

Parametric modulation of error-related ERP components by the magnitude of visuo-motor mismatch

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ABSTRACT

Errors generate typical brain responses, characterized by two successive event-related potentials (ERP) following incorrect action: the error-related negativity (ERN) and the positivity error (Pe). However, it is unclear whether these error-related responses are sensitive to the magnitude of the error, or instead show all-or-none effects. We studied error-monitoring with ERPs while healthy adult participants performed ballistic pointing movements towards a visual target with or without optical prisms, in alternating runs. This allowed us to record variable pointing errors, ranging from slight to large deviations relative to the visual target. Behavioural results demonstrated a classic effect of prisms on pointing (i.e. initial shifts away from targets, with rapidly improving performance), as well as robust prismatic after-effects (i.e. deviations in the opposite direction when removing the prisms after successful adaptation). Critically, the amplitude of both ERN and Pe were strongly influenced by the magnitude of errors. Error-related ERPs were observed for large deviations, but their amplitudes decreased monotonically when pointing accuracy increased, revealing a parametric modulation of monitoring systems as a function of the severity of errors. These results indicate that early error detection mechanisms do not represent failures in an all-or-none manner, but rather encode the degree of mismatch between the actual and expected motor outcome, providing a flexible cognitive control process that can discriminate between different degrees of mismatch between intentions and outcomes.

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1. Introduction

The rapid detection of errors is crucial for adaptive and flexible behaviours. Accordingly, the recording of event-related brain potentials (ERPs) has revealed the existence of rapid error monitoring systems in the human brain, centered on the anterior cingulate cortex (Bush, Luu, & Posner, 2000; Dehaene, Posner, & Tucker, 1994). Following erroneous action, two ERP specific components have been consistently identified in many studies, across various tasks and stimuli (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). First, the error-related negativity (ERN) peaks 0–100 ms after the occurrence of an incorrect response (either a false alarm or a discrimination error), with a maximum amplitude over fronto-central leads (see Gehring, Coles, Meyer, & Donchin, 1990). Next, the positivity (Pe) peaks 150–200 ms after the incorrect response, with a maximum amplitude over central leads

(Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991). Whereas the ERN is thought to reflect an early automatic detection of errors (i.e. the rapid appraisal of a mismatch between the actual and intended motor response), the Pe is assumed to reflect higher-order behavioural or motivational processes associated with the subsequent adjustment of performance (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996).

Typically, the ERN and Pe components are recorded while participants perform interference tasks such as Stroop, Flanker, or go/nogo tasks, and occasionally make unwanted response errors. In the two former cases, discrimination errors may arise, whereas false alarms are frequently produced in the latter case; but in all these instances, comparing ERPs to incorrect vs. correct responses classically reveals prominent ERN and Pe components. However, because of this dichotomous distinction between correct and incorrect responses in most of the error monitoring studies, it is unclear if these ERP components may also code for the degree or “severity” of mismatch between the actual and intended motor response. Errors can be either large or small, and thus require different degrees of behavioural adjustment. Presumably, early error-detection systems, as reflected by the ERN and Pe ERP components, might be sensible to this factor, enabling flexible cognitive control and learn-

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ing mechanisms. However, a simple comparison between correct vs. incorrect responses does not allow to test this prediction.

According to the dominant error detection theory of Falkenstein et al. (2000), the greater the amount of mismatch between the executed and intended correct response, the larger the probability to detect this error, and by extension, the larger the ERN amplitude, since this component is directly related to the detectability of errors (Maier, Steinhauser, & Hubner, 2008). However, it has been shown that undetected (i.e. unconscious) errors can also elicit a sizeable ERN component in the absence of any Pe component (Endrass, Reuter, & Kathmann, 2007; Nieuwenhuis et al., 2001; Scheffers & Coles, 2000), suggesting that the early ERN may reflect an automatic (perhaps subcortical) “all or nothing” error signal associated with alerting and learning mechanisms. Alternatively, a residual ERN to unconscious errors might indicate that some detection of mismatch may still operate on sensorimotor representations unavailable to conscious awareness. By contrast, the Pe was ascribed to a conscious (cortical) remedial action process, pointing to a functional dissociation between the ERN and Pe components (Nieuwenhuis et al., 2001). Thus, until now, ERP results have remained inconclusive regarding the link between the ERN and the degree of error detectability (Maier et al., 2008).

It is noteworthy that recent models of error monitoring have postulated that the ERN might reflect reinforcement learning signals mediated by dopaminergic pathways (Holroyd & Coles, 2002; Nieuwenhuis, Holroyd, Mol, & Coles, 2004). In both humans and primates, there is evidence that the phasic firing rate of midbrain dopamine neurons encode the difference between the predicted and experienced reward of an event, consistent with a prediction error model (Schultz, Tremblay, & Hollerman, 2000). Moreover, this dopaminergic signal shows a linear response according to the extent to which expectations are violated (see Abler, Walter, Erk, Kammerer, & Spitzer, 2006 for human fMRI evidence; Fiorillo, Tobler, & Schultz, 2003), such that larger deviations from expectations lead to larger firing bursts of dopaminergic neurons. On the other hand, because errors correspond to events that turn out to be worse than anticipated, they are thought to induce a phasic suppression of dopamine, which can in turn produce a transient release of activity in the dorsal ACC activity, and thus generate the ERN/Ne (Holroyd & Coles, 2002). Based on these data (Abler et al., 2006; Fiorillo et al., 2003), one may therefore predict a linear relationship between the magnitude of errors and the amplitude of the ERN/Ne component (being under dopaminergic influences), although this hypothesis has received no empirical validation so far.

In the current study, we addressed this question using a novel method. We recorded response-related ERPs while healthy participants were asked to perform a simple visuo-motor pointing task in which variable errors could be systematically induced by prism goggles. Prism goggles create a compelling deviation of the visual field that does not correspond anymore to the motor space (Redding & Wallace, 1996; Rossetti, Koga, & Mano, 1993). As a result, pointing movements towards a seen target become inaccurate and deviate away from the actual target location. To counteract this optical displacement induced by prisms, adaptive visuo-motor processes take place so as to progressively correct the pointing movements (see Redding, Rossetti, & Wallace, 2005 for a review). This so-called prismatic adaptation effect can fully restore pointing accuracy after a few trials (~15 on average). But once adaptation has occurred, the active nature of this adjustment will lead to a deviation in the opposite direction when prisms are removed (the so-called aftereffect). These effects suggest rapid plasticity mechanisms and cerebral reorganisation in response to the visuo-motor mismatch. Interestingly, a recent fMRI study of prism adaptation in healthy participants (Luu et al., 2009) reported that the magnitude of error produced by prismatic shift was correlated with activation of the ACC, consistent with the

notion that this region plays crucial role in error detection (Kerns et al., 2004).

