicited by errors indicating the failure to meet a system goal. However, the role of the ERN in the modification of motor behaviour remains unclear. Given that the ERN is thought to reflect a reinforcement learning signal utilised to modify future behaviours (Holroyd & Coles, 2002), it is possible that the ERN observed following high-level motor errors reflects a reinforcement learning signal utilised to improve future motor performance. Experiment One provided electroencephalographic evidence supporting this contention. Specifically, the results indicated that a negative waveform with a parietal scalp distribution followed after the ERN. We propose that this parietal negativity may reflect the impact of a reinforcement learning signal sent from either the basal ganglia or anterior cingulate cortex to posterior parietal cortex to improve the performance of this system on future trials. In other words, following the evaluation that a system goal has not been achieved by the medial-frontal system, it sends a learning signal to other neural systems (i.e., posterior parietal cortex) to improve future behaviours. Interestingly, and in line with this proposal, recent computational research has demonstrated that a temporal difference prediction error signal can be used to train a forward model for predictive motor control (Branning et al., 2005).

## *A Framework for Hierarchical Error Processing*

One of the goals of the present research was to develop a theoretical model for hierarchical error processing in the human brain (see Figure 19). The model is biologically plausible, can account for movement planning and control processes, and includes systems which evaluate low-level and high-level motor errors. Furthermore, the

