ERP Components of Motor Adaptation

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ABSTRACT

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While the phenomena of motor adaptation and motor aftereffects are well documented, a full picture of the neural substrate underlying these phenomena remains elusive. The motor aftereffects generated when a force is removed that a subject has adapted to can be thought of as a motor error. The aim of this study was to determine whether cortical event-related potentials for motor errors could be detected. Participants made reaching movements and slowly adapted to a force that perturbed their reaches. On some trials the perturbing force was turned off without warning, and we found that this caused motor aftereffect errors. We compared the reaching movements made before the influence of a perturbing force to reaching movements made when the participant had fully adapted to a perturbing force and the force has been turned off. We found representative ERPs occurring at 255ms and 182ms for negativities and 345ms and 292ms for positivities at electrodes FCz and Cz. Amplitudes at these electrodes ranged
from -41.4mV to -36.8mV for negativities to 32.7mV to 35.1mV for positivities. All subjects showed error-related negativities at FCz and Cz and 62.5% and 75% of subjects showed error-related positivities at FCz and Cz respectively. Error-related negativities for these modalities occur earlier. It is unclear whether this reflects a difference in error processing between the motor system and other modalities or a limitation in our method.
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1 ERP Components of Motor Adaptation

The feed-forward control of reaching movements by the central nervous system is a well-documented phenomenon, one that allows for adaptation and compensation to disturbances that initially perturb the trajectory of a reach but no longer cause perturbations after performing a number of reaches (Jordan 1993). Disturbances that affect the trajectory of a reach can be the result of Coriolis forces due to body rotation (DiZio and Lackner 1994) or the result of velocity dependent force delivered through mechanical contact on the hand as reaching movements are attempted (Scheidt 2000). Two commonalities among these paradigms and results are that 1) no cues to the presence of the forces are available until after movement onset and 2) over the course of many reaches subjects learn to adapt to perturbations and regain reaching accuracy. This produces kinematic aftereffects as a result of expectation of a force that does not come (Shadmehr 1994). These aftereffects tend to be in the opposite direction of the force previously applied and persist for a short number of reaches before abating.
While motor adaptation and motor aftereffects are both well documented phenomena, a full understanding of the cortical processing underlying these phenomena has not been achieved. Evidence from physiological studies of the sensorimotor system suggests that cerebellar mechanisms are sufficient for error-based adaptation that occurs quicker than delays in the cortical sensorimotor system would allow (Shadmehr 2010). It is understood that the central nervous system plays a role in the storage and selection of “internal models” that mediate context-specific adaptations to motor demands. However the neural mechanisms under which these internal models are modified or the experience necessary to modify these models have not been sufficiently investigated. Understanding the cortical processing that occurs during motor adaptation and motor aftereffects has important implications for a fully developed theory of motor control and can help explain the link between the sensorimotor system and conscious/unconscious processing in cortex. The specific question addressed here is whether cortical correlates of motor errors can be observed in surface EEG recordings.

The motor aftereffects generated after a subject has adapted to a force and that force is removed can be thought of as a motor error. Removal of a force to which a subject has adapted causes errors in both trajectory and endpoint (DiZio and Lackner 1994). If characteristic event-related potentials (ERP) for motor errors could be uncovered, it would tell us a great deal about the timing of cortical processing as well as the neural substrate underlying these phenomena. Characteristic error-based ERP components have been found for mismatch errors in both auditory matching and visual matching tasks and are similar enough to suggest that this error-based ERP exists
irrespective of modality. (Falkenstein 2000) Researchers have identified a negative error-based ERP (Ne) occurring 80ms post-error component that is thought to represent response checking or error detection and a positive error-based ERP (Pe) occurring between 200 and 500ms post-error component that is thought to represent additional processing after errors that is functionally different from error detection. Ne components are shown in response to many different types of errors and across modalities, varying in time course but typically occurring within a range of 80-150ms post-stimulus presentation (Yeung 2004, Coles 2001). The Pe component was found to vary differentially from Ne suggesting that it doesn’t merely represent the end of the Ne. While it seems intuitive to reason what this Pe might actually represent, evidence from studies on older adults suggest that Pe is not conscious error recognition or adjustment of response strategy, or even emotional error assessment. (Falkenstein 2000) This leaves considerable uncertainty as to what Pe might represent, but what is evident is that both Ne and Pe can be used as an index of error-dependent brain activity across modalities. There is support for the existence of these error-dependent ERP components for visual and auditory errors but there is little empirical data on the use of error-based ERPs in motor control experiments.