Here, to determine whether such recruitment of dorsal ACC during prism adaptation may correspond to typical error monitoring systems observed in ERPs, we recorded ERP in healthy participants who performed a similar pointing task with ballistic movements towards a visual target, with or without prisms in alternating runs. This enabled us to collect many pointing errors with variable magnitudes of deviation, and hence determine whether the amplitude of the ERN and Pe components may vary with the severity of deviations. We tested the prediction that the ERN (and Pe to a lesser extent) might reflect error detection mechanisms under the influence of reinforcement learning signals (perhaps related to dopaminergic midbrain structures; see Holroyd & Coles, 2002), whose degree of recruitment should be modulated by the amount of mismatch between the actual and intended motor response. In other words, the larger the deviation, the larger the amplitude of the error-related ERPs should be.

2. Method

2.1. Participants

Twenty-one healthy participants (10 women; 2 left-handed) with a mean age of 27 years ($SD = 2$) took part in the study. They reported no history of neurological or psychiatric disease and normal or corrected-to-normal vision. The study was approved by the local university ethical committee.

2.2. Stimuli

Visual stimulus consisted of a black dot (of 2 cm diameter; subtending 2.9° visual angle at a 40 cm viewing distance) that was presented against a uniform white homogenous background.

2.3. Procedure

Participants were seated in front of a 17-in. touch screen LCD monitor. The position of their head was restrained using a chin rest positioned at 40 cm in front of the screen, with the eye level aligned to the center of the screen. At the beginning of each trial, participants were asked to place their right hand (and right upper limb) in a fixed initial position on the table. They had to start with their arm and hand perpendicular to the virtual axis formed by their head and the touch screen. We used an opaque cover attached to the chin rest to prevent participants to see their right arm in this initial position, while ensuring good vision of the full screen and final pointing location. Participants could only see their hand when it approached the screen. They were instructed to make a rapid, ballistic movement towards the location of the target dot, and hit the position of the dot by touching it with their index finger. Immediately after having completed their pointing, participants were instructed to place their right arm back in the initial starting position. Participants were discouraged to perform slow or corrective pointing movements. The use of online corrective pointing movements was further prevented by refraining vision of their arm during movements.

Each trial started with the appearance of the dot on the screen. To avoid motor habituation and stereotypic pointing movements, the position of the dot varied across trials. The resolution of the touch screen was 0–640 pixels on the horizontal axis (0 corresponding to the leftmost position) and 0–480 pixels on the vertical axis (0 corresponding to the highest position). For each trial, the dot appeared randomly in one location whose coordinates (as measured for the center of the dot) could range from 160 to 480 pixels on the horizontal axis and from 120 to 360 pixels on the vertical axis. The dot appeared equally often in the four visual quadrants. This ensured that visuo-motor adaptation was not limited to one specific target location. Touching the screen caused disappearance of the dot. We used a variable inter-trial interval (from 1500 to 2500 ms), consisting of a uniform blank screen.

Participants started with an initial block composed of 12 trials. During the second block, they performed again 12 successive pointing movements, but now wearing prism goggles that shifted binocular vision along the horizontal axis (Rossetti et al., 1998). We selected prisms producing a shift of 10 degrees of the visual field towards the right (a more pronounced deviation often caused pointing to fall outside the touch screen). As a result, pointing was systematically deviated away from the target, towards the right side. During the course of this block (12 trials), a significant visuo-motor adjustment took place in such a way that participants performed almost without error at the end of the block (see Fig. 2). This reflected an early phase of prismatic adaptation, although it must be noted that true prismatic adaptation is typically found in studies using many more pointing movements (Redding et al., 2005), when there is a stable recalibration of visuo-motor processes. This procedure (i.e. an initial block without prisms, immediately followed



Fig. 1. Graphical representation of the four possible magnitudes of pointing errors, relative to the target position. Each level of deviation was defined in terms of the radial distance (1 cm) from the central target location. Note that the actual target position on the screen was randomized from trial to trial and unpredictable. Final motor responses made between 1 and 2 cm from the center of the target were arbitrarily classified as edge responses (in white), between 2 and 3 cm as slight deviations (in yellow), between 3 and 4 cm as mild deviations (in orange), and above 4 cm as large deviation (in red). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

by a block under prisms) was repeated 10 times to eventually obtain 240 different pointing movements (i.e. 10 blocks with prisms and 10 blocks without). Because of the prismatic effect, the blocks without prisms following those performed with the prisms also contained substantial deviations, corresponding to the prismatic aftereffect (see Rossetti et al., 1993 and Fig. 2). The latter errors were in the opposite direction (i.e. leftward of the target location) as compared with errors performed during blocks under prisms. Thus, this on-off prism manipulation yielded many pointing errors in all participants, with variable magnitudes and variable directions. The recording session lasted approximately 13 min.

2.4. Analyses of pointing movements

For each pointing trial, we recorded the actual coordinates of the final touch location on the screen and calculated, using standard trigonometric/Euclidean geometry rules, the deviation (in cm) from the target dot location. Stimulus presentation and response recording were controlled using E-Prime software (V1.1, <http://www.pstnet.com/products/e-prime/>).

For both pointing conditions (with prisms vs. without), we computed the mean deviation for each of the 12 trials in the successive blocks (i.e. mean deviation of 10 pointing movements). We then submitted these data to a 12 (trial order) \times 2 (condition) repeated-measure ANOVA.

Finally, pointing errors were binned according to five different categories, depending on the degree of objective deviation between the actual response on the touch screen and the position of the dot (target). We took the radius of the target (= 1 cm) as an initial reference point. The five categories of errors corresponded to the following final touch locations: (1) target (from 0 to 1 cm), (2) edge (from 1 to 2 cm), (3) slight deviation (from 2 to 3 cm), (4) mild deviation (from 3 to 4 cm), and (5) large deviation (more than 4 cm). To obtain a sufficient number of ERP trials in each category, we collapsed trials made with and without prisms according to the same binning procedure (see Fig. 1).

