Hierarchical Error Evaluation: The Role of Medial-Frontal Cortex in Postural Control

Cameron D. Hassall, Stephane MacLean & Olave E. Krigolson

Published online: 10 Sep 2014.
RAPID COMMUNICATION
Hierarchical Error Evaluation: The Role of Medial-Frontal Cortex in Postural Control
Cameron D. Hassall, Stephane MacLean, Olave E. Krigolson
Psychology and Neuroscience, Dalhousie University, Halifax, Nova Scotia.

ABSTRACT. Motor error evaluation appears to be a hierarchically organized process subserved by 2 distinct systems: a higher level system within medial-frontal cortex responsible for movement outcome evaluation (high-level error evaluation) and a lower level posterior system(s) responsible for the mediation of within-movement errors (low-level error evaluation). While a growing body of evidence suggests that a reinforcement learning system within medial-frontal cortex plays a crucial role in the evaluation of high-level errors made during discrete reaching movements and continuous motor tracking, the role of this system in postural control is currently unclear. Participants learned a postural control task via a feedback-driven trial-and-error shaping process. In line with previous findings, electroencephalographic recordings revealed that feedback about movement outcomes elicited a feedback error–related negativity: a component of the human event-related brain potential associated with high-level outcome evaluation within medial-frontal cortex. Thus, the data provide evidence that a high-level error-evaluation system within medial-frontal cortex plays a key role in learning to control our body posture.

Keywords: balance, ERP, fERN, outcome evaluation, posture, reinforcement learning

Maintaining an equilibrium position—standing, sitting, or otherwise—is of critical importance for everyday function. To maintain equilibrium the motor system needs to continually evaluate and correct postural errors—small deviations from balance equilibrium. These deviations may be induced externally, such as tripping over an obstacle (postural perturbations; see Jacobs & Horak, 2007), or internally (e.g., neuromotor noise; Meyer, Abrams, Kornblum, Wright, & Smith, 1988). In addition to mediating these low-level errors the motor system also has to monitor and evaluate the movement goal itself (e.g., maintaining a target posture) for high-level errors (e.g., a fall). Thus, through the successful detection and correction of low-level movement errors a high-level postural goal such as standing upright may be achieved. Conversely however, if low-level errors are not resolved a high level will result—for instance a fall or other undesired posture.

Although existing research suggests that postural control is subserved by midbrain systems (Magnus, 1926; Takakusaki, Saitoh, Harada, & Kashiyayanagi, 2004; Visser & Bloem, 2005) recent evidence suggests that regions of cortex also play a role in maintaining balance (Jacobs & Horak, 2007; Mochizuki, Boe, Marlin, & McIlroy, 2010; Slobounov & Newell, 2009). For example, stroke patients with cortical damage but intact brainstems show postural deficits while standing (Geurts, de Haart, van Nes, & Duyssens, 2005). In particular, the parietotemporal junction appears to play a crucial role in postural control, suggesting that normal sensory processing is needed in order to maintain balance (Bonan et al., 2004; Geurts et al., 2005; Pérénou et al., 2000). Behaviorally, and in line with the previous results targeting the parietotemporal junction, older adults who perform an attentional task simultaneously with a postural task exhibit decreased postural stability (Woollacott & Shumway-Cook, 2002).

In addition to the attentional system mentioned previously, another potential candidate cortical region that could play a role in postural control is the medial-frontal cortex. In a series of experiments, Krigolson and colleagues (Krigolson & Holroyd, 2006, 2007a, 2007b; Krigolson, Holroyd, Van Gyn, & Heath, 2008) demonstrated that when participants failed to achieve a movement outcome—either failing to reach a movement target or crashing during a continuous tracking task—a feedback error–related negativity (fERN) was elicited. The fERN is a component of the human event-related brain potential (ERP) with a frontal-medial scalp topography, occurring 200–350 ms after feedback. Source localization suggests that the fERN is generated within anterior cingulate cortex (Miltner, Braun, & Coles, 1997) and one influential account posits that the fERN reflects a reinforcement learning prediction error generated by the aforementioned medial-frontal system (Holroyd & Coles, 2002). In other words, the fERN may reflect a signal that is used to improve motor performance (i.e., motor learning). However, to date there is no evidence that demonstrates that the medial-frontal reinforcement learning system plays a role in the evaluation of high-level postural errors.