There is, however, recent evidence that suggests a context-dependent mediator of motor responses exists that may have cortical representation during or after errors. (Frucht, Dizio, and Lackner 2012) This cortical representation of errors in the motor domain may be a shared mechanism of error recognition and error processing across all modalities or it may be unique for the motor dimension. If error-based ERPs accompany motor errors, they would provide evidence that the cortex is responsible for
processing motor error. It is, however, possible that error-based ERPs as described in visual and auditory modality research are not present in motor error, as the errors described in visual/auditory research are typically choice errors where a participant is making a choice between two options (Yeung, 2004), while for motor error there are many possible “incorrect” movements that may need be adjusted by cortical systems.

The current research attempts to identify error-dependent ERP components for motor error. Participants grasped the handle of a robot device that delivered velocity-dependent forces to perturb their reaches. On some trials the perturbing force was turned off without warning and the participant made motor aftereffect errors. We aimed to determine whether reaching movements made before the influence of a perturbing force would show characteristically different patterns of ERP compared to reaching movements made when a participant who has been fully adapted to a perturbing force made a reaching movement when the force has been turned off. The study sought to provide evidence to assess whether the ERP components of motor error take the same characteristic form as those ERP components shown for errors in the visual and auditory modalities or whether the motor errors generate distinct cortical activity. If there is a shared neural mechanism for error across modalities we could expect to see the components shown for error-dependent activity in other modalities: a Ne component occurring between 80 and 150ms and a Pe component occurring between 200 and 500ms. If the motor error ERP components differ significantly from these previously described error-related ERPs, it would suggest a difference between error processing in the motor system and error processing in visual and auditory modalities.
2 Method

Subjects

10 undergraduate and graduate students at Brandeis University were compensated for their participation in the hour long session. All were right handed and had no difficulty performing reaching movements. No significant gender bias was present in the sample (N of female = 6). All subjects gave informed consent and the experimental protocol was approved by the IRB.

Apparatus

The present study uses a robotic manipulandum (PHANTOM robot) to produce a velocity dependent force perpendicular to the direction of movement. The PHANTOM is a lightweight two-link robotic arm, which can be driven by torque motors to create forces in three dimensions. The subjects interact with the robot arm by grasping a handle attached to its terminus. The arm contains encoders from which the position of the tip and its derivatives can be computed at a high enough rates to generate movement-contingent forces at greater than 1200 Hz.
The reaching environment consists of a horizontal square 9.5” x 16.5” two-dimensional surface with LEDs at fixed intervals and an LED illuminated start button at one end. The environment is constructed so that a participant can reach the 20cm between start and end LEDs comfortably by only extending their arm and keeping their shoulder in a fixed position. A second LED that can serve as a target is located the same distance from the start button as the primary target but 5 cm to the left.

A g.tec 64-channel electroencephalography (EEG) system was used in the experiment. It was composed of 4 multi-modal biosignal amplifiers (model g.USBamp) with appropriate electrode interface boxes (model g.GAMMAbox) and power supply, 54 active biosignal electrodes (model g.LADYbird) mounted on standard 10-20 EEG positions on a fitted cap (model g.GAMMAcap), 2 active electromyography (EMG) electrodes positioned over the deltoid muscle of the reaching arm, and 1 ear clip electrode as the reference for the system. The PHANTOM data were collected at 100Hz and the EGG data at 256Hz to two different computers. A signal from the start button of the reaching target array was recorded in the EEG data stream to allow for synchronization of the reaching and EEG data.