2.5. EEG acquisition

Continuous EEG was acquired at 2048 Hz (0–417 Hz band-pass) using a 64-channel (pin-type) Biosemi ActiveTwo system (<http://www.biosemi.com>) referenced to the CMS–DRL ground (driving the average potential across the montage as close as possible to the amplifier zero). Details of this circuitry can be found on the Biosemi website (<http://www.biosemi.com/faq/cms> and <http://drl.htm>). Electrodes were evenly distributed over the scalp according to the extended 10–20 EEG system. ERPs of interest were computed offline following a standard sequence of data transformation (Picton et al., 2000): (1) common average reference, (2) ocular correction for blinks (Gratton, Coles, & Donchin, 1983) using the electrode FP1, (3) –100/+400 ms epoching around the motor response onset time, (4) pre-response interval baseline correction (from –100 ms to motor response), (5) artifact rejection (mean of $-72.5/+72.5$ μ V amplitude scale across participants), (6) averaging for each of the five levels of pointing deviation (center, edge, slight deviation, mild deviation and large deviation), and (7) 1–30 Hz digital filtering of the individual average data. In a control analysis, we also assessed whether blinks (and other lateral eye movements) occurred more frequently or were more ample during the critical ERP event (i.e. –100 ms/+400 ms epochs around the touch on the screen), but results showed that almost no blinks were recorded during this short-time interval. Moreover, we

did not find any evidence for any differential pattern of eye movements (or ocular corrections in EEG) between the conditions with prisms vs. without prisms when focusing on data time-locked to the critical event of interest (i.e. touch on the screen).

2.6. ERP analyses

To identify differences in the ERP waveforms between correct responses (pointing on target) and each of the four deviation levels (edge, slight, mild, and large), we computed subtraction waveforms, in line with previous studies on the ERN and Pe components (Dehaene et al., 1994; Falkenstein et al., 1991). By computing the difference waves, we could remove any (unwanted) residual motor and/or proprioceptive activities related to the execution of the pointing movement itself (and/or actual touch on the screen), which otherwise may have contaminated error-related ERP activities. As a result, ERP activities specifically related to error monitoring were best revealed by difference waves. Moreover, the use of difference waves was globally compatible with the assumptions put forward by van Boxtel, van der Molen, Jennings, and Brunia (2001) for this approach. For the four levels of deviation (edge, slight, mild, large), in each participant, the ERPs for correct responses (target) were subtracted from the ERPs obtained in the specific deviation condition so as to isolate ERP components generated by the different error magnitudes. In keeping with the analyses of behavioural data described above, we first collapsed trials performed with and without prisms, before computing ERPs for the different deviation conditions, in order to ensure a sufficient number of trials and obtain reliable ERP waveforms in each deviation condition.

Next, we performed a conventional peak analysis (Picton et al., 2000). The ERN/Ne was defined as the negative peak amplitude during the 30–130 ms interval post-response onset at electrode FCz. The Pe component was defined as the positive peak amplitude during the 130–280 ms interval post-response onset at the same electrode FCz. The selection of this specific scalp location was based on the topographic properties of the present dataset (both components reached their maximum amplitude on this electrode, see Fig. 4). Note that for the Pe, we did not find any evidence for a later positivity subcomponent (e.g. during the 200–400 ms post-response interval) with a more posterior/parietal scalp distribution (e.g. maximum amplitude at Pz or POz; see Fig. 3C), unlike some ERP studies that have occasionally reported two different sub-components for the Pe (i.e. an early fronto-central positivity like here, followed by a later parietal activity; see Van Veen & Carter, 2002; O'Connell et al., 2007). Accordingly, the Pe component recorded in our study most likely corresponded to the former subcomponent (see Fig. 4), and no measure could be obtained for the later parietal component. Statistical analyses were performed on the amplitude of each component using a one-way ANOVA with the severity of deviation as factor (with four levels: edge, slight, mild, large) as well as posthoc pairwise Student *t*-tests between conditions, with a significance threshold set to $p < .05$. The latency of the two ERP components was also calculated and analyzed using the same statistical tests, but these analyses failed to reveal any significant effect and are not reported hereafter.

3. Results

3.1. Behavioural results

As expected, participants made a high number of pointing errors (accuracy: $27.62 \pm 9.56\%$), with a variable magnitude of mismatch between the target location and final motor response. Mean RTs were 520.66 ms (± 40.5 ms) for pointings on the target, 513.35 ms (± 31.11 ms) for edge responses, 523.04 ms (± 62.31 ms) for slight deviations, 514.06 ms (± 42.93 ms) for mild deviations, and 525.56 ms (± 34.8 ms) for large deviations. A repeated-measure ANOVA indicated that the mean RTs of pointing movements were similar for all four levels of deviation [$F(3,57) = 1.01$, $p = 0.39$]. No reliable speed/accuracy tradeoff effect was found ($r = .123$, $p = .596$).

Wearing the prism goggles produced systematic deviations towards the right (direct effect), whereas smaller deviations occurred in the opposite (left) direction during blocks without prisms (aftereffect). A smaller size of the aftereffect relative to the direct effect is the hallmark of prismatic adaptation (see Cohen, 1966; Redding & Wallace, 1988 for examples). In both conditions, motor accuracy reliably improved throughout the block (see Fig. 2). An ANOVA on the mean deviation values across trials confirmed significant changes throughout the block (main effect of trial order; $F(11,9) = 26.641$, $p < .001$). Moreover, errors were larger under prisms than without prisms ($F(1,19) = 43.267$, $p < .001$). There was also a significant interaction between condition (prisms vs. no prisms) and trial order ($F(13,7) = 13.630$, $p < .001$), reflecting a

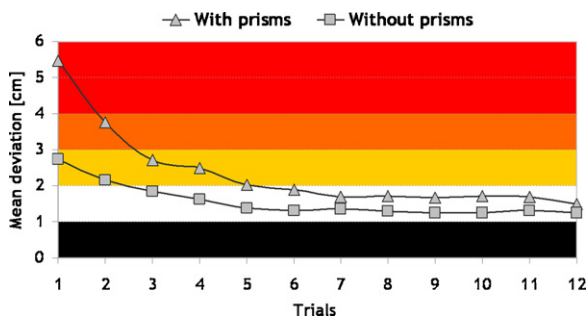


Fig. 2. Evolution of the mean pointing deviation from the first to the twelfth (and last) trial during blocks with or without prisms. For both conditions, larger errors were made at the beginning of the block. Under prisms, a progressive adaptation led to accurate pointing after 6–7 trials. Without prisms, the deviation (aftereffect) was less pronounced and disappeared after 5–6 trials. The different classes of deviation are color-coded as follows: responses on the target are represented in black; edge responses in white, slight deviations in yellow; mild deviations in orange; and large deviations in red. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

more rapid implementation of corrective movements during the after-effect, relative to the direct effect.