Our goal here was to provide novel evidence that high-level postural errors elicit a medial-frontal response that is typical of high-level movement outcome evaluation, namely the fERN. To do this we had participants perform a learnable postural control task while electroencephalogram (EEG) data were recorded. Participants controlled an on-screen cursor by shifting their center of pressure (COP), and were asked to move the cursor into one of four different target areas on each trial. Following each trial, participants
were given visual feedback indicating that they had hit or missed the target region. In a key manipulation, target size was adjusted as participants learned to hit targets with increasing accuracy. We hypothesized that as participants improved their postural control by hitting increasingly smaller balance targets, error feedback would elicit a fERN, suggesting the involvement of the medial-frontal system in the evaluation of postural movement goals.

Method

Participants

We tested 15 Dalhousie University students (12 women, 3 men; $M_{age} = 22$ years, $SD = 3$ years; age range = 18–27 years) with no known neurological impairments, and with normal or corrected-to-normal vision. All of the participants were volunteers who received credit points in an undergraduate psychology course for their participation. The study was conducted in accordance with the ethical standards prescribed in the Declaration of Helsinki and approved by the Health Sciences Research Ethics Board at Dalhousie University.

Apparatus and Procedure

Participants stood on a Wii Balance Board (WBB; Nintendo, Kyoto, Japan), 100 cm in front of a computer display with a 38 cm wide by 30 cm high viewable area. The WBB contains four force transducers arranged to measure force distribution, from which any subsequent changes in COP may be estimated with a reasonable level of reliability and validity (Clark, Bryant, Pua, McCrory, Bennell, & Hunt, 2010). The center of the display was elevated 110 cm off the ground. Participants used the WBB to perform a computerized targeting task (written in MATLAB [Version 7.14, the MathWorks, Natick, MA] using the Psychophysics Toolbox Extension [Version 3.0]; Brainard, 1997). OSCulator software (Version 2.13, Wildora, Sucy-en-Brie, France) converted the WBB force sensor values into two COP displacement values (anteroposterior and mediolateral) that were communicated to the MATLAB program. Participants were shown that their COP moved the cursor into a central target box (a 40 mm square) in the middle of the screen. Shortly after their COP was centered (1000 ms), one of the four target locations appeared on the screen. Each target was initially a 70 mm by 70 mm square located in one of the four corners of the display. After a random delay of 900 to 1100 ms, both the target and the cursor were occluded. Importantly, this manipulation forced participants to make a memory-guided postural movement to the target location (cf. Krigolson, Heinekey, Kent, & Handy, 2012). Shortly after target and cursor occlusion (400–600 ms), an auditory tone cued participants to initiate their postural movement. If the participant’s cursor left the central target box before the auditory tone occurred, the trial ended immediately and the message Too Fast was displayed. Following movement completion, participants were asked to hold the target position for 2500 ms. If participants were successful in maintaining the target posture for 2500 ms, they were shown a feedback screen with a checkmark indicating successful completion of the postural aiming movement. If participants were unable to enter the target boundary within 2500 ms, or if they left the target area early, they were shown a feedback screen with an X indicating an unsuccessful postural movement. In all cases (hits and misses) a delay was added such that the total time between the auditory tone and the feedback was between 5400 ms and 5600 ms. This was done so that the delay between the tone and feedback was the same, regardless of condition. See Figure 1 for an overview of the trial timing details. Following successful trials, the size of the target was decreased by 5%, and following unsuccessful trials it was increased by 5%. The change in target size following correct and incorrect feedback was done in order to equalize the total number of correct and incorrect trials, so that neither feedback type was more unexpected than the other, thus avoiding frequency-related N200 contamination of the fERN (Krigolson & Holroyd, 2006; Miltner et al., 1997). In total, participants completed six blocks of 25 trials each (blocks were separated by brief rest periods). During a training phase prior to the actual experiment (10 trials), participants practiced postural aiming movements where both the target and the cursor remained on the screen throughout the trial.