Procedure

This procedure is adapted from studies performed in the Graybiel Spatial Orientation Laboratory (Panic 2011) and other motor control studies demonstrating adaptation and aftereffects (Scheidt 2000). Participants were seated in a comfortable chair in front of the reaching environment in such a way that their shoulder lined up with an LED button that served as the starting point for all reaching movements during the
task. The participants grasped the pointing end of the PHANTOM robot with their index finger extended and were told to “imagine the tip of the pointer is an extension of your index finger” and make goal directed reaching movements in a straight line towards an illuminated target. The target light was located 20cm from the starting point. The lights in the experimental room were kept dim to ensure the participants were only focusing on the start points and end points of reaching movements. The participants pressed a button that served as the starting point of the reaching movement, waited 500ms for the target to become illuminated, extended their arm at the elbow to reach the target light, and then returned to the starting point within 3 seconds.

Participants performed a block of 8 practice trials to ensure they were comfortable with the reaching movement and were moving so that peak velocity was approximately 600mm/s. They then performed 2 blocks of reaches to a target offset left of the straight line target, approximately where their trajectory would have ended up during aftereffect trials following adaptation to motor perturbations. Then participants performed 2 blocks of reaches to the standard straight line target (baseline trials). Following this, a velocity-dependent force of 0.006N x velocity perpendicular to the direction of motion for outward reaches was turned on and participants reached to the straight line target for 4 blocks of 8 trials each. It was expected that participants would exhibit either endpoint or trajectory errors but over time would regain reaching accuracy to near baseline levels, showing evidence of motor adaptation. After this series of adaptation trials, there were 16 blocks during which one of the 8 trials was without a force present. These force-absent “catch” trials were designed to detect motor aftereffects. These aftereffect trials occurred randomly within each block but couldn’t be
the first or last trial of a block to ensure that participants did not get two aftereffect trials in a row.

**Kinematic Analysis**

Data about the planar position of the robot pointer were transmitted during each trial to the computer to allow trial by trial analysis of reaching movements and to allow each trial to be time-locked to an EEG signal. Participant's reaches were grouped by trial type and the trajectories were inspected to make sure they had bell shaped velocity profiles. Peak trajectory and endpoint deviations were measured and analyzed across trial types to determine whether there were kinematic differences due to trial type. Peak trajectory deviation was determined as the largest lateral deviation from the straight line connecting the beginning and end of the reach, and endpoint deviation was determined as the lateral displacement of where the participant ended compared to where the target was. Baseline trials, which normally are straight reaches from button to target, were compared to 1) adaptation trials, which typically show trajectory and endpoint errors that decrease over time to resemble baseline reaches, and 2) aftereffect trials, which should have endpoint and trajectory errors in the direction opposite the force perturbation direction. Those participants who did not show evidence of motor adaptation or motor aftereffects due to lack of significant differences in error magnitude across trials were not included in subsequent analyses, because without evidence of adaptation it is not possible to induce motor errors or to show an error-related ERP of interest.

**EEG Analysis**
The EEG signal processing was time locked to release of the LED button which also signaled the start of the movement. Analysis was limited to 5 midline electrodes spanning the anterior to posterior cranial locations: Fz, FCz, Cz, CPz, Pz. Epochs of data for each trial and channel were extracted for .5 seconds before button release and 1 second after button release. The 16 baseline and 16 aftereffect trials were evaluated and compared, as they are congruent in terms of the lack of force but differ in the degree of expectation of the null force. In baseline trials, the lack of force is expected but in aftereffect trials the lack of force is unexpected and causes a motor error—a reach in the wrong direction—that might have a cortical response from an error comparison/recognition system. EEG grand averages for aftereffect trials and baseline trials were constructed, and a difference waveform was constructed by subtracting baseline grand averages from aftereffect grand averages to allow elucidation of the ERP components. Difference waveform construction is advantageous as it allows for the suppression of mutual response-locked activity between aftereffect and baseline trials (Falkenstein 2000). A 95% confidence interval was constructed around the difference waveform, and we searched for the minimum and maximum of the difference waveform and noted their latencies from movement onset, the signs and magnitude of the differences at those points, and whether they were in regions that were significantly different from zero.