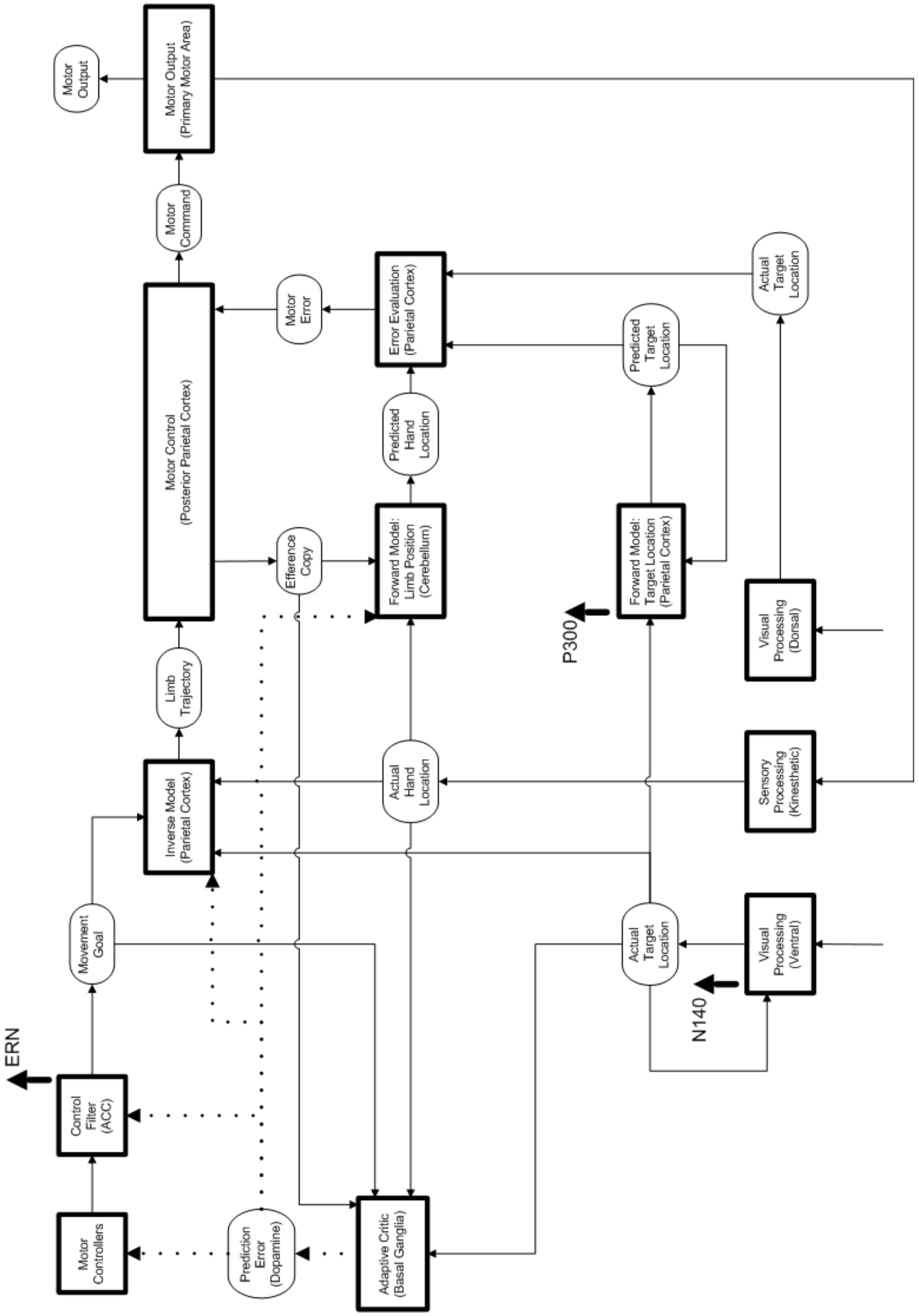

**Figure 19. The neural model for hierarchical error processing in the human brain.**

model accounts for the ERP components observed in the experiments which comprise the present research. During movement planning, it is thought that the motor system utilises an inverse model to determine the motor command which will achieve the desired system goal (Baud-Bovy & Viviani, 1998; Haggard, Leschziner, Miall, & Stein, 1997; Kawato, 1999; Wolpert & Ghahramani, 2000; Wolpert et al., 1998). Within the framework of the hierarchical model, the inverse model unit receives input from frontal systems specifying the movement goal. Note that within the hierarchical model detailed here, these frontal systems are simplified to two units – the motor controllers which reflect differ movement strategies and the control filter which selects the appropriate controller for a given action. Following reception of a movement goal, the inverse model unit utilises proprioceptive and visual information from sensory systems about the location of the movement effector and the target to determine the appropriate motor command (e.g., the limb trajectory). Following this and just prior to movement initiation, the motor system generates the actual motor command to perform the desired movement.

Unfortunately, the initial motor command is not always sufficient to achieve the desired movement goal. For instance, neuromotor noise at the time of movement planning and/or execution may result in reduced movement accuracy. In a similar fashion, sudden, unexpected change in the movement environment following movement onset can also invalidate the initial motor command. In these instances, the motor system has to be able to evaluate errors within an ongoing movement to ensure successful execution of goal-directed actions. Within, the hierarchical model, these errors which reflect a discrepancy between the actual and the desired motor command are considered to be “low-level” and are corrected via online control processes.

To correct low-level errors, the current motor output is continually monitored by a system that includes posterior parietal cortex and the cerebellum following movement initiation (Desmurget et al., 1999; 2001; Desmurget & Grafton, 2000; Kawato, 1999; Schweighofer, Arbib, & Kawato, 1998; Schweighofer, Spoelstra, Arbib, & Kawato, 1998; Wolpert & Ghahramani, 2000; Wolpert et al., 1998). More specifically, at each time step a forward model within the cerebellum utilises the efference copy of the current motor command and information about the actual system state derived from the dorsal visual stream to predict the end state of the current movement. Continual comparisons are made between the predicted end state and the desired end state to gauge the correctness of the current motor command (Desmurget & Grafton, 2000; Miall et al., 1996; Nowak, Topka, Timmann, Boecker, & Hermsdorfer, 2007). If a discrepancy is detected between the predicted and desired states, an online control unit within posterior parietal cortex attempts to resolve the discrepancy between the desired and predicted end states by modifying the current motor command. By allowing continual comparisons between the predicted end state and the desired end state, the forward model provides the motor system with the ability to implement corrective sub-movements without the delays associated with the processing of sensory feedback (Desmurget & Grafton, 2000; Wolpert & Ghahramani, 2000). Although a forward model for limb position is included in the hierarchical model, the structure of neurons within the cerebellum prevents cerebellar activity from being detectable with ERPs. Indeed, the effectiveness of predictive control systems combined with the inability of ERPs to detect cerebellar activity may explain why the present research found limited evidence for an ERP signature for the resolution of low-level motor errors in Experiments One and Four.