Data Collection

Our experimental program recorded the position of the COP throughout the trial, the outcome of each trial (successful or unsuccessful), and the target location and size. The EEG was recorded from 64 electrode locations using BrainVision Recorder software (Version 1.20, Brain Products, GmbH, Munich, Germany). The electrodes were mounted in a fitted cap with a standard 10–20 layout and were recorded with an average reference built into the amplifier. The vertical and horizontal electrooculograms were recorded from electrodes placed above and below the right eye, and on the outer canthi of the left and right eyes.
Electrode impedances were kept below 20 kΩ. The EEG data were sampled at 1000 Hz and amplified (Quick Amp, Brain Products, GmbH, Munich, Germany).

Data Analysis

Following data collection, the EEG data were filtered through a (0.1–25 Hz pass band) phase shift-free Butterworth filter and rereferenced to the average of the two mastoid channels. Next, ocular artifacts were corrected using the algorithm described by Gratton, Coles, and Donchin (1983), and all trials were baseline corrected using a 200 ms epoch prior to stimulus onset. Finally, trials in which the change in voltage in any channel exceeded 10 µV per sampling point or the change in voltage across the epoch was greater than 100 µV were discarded. In total, 9% of the data were discarded due to artifacts.

To test our hypothesis that high-level postural errors engaged the midbrain outcome evaluation system, we created ERP waveforms by averaging the EEG epochs for each event of interest (correct feedback, incorrect feedback) for each channel and participant. Based on an observation of the grand average waveform and previous work (Holroyd & Coles, 2002; Holroyd & Krigolson, 2007; Miltner et al., 1997) we quantified the fERN as the peak (i.e., minimum) of the difference wave (incorrect feedback waveform minus correct feedback waveform) 200–325 ms following feedback onset. The peak detection analysis focused on electrode Cz, based on the topography of the peak difference and previous research (Holroyd & Coles, 2002; Krigolson & Holroyd, 2006, 2007a, 2007b; Krigolson, Holroyd, Van Gyn, & Heath, 2008; Miltner et al., 1997).

All analyses were done in BrainVision Analyzer (Version 2.0.4, Brain Products, GmbH), and in MATLAB (Version 7.14, the MathWorks) using custom scripts. Peak velocities and target sizes were computed for each successful trial and averaged by block and participant. Repeated measures analyses of variance (ANOVCs) were used to determine if there was an effect of block number (1–6) on both target size, \(F(1, 14) = 7.0, p < .001\), and peak velocity, \(F(1, 14) = 2.7, p = .02\) (see Figure 2). Finally, there was a significant Pearson

Results

Since the size of the visually presented target was adapted for each participant on a trial-by-trial basis, there was no difference between the total number of successful trials and the total number of unsuccessful trials, \(t(14) = 1.1, p = .15\). Furthermore, repeated-measures ANOVAs revealed effects of block (1–6) on both target size, \(F(1, 14) = 7.0, p < .001\), and peak velocity, \(F(1, 14) = 2.7, p = .02\) (see Figure 2). Finally, there was a significant Pearson
correlation across participants between mean peak velocity and mean targets size, \( r(13) = .54, p = .037 \) (see Figure 3).

An analysis of difference waveforms locked to the onset of feedback revealed an ERP component with a latency (268 ± 9 ms) consistent with the fERN (tested against zero): \( t(14) = 6.4, p < .001 \) (see Figure 4; also see Holroyd & Coles, 2002; Miltner et al., 1997). To verify that the fERN was maximal at electrode Cz, we conducted two repeated measures ANOVAs with data from the peak detection analysis—one on peak data from the electrodes along the azimuth line (e.g., Fpz, AFz, Fz) and the other on peak data along the coronal midline (e.g., C3, Cz, C4). Polynomial fits to the fERN along these lines revealed quadratic best fits, azimuth: \( F(1, 14) = 20.9, p < .001 \); coronal: \( F(1, 14) = 23.8, p < .001 \). The results of these analyses affirm that the fERN was indeed maximal at electrode Cz, in line with previous findings (Holroyd & Coles, 2002; Mathewson, Dywan, Snyder, Tays, & Segalowitz, 2008; Miltner et al., 1997; Ruchshow, Grothe, Spitzer, & Kiefer, 2002). To further validate these findings, we implemented a wavelet analysis for correct and incorrect trials that revealed scaled frontal-medial activity (correct < incorrect: see Figure 5), \( t(14) = 2.2, p = .03 \), in the lower theta range (3–6 Hz) 200–500 ms post feedback at electrode FCz, where activity in this range was maximal. This frequency difference mirrored our ERP result, and was consistent with previous work revealing greater frontal-medial theta power following incorrect feedback compared to correct feedback in this time range and at this scalp location (Cavanagh, Frank, Klein, & Allen, 2010; Hajihosseini & Holroyd, 2013).