Typically 6-8 trials are needed to get reliable and accurate error-based ERPs for a flanker task, so having 16 coupled trials was estimated to be sufficient to allow us to generate robust and accurate ERPs for each force condition and give enough statistical power to analyze cross-condition (Pontifex 2010).
3 Results

Kinematic Results

For first pass analysis, trajectory and endpoint errors were summed into a single aggregate measure of reaching error. Total error of baseline and aftereffect trials were compared. Overall, participants were found to have significantly different error magnitudes between aftereffect and baseline trials, $t(9) = 4.2377, p < .05$ This is evidence that the paradigm was able to reliably induce motor errors (see Fig. 1).

![Figure 1. Trajectory deviation by trial type. Baseline refers to the average of the last 16 baseline trials, 1st adaptation refers to the first adaptation trial, last16 adapt refers to the average of the last 16 adaptation trials, and 16 catch refers to the average of the 16 aftereffect trials.](image-url)
Further analysis was conducted to assess adaptation on a participant-level and the presence of aftereffect errors. Endpoint errors and trajectory errors were compared separately across conditions to detect any significant differences in error magnitudes and error type. It was found that of the 10 participants, 7 had significantly different peak trajectory magnitudes between aftereffect and baseline trials, 1 had significantly different endpoint error magnitudes between aftereffect and baseline trials, \( t(15)=3.866, p<.05 \), and 2 participants showed no significant differences in error at all. EEG analysis and difference wave construction accordingly only focused on the 8 subjects that had significant differences in reaching error magnitude across trial conditions. Among these 8 subjects, participants had a 10.9mm difference in total error magnitude between aftereffect and baseline trials. On average there was a 0.5mm difference in endpoint error between aftereffect and baseline trials, however for the subject who had significant endpoint error differences, the difference between endpoint error during adaptation and baseline trials was 3.2 mm. Participants had on average an 8.6mm difference in trajectory magnitude between aftereffect and baseline trials. Average peak velocity of reaching movements was recorded as 442mm/s with an average latency of 281.0ms.

**EEG Results**

Grand averages of baseline trials were constructed by averaging the baseline EEG response across trials for each subject at each electrode of interest. This average baseline EEG response was subtracted from each catch trial separately to produce 16 individual difference waves per subject and per electrode. These difference waves were then averaged across trials to produce a single difference waveform at each electrode...
for each participant. Figure 2 shows a difference waveform at electrode Cz with sample-wise standard error band for subject 2. In this figure, darker shaded areas represent regions that have been identified as significantly different from 0. Electrodes Fz, FCz, Cz, CPz, and Pz are the foci of Ne and Pe component analysis. The difference waveform was examined at regions where there exist significant differences from zero, using sample-wise t-tests, for the absolute minimum and maximum amplitude points and their latencies. If these latencies fell within the regions identified by sample-wise t-tests as being significantly different between aftereffect and baseline, they were considered as a legitimate ERP component and included in our analysis. The amplitude and latency for the maxima and minima were then averaged across participants.

Figure 2. EEG difference waveform for a typical subject. Darker shaded regions indicate significant deviation from zero. The minimum and maximum are highlighted with a dot.
Components identified for Fz, FCz, Cz, CPz, and Pz electrodes can be organized by location of the electrode and whether that electrode showed a positivity (Pe), a negativity (Ne), or both. Electrodes Fz, FCz, Cz, and CPz show first a negativity and later a positivity, while electrode Pz shows a positivity occurring at or before a subsequent negativity.

The ERP results can be compared to the kinematics to better understand when these positivities and negativities are occurring relative to the movement.
peak velocity on average occurs at 281.0 ms after button release, which indicates that many negativities occur at or just before peak velocity is reached. Error-based negativities and positivities were observed in a number of participants across electrodes. At electrode Fz, 87.5% of subjects who adapted showed a significant error-based negativity and 62.5% showed a significant positivity. At electrode FCz, 100% of subjects who adapted showed a significant error-based negativity and 62.5% showed a significant positivity. At electrode Cz, 100% of subjects showed a significant error-based negativity and 75% showed a significant positivity. At electrode CPz, 87.5% of subjects showed a significant error-based negativity and 62.5% showed a significant positivity. At electrode Pz, 75% of subjects showed a significant error-based negativity and 50% showed a significant positivity. For these subjects who showed significant ERP components, these components were averaged to generate an average amplitude and latency of negativity at each electrode. At electrode Fz, a negativity of -44.7mV (SD=38.72) occurring at 225.4ms (SD=168.7) after movement onset was observed. At electrode FCz, the negativity had an average amplitude of -36.8mV (SD=20.3) with an average latency of 254.9ms (SD=139.6). At electrode Cz the negativity had an average amplitude of -41.4mV (SD=37.0) with an average latency of 182.1ms (SD=181.5). At electrode CPz, the negativity had an average amplitude of -43.4mV (SD=29.0) with an average latency of 253.9ms (SD=147.8). At electrode Pz, the negativity had an average amplitude of -44.4mV (SD=32.0) with an average latency of 256.5ms (SD=162.9).

