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THE GRASPING SIDE OF POST-ERROR SLOWING

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LIST OF ABBREVIATIONS

AIC: Akaike’s Information Criterion

ACC: Anterior Cingulate Cortex

AIP: Anterior Intraparietal Sulcus

BF: Bayes Factor

BG: Basal Ganglia

CE: Cardiac Effort

CST: Corticospinal Tract

CMT: Cognitive Monitoring Theory

DLPFC: Dorsolateral Prefrontal Cortex

EMG: Electromyography

ERN: Error-Related Negativity

ERP: Event-Related Potential

FDI: First Dorsal Interosseous

FFA: Fusiform Face Area

fMRI: Functional Magnetic Stimulation

GLME: Generalized Mixed-Effect

IFC: Inferior Frontal Cortex

ITI: Inter Trial Interval

LME: Linear Mixed-Effect

M1: Primary Motor Cortex

MEP: Motor Evoked Potential

MFC: Medial Frontal Cortex

MGA: Maximum Grip Aperture

MIP: Medial Intraparietal Area

ML: Maximum Likelihood

Ms: Milliseconds

MT: Movement Time

oERN: Observational Error-Related Negativity

oPES: Observational Post-Error Slowing

OSL: Optimal Scalp Location

OR: Odd Ratio

PCA: Principal Component Analysis

PES: Post Error Slowing

PFC: Prefrontal Cortex

PG: Precision Grip

PIA: Post Error Improvement of Accuracy

PMC: Postero-Medial Cortex

PMd: Dorsal Premotor Cortex

PMv: Ventral Premotor Cortex

rMT: Resting Motor Threshold

RLA: Reinforcement Learning Account

RSI: Response Stimulus Interval

RSVP: Rapid Serial Visual Presentation

RT: Reaction Time

SD: Standard Deviation

SMA: Supplementary Motor Area

STN: Sub Thalamic Nucleus

SPOC: Superior Parietal Occipital Cortex

spTMS: Single Pulse TMS

tDCS: Transcranial Direct Current Stimulation

TMS: Transcranial Magnetic Stimulation

VCH: Visuomotor Channel Hypothesis

SYNOPSIS

"Errare humanum est, sed in errore perseverare diabolicum" is probably one of the most famous quote by Seneca. The central message of this quote is that humans are error-prone by nature, and that immediate steps need to be taken to ensure that the same errors are avoided in the future. Indeed, reacting efficiently to our errors is a fundamental ability to interact with the outside world. For this reason error reactivity is the focus of a growing body of research in cognitive neuroscience. In this respect, the study of the brain mechanisms underlying error processing has greatly advanced in the last few years. The discovery of specific event-related potentials, such as the error-related negativity (ERN), and of neural networks associated to error commission have contributed to our understanding of the processes taking place after error commission.

If, on the one hand, the findings obtained in neuroimaging studies on error processing seem to be robust and consistent, on the other hand how such neural activity translates into a differential movement pattern has yet to be fully understood. So far what is known is that people tend to respond more slowly after making an error, an effect termed *post-error slowing* (PES; Rabbitt, 1966). PES has been traditionally hypothesized to reflect a strategic increase in response caution, aimed at preventing the occurrence of new errors. This interpretation of PES, however, has been challenged on multiple fronts. Firstly, recent investigations have suggested that errors may produce a decrement in performance accuracy and that PES might occur because error processing has a detrimental effect on subsequent information processing. Secondly, this research has been criticized because of the limited ecological validity of speeded RT tasks.

The present work aims to extend previous literature by investigating for the first time the post-error effect in the context of realistic goal-directed actions. For this purpose I examined the effect of errors on both the preparation and the execution of reach-to-grasp movements. The introductory section of this thesis will focus on some of the most important empirical data acquired during the last years of research in experimental psychology and cognitive neuroscience regarding error-reactivity. In Chapter 1, after a detailed introduction to behavioral adjustments following errors (sections 1.1 and 1.2), a set of neuroimaging studies investigating the neural mechanisms underlying error-reactivity will be exposed (section 1.3). Then, I shall review the most important theoretical accounts of error-reactivity (section 1.5). In Chapter 2, I shall introduce the experimental window used to explore PES in the context of goal directed actions, namely the reach to grasp movement. The second part of the present thesis concerns the experimental work I undertook. Chapter 3 provides a description of the general methodology common to the entire experimental work. In the first experiment (Chapter 4) a novel task to study error-reactivity will be described. Participants were asked to reach out and grasp a steel ball positioned upon a wooden support, without knock it over. In addition to RTs, a kinematical analysis was performed in order to asses if error-reactivity extends at the level of movement execution. In a subsequent experiment (Chapter 5), a similar paradigm was adopted, but the task was chiefly concerned with a reaching movement. This experiment was ran with the specific aim to investigate whether error-reactivity has a different impact on the grasping and the reaching components or whether it produces an unspecific slowdown of the whole movement. In Chapter 6, I shall describe an experiment in which kinematical analysis was coupled with transcranial magnetic stimulation (TMS) in order to measure corticospinal excitability after an error and whether behavioral and neural measures do correlate in such circumstances. In a further experiment (Chapter 7) the effect of error observation on the reach-to-grasp movement was examined via kinematical analysis. A general discussion (Chapter 8), contextualizing the results obtained by the studies presented in the present thesis will follow. Overall, these studies will help to understand how error-reactivity influence our behavior and the way errors are interpreted and evaluated by the human mind.

1. POST-ERROR ADJUSTMENTS

Error commission is usually followed by several physiological and behavioral changes. For instance, heart rate deceleration (Danev & de Winter, 1971), pupil dilation (Critchley at al., 2005) and an increase of skin conductance response (O'Connell et al., 2007) have been observed after an error. From a behavioral standpoint, the most reported adjustments following an error are the *post-error slowing* and the *post-error improvement of accuracy*. In the following sections these post-error adjustments will be exposed in detail.

1.1. Post-error slowing

In the mid-1960s Rabbitt observed for the first time the so called *post-error slowing effect* (PES) (Figure 1), a slowing down of the onset of actions subsequent to erroneous actions (Rabbitt, 1966). Rabbitt (1966) investigated error-reactivity using a 4-choice and a 10-choice task. The task required that participants responded to a light signal by touching a corresponding position within a grid with the index finger. This early experiment demonstrated that responses following an error were slower than average correct RTs. In a subsequent study, Laming (1968) further examined error related RT changes, by means of a line judgement task. At the beginning of each block participants were shown two initial lines. During the experimental session, a single line was presented and participants had to decide at which of the two initial lines it corresponded. Laming (1968) observed that post-error trials were slower than post-correct trials and that responses were more accurate after an error. In a similar study, Desmet and colleagues (2012) asked participants to judge the accuracy of simple multiplication equations. Reaction times were slower after making a wrong judgement than after making a correct judgement.

Since the pioneering work of Rabbit (1966) and Laming (1968) the study of error-reactivity advanced enormously, and many interesting features of the PES effect have been unveiled.

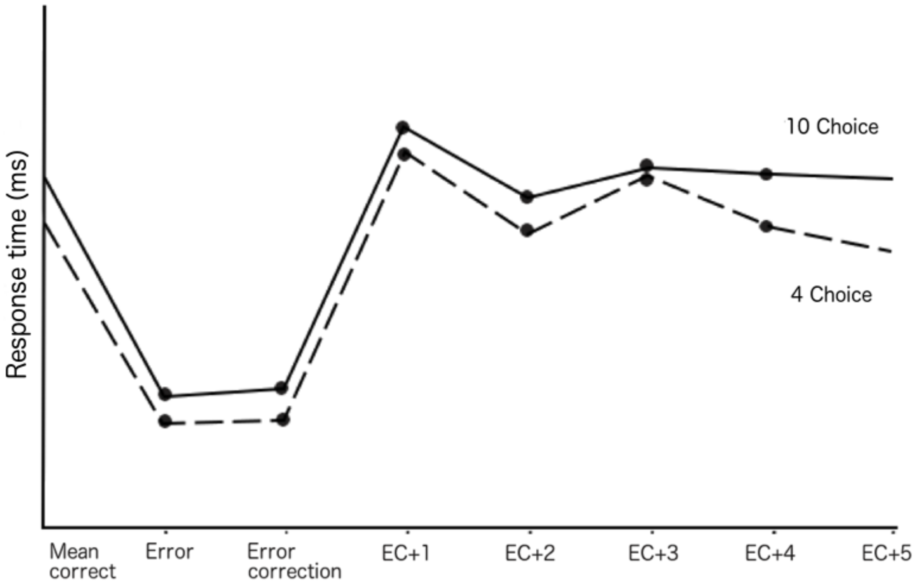

Figure 1. Response times as a function of errors, error corrections and correct responses for both a 10 choice and a 4 choice task. Post-error slowing consists in slower reaction times in trials following the error (EC + N) than the mean correct responses. From Rabbitt (1966).