**Discussion**

In the present experiment we found that feedback indicating that participants failed to achieve their movement goal in a postural aiming task elicited a fERN. Specifically, an analysis of the difference waveform derived by subtracting correct from incorrect feedback revealed a medial-frontal negativity with a timing and scalp topography consistent with the fERN (Krigolson & Holroyd, 2006, 2007a, 2007b; Krigolson et al., 2008; Miltner et al., 1997). Further, an analysis of our behavioral data revealed that participants improved at the postural aiming task—the size of the movement target decreased over the course of the experiment, indicating that participants were successfully moving their COP to the target locations. Further, participants’ peak velocity increased as participant skill improved—a result indicative of improved task ability (Elliott, Hansen, Mendoza, & Tremblay, 2004). Importantly, the fact that high-level postural errors elicited a fERN suggests that a reinforcement learning system within medial-frontal cortex (cf. Holroyd & Coles, 2002; Krigolson & Holroyd, 2006, 2007a, 2007b) plays a role in evaluating the outcome of postural aiming movements. Recall that previous studies examining the role of...
the medial-frontal system in movement outcome evaluation demonstrated that tracking outcome errors (Krigolson & Holroyd, 2006, 2007b) and aiming outcome errors (Anguera, Seidler, & Gehring, 2009; Krigolson & Holroyd, 2007a; Krigolson et al., 2008; Vocat, Pourtois, & Vuilleumier, 2011) elicited a fERN. It is important to note that the motor systems that underlie postural control are more typically associated with midbrain structures and systems (Magnus, 1926; Takakusaki et al., 2004; Visser & Bloem, 2005). While this of course is true given the overwhelming evidence to date, more recent evidence highlights the contribution of cortical regions to postural control (Jacobs & Horak, 2007; Slobounov & Newell, 2009). In line with this research, our results suggest that the medial-frontal outcome evaluation system may also contribute to postural control by monitoring these midbrain structures to provide signals indicating the success or failure of a given movement goal.

Finally, the results of the present study are consistent with the hierarchical error-processing hypothesis.

FIGURE 4. Medial-frontal response to correct and incorrect feedback: (a) grand average event-related brain potential waveforms at electrode Cz averaged to feedback onset; (b) scalp topography of the peak feedback error–related negativity difference.

FIGURE 5. Frequency response (power) to correct and incorrect feedback at electrode FCz. There was an enhancement in the theta frequency range following incorrect feedback compared to correct feedback. Inset topographies show the power distribution at 5.5 Hz and 300 ms post feedback, where the response was maximal.
(Krigolson & Holroyd, 2006, 2007a). The hierarchical theory states that movement evaluation is the province of at least two distinct levels of systems—a lower level system responsible for mediating low-level errors in the motor command or changes in the environment within a given movement, and a higher level system responsible for evaluating high-level errors: the success or failure of a given movement. Here, we provided evidence of high-level postural error evaluation. It should be noted that since we did not examine responses prior to visual feedback, our data do not shed any light on the evaluation of low-level postural errors as others have (e.g., Mochizuki et al., 2010). Specifically, our data suggest that higher level errors in postural control are evaluated by the medial-frontal system. Thus, one possible role of the medial-frontal system is to train the postural system via the evaluation of high-level errors (Holroyd & Coles, 2002; Krigolson & Holroyd, 2007a).

**FUNDING**

This research was supported by the Nova Scotia Health Research Foundation (Cameron D. Hassall) and by a Discovery Grant from the Natural Sciences and Engineering Research Council of Canada (Olave E. Krigolson).

**REFERENCES**


*Received November 16, 2013*

*Revised April 14, 2014*

*Accepted April 18, 2014*