Although the predictive forward model is highly effective, in some instances it can fail (e.g., if there is an unexpected change in the movement environment), forcing the motor system to adapt an online control strategy where real-time feedback based adjustments are made to the current motor command (Elliott et al., 1995a, 1995b; Elliott et al., 2001; Heath, 2005; Heath et al., 1998; Milner & Goodale, 1993, 1995). Essentially, the lack of input from the forward model results in comparisons being made between the actual state of the limb (instead of the predicted end state) and the desired state with the low-level error reflecting the discrepancy between these inputs (Desmurget & Grafton, 2000). As outlined above, once the low-level error is determined, the motor system (i.e., posterior parietal cortex) adjusts the current motor command accordingly. It is generally assumed that these feedback based modifications are generally slow (≈ 100 to 150 ms: (Desmurget & Grafton, 2000; Jeannerod, 1988; Paillard, 1996) and occur during the later half of a reaching movement (Chua & Elliott, 1993; Elliott et al., 2001; Heath et al., 1998; Khan et al., 2006). As noted above, experimental evidence suggests that the mediation of low-level motor errors occurs within posterior parietal cortex (Desmurget et al., 1999; Desmurget et al., 2001; Grea et al., 2002; Pisella et al., 2000).

In the present research the target perturbations in Experiments Two and Three would have negated that effectiveness of the predictive control system and forced feedback-based movement modifications. Interestingly, in Experiments Two and Three the target perturbations elicited a P300 ERP component (and an N140 component in Experiment Two). While the P300 component has a parietal scalp distribution in line with research associating this region with the online control of movement (see above), the behavioural results from Experiment Three suggest that the P300 is too slow to be representative of an online control system. Instead, within the hierarchical model the P300 is thought to reflect the updating of a forward model responsible for predicting the behaviour and location of the target. Specifically, within the hierarchical model a forward model utilises visual input to predict target behaviour and location at movement end. The output of the forward model is sent to parietal cortex to facilitate online control, and is also sent back to the unit for comparisons with subsequent input. When the target location changes suddenly (as in Experiments Two and Three), a P300 is evoked when the forward model detects the discrepancy with the previous output and updates its model of the environment (i.e., the context-updating hypothesis of the P300, Donchin & Coles, 1988). Importantly, the updating of the forward model for target behaviour and location would allow the motor system to better prepare subsequent movements in an uncertain environment. Furthermore, this hypothesis is also in line with theories which posit posterior parietal cortex has predictive capabilities (Desmurget & Grafton, 2000). As with the P300, the N140 evoked by target perturbations in Experiment Two was too slow to represent online control processes and furthermore was not elicited by target perturbations in Experiment Three. Consistent with previous accounts of the N100, within the hierarchical model the N140 is associated with the focusing of visuo-spatial attention (e.g., Clark & Hillyard, 1996; Kremlacek, Kuba, Chulbnova, & Kubova, 2004; Shoji & Ozaki, 2006; Van Velzen & Eimer, 2003).

In spite of the online control processes outlined above, not all motor output achieves the desired result. Thus, the motor system has to evaluate errors which indicate the failure of a system goal. For instance, if one reaches for a target and misses, the motor system needs to be able to determine that the movement was not successful and then use this information to modify the next movement. The results of the present research demonstrate that a variety of high-level motor errors elicited an ERN. Importantly the ERN is thought to be representative of a temporal difference prediction error that is generated by the impact of a dopaminergic reinforcement learning signal sent from the basal ganglia to anterior cingulate cortex. Within this framework, the basal ganglia is thought to be an adaptive critic which evaluates whether or not an error has occurred and anterior cingulate cortex is thought to be a control filter for selecting the appropriate motor behaviour. The hierarchical model includes these units, and the projections that associate these units with the motor system. Importantly, the basal ganglia - anterior cingulate system is an ideal candidate for the processing of high-level motor errors as the reinforcement learning signals generated by this system could be used to improve future motor output. As mentioned previously, a recent computational model demonstrates just this – a temporal difference prediction error was used to train a forward model of control (Branning et al., 2005).