An important distinctive characteristic of the PES is that decays over time. Jentzsch and Dudschig (2009) investigated the time course of PES by manipulating the inter-trial interval (ITI) in a categorization task. The ITI was either short (500 ms) or long (1000 ms). The PES effect was present in both conditions, but it was much larger under short ITI condition. Danielmeier and Ullsperger (2011) obtained similar results using a flanker task, showing that long ITIs (> 1000 ms) tend to decrease the PES amplitude. Overall, these studies indicate that the PES effect is a *micro-adjustment* consisting in trial-by-trial modifications determined by the commission of errors, rather than a *macro-adjustments* producing long-term strategic modifications (Botvinick et al., 2001).

Another common finding is that the PES effect can be observed after unconscious errors. Cohen, Van Gaal, Ridderinkhof and Lamme (2009) using a Go-Nogo task, compared two conditions: the first condition included visible No-Go cues, and participants were driven to commit “conscious errors”, whereas in the second condition No-Go cues were masked and subjects committed “unconscious errors”. Although the effect size was very small (3.1 ms), results showed a PES effect after unnoticed No-go errors, which suggests that error-reactivity effects are probably dissociated from the conscious experience of error detection.

The PES effect is also present after the so called *partial errors* (Figure 2). Some EMG studies have observed muscle twitches from the hand related to the erroneous response in about 15-20% of overt correct responses (Burle at al., 2002; Allain, Burle, Hasbroucq, & Vidal, 2009). These covert activations are supposed to be partial errors that once detected are aborted and corrected before reaching the overt response threshold. The fact that correct responses preceded by partial errors tend to be slower than those which are not, provides supporting evidence for the existence of an on-line control process that corrects error response tendencies and implements strategic adjustment.

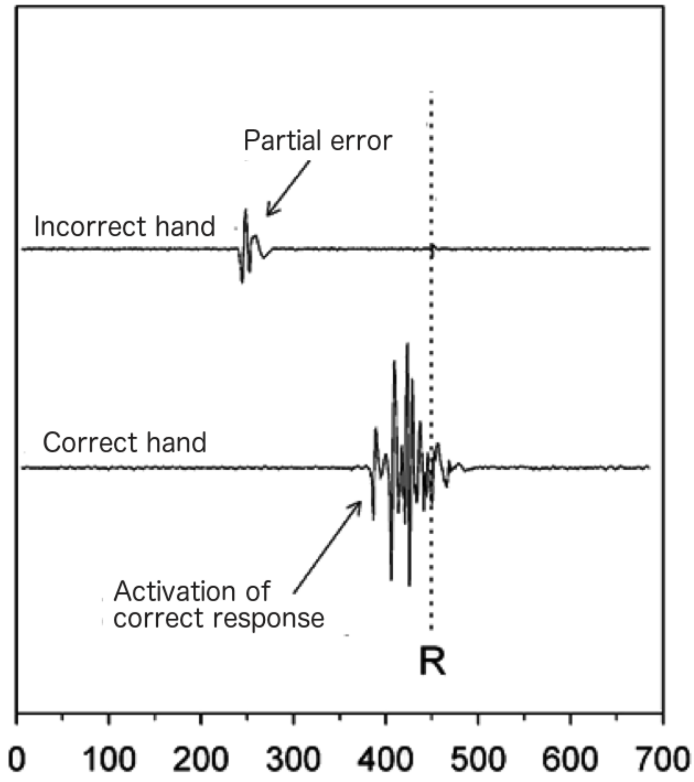

Figure 2. Vertical dashed line indicates the mechanical response. The bottom trace presents the EMG activity of the muscle involved in the correct response. The top trace shows a partial error, that is a small burst on the incorrect muscle, preceding the mechanical response. From Burle et al. (2002).

1.2. Post-error improvement of accuracy

In addition to RTs, post-error accuracy has been used to investigate behavioral adjustments following an error. Several studies have reported that fewer errors are made after an error compared to after a correct response (e.g., Laming, 1968; Maier, Yeung, & Steinhauser, 2011; Danielmeier et al., 2011). However, behavioral findings are not clear with respect to *Post-Error Improvement of Accuracy* (PIA). Indeed, some studies did not found any difference between post-error and post-correct trials in terms of accuracy (e.g., Hajcak, McDonald, & Simons, 2003; King et al., 2010). For instance, Hajcak and colleagues (2003), used a modified version of the Stroop task and asked participants to respond to whether or not the color and name of the stimulus matched. Results did not show any difference between post-correct and post-error trials in terms of accuracy. Other research reported a decrease in accuracy after an error (e.g., Rabbitt & Rodgers, 1977; Fiehler, Ullsperger, &Von Cramon, 2005). Rabbitt and Rodgers (1977) investigated the post-error accuracy in an a choice task. Participants were requested to identify some numbers presented on the computer screen by a bottom-press. Results showed that post-error responses were not only slow, but also inaccurate. Other investigations reported that the PIA effect is present only after aware error, but not after unnoticed errors. This evidence was observed for the first time by Klein and colleagues (2007) using an antisaccade task. Subjects were asked to fixate a central point on the screen and to look away from an abruptly appearing peripheral stimulus. When participants did not succeed in disengaging the gaze from the peripheral stimulus the trial was classified as an error. To distinguish consciously perceived errors from unperceived errors, after each trial, subjects had to indicate whether or not they had noticed an error. Results indicated that performance accuracy tended to improve after an aware error, whereas no evidence of PIA was observed after unperceived errors.

A reason of why PIA results are not unequivocal, is that post-error accuracy can be biased by both inter-individual differences and task difficulty. In particular, when participants commit too many errors, or when the error rate is too low, there is a reduced chance to improve the performance after an error, regardless the adoption of a more cautious response criterion (Danielmeier & Ullsperger, 2011). Likewise, if the task is too difficult participants do not have any chance to improve their accuracy. In these situations, post-accuracy is a misleading measure of post-error performance.

A core topic in error-reactivity literature concerns the relation between PES and PIA. Some investigators contend that the PES and PIA might represent different processes, as they do not necessarily occur together (Danielmeier & Ullsperger, 2011). An evidence in favor of this hypothesis is that, contrary to the PES effect, short inter-trial intervals (ITIs) (< 500 ms) are usually associated with a post-error decrease in accuracy (Jentzsch & Dudschig, 2009; Danielmeier & Ullsperger, 2011). Danielmeier and Ullsperger (2011), using an arrow version of the flanker task, investigated post-error accuracy in four conditions, in which the RSI was 200, 750, 1500, or 3000 ms, respectively. Results showed that post-error accuracy tended to decrease at short RSIs. Instead, long ITIs (> 750) tend to elicit a post error increase in accuracy (e.g., Marco-Pallares, Camara, Munte, & Rodriguez-Fornells, 2008; Maier, Yeung, & Steinhauser, 2011). Marco-Pallares and colleagues (2008) investigated post-error accuracy by means of a modified version of the Eriksen flanker task. Participants were instructed to identify a central letter on a screen, flanked by four distractors, with either their right (letter H) or left (letter S) hand. Distractors could be either congruent, that is requiring the same response as the target (SSSSS, HHHHH), or incongruent (HHSHH, SSHSS), that is priming the erroneous response. The ITI was set to 1000 ms. Results showed a large PIA effect, whereas the effect size of PES was reduced.

In addition to behavioral methods, several studies have investigated error reactivity by means of neurophysiological technics. These studies are important because some of the main theories put forward to explain post error behavior heavily rely on the neurophysiological changes following an error. In the ensuing section, I will outline the neural mechanisms underlying error reactivity.

1.3. Neuroanatomical basis of error-reactivity

A first description of the neural mechanism involved in error processing stems from experiments using *event related potentials* (ERPs; Danielmeier & Ullsperger, 2011). ERPs are small transient electrical voltages originating from the brain in response to specific stimuli or events. They are measured recording the electrical activity by means of a number of electrodes, placed on the scalp according to international standards for electrode placement (Luck, 2014), such as the *International 10/20 System* (Figure 3A). ERPs reflect the postsynaptic activity of pyramidal neurons during information processing (Peterson et al*.,* 1995), and therefore each ERP is associated with the brain activity elicited by specific mental operation (Luck, 2014). ERPs studies have revealed two main components which consistently emerge after an error: the error related negativity (ERN) and the error positivity (P_E) (Figure 3B).