For the blocks performed without prisms, the mean deviation of the first trial was 2.74 cm (SD=0.74 cm). Posthoc (pairwise) comparisons confirmed that deviation was significantly larger for first than second trial [0.58 cm, $t(20)=4.497$, $p<.001$], larger for second than third trial [0.32 cm, $t(20)=3.873$, $p=.001$], larger for third than fourth [0.21 cm, $t(20)=2.661$, $p=.015$], and larger for fourth than fifth [0.25 cm, $t(20)=2.838$, $p=.010$]. However, following the fifth trial, deviations were not statistically different from each other (all $t<.800$, all $p>.418$). For blocks with prisms, the mean deviation of the first trial was 5.46 cm (SD=1.31 cm). Deviations were significantly larger for the first trial than second (1.70 cm, $t(20)=10.446$, $p<.001$), second than third (1.05 cm, $t(20)=10.934$, $p<.001$), third than fourth (0.23 cm, $t(20)=2.755$, $p=.012$), fourth than fifth (0.46 cm, $t(20)=5.255$, $p<.001$), sixth than seventh (0.19 cm, $t(20)=2.498$, $p=.021$), and eleventh than twelfth (0.19 cm, $t(20)=2.239$, $p=.037$). Other successive trials (7–11) did not significantly differ (all $t<.1470$, all $p>.157$).

We also tested whether a general learning effect occurred across successive blocks, despite the fact that target location was randomized on each trial within each block. However, the mean size of deviations were similar across the 10 blocks during the whole session [with prism, $F(9,11)=.884$, $p=.541$; without prism, $F(9,11)=1.425$, $p=.179$], suggesting no significant facilitation of adaptation (or deadaptation) over time.

Finally, comparing the different levels of deviation (pooling the two conditions with and without prisms) indicated that large (mean number: 18.9; percentage: $8\pm 6\%$) and mild deviations (20.57; $9\pm 4\%$) were less frequent ($6.03 < t(20) < 15.40$, $p<.001$) than slight deviations (47.9; $20\pm 5\%$), while slight deviations were also less frequent than the two smallest deviation levels (target: 64.5; $28\pm 9\%$; edge: 81.9; $35\pm 6\%$). Importantly, however, these differences in trial number could not account for the distinct ERP amplitudes found in each condition (as further described below).

3.2. ERP results

Analyses of ERP data showed that pointing errors were associated with two conspicuous components (Fig. 3A) following the touch on the screen. First, we observed an early negative deflection, with a maximum negative amplitude at electrode FCz (around 76 ms), immediately followed by a broad positive potential with a maximum amplitude at the same electrode position (around 185 ms). The electrophysiological properties of these

early ERP components (early latency following motor execution, marked polarity and fronto-central topography) are compatible with the two standard error-related ERP components, as typically described in the literature, namely the ERN/Ne and Pe components (Falkenstein et al., 2000). As can be seen from Fig. 3A, ERP results showed a clean pre-touch baseline (starting 100 ms before the touch on the screen), with no effect of the different error conditions. Given that participants had to perform a rapid/ballistic pointing movement and that the vision of their arms was blindfolded, they could essentially monitor their performance only by seeing their finger when it touched the screen. In these conditions, the actual pointing movement (and the degree of response conflict) was balanced between the different conditions (and deviations).

Remarkably, these two components were clearly visible for the slight, mild, and large deviations, but not for the edge condition (see subtraction waveforms in Fig. 3A; and topographic maps in Fig. 4). More importantly, there was a clear modulation of the amplitude of these two ERP components as a function of the magnitude of errors between the actual motor response and the target location: the larger the deviation, the larger the amplitude of both the ERN and the Pe components. This was confirmed by an ANOVA performed on the mean amplitude of the each component separately. This ANOVA revealed a significant effect of deviation magnitude both for the ERN amplitude [$F(3,17)=9.215$, $p<.001$] and for the Pe amplitude [$F(3,17)=18.387$, $p<.001$]. For each component, the linear trend (edge–slight–mild–large) was highly significant [ERN: $F(3,17)=27.562$, $p<.001$; Pe: $F(3,17)=53.522$, $p<.001$]. The mean amplitudes of the ERN and Pe activity, obtained from the subtraction waveforms (see Section 2), were, respectively, $-0.28\ \mu\text{V}$ and $0.34\ \mu\text{V}$ for edge responses; $-0.88\ \mu\text{V}$ and $0.75\ \mu\text{V}$ for slight deviations; $-1.15\ \mu\text{V}$ and $1.87\ \mu\text{V}$ for mild deviations; and $-1.59\ \mu\text{V}$ and $3.21\ \mu\text{V}$ for large deviations.

Direct pairwise comparisons confirmed that both the ERN and the Pe component monotonically increased as a function of the amount of mismatch between the actual motor response and target location. Their amplitude was larger for slight deviations compared to edge responses [ERN: $t(20)=5.440$, $p<.001$; Pe: $t(20)=-2.612$, $p=.017$], larger for mild compared to slight deviations [ERN: $t(20)=2.439$, $p=.024$; Pe: $t(20)=-4.584$, $p<.001$], and larger for large compared to mild deviations [ERN: $t(17)=2.252$, $p=.039$; Pe: $t(17)=-4.125$, $p=.001$]. Topographic analyses also corroborated these observations (Fig. 4): as can be seen in Fig. 4, scalp topographies for both the negative (ERN, top row) and the positive (Pe, bottom row) activity remained unchanged (i.e. the electric field configuration did not vary with the severity of errors), but only the amplitude of these ERP signals monotonically increased with larger deviations.