A similar averaging was done for those subjects showing significant positivities. At electrode Fz, the positivity had an average amplitude of 29.7mV (SD=8.9) with an average latency of 319.5ms (SD=172.3). At electrode FCz, the positivity had an average
amplitude of 32.7mV (SD=15.5) with an average latency of 345.3ms (SD=150.4). At electrode Cz, the positivity had an average amplitude of 35.1mV (SD=30.6) with an average latency of 292.3ms (SD=127.5). At electrode CPz, the positivity had an average amplitude of 27.8mV (SD=14.7) with an average latency of 288.3ms (SD=132.6). At electrode Pz, the positivity had an average amplitude of 32.1mV (SD=13.9) with an average latency of 238.3ms (SD=81.5).

Figure 3. Latency of ERP component minima and maxima by electrode. Latency indicates time after button release.
Examining how component latency and amplitude differs as a result of scalp position can reveal the timing and strength of processing as a function of electrode location. The latency of Ne increases moving from Fz to FCz, is at its shortest latency at Cz, and then increases again for posterior electrodes. The latency of Pe increases from Fz to FCz, then decreases at more posterior electrode positions. Amplitudes of both Ne and Pe remain relatively stable across electrodes, with a slight decrease in absolute amplitude from Fz to FCz for Ne and a decrease in amplitude from Cz to CPz for Pe. Thus while component amplitudes are relatively stable across electrodes, the latencies for Ne components are shortest at center scalp electrodes and the latencies for Pe components decrease posteriorly.
4 Discussion

Kinematic results show significant differences in error magnitude between aftereffect and baseline trials. This indicates that overall participants are adapting to the force in our paradigm and producing motor aftereffects when this force is removed. This can be taken as evidence that the paradigm reliably generated motor aftereffects. The majority of participants had significant error magnitudes that came mainly as a result of trajectory errors; however it is important to note that one participant had significant endpoint errors but not trajectory errors. It is likely that the paradigm described above is biased in inducing trajectory errors over endpoint errors. This may be due to a relatively long plane of reaching that allows subjects to correct their trajectory when it becomes perturbed or perhaps due to differing motor strategies used by participants. If the length of the plane of reaching impacts the type of error generated using the same motor strategy, a longer plane should reliably induce trajectory errors and a shorter plane should reliably induce endpoint errors. Another possibility is that those participants generating endpoint errors rather than trajectory errors may not be consciously aware that their reach has been perturbed. Trajectory errors result in a deviation that is corrected by the time the participant reaches the endpoint, so subjects who generate endpoint errors may not be correcting the deviation of their reach during the movement.
Further experiments are needed to test this hypothesis and to verify whether the differences in error type have differing neural signatures.