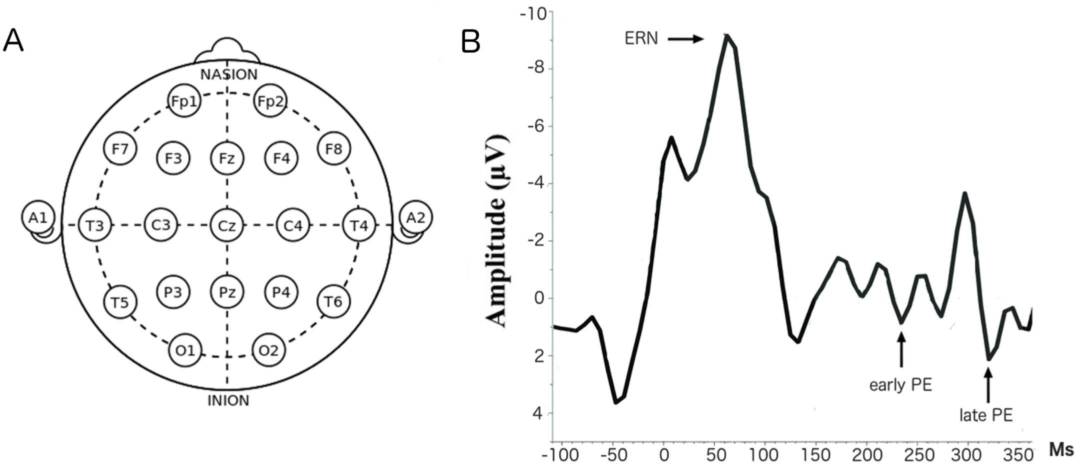

Figure 3. A) International 10/20 System. B) Graphical representation of the error-related negativity (ERN) and positive error (Pe).

The ERN (also termed as N_e) and the P_E were observed for the first time by Falkenstein and colleagues (1991) in a speeded choice RTs task. Participants were presented on a screen the letters “J” or “F”, in random order. They were asked to respond to each letter by pressing the corresponding key on a keyboard, as fast and accurately as possible. Falkenstein and colleagues (1991) observed that errors gave rise to a ERN, that is a response-locked negative deflection, peaking between 50 and 100 ms after the execution of errors, maximal over fronto-central scalp locations (typically the 10-20 location Fz). Falkenstein and colleagues (1991) observed that the ERN was followed by the P_E , a slow positive wave with a diffuse parietal scalp distribution and a maximum amplitude between 200 and 500 ms from the erroneous response. Two temporally independent peaks are involved in the P_E (Figure 3B): an early P_E occurring between 200-400 ms after error commission, and a late P_E occurring after 300-600 ms (Falkenstein et al., 1991).

Several studies have tried to understand the functional meaning and the peculiarities of these two ERPs. The ERN is considered to reflect error-reactivity processes. Indeed, the ERN amplitude is correlated to compensatory processes, such as the PES (West & Travers, 2008). West and Travers (2008) investigated the relation between the ERN amplitude and PES using a *counting Stroop task*. The task required to indicate the number of digits appearing on a screen. In the congruent condition the number of digits was equal to the identity of digits (e.g., 4444), whereas in the incongruent condition the identity and the number of digits were different (e.g., 444). West and Travers (2008) found a strong correlation between the ERN and PES and suggested that processes reflecting the ERN modulate the behavioral effects following errors. Another important characteristic of the ERN is that is has been observed irrespective of the response modality (Holroyd, Dien, & Coles, 1998). Holroyd and colleagues (1998) using an Eriksen Flankers task, in which subjects were asked to respond with either their hands or their feet, demonstrated that the ERN could be observed in both response conditions. These findings suggest that the ERN is effector-independent and can be generated by the activity of a generic error-processing mechanism (Holroyd et al., 1998). Moreover, the ERN amplitude is modulated by the degree of mismatch between the error and the correct response (Vocat, Pourtois, & Vuilleumier, 2011). Indeed, Vocat and colleagues (2011) asked participants to reach and touch with their index finger a target dot on a touchscreen. Interestingly, the amplitude of ERN was modulated by the severity of errors. In particular, the ERN amplitude increased when pointing accuracy decreased, indicating that the error-detection mechanisms, reflected by ERN, encode the degree of mismatch between the correct response and the response given by participants.

With regard to the P_E, Falkenstein and colleagues (1991) proposed that it reflects conscious error recognition. This hypothesis was partially confirmed by Endrass and colleagues (2007). They asked participants to indicate the position of a peripheral circle appearing on a screen, and then to rate with a button press if their response had been correct or incorrect. Results showed that whereas the late P_E (400-600 ms) shows an increased positivity for aware compared with unaware errors, the early P_E (200-300 ms) shows no dissociation between aware and unaware errors (Endrass et al., 2007).

Dipole modelling have located the source of the ERN and P_E in the medial frontal cortex (MFC) (Botvinick et al., 2001). In particular, the neural generator of these components is supposed to be the anterior cingulate cortex (ACC), an important hub located in the medial wall of the frontal lobes, that regulates thought and action at the service of achieving goals and context demands (Botvinick et al., 2001). The ACC can be divided into three subregions: the dorsal ACC (dACC), the perigenual ACC (pgACC), and the subgenual ACC (sgACC). While the pgACC, and the sgACC are implicated in emotional functions (Etkin, Egner, & Kalisch, 2011; Bush, Luu, & Posner, 2000), the dACC is considered a crucial area for cognitive control functions (Botvinick et al., 2001). Indeed, the dACC is engaged by tasks in which competing responses must be overcome (e.g., the Stroop Task) and an increase of cognitive control is necessary to minimize the processing of distractor information (Botvinick et al., 2001). Several neuroimaging studies have confirmed that the dACC is involved in cognitive control. For instance, Metzler-Baddeley and colleagues (2012) showed that subjects with amnestic mild cognitive impairment presented a reduction of the white matter in the anterior cingulum bundle, and performed worse on control-demanding tasks. The involvement of the ACC in error monitoring was observed for the first time by Carter and colleagues (1998), that used fMRI to determine the neural areas associated with incorrect responses in an *AX continuous performance task.* Participants were shown a stream of letters, with the instruction to identify the letter "X", preceded by the letter "A". Results showed that erroneous responses were accompanied by a specific activation of ACC. Other studies observed that the activation of ACC is often inversely related to the actual number of errors committed (Holroyd & Coles, 2002; Yeung et al., 2004), such that a reduced ACC activity coincides with increasing errors. These findings gave rise to the idea that the ACC plays a key-role in error processing and in error detection (Kiehl, Liddle, & Hopfinger, 2000; Menon et al., 2001). This hypothesis is supported by a series of studies in patients with unilateral focal lesions of the ACC showing impairments in post-error adjustments and a reduced ERN amplitudes (e.g., Di

Pellegrino, Ciaramelli, & Ladavas, 2007) and by some single-unit studies in monkeys that observed a post-error increased activity in the anterior cingulate cortex (e.g., Ito, Stuphorn, Brown, & Schall, 2003).

Several fMRI studies pointed out that, in addition to the ACC, a post-error increased neural activity can be observed in the dorsolateral prefrontal cortex (dPFC). The dPFC is located in the lateral surface of the frontal lobes and it is involved in a variety of higher order executive functions essential for goal-directed behavior, such as action planning (Mushiake et al., 2006) and self-control (Hare et al., 2009). Interestingly, the dPFC activity seems to increase when more top-down control is needed to accomplish the task (MacDonald, Cohen, Stenger, & Carter, 2000). For instance, MacDonald and colleagues (2000) observed that the Stroop interference effect was smaller when the dPFC activity increased, which suggests that the dPFC activity leads to the implementation of more top-down control reducing interference effects in incongruent trials. The role of the dPFC in error-reactivity is far to be clear. It was proposed that while the ACC monitors the occurrence of errors, acting as an error-monitoring system, the dPFC elicits an increase of top-down support after an error and leads to the implementation of post-error adjustments (Botvinik et al., 1999). Other studies have associated dPFC activity with error awareness. Harty and collegues (2014) stimulating the dPFC by means of the anodal *transcranial direct current stimulation* (tDCS) found that participants significantly improved error awareness. This finding indicated the presence of a causual relation between the activity of the dPFC and self-awareness of making errors. It is noteworthy, however, that recent studies failed to replicate these results. For instance, Masina and colleagues (2018) using an online single-pulse TMS paradigm, did not confirm the involvement of the dPFC in error awareness.