A potential concern might be that, due to the time course of prismatic effect, mild and large deviations were less frequent than edge responses and slight deviations. Likewise, the two former deviations occurred more often during the beginning of the blocks, whereas edge responses were more frequent at the end. These factors might potentially confound some of the amplitude ERP differences found for the ERN and Pe components across the four levels of deviation. However, note that any influence of the number of trials should lead to stronger amplitudes for the edge and slight deviation conditions, exactly opposite to our findings. Moreover, we performed several control analyses to directly rule out these alternative accounts. For each level of deviation, we selected a fixed number of 25 trials per participant, corresponding to the first of each condition occurring during each block, such that any possible effect of trial order and frequency was controlled for. We then re-computed new waveforms (Fig. 3B) and performed new peak analyses, each based on 25 trials per participant and per condition (now with a similar early position during a block). An ANOVA run on these new amplitude values confirmed that the effect of deviation

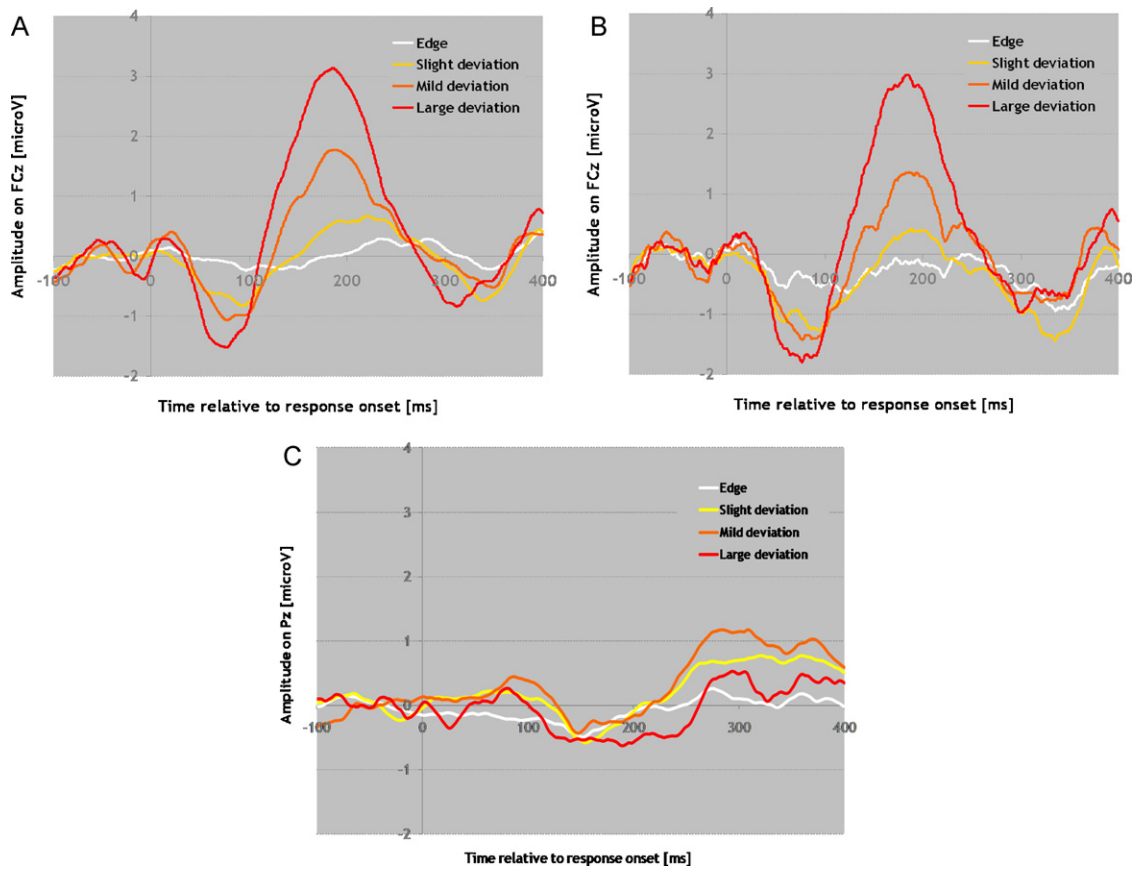


Fig. 3. (A) Response-locked ERPs (electrode FCz) associated with each level of deviation (edge, slight, mild, and large). ERPs from target responses were subtracted to cancel out the sensorimotor components and keep only the error-related activity. (B) ERPs obtained at the same electrode (FCz) after selecting the first 25 trials for each deviation condition (all blocks pooled together), showing that ERP differences could not be simply accounted by different trial number or different trial order. (C) Response-locked ERPs (electrode Pz) associated with each level of deviation (edge, slight, mild, and large), showing that no clear late Pe (normally associated with more posterior topography, see Van Veen & Carter, 2002) was observed following the ERN and early phase of the Pe (associated with a more anterior scalp distribution, see A and Fig. 4).