One possible interpretation of the posterior waveform which followed the ERN in Experiment One is that it reflects the impact of a reinforcement learning signal on posterior parietal cortex - a result that has been replicated in a subsequent study which examined the impact of predictive cues on the basal ganglia – anterior cingulate system (Krigolson & Holroyd, 2007a). In this study, two experiments were conducted in which participants completed a base tracking task similar to the one employed in Experiment One. Recall that during performance of the base tracking task in Experiment One participants encountered two types of “difficult corners”: one during which participants inevitably committed a tracking error (*unlocked difficult corners*) and one during which participants could not commit a tracking error as the computer briefly controlled the participants' cursor (*locked difficult corners*). In the first experiment of the 2007a study, one half of the unlocked and locked difficult corners were preceded by predictive visual cues which indicated that a difficult corner was about to occur (500 ms warning; colour indicated type of corner). Note that participants were not told the meaning of these cues and had to determine their relevance for themselves. As in Experiment One, the results revealed that unpredicted tracking errors elicited an ERN. Interestingly, the experimental results also revealed that an ERN was elicited by cues predicting a subsequent tracking error. Furthermore, the latency of the ERN propagated back in time over the course of the experiment towards the onset of the predictive cue – a result possibly tied to participants learning the meaning of the predictive cue as the experiment progressed (see below). As noted above, the experimental results also revealed a parietal negativity which peaked shortly after the ERN for both the unpredicted and predicted tracking errors – a waveform which we interpreted as the impact of a reinforcement learning signal of posterior parietal cortex. The results of the second experiment were similar in that we found that another type of predictive cue which indicated a subsequent tracking error (loss of joystick control) also elicited an ERN. As with the first experiment, in the second experiment the latency of the ERN propagated back in time towards the onset of the predictive event during the course of the experiment. However, in this second experiment we only found a parietal waveform following the ERN for unpredicted tracking errors.

The reinforcement learning theory of the ERN predicts that an ERN is elicited at the earliest indicator that events are worse than expected. Within the hierarchical model, an ERN is elicited at the earliest indication that a system goal has not been achieved. The present research provided strong evidence for this contention. In Experiment One tracking errors were found to elicit an ERN. In Experiment Two an ERN was elicited when corrective joystick movements were blocked – a manipulation that made it impossible to achieve the desired system goal. In Experiments Three and Four an ERN was elicited when participants finished a trial off-target – again, a behavioural result indicating the failure of a system goal. Providing further support to the hierarchical hypothesis, the results of the present research also indicated that target perturbations did not elicit an ERN. Within the hierarchical framework this makes sense at the target perturbations only construed a low-level error – their occurrence did not indicate that a system goal had not been achieved – it only indicated that the current motor command needed to be changed.

### *Conclusion*

Human error processing during motor control appears to be reliant upon a series of hierarchically organised systems. Low-level errors indicating a discrepancy between a desired and an actual motor command appear to be mediated by an online control system that utilises predictive forward models and external feedback. The results of the present research revealed two ERP components that may be associated with the processing of low-level motor errors – the N140 and the P300. The N140 appears to reflect the focusing of visuo-spatial attention, but may be representative of online control processes. The P300 was also evoked by target perturbations, and the present results support its proposed relationship with the updating of a forward model of the environment. While not directly related to the online control of movement, this internal updating process may be utilised in a feed-forward manner to modify future movements. The results of the present research also revealed greater parietal activity during visually-guided reaching movements, a result that may be related to neural processing in posterior parietal cortex associated with the online control of movement.

The results of the present study also provide direct evidence that high-level motor errors indicating the failure of a system goal are evaluated by a reinforcement learning system involving the basal ganglia and anterior cingulate cortex. Specifically, the experimental results revealed that an ERN was elicited by high-level errors. Interestingly, the ERN is thought to represent a temporal difference prediction error signal and thus, it could be representative of the motor learning process. Here, I have also provided a model that unifies the low-level and high-level system. Specifically, this model brings together a typical model for motor planning and control and puts it within the framework of the reinforcement learning model of the ERN. The model has the potential to explain the ERP results from the experiments reported here, in addition to suggesting a unified framework for motor learning and control within the human brain (c.f., Doya, 2000, Doya et al., 2001).

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