Together these findings indicate that the MFC and dPFC are involved in error-reactivity. In particular, the role played by these areas in terms of error-reactivity becomes important for the development of theories to explain PES as outlined below.

1.4. Theories of error-reactivity

To explain PES two theoretical accounts have been put forward, namely *functional* and *non-functional* (Houtman & Notebaert, 2013). Functional accounts, such as the *conflict monitoring* (Botvinick et al., 2001), the *inhibition* (Ridderinkhof, 2002), and the *reinforcement learning* (Holroyd & Coles, 2002) theories, propose that PES is the byproduct of a compensatory control mechanism serving the purpose of improving subsequent performance. PES is thus interpreted as the result of a more cautious response strategy aimed at producing a PIA. However, PES might not necessarily be the expression of an adaptive mechanism. In this perspective, *Non-functional accounts,* such as *the Orienting account* (Notebaert et al., 2009), and the *Bottleneck theory* (Dudschig & Jentzsch, 2009) suggest that error-monitoring has a detrimental effect on the subsequent information processing and predict a post-error decrease of accuracy.

The following paragraphs summarize the principal theory explaining PES as a functional or a non-functional effect.

1.4.1. Functional accounts

One of the most prominent theory of error reactivity is the *conflict monitoring theory* (CMT; Botvinick et al., 2001), according to which the PES is an adaptive strategy aimed to avoid future errors. The CMT contends that cognitive control is recruited by a mechanism that detects incompatible action tendencies coexisting in our cognitive system. In this framework, two components are supposed to be involved: a conflict monitor process, associated with the activity of the dACC, and an implementation system, located in the dPFC. Once conflict is detected, the conflict monitoring process modulates the activity of the dPFC, calling for an increase in response threshold, which produces slower and more accurate performance. Consistent with the monitoring-control loop perspective, several behavioral research found that distractor effects are abolished in trials following a response conflict. For instance, Gratton and colleagues (1992) observed that in a flanker task congruency effects tend to decrease after incongruent trials compared to congruent trials. In line with the CMT, the *Gratton effect* is produced by the response conflict from the previous trial, which signals a *need for control* that modulates response times and error rates on the subsequent trial (Botvinick et al., 2001). Analogous effects have been observed in the Stroop task. Kerns and colleagues (2004) found that the Stroop effect was smaller after trials with incompatible word-color pairing than after trials with compatible pairings. Moreover, they observed a negative correlation between the ACC activity and the level of interference on the subsequent trial, which supports the idea that a strong ACC engagement leads to an increase of top-down control. Further support for the CMT comes from several fMRI studies showing that in high-conflict trials the amplitude of dPFC activity predicts the degree of behavioral adjustment on the subsequent trial (e.g., Egner & Hirch, 2005). As aforementioned in the previous section, when a response is executed, a simultaneous activation of correct and incorrect responses can be observed in EMG trace. In line with this evidence, error can be interpreted as a particular form of highly conflictual response occurring when incorrect response tendencies (i.e., partial errors) are not successfully overridden. Therefore, according to CMT, the PES effect occurs because of response conflict associated with errors. The idea that the PES is a post-conflict effect has been supported by some fMRI studies (e.g., Garavan, Ross, Murphy, Roche, & Stein, 2002; Kerns et al., 2004; Chevrier & Schachar, 2010). For instance, Kerns and colleagues (2004), using the Stroop color-naming task observed that the ACC activity predicts the dPFC activity, which in turn, showed a positive correlation with both the amplitude of adjustments following incongruent trials as well as the magnitude of PES. In accord with the CMT, this result supports the view that the ACC is involved in conflict monitoring, whereas the PFC implements a more conservative response strategy after highly conflict trials as well as after erroneous responses.

In contrast to the CMT, the *inhibition account* (Ridderinkhof, 2002; Marco-Pallares et al., 2008) suggests that the dPFC activity and PES are spuriously correlated. In particular, the inhibition account posits that the error monitoring system implements post-error adjustments by inhibiting the primary motor cortex (M1). As a result, motor activation will reach response threshold later, leading to longer RTs, but also to higher accuracy in trials following an incorrect response. To test this hypothesis, Danielmeier and colleagues (2011) examined neural activity in sensory color and motion-related brain areas after error commission. Participants were showed a cloud of colored dots moving to the left or to the right side of the monitor. The task required to identify the color of the dots, ignoring the motion direction. Danielmeier and colleagues (2011) found that after an error an attentional shift toward task-relevant stimulus features and away from task-irrelevant features occurred. In particular, results showed that after an error, the activity in perceptual areas that encoded colors increased, whereas the processing of distracting stimuli features tended to decrease. Crucially, the PES effect correlated with a decrease of motor system activity. Similar results were obtained by King and colleagues (2010). He measured the activation of the fusiform face area (FFA, Kanwisher et al., 1997) after an error in a face discrimination task. King and colleagues (2010) instructed participants to identify the gender of male and female faces presented on the left or on the right side of the monitor. They found a post-error increased activity of FFA, which suggested that faces received more attention after an error. Again, a negative correlation between PES and response-related sensorimotor cortex was observed. Further support to the inhibition account was also given by Marco-Pallares and colleagues (2008) who examined oscillatory electrical brain activity after an error in a flanker task. Results showed that PES correlated with an increased beta band power, which reflects motor inhibition and motor stopping (Kuhn et al., 2004; Marco-Pallares et al., 2008). The studies summarized above support the hypothesis that motor inhibition plays a crucial role for PES, but it does not rule out the hypothesis that the post-error motor inhibition could be triggered by other cognitive process, as suggested by the CMT (Danielmeier & Ullsperger, 2011).

A third functional explanation of error reactivity was provided by the *reinforcement learning account* (RLA; Holroyd & Coles, 2002). This theory draws on artificial intelligence models of learning (Sutton & Barto, 1988) and on physiological evidence from the field of reward-guided learning in primates. According to RLA, the basal ganglia (BG) is the location of an error monitoring system computing predictions on whether the outcomes associated with the ongoing action are favorably or unfavorably. When events are worse than expected, as in the case of errors, the midbrain dopamine system sends error signals to the ACC, which processes this error information and improves task performance by implementing strategic adjustments. Error signals, conveyed from the BG, consist in phasic decreases of the mesencephalic dopaminergic system activity (Holroyd & Coles, 2002). Converging lines of evidence support a role for dopamine in error-reactivity. Patients with neurological disorders characterized by a dopaminergic dysfunction (e.g., Parkinson's and Huntington's disease) exhibit an abnormal PES effect (Stemmer et al., 2007). Moreover, some studies showed that antidopaminergic drugs (e.g., Haloperidol) tend to attenuate the PES effect, whereas amphetamine, which blocks dopamine uptake and promotes its release, enhances the PES amplitude (de Bruijn et al., 2004). In this respect, the PES may be considered a marker of dopaminergic dysfunction in neurological and psychiatric disorders (Wardle, Yang, & de Wit, 2012). Although the RLA offers an interesting framework to understand error-reactivity, neurochemical mechanisms underlying this theory are difficult to investigate in humans (Ullsperger, Danielmeier, & Jocham, 2014). Indeed, many hypotheses of this theory can be tested only using invasive methods, such as drug administration or intra-cortical recording. For this reason, only a paucity of studies have investigated RLA predictions about error-reactivity.