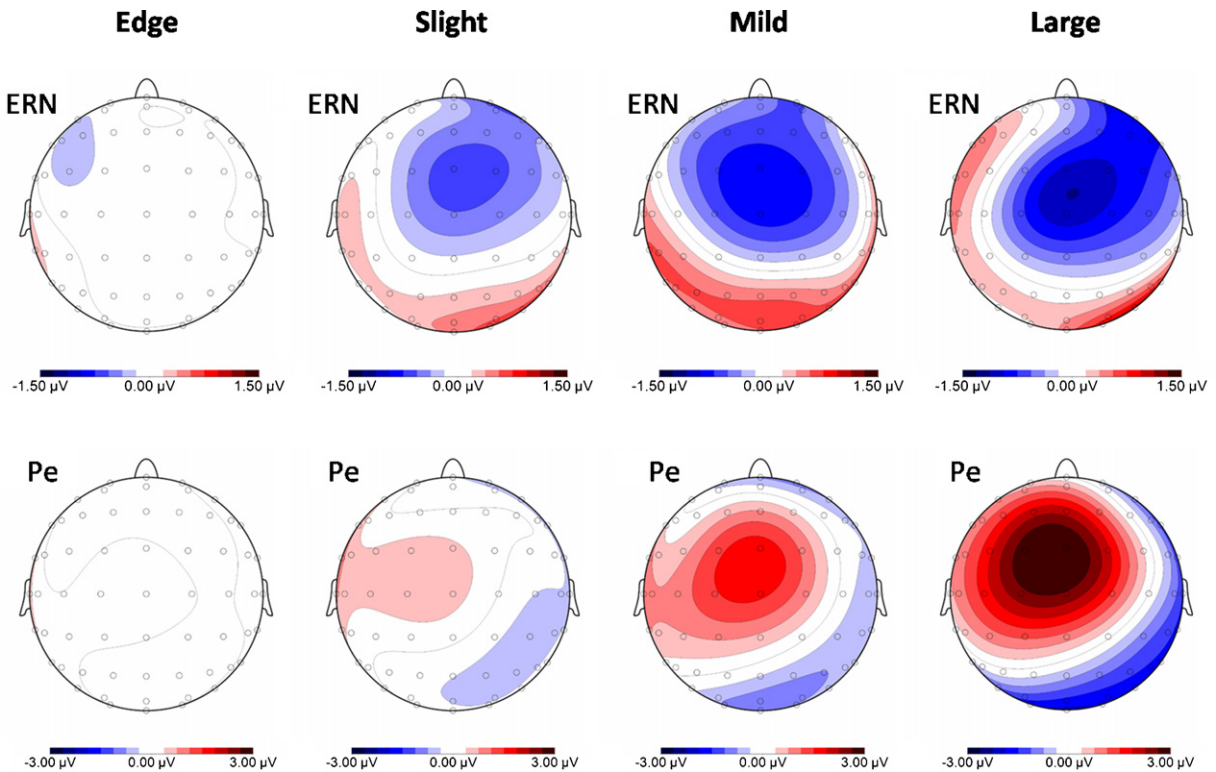


Fig. 4. Horizontal topographical maps of the electrical field generated on the scalp during the ERN and the Pe time-window, for each level of deviation (edge, slight, mild, and large). As for waveforms, topographical maps associated with errors were calculated by subtracting the activity recorded for correct target responses.

magnitude remained significant, both for the ERN [$F(3,17)=4.212$, $p=.024$] and for the Pe component [$F(3,17)=12.159$, $p<.001$]. Direct pairwise comparisons also confirmed larger amplitudes for slight deviations than edge responses [ERN: $t(20)=2.769$, $p=.012$; Pe: $t(20)=-3.222$, $p=.004$] and for large than mild deviations [ERN: $t(17)=2.197$, $p=.042$; Pe: $t(17)=-3.680$, $p=.002$]. The Pe amplitude was larger for mild than slight deviations [$t(20)=-2.877$, $p=.009$], though this difference did not reach significance for the ERN [$t(20)=1.623$, $p=.120$]. Thus, these control analyses confirmed that the magnitude of pointing errors, rather than their frequency or position during the block, was the critical factor that modulated the size of these two error-related ERP components.

Finally, for completeness, given the putative role of the ERN and Pe in learning and performance adjustment (Falkenstein et al., 2000; Vocat, Pourtois, & Vuilleumier, 2008), we tested for any relation between these components and behavioural measures. Across participants, we found that the mean amplitude of the Pe (light, mild and large deviations pooled all together) was significantly correlated ($r=.443$, $p=.044$) with the overall accuracy of pointing (total number of hits in the center position). On the contrary, the amplitude of the ERN was not related to accuracy ($r=-.102$, $p=.660$).

4. Discussion

The goal of this study was to test if early error-related brain responses may be sensible to the amount of discrepancy between the actual and intended motor action, consistent with the hypothesis that these early error-related ERPs may reflect reinforcement learning mechanisms that entertain a direct relationship with prediction error magnitude (Fiorillo et al., 2003). For this purpose, we used a standard pointing task performed either with or without prisms, in alternation, enabling us to record many visuo-motor errors that ranged from slight to large deviations relative to the target location. This task therefore allowed us to go beyond the traditional dichotomy between correct and incorrect responses, and to determine whether the error-detection mechanisms classically investigated by ERPs encode quantitative information about the severity of action failures, or simply constitute an all-or-none signal about such failures.

As expected, our task was successful in producing a large number of pointing errors, with a range of different magnitudes. Pointing performance was significantly disrupted by wearing prism goggles, but then progressively improved over successive trials, consistent with the rapid visuo-motor adaptation that typically takes place during such task (Cohen, 1966; Redding & Wallace, 1988 for example). Moreover, once the prisms were removed, participants showed a classical after-effect, reflected by new pointing errors in the opposite direction. Both the direct effects and the after-effects induced by our prism manipulation were short lived, as pointing performance was very accurate again after 6 or 7 trials. Previous studies have shown that, on average, 15 trials are needed for a reliable correction of the prism-induced shift (Redding & Wallace, 1993; Rossetti et al., 1993). Here, the shorter adaptation effect reported here might be explained by the fact that we used prisms with a relatively small optic shift of only 10 degrees, but the frequent alternation between short blocks of 12 trials with and without prisms ensured that no stable visuo-motor recalibration was achieved and that adaptation processes were similarly recruited during successive blocks during the whole experimental session (see Luaute et al., 2009). Accordingly, we found no significant learning effect across the successive blocks.

The critical new result of our study was that the two early error-related components in EEG (namely the ERN and Pe) were strongly influenced by the amount of mismatch between the motor response and actual target location: the larger the deviation, the

larger the amplitude of the ERN and Pe components. Remarkably, these results were obtained for ERPs time-locked to the physical touch on the screen (as opposed to an erroneous key press, as classically reported in the literature for errors during Stroop or Flanker tasks, see Falkenstein et al., 2000). Hence, these results suggest that internal error detection mechanisms were rapidly activated (mean peak latency = 76 ms and 185 ms, respectively), as soon as a spatial mismatch between the position of the finger on the screen and the location of the visual target was detected.

Importantly, these findings reveal for the first time to our knowledge that error-detection mechanisms associated with both ERN and Pe activity do not function using an “all or nothing” rule, but instead, precisely monitor the degree or severity of mismatch between motor intention and actual output, providing an efficient control process capable of gauging the importance of errors and thus appropriately modulating subsequent adjustment mechanisms. As such, these new results are compatible with the assumption that the error-related components (including Pe, whose amplitude was related to the overall pointing accuracy) may reflect a reinforcement learning process, associated with dopaminergic midbrain structures that project to the striatum and dorsal ACC (Holroyd & Coles, 2002; Nieuwenhuis et al., 2004), even though the exact relation of these ERP components to learning rate and accuracy changes remain to be fully clarified. Previous animal studies have disclosed a linear relationship between prediction error and phasic responses of dopamine neurons (Fiorillo et al., 2003), enabling a precise and flexible learning mechanism by which actions may acquire specific values that eventually guide future behaviours (Montague, Dayan, & Sejnowski, 1996). Our new results therefore suggest that the early comparison between the actual and intended motor response may not solely involve a strict categorical coding (i.e. the action is either correct or incorrect), or simple threshold process (if the action is incorrect, then trigger remedial action). Instead, a precise information about the magnitude of discrepancy between the goal and actual response is rapidly available to the action monitoring systems, within less than 100 ms post motor execution onset, probably prior to conscious visual feedback and possibly under the influence of dopaminergic drives. Such rapid and quantitative error monitoring mechanisms might be critical to swiftly adjust the accuracy of motor behaviour in parallel to action planning, and accords with behavioural evidence that reaching movement can be corrected “online” without conscious awareness (Pisella et al., 2000).