ERP data shows that there may be differences in the ERP components found across the scalp. Changing how the EEG signal is time-locked can significantly alter the conclusions drawn. Our analysis focuses on latency of components that result from time-locking to button release, which signals the start of the reaching movements. This was done because the research is exploratory in nature, and we prioritized capturing all components of the ERP over finding the exact latencies. This means that the latencies described above are larger and more variable than they would be to represent the same type of processing in other modalities. Latency of error-based ERP components occur when the error is detected (Coles 2001), for motor errors this happens when a movement crosses the threshold deviation to be noticed. In other modalities, the latency between stimulus presentation and error detection is much shorter and less variable than for the motor domain. For motor error generated using velocity-dependent perturbations, subjects must be moving with sufficient velocity to generate significant force to perturb reaches. If peak velocity occurs at 281.0ms on average, this means that subjects are detecting the perturbation of their reaches before 281ms but not before they reach some percentage of their peak velocity. Thus latencies observed that occur after 281ms in our experiment would still be in the range of Ne found in other modalities if properly time-locked to a percentage of maximum velocity.
Current component analysis reveals evidence that an error-driven negativity is present and occurs before a subsequent positivity in all but the most posterior electrode. In most cases, this negativity occurs at or just before participants reach peak velocity, which suggests that this negativity may represent error detection. A subsequent positivity may reflect later stage processing of some sort. The fact that more participants showed significant negativities than corresponding positivities is interesting and suggests that the processing represented by the negativity is more stable and occurs in all participants while the processing represented by the positivity may be more variable and not occur in every participant. This may indicate that both the Ne and Pe are localized over anterior electrodes as they are not as frequently observed in posterior electrodes, especially Pz. This fits with a biological interpretation of both motor control and neural correlates of error-related negativities found in auditory and visual modalities. Both primary and supplementary motor areas are located in more frontal and central regions of the brain (Graziano 2009). Thus processing of motor errors would most likely be located in these frontocentral areas, corresponding to electrodes Fz, FCz, and Cz (Yeung 2004). Error-related negativities for visual and auditory modalities have also been described as possessing more frontal localization, suggesting that processing of error may occur frontally. This corroborates the finding of more stable Ne and Pe components on frontal electrodes, especially at Fz, FCz, and Cz. Further experiments investigating Pe are necessary to understand why more subjects showed Ne components, even at frontal electrodes.

While the error-related ERPs observed do seem to be generated as result of the violation of participant’s expectations, there still remains the possibility that the neural
activity recorded is the result of sensory, rather than cognitive processing. When the force is turned off unexpectedly, unexpected sensory information (lack of force) is delivered to the arm along with the evaluation of error thought to be represented by error-related negativities. Future experiments must include conditions where a force is turned on unexpectedly to generate a motor error in addition to conditions where a force is turned off. If the same neural signature is present in both conditions, it provides evidence that the cortical activity is a result of processing of error, not processing of sensory information.

Limitations
There are some aspects of our study that limit the interpretation of the data and the generalizability of this study. Participants in our study typically generated larger trajectory than endpoint errors during adaptation and aftereffect trials. This could be due to the strategy they used to adapt to the force or it could be due to task design being biased to induce one type of error over the other. For continuity and to ensure that motor errors as a whole could be assessed, trajectory and endpoint errors were combined into a single dimension but there still remains the possibility that the neural substrate of endpoint and trajectory errors are fundamentally different. If this is the case, the current paradigm is not sensitive enough to detect these differences. Future studies should check whether there are systematic differences in the ERP components of endpoint error and trajectory error.

Another limitation of our study is that it is hard to tell whether all participants are using the same motor adaptation strategy, i.e. that they are adapting in the same way. It is possible that in some cases participants learn to tense up their arm and shoulder
muscles which would prevent the perturbations from deflecting their arm rather than
learn to adapt by outputting an opposite vector to the force being applied. Both of these
types of ‘adaptation’ would look similar behaviorally but have different neural substrates.
Post-experiment interviews with participants checked that participants were responding
to the force and attempting to overcome it but this does not ensure that adaptation
strategy was constant across participants.

A third limitation of the current study is that of the study population. Participants
were undergraduate or graduate university students, and elderly and juvenile
populations are not well represented. Given that ERP data can vary widely across age
ranges, this means our results may not generalize to populations not represented in our
study. Further experiments should attempt to link brain and motor system development
to behavioral and ERP component analysis to get a true picture of how the neural
substrate identified in young adults changes throughout the lifespan.
References:


Motor adaptation to dynamic non-contacting force (Lackner and DiZio, 1994)

Motor Adaptation to dynamic contacting force (Shadmehr and Mussa-Ivaldi, 1994)


Context Specific motor adaptation to contacting and non-contacting forces (Panic, 2011, Frucht et al., in preparation)