1.4.2. Non-functional accounts

Over the last 10 years, functional accounts of error reactivity have been challenged on multiple fronts. In particular, the evidence that PIA and PES are dissociated, and the results of some studies showing a decrease of accuracy after an error (e.g., Rabbitt & Rodgers, 1977; Fiehler et al., 2005) gave rise to non-functional accounts, interpreting the PES as a non-adaptive effect. Notebaert and colleagues (2009) proposed the *Orienting account.* This theory suggests that PES is produced by the interference caused by an error-evoked orienting response. Since errors are unexpected events, they divert attention from the task, producing both a PES and a *decrease* in post-error accuracy. Therefore, according to this account, it is not the error itself that elicits the slowing, but rather the attentional orientation toward that event. To test this hypothesis, Notebaert and colleagues (2009) compared a condition in which errors were infrequent with a condition where, instead, correct trials were infrequent. The task required to identify the color of a square presented on the screen. The brightness of the colors was adjusted in order to manipulate the difficulty of the task and to define three conditions, characterized by a different level of accuracy (35, 55, 75% accuracy). Results showed that in conditions where errors were infrequent, a PES effect was present. However, when participants committed many errors and correct trials were rare, a *post-correct slowing* was observed*.* The orienting account posits that a lower level of surprise is expected for participants who make more errors and this, in turn, reduces the PES effect (Notebaert et al., 2009; Notebaert & Verguts, 2011; Steinborn, Flehmig, Bratzke, & Schroter, 2012; Wessel, Danielmeier, Morton, & Ullsperger, 2012). In order to corroborate this hypothesis, Notebaert and colleagues (2009) compared the reaction to erroneous responses with the reaction to oddball stimuli. Crucially, results showed that RTs tended to increase both on trials following infrequent acoustic signal and on trials following an erroneous response, which supports the idea that errors act as "oddballs" that divert attentional resources from the task and, thus, impair subsequent performance. Steinborn and colleagues (2012) tested the *Orienting* account using a *serial mental addition and comparison task*. The task required to solve an addition, compare the solution with an adjacent number, and indicate the larger number value by means of a button-press. After the task, participants were classified into three groups differing in overall accuracy. Steinborn and colleagues (2012) found that less accurate participants presented a decreased PES. Similar results were obtained by Fiehler and colleagues (2005). Participants were assigned to two groups. The first group was instructed to correct errors by pressing the correct button right after the response (correction-instructed group). Instead, the second group was not informed of the possibility to correct errors (non-instructed group). The non-instructed group was more cautious and committed relatively fewer errors than correction-instructed group. In line with the *Orienting* account, Fiehler and colleagues (2005) observed a PES effect only for the non-instructed group, whereas no post-error effects were found in the correction-instructed group. The Orienting account is also supported by some ERPs studies. Nunez Castellar, Kuhn, Fias and Notebaert (2010) observed that the P3 amplitude correlates with the PES. The P3 reflects, among other things, the processing of an orienting response (Friedman et al., 2001). Van Der Borght, Schevernels, Burle, and Notebaert (2016) examined the effect of errors on early visual ERP components, using a flanker task followed by a visual discrimination task. In the flanker task participants had to focus on a central letter and ignore surrounding letters, whereas in the visual discrimination task they had to categorize a target-stimulus, which could be a diamond or a triangle. The main result of this study was that, after an erroneous response in the flanker task, the amplitude of the N1 decreased in the visual discrimination task. Since the N1 component is associated with focal attention processes (Luck, 2014), Van Der Borght and colleagues (2016) argued that after an error an attentional drop occurs, which makes more difficult to focus attention on the stimulus of the visual discrimination task.

However, the detrimental effect of errors on subsequent information processing may not necessarily depend on an error-evoked orienting response. In contrast with the Orienting account, the *Bottleneck theory* (Dudschig & Jentzsch, 2009) suggests that the PES effect is the byproduct of a "processing overflow". Indeed, error processing consists in the detection of an error and in the identification of what determined it. Such elaboration subtracts cognitive resources from a limited central information processor. As a result, when an error occurs, if the RSI is not long enough, its elaboration can interfere with performance of the following trial, because fewer resources are available. Support for the bottleneck theory comes from the evidence that PES is significantly larger when the ITI is short, and it is reduced when more time is given to process an error (e.g., Danielmeier & Ullsperger, 2011; Dudschig & Jentzsch, 2009). In a recent study, Houtman and Notebaert (2013) tried to dissociate between the Bottleneck and Orienting account. For this purpose, at the beginning of each trial participants were shown an irrelevant signal, consisting in a green J standing for "correct" or a red F letter standing for "incorrect". Red signals determined similar effects to errors on the subsequent performance, as red color is associated with danger and evokes avoidance motivation (Elliot et al., 2007). The frequency of these two signals was manipulated, so that in one group the letter J was more frequent and the letter F infrequent, and viceversa. Afterward, the task required to perform a *rapid serial visual presentation* (RSVP) task. This task consists in identifying a target letter, placed inside a stream of numbers presented in quick succession. The Orienting account would predict an interaction between the frequency of the two signals and the target detection accuracy. Instead, the Bottleneck account would predict a performance deterioration only after the error-like red signals. Results showed that, after an irrelevant red signal, target detection was less accurate, irrespective of frequency, providing support for the Bottleneck theory (Houtman & Notebaert, 2013).

To conclude, there appears to be evidence for both functional and non-functional accounts of error reactivity and that these accounts are not mutually exclusive (Danielmeier & Ullsperger, 2011). Indeed, the functional and non-functional aspects of error reactivity may follow different time courses. Errors may first elicit an orienting response, and then, if enough time is available, trigger strategic adjustments aimed at improving the subsequent performance. It is noteworthy that realistic actions have slower time course that choice RT tasks, and thus more opportunity to benefit from strategically implemented control.

2. THE EXPERIMENTAL WINDOW: THE REACH-TO-GRASP MOVEMENT

The reach to grasp movement is amongst the most important daily-life movement. For this reason, its kinematical organization and the neural bases underling it have been extensively investigated by cognitive neuroscientists. In the following paragraphs I will review the principal studies that paved the basis of our current understanding of human prehension and why it is suitable to test my experimental hypotheses.

2.1. The static and dynamic characteristics of the reach to grasp movement

The modern study of human hand movements has been pioneered by the British biologist John Napier (1960) that provided the first taxonomic description of human handgrips by describing two main types of grasping, namely *power* and *precision grip* (Figure 4). With a power grip the object is clamped by means of a flexion of the fingers toward the palm, with the thumb acting as a reinforcing agent to enable large force to be applied. Instead, the precision grip consists in a thumb opposition to one or more fingers, and it is usually employed for accurate movements.

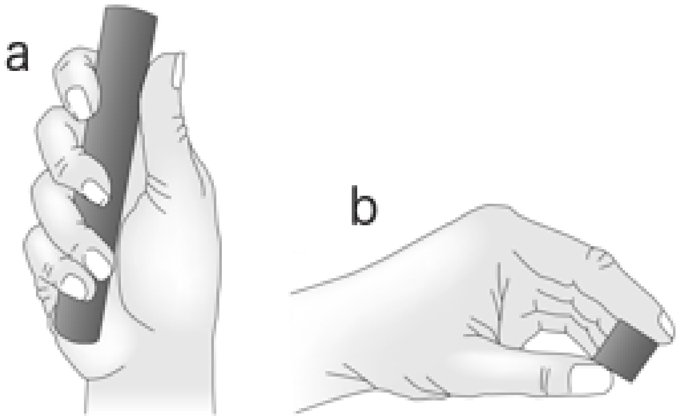

Figure 4. Examples of power grip (a) and precision grip (b). From Castiello, 2005.

Napier provided a qualitative descriptions of handgrips using photographs of the hand. It is noteworthy, however, that the final grip is preceded by a dynamic *pre-shape phase*, during which the fingers shape in order to prepare the hand to match with the features of the to-be-grasped object. These dynamic aspects were first examined by Marc Jeannerod (1981; 1984) who, using high speed cinematographic techniques, provided a quantitative description of the reach to grasp movement. Jeannerod (1981) identified two major components of the prehension: the reaching component, concerning the transport of the hand near the target object, and the grasping component, consisting in the opening of the hand followed by a gradual closure of the grip. In order to provide a quantitative description of the reaching component, Jeannerod (1984) measured the position of the wrist during the movement, considering each frame of the video, and then computed the tangential velocity and acceleration of the wrist trajectory. Instead, the grasping component was measured considering the distance between the index finger and the thumb during the movement. Jeannerod (1981; 1984) observed that the reaching movement was represented by a right-skewed velocity profile, characterized by a ballistic pre-planned phase followed by a low-velocity phase controlled by visual guidance (Jeannerod, 1981). As regards the grasping movement, Jeannerod (1981) observed that the maximum grip aperture (MGA) exceeded of about 20% the size of the object being picked up, and occurred at around 50-70% of the movement duration (Jeannerod, 1984). Marteniuk and colleagues (1990) obtained similar results, founding that the amplitude of MGA increased by 0.77 cm for each increase of 1 cm of the object size.

Beside describing the principal kinematical characteristics of the reach to grasp movement, Jeannerod (1981) investigated the underlying visuomotor processes, proposing that the reaching and grasping component were based on independent and separate visuomotor mechanisms. This hypothesis, known as *visuomotor channels hypothesis* (VCH)*,* has been the subject of intensive investigations and will be discussed in the following paragraph.