There are some restrictions to the present study. Because larger deviations were unavoidably obtained when participants wore the prismatic goggles, the severity of errors computed in our study (and the four corresponding deviation levels) is not fully orthogonal to the prism manipulation (i.e. task performed with vs. without prisms). However, the primary goal of our study was to study error monitoring functions and determine whether they were sensitive to the severity of errors; but we had to hypothesize concerning the effects of wearing prism goggles per se. In addition, because we had to interleave blocks with prisms and blocks without prisms to maximize the occurrence of variable errors throughout the experiment, and thus avoid a sustained adaptation/learning effect, we did not collect a sufficient number of trials per condition to compute ERP waveforms separately for the conditions with prisms vs. without prisms. However, given this alternation of blocks and the highly significant modulation by error magnitude irrespective of this factor, it seems very unlikely that our ERP results could simply be accounted by the fact of wearing goggles.

Although we performed a control analysis to establish that the number of trials did not account for the reported effects, another possible caveat might concern a more general effect of time. This effect could presumably manifest with larger deviations at the beginning of each block, and/or reflect the fact that larger devia-

tions probably occurred at the beginning, relative to the end of the experiment. However, given the progressive nature of learning and adaptation, there is no simple solution to fully control for this general effect of time. Nevertheless, we performed an auxiliary analysis to assess whether unspecific effects of time may have played any role (besides the effect of trial number). This analysis confirmed that the effects of prisms were uniform across the 10 blocks, ruling out the possibility that a general effect of time could account for the observed ERP differences.

Finally, another limitation is that with scalp ERPs, the exact cerebral localization of these error-related activities remains unclear. Many previous ERP studies have, however, pointed to the dorsal ACC as the main generator of the ERN (Dehaene et al., 1994; O'Connell et al., 2007; Van Veen & Carter, 2002; Vocat et al., 2008), and accordingly, there are good reasons to ascribe the intracerebral generators of the ERN recorded in this study to the dorsal ACC. In a previous source localization study using dipole modelling (Van Veen & Carter, 2002), the authors reported an interesting dissociation between the caudal and the rostral division of the ACC during early error monitoring: whereas the former was found to contribute both to the ERN and to the early phase of the Pe, the latter was involved in the late phase of Pe. Because the rostral ACC is typically associated with affective processing (Bush et al., 2000), these ERP results suggest that the late phase of the Pe might reflect a conscious/subjective emotional appraisal of errors (see also Falkenstein et al., 2000). Here we failed to find a late Pe component, suggesting that visuo-motor mismatches produced in this pointing task may be qualitatively different, relative to more conventional errors committed during go/nogo or interference tasks (see also Vocat et al., 2008), and hence that no comparable conscious emotional appraisal of "errors" was elicited in our visuo-motor task. However, the current findings indirectly support a dissociation between these ERP subcomponents. Moreover, recent fMRI results have also revealed an involvement of the anterior intraparietal sulcus (IPS), besides the dorsal ACC, in the online detection of a visuo-motor mismatch during pointing movements with prisms (see Luaute et al., 2009). Since this activation of anterior IPS also correlated with the magnitude of pointing error in this fMRI study (Luaute et al., 2009), this region may be responsible for providing the dorsal ACC with diagnostic information about the spatial discrepancy between the actual finger position and the real visual target. Further studies are needed to gain further insights into the possible interactions between dorsal ACC and anterior IPS during action monitoring in spatial and non-spatial tasks.

Our new results converge with previous ERP results (Carter et al., 1998; Gehring, Goss, Coles, Meyer, & Donchin, 1993) suggesting that the ERN/Ne is too rapid to be linked with sensory or proprioceptive feedback, and seem rather to result from some internal monitoring processes operating on a representation of the action command. In addition, however, we show that these monitoring processes receive rapid inputs about precise metric parameters necessary to evaluate ongoing actions. More generally, these findings are in line with the error detection theory of Falkenstein et al. (2000), suggesting that the ERN (and to some extent the Pe component) encodes the mismatch between an executed and intended response. Consistent with the main prediction of this theory, and the putative implication of dopaminergic systems, the larger this response mismatches, the larger the error signal, and thus the larger the amplitude of the ERN was. More generally, because similar effects were observed for the ERN and Pe amplitude in our study, it remains difficult to propose a clearly distinct role for each component during error monitoring based on the current data alone. However, the observation that the mean amplitude of the Pe (and not the ERN) was significantly correlated with the overall accuracy of pointing is consistent with the idea that this later ERP component may reflect a more elab-

orate (perhaps conscious) stage of error detection, as previously suggested (Nieuwenhuis et al., 2001; Ridderinkhof, Ramautar, & Wijnen, 2009). By contrast, the ERN might reflect the detection of a mismatch between sensori-motor representations, which remains unavailable to conscious awareness.

Importantly, we could rule out the possibility that this graded error response may have been confounded by differences in the frequency of errors between the four critical mispointing conditions (edge, slight, mild, or large deviations). Our ERP results were unchanged when we corrected the waveforms for the number of trials (as well as their position within the block). Moreover, in our experiment, mild and large deviations had roughly the same frequency but large deviations elicited reliably larger ERN and Pe components than mild deviations, corroborating the assumption that the magnitude of deviations (rather than their frequency) was the critical dimension accounting for the ERP amplitude differences. Furthermore, edge responses were the most common overall, but produced no reliable ERN or Pe.

These new results have important implications for neurocognitive models of error monitoring. By demonstrating that the ERN and Pe do not simply code for the occurrence of an error, but contain finer information about the amount of visuo-motor mismatch during pointing (at a very early latencies), we show that the corresponding neural processes do not represent non-specific alert signals – but rather seem to code the importance of the action failure, and thus also the importance of the necessary adjustment (Holroyd & Coles, 2002). In this view, errors provide the organism with important learning signals, which incorporate the amount of discrepancy between the actual and desired motor outcome. This fine-tune control mechanism may permit rapid corrections, possibly generated automatically in parallel with motor commands. More research is needed to determine whether this modulation of ERN and Pe is also related to trial-by-trial learning and improvement of performance.