2.2. The visuomotor channels hypothesis

The core idea of the VCH is that each component of the prehension arises from two discrete and independent *visuomotor channels*. In this view, a visuomotor channel is a specialized mechanism that generates appropriated motor responses by processing a limited number of features of the object. The VCH holds that the reaching movement involves the *transport channel,* which would be sensitive to the *extrinsic properties* of the object, such as the object's location (Jackson, Jackson, & Rosicky, 1995) and distance from the object to the observer (Chieffi & Gentilucci, 1993; Jeannerod, 1984). Instead, the grasping component would involve the *manipulation channel,* which would be based on the *intrinsic properties* of the object, such as fragility (Savelsbergh, Steenbergen, & van der Kamp, 1996), size of the contact surface (Bootsma, Marteniuk, MacKenzie, & Zaal, 1994; Castiello, Bennett, & Stelmach, 1993), texture (Weir, MacKenzie, Marteniuk, & Cargoe, 1991) and weight (Gordon at al., 1991).

In order to accomplish successfully a reach to grasp movement, the transport and manipulative components must be temporally synchronized. In this respect, Jeannerod (1984) noted that the time at which fingers starts to open was correlated with the beginning of the reaching movement (Jeannerod, 1984). Moreover, the time of the maximum grip aperture is correlated with the time of wrist peak deceleration (Jeannerod, 1984). On the basis of these results, Jeannerod (1984) proposed that reaching and grasping are temporally coupled and their organization is programmed by a central plan, which ensured the execution of these independent components in a single coordinated act. This idea was confirmed and data replicated only by Wallace, Weeks and Kelso (1990), that studied the relation between the MGA and the velocity of the movement. Participants were assigned to two conditions: the *comfortable condition,* in which participants had to reach out and grasp a wooden dowel at normal speed, and the *fast condition,* in which they had to perform the reach to grasp movement as fast as possible. Results showed that the relative time of MGA, occurring within 60–70% of the duration of the movement, was consistently invariant despite the speed of the movement.

Another fundamental assumption of the VCH is that the transport and manipulation channel are informationally distinct, in the sense that the information used by one channel is not available to the other. Jeannerod (1981) investigated this prediction, using a *visual perturbation paradigm.* Initially, participants were showed a spherical object. As soon as they moved the hand to grasp it, an oval-shaped object was unexpectedly superimposed on the sphere. Jeannerod (1981) observed that the shape-perturbation effect was confined to the grasping component, whereas no effects for the reaching component were found. In a following study, Paulignan and colleagues (1991) presented to subjects concentric objects consisting of an inner dowel, surrounded by a larger cylinder. These objects, made of translucent material, could be illuminated independently by means of some LEDs positioned below them. Each trial began with illumination of the dowel or the cylinder. In perturbed trials, the light illuminating the dowel was unexpectedly shifted to the cylinder, creating the illusion of a sudden increase of the dowel size. Instead, when the light was shifted from the cylinder to the dowel, participants had the impression of a sudden decrease of the cylinder size. Results showed that the perturbation of the object’s size did not affected the wrist kinematic landmarks, but only the grasping component, supporting the hypothesis that the reaching component is not influenced by the intrinsic proprieties of the object. Further evidence in favor of the VCH was provided by Chieffi and colleagues (1992). Participants were asked to grasp a plastic sphere positioned on a carrier driven by an electrical motor. The sphere moved toward the subject at four constant velocities. Results showed that the time and amplitude of the maximum peak velocity of the wrist were lower when the plastic sphere was faster. However, no effect was found for the grasping component. Chieffi and colleagues (1992) argued that, as the object velocity is an extrinsic property of the object it could only influence the transport component, leaving the grasping component unaffected.

In addition to behavioral evidence, several neurophysiological, neuropsychological and neuroimaging studies have confirmed that the two components of prehension are anatomically separable though interconnected. These studies will be described below.

2.3. Neural pathways underlying reaching and grasping

Knowledge of the neural network underlying reaching and grasping have gained a great advance in the last 20 years. Monkey neurophysiological studies and neuropsychological studies on humans have provided converging evidence in support of the VCH. In the next sections, I will make an overview of the principal findings that resulted from these studies.

2.3.1. Macaques studies

The studies conducted on macaques have revealed different neural pathways involved in reaching and grasping (Figure 5). The anterior intraparietal sulcus (AIP) and the premotor cortex (F5) have been found to be related to the grasping movement (Castiello, 2005). In monkeys, the AIP is located in the rostral part of the posterior bank of the intraparietal sulcus and it is a key-area for the visual guidance of hand movements (Baumann et al., 2009; Gallese et al., 1994). Single-cell recording studies showed that these neurons are responsive to shape, orientation and size of object (Murata et al., 2000). Gallese and colleagues (1994) found that a focal lesion of the AIP produces deficits of hand orientation and hand preshaping. Therefore, the AIP seems to be involved in the visual guidance of hand movements (Gallese et al., 1994) and in particular, in the extraction of visual and spatial characteristics of graspable objects (Murata et al., 1997).

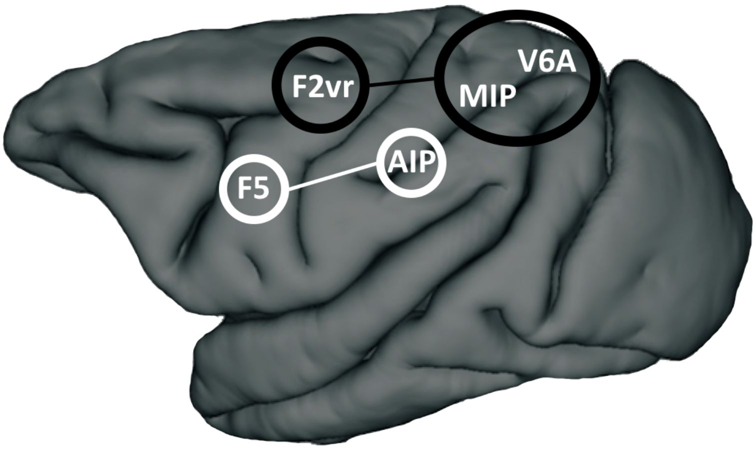

Figure 5. Anatomical localization of grasping (white circles) and reaching (black circles) neural regions in monkeys. From Turella & Lingnau (2014).

The AIP is richly connected with the F5 area (Luppino et al., 1999; Stark et al., 2007). This area, located in the rostral part of the ventral PMC, is supposed to encode specific grip types depending on the object to grab and on the goal of the action (Stark et al., 2007; Murata et al., 1997; Fluet et al., 2010). Fogassi and colleagues (2001) found that the inactivation of F5, in particular a lesion of the posterior bank of the inferior arcuate sulcus, determines an abnormal hand shaping and produces inappropriate hand postures for the object size and shape.

For the reaching component three crucial areas have been found in monkeys: V6A (Bosco et al., 2010), MIP (Johnson et al., 1996), and the dorsal premotor cortex (F2vr; Caminiti et al., 1991). Area V6A is located in the anterior bank of the parieto-occipital sulcus. This area includes visual neurons and transmits visuospatial information to frontal areas for the visual control of the reaching movement (Bosco et al., 2010). A lesion to V6A disrupts the capacity to correctly plan the trajectory of the reach movement and to adjust online the position of the arm (Bosco et al., 2010).

The medial intraparietal area (MIP) is supposed to process the spatial coordinates of the target, and to transform them into a representation used by the motor system in order to define the direction and trajectory of the reach movement (Cohen & Andersen, 2002). Rushworth and colleagues (1997) observed that the inactivation of the MIP produces a disruption of the ability to coordinate the arm and an abnormal coordination of the hand in shoulder-centered space.

2.3.2. Human studies

The VCH is not only supported by monkey neurophysiological studies but also by neuropsychological and neuroimaging technics (Turella & Lingnau, 2014), which have found a similar dissociation between reaching and grasping in humans (Figure 6). The first neurophysiological evidence supporting the VCH came from lesion studies, investigating the motor deficits induced by parietal lesions (e.g., Jeannerod, Decety, & Michel, 1994; Binkofski et al., 1998; Davare et al., 2006). Jeannerod and colleagues (1994) described a 35year-old patient (A.T.) with an extensive posterior parietal lesions, presenting a specific bilateral deficit in grasping. A.T. had abnormal anticipatory opening of fingers resulting in a clumsy and inaccurate grasping, whereas reaching was much less impaired. A similar *misgrasping* has been observed in patient suffering from *optic ataxia*, a visuomotor disorder following lesions of the posterior parietal cortex. Jeannerod found that patients with optic ataxia showed abnormal finger grip aperture and did not present the usual correlation between the maximum grip aperture and object size (Jeannerod, 1986).

With the advent of neuroimaging, particularly fMRI, considerable progress has been made in understanding the neural substrates of reaching and grasping. In particular, two distinct parietofrontal circuits have been observed to contribute to the control of prehension: a dorsolateral parietofrontal circuit (*grasp network*), controlling the distal musculature involved in the grasping component, and dorsomedioal parietofrontal circuit (*reach network*), controlling the proximal muscles involved in reaching component.

The grasp network involves the anterior part of the intraparietal area (AIP) and the ventral premotor cortex (PMv; Godschalk, 1991; Raos et al., 2004; Begliomini et al., 2007). AIP is supposed to process information about the intrinsic proprieties of the object (Davare, Rothwell & Lemon, 2010). Binkofski and colleagues (1998) observed that a lesion of the AIP causes a delayed and inappropriate aperture formation. In line with these results, Davare and colleagues (2006) examined the involvement of the AIP-PMv circuit in the reach to grasp movement, inducing a *virtual lesion* by means of TMS. In conventional virtual lesion paradigms, quick repetitive burst of magnetic stimulation are applied in order to transiently disrupt the functioning of the cortical area of interest. Davare and colleagues (2006) reported that a *virtual lesion* of the AIP alters the preshaping of the hand, whereas the reaching movement remains intact. The PMv seems to play a key-role in the preshaping of the hand (Davare et al., 2006). A virtual lesion in PMv disrupts the correct positioning of fingers on the object, suggesting that this area is also implicated in contact point selection (Davare et al., 2006).

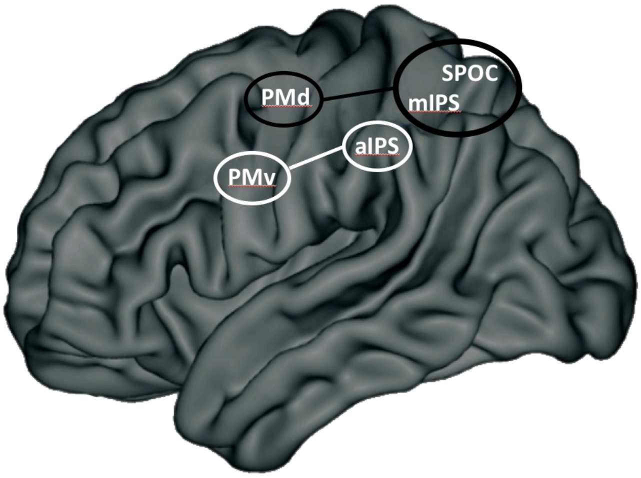

Figure 6. Anatomical localization of grasping (white circles) and reaching (black circles) neural regions in humans. From Turella & Lingnau (2014).

The reach network includes the medial intraparietal area (MIP; Andersen & Cui, 2009), the dorsal premotor cortex (PMd; Sakata & Taira, 1994) and the superior parietal occipital cortex (SPOC; Gallivan, Cavina-Pratesi & Culham, 2009). MIP is involved in the encoding of the visual coordinates of the reach object (Torres, Quiroga, Cui & Buneo, 2013).

A virtual lesion in MIP leads to errors in the initial direction of movements (Davare, Zénon, Desmurget & Olivier, 2015). PMd is thought to plan high level kinematic parameters such as the speed and direction of the reach movement (Torres et al., 2013). PMd lesions disrupt the temporal and kinematical organization of the reaching movement (Davare et al., 2015). As regard the SPOC, this area is more activated when subjects are showed reachable objects than objects beyond the reach of the hand (Gallivan et al., 2009). This may suggest that the SPOC may control the visual control of the reaching movement (Gallivan et al., 2009). The primary motor cortex (M1) is a downstream target of the grasp and reach network. The M1 is located in the precentral gyrus of the frontal lobe (Brodmann areas 4,6,8) and contains a somatotopical representation of each part of the body (Figure 7). The arm and the hand are represented in two distinct areas at the level of the M1 and are dorsolaterally located between the trunk and the face area.

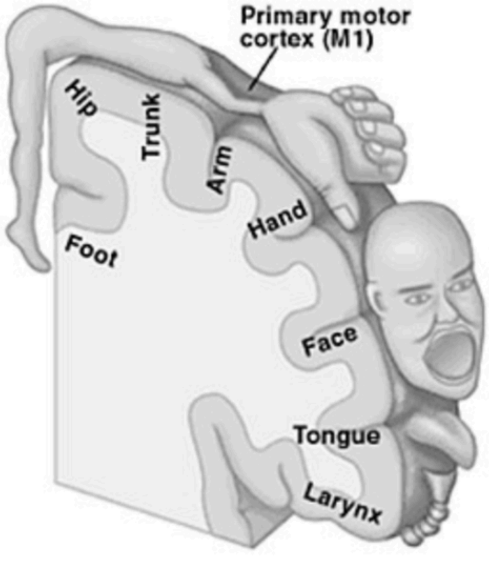

Figure 7. The motor homunculus in primary motor cortex. (Source: http://brainconnection.positscience.com).

The above-mentioned studies demonstrate that the existence of two independent visuomotor mechanisms is well supported by behavioral, neuropsychological and neuroimaging evidence. However, some studies have challenged the VCH on both theoretical and empirical grounds. This body of data will be discussed in the next paragraph.

2.4. Limitation of the visuomotor channels hypothesis

A crucial prediction of the VCH concerns the impermeability of visuomotor channels. In other words, changing an extrinsic feature (e.g., the position) of an object should impact only on the reaching component, whereas modifying an intrinsic feature of the object (e.g., the size) should have a specific influence on the grasping component (Jeannerod 1981, 1988; Paulignan & Jeannerod, 1996). This critical prediction has been tested by several studies, and some of them provided evidence that questioned the VCH, showing that, in some circumstances, reaching and grasping are influenced at the same extent by both the intrinsic and extrinsic proprieties of the object (Castiello et al., 1993; Jakobson & Goodale, 1991; Marteniuk, Leavitt, MacKenzie, & Athenes, 1990; Chieffi et al., 1992; Gentilucci et al., 1991). For instance, some investigations observed that object size can affect the transport component (Marteniuk et al., 1990; Jakobson & Goodale, 1991; Gentilucci et al., 1991). Marteniuk and colleagues (1990) conducted an experiment in which participants were required to reach out and grasp ten disks differing in size, by means of a precision grasping. For the grasping component, results showed an anticipation of the MGA time as the disk size decreased. Crucially, for the reaching component, the movement time (MT) tended to increase as the object size decreased. Using a similar task, Jakobson and Goodale (1991) confirmed these results, and further noted that the maximum height of the wrist tended to increase with larger objects. Gentilucci and colleagues, (1991) investigated whether the reaching component was influenced by the handgrip used to grasp the object. Participants were required to grasp a large cylinder by means of a power grasping or a small sphere by using a precision grasping. Results showed that the transport time was longer and peak velocity was higher when participants used the power grasping respect to when they used the precision grasping, again demonstrating a dependence between reaching and grasping. Coherently, the manipulation of the extrinsic features of the object have been observed to influence the manipulative component. Paulignan and colleagues (1990) presented to participants three identical dowels, positioned concentrically at 10, 20, 30 degrees from the mid-sagittal plane of the participant. These objects, made of translucent material, could be independently illuminated by means of some LEDs, placed below them. Participants were required to reach out and grasp the central illuminated dowel. However, in perturbed trials, at the onset of the movement, the light quickly shifted from the central dowel to the one placed to the right or to the left, giving the impression of a sudden shift of the object position. Results showed that after the perturbation the grip aperture tended to slightly decrease and then resumed a normal preshape in order to grasp the object. Haggard and Wing (1991), replicated these results, finding that if the arm is perturbed during the reach to grasp movement, compensatory adjustments are implemented for both the wrist trajectory and grip aperture. In their experiment, a mechanical device was attached to the participant’s arm. During the reach to grasp movement the device delivered mechanical perturbations, by pulling on the participant’s arm for about 250 ms. This perturbation elicited a rapid adjustment of the wrist, consisting in a reacceleration of the arm during the reaching of the object location. In addition, the grip aperture showed a double peak indicating a reorganization of the grasping component following the perturbation.

At neurophysiological level, some studies challenged the strict neural dichotomy between reaching and grasping (e.g., Battaglini et al., 2002; Fattori et al., 2010). Battaglini and colleagues (2002) investigated the effect of lesions to V6A area in monkeys. Results showed that V6A lesions did not specifically impair the reaching movement, but also provoked a deficit in wrist orientation and grasping. These findings were confirmed and extended by Fattori and colleagues (2010). They trained two macaque monkeys to reach and grasp five objects: a ball with by means of a whole-hand prehension, a handle by inserting the fingers in the gap behind it (finger prehension), a ring by inserting the finger into it (hook prehension), a plate by opposing the thumb to the other fingers (primitive precision grip), and a small object in a container by means of a precision grip (Figure 8).