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References

- Abler, B., Walter, H., Erk, S., Kammerer, H., & Spitzer, M. (2006). Prediction error as a linear function of reward probability is coded in human nucleus accumbens. *Neuroimage*, 31(2), 790–795.
- Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences*, 4(6), 215–222.
- Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, 280(5364), 747–749.
- Cohen, H. B. (1966). Some critical factors in prism-adaptation. *American Journal of Psychology*, 79(2), 285–290.
- Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error detection and compensation. *Psychological Science*, 5(5), 303–305.
- Endrass, T., Reuter, B., & Kathmann, N. (2007). ERP correlates of conscious error recognition: Aware and unaware errors in an antisaccade task. *European Journal of Neuroscience*, 26(6), 1714–1720.
- Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. *Electroencephalography and Clinical Neurophysiology*, 78(6), 447–455.
- Falkenstein, M., Hoormann, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: A tutorial. *Biological Psychology*, 51(2–3), 87–107.
- Fiorillo, C. D., Tobler, P. N., & Schultz, W. (2003). Discrete coding of reward probability and uncertainty by dopamine neurons. *Science*, 299(5614), 1898–1902.
- Gehring, W. J., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1990). The error-related negativity: An event-related brain potential accompanying errors. *Psychophysiology*, 27, S34.

- Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A neural system for error-detection and compensation. *Psychological Science*, 4(6), 385–390.
- Gratton, G., Coles, M. G., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology*, 55(4), 468–484.
- Holroyd, C. B., & Coles, M. G. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, 109(4), 679–709.
- Kerns, J. G., Cohen, J. D., MacDonald, A. W., 3rd., Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science*, 303(5660), 1023–1026.
- Luaute, J., Schwartz, S., Rossetti, Y., Spiridon, M., Rode, G., Boisson, D., et al. (2009). Dynamic changes in brain activity during prism adaptation. *Journal of Neuroscience*, 29(1), 169–178.
- Maier, M., Steinhauser, M., & Hubner, R. (2008). Is the error-related negativity amplitude related to error detectability? Evidence from effects of different error types. *Journal of Cognitive Neuroscience*, 20(12), 2263–2273.
- Montague, P. R., Dayan, P., & Sejnowski, T. J. (1996). A framework for mesencephalic dopamine systems based on predictive Hebbian learning. *Journal of Neuroscience*, 16(5), 1936–1947.
- Nieuwenhuis, S., Holroyd, C. B., Mol, N., & Coles, M. G. (2004). Reinforcement-related brain potentials from medial frontal cortex: Origins and functional significance. *Neuroscience and Biobehavioral Reviews*, 28(4), 441–448.
- Nieuwenhuis, S., Ridderinkhof, K. R., Blom, J., Band, G. P., & Kok, A. (2001). Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. *Psychophysiology*, 38(5), 752–760.
- O'Connell, R. G., Dockree, P. M., Bellgrove, M. A., Kelly, S. P., Hester, R., Garavan, H., et al. (2007). The role of cingulate cortex in the detection of errors with and without awareness: A high-density electrical mapping study. *European Journal of Neuroscience*, 25(8), 2571–2579.
- Picton, T. W., Bentin, S., Berg, P., Donchin, E., Hillyard, S. A., Johnson, R., Jr., et al. (2000). Guidelines for using human event-related potentials to study cognition: Recording standards and publication criteria. *Psychophysiology*, 37(2), 127–152.
- Pisella, L., Grea, H., Tilikete, C., Vighetto, A., Desmurget, M., Rode, G., et al. (2000). An 'automatic pilot' for the hand in human posterior parietal cortex: Toward reinterpreting optic ataxia. *Nature Neuroscience*, 3(7), 729–736.
- Redding, G. M., Rossetti, Y., & Wallace, B. (2005). Applications of prism adaptation: A tutorial in theory and method. *Neuroscience and Biobehavioral Reviews*, 29(3), 431–444.
- Redding, G. M., & Wallace, B. (1988). Components of prism adaptation in terminal and concurrent exposure: Organization of the eye–hand coordination loop. *Perception and Psychophysics*, 44(1), 59–68.
- Redding, G. M., & Wallace, B. (1993). Adaptive coordination and alignment of eye and hand. *Journal of Motor Behavior*, 25(2), 75–88.
- Redding, G. M., & Wallace, B. (1996). Adaptive spatial alignment and strategic perceptual-motor control. *Journal of Experimental Psychology: Human Perception and Performance*, 22(2), 379–394.
- Ridderinkhof, K. R., Ramautar, J. R., & Wijnen, J. G. (2009). To P(E) or not to P(E): A P3-like ERP component reflecting the processing of response errors. *Psychophysiology*, 46(3), 531–538.
- Rossetti, Y., Koga, K., & Mano, T. (1993). Prismatic displacement of vision induces transient changes in the timing of eye–hand coordination. *Perception and Psychophysics*, 54(3), 355–364.
- Rossetti, Y., Rode, G., Pisella, L., Farne, A., Li, L., Boisson, D., et al. (1998). Prism adaptation to a rightward optical deviation rehabilitates left hemispatial neglect. *Nature*, 395(6698), 166–169.
- Scheffers, M. K., & Coles, M. G. (2000). Performance monitoring in a confusing world: Error-related brain activity, judgments of response accuracy, and types of errors. *Journal of Experimental Psychology: Human Perception and Performance*, 26(1), 141–151.
- Scheffers, M. K., Coles, M. G., Bernstein, P., Gehring, W. J., & Donchin, E. (1996). Event-related brain potentials and error-related processing: An analysis of incorrect responses to go and no-go stimuli. *Psychophysiology*, 33(1), 42–53.
- Schultz, W., Tremblay, L., & Hollerman, J. R. (2000). Reward processing in primate orbitofrontal cortex and basal ganglia. *Cerebral Cortex*, 10(3), 272–284.
- van Boxtel, G. J., van der Molen, M. W., Jennings, J. R., & Brunia, C. H. (2001). A psychophysiological analysis of inhibitory motor control in the stop-signal paradigm. *Biological Psychology*, 58(3), 229–262.
- Van Veen, V., & Carter, C. S. (2002). The timing of action-monitoring processes in the anterior cingulate cortex. *Journal of Cognitive Neuroscience*, 14(4), 593–602.
- Vocat, R., Pourtois, G., & Vuilleumier, P. (2008). Unavoidable errors: A spatio-temporal analysis of time-course and neural sources of evoked potentials associated with error processing in a speeded task. *Neuropsychologia*, 46(10), 2545–2555.