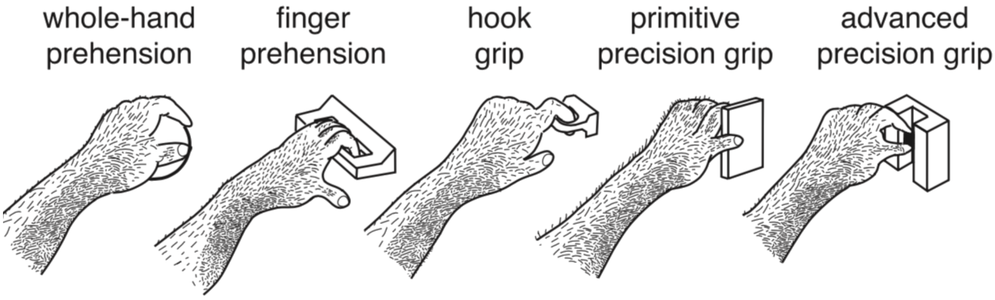

Figure 8. Stimuli and grip types used by Fattori and colleagues (2010).

Using a single-cell recording method, Fattori and colleagues (2010) observed that some neurons in V6A area were selectively activated depending on the grip used in the task. On the basis on this result, they suggested that V6A neurons are not only involved in the reaching movement, but they also play a key-role in grip selection (Fattori et al., 2010). Similarly, some fMRI study in humans suggest that reaching and grasping are underpinned by overlapping areas. Gallivan and colleagues (2011) asked participants to grasp the top and the bottom of a cube, or to reach and touch the side of the cube. They found similar parieto-occipital and dorsal premotor activation for reaching and grasping movements.

The data reported above certainly weaken the proposal of two independent channels and suggest that, to some extent, there is a cross-talk between the two components of prehension (Castiello et al., 1993; Chieffi & Gentilucci, 1993). Although not definitive, the results of these studies have fueled a vast body of research that has contributed to understand the influence of object features on human prehension. It is noteworthy, however, that reaching and grasping are not only constrained by the extrinsic and intrinsic properties of the object but also by the characteristics of the previous movement. In the next paragraph, I will describe one of the most reported sequential effects, that is the *hysteresis effect* (Kelso, Buchanan, & Murata, 1994).

2.5. The hysteresis effect

The way human beings reach and grasp an object is influenced by preceding motor events. Indeed, even simple actions, such as reach out and grabbing a glass, are based on a motor plan. Motor plans are not necessarily created from scratch, but are often based on the previous ones, which can be, at least in part, readopted again (Dixon, McAnsh, & Read, 2012). This phenomenon, known as hysteresis effect, was observed for the first time by Rosenbaum and Jorgensen (1992) using a *grip preference task*. Participants stood in front of a bookshelf with 14 shelves vertically arranged. They were asked to reach out and grab an horizontal rod positioned on a stand, either approaching the rod from above by using an overhand grip, or approaching the rod from below by using an underhand grip. After grabbing the rod, participants had to place the marked side of the rod against one the14 targets positioned on each shelf, and then return the bar to the cradle. This procedure was repeated for each target, once from top to bottom, once from bottom to top. Rosenbaum and Jorgensen (1992) observed that participants tended to use an overhand grip for upper target and a underhand grip for lower target. However, the type of grip used for the central targets varied as a function of the grip used in the previous trial. In other words, participants tended to perseverate the grip adopted in the previous trial until the posture was not so uncomfortable to compromise the correct accomplishment of the task. The hysteresis effect has been observed in a great variety of task. For instance, Jax and Rosenbaum (2007) using an *obstacle avoidance task* investigated whether the curvature of recent hand paths influenced the hand paths on the subsequent trial. Participants were seated in front of a screen, which showed the position of their arm and hand, tracked by means of infrared markers. A target appeared on the screen, and participants had to reach it as fast as possible, avoiding an obstacle, if one was present. Jax and Rosenbaum (2007) observed that when participants avoided successfully an obstacle to reach a target, they tended to use the same trajectory in the following trial, even if the obstacle was not present. Similar hysteresis effects have been found for the grip aperture. Dixon and Glover (2009) asked participant to reach out and grasp some disks placed in front of them, differing in size. Results showed a potent tendency to perseverate in grip aperture during the latter portion of a movement to grasp a disc.

To conclude, the hysteresis effect is consistent with the idea that motor control is a very efficient process, which, instead of creating a new motor plan for every action, economizes resources recycling recently executed motor parameters. In this sense, a prehension movement cannot be investigated exclusively considering the proprieties of the object, but it is also important to consider its recent motor history. A point worth noting is that until now research on hysteresis has focused the investigation on sequential effects arising from sequences of correct movements. In daily life, however, carrying out a task does not always run smoothly and people can fail to perform a reach-to-grasp movement, which begs the question - how and to what extent the failure to grasp an object influences the following movement? This is the main question that drove my entire experimental work, which will be presented in the next section.

2.6. The present research

Generally, with just a few exceptions (see Anguera, Seidler, & Gehring, 2009; Krigolson & Holroyd, 2007; Vocat, Pourtois, & Vuilleumier, 2011), error reactivity has been investigated by means of speeded reaction time (RT) tasks and most studies have measured only arbitrary button-press responses (Gehring, Liu, Orr, & Carp, 2011). However, as pointed out by Gehring and colleagues (2011), most daily life movements have a slower time course than speeded RT response, and more realistic and ecologically valid tasks may afford a better opportunity to investigate error-reactivity. Moreover, since the functional meaning of PES is yet unclear, it might be useful to explore error-reactivity by using richer measures than RT, which limits the investigation to pre-movement processes. For instance, the consequences of self-generated errors on the kinematics of goal directed actions has yet to be investigated at the time the present thesis work started.

In light of these consideration, the overarching aim of my thesis was to investigate the consequences of errors in the context of goal directed actions. To do this, I explored error reactivity effects on both the preparation and the execution of reach-to-grasp movements. Movement preparation includes the relevant sensory and perceptual processes preceding movement initiation (Haith, Pakpoor, & Krakauer, 2016). Traditionally, it is assessed through the measurement of RT (Wong, Haith, & Krakauer, 2015). Instead, movement execution is customarily assessed via kinematical analysis and allows investigating the added benefit of monitoring and occasionally adjusting motor programs in flight (Erlhagen & Schöner, 2002). A further aim of my thesis was to verify whether error-reactivity has a different impact on the grasping and reaching components or whether it produces an unspecific slowing of the whole movement execution. For this purpose, in Experiment 1 (Chapter 4) I investigated the effects of errors on the planning and the execution of the prehension movement. Participants were asked to reach out and grasp a steel ball positioned on a wooden support, without knocking it over. The tracking of the movement was performed by means of six infrared cameras recording the position three infrared reflective markers taped to the wrist, the index finger and thumb. The index finger and the thumb markers were used to measure the grasping component of the movement, whereas the wrist marker was used to measure the reaching component.

In Experiment 2 (Chapter 5), in order to clarify some aspects concerned with the results obtained in Experiment 1, I adopted an “only reach” task. This was specifically done in order to investigate the effect of errors on the reaching component when it is not embedded in a reach to grasp movement. The task required participants to reach out and touch a wooden support without dropping it. The kinematical analysis was performed by tracking the position of the wrist marker during the movement.

In Experiment 3 (Chapter 6), I combined kinematics with TMS. In particular, I compared the amplitude of the motor-evoked potential (MEP) after correct and erroneous reach to grasp movements. Indeed, the amplitude of the elicited MEP has been considered as a marker of excitability of the corticospinal tract (CST). Therefore, the use of a MEPs analysis can be an useful tool to measure the post-error inhibition of motor planning.

Recently, some evoked related potential (ERP) studies have shown that when participants monitor errors made by other individuals a negative-going ERP component, similar to the error-related negativity (ERN), can be recorded in the observer (oERN; Bates, Patel, & Liddle, 2005; van Schie et al., 2004). However, the effects of error observation on the execution of a goal-directed movement has never been investigated before. Thus, in Experiment 4 (Chapter 7), I examined whether the vicarious experience of errors influences the observer's prehension movement. In this experiment, the task required that participants and a confederate alternated in reaching out and grasping a steel sphere positioned on a wooden support.

3. GENERAL METHODS

In this chapter will be described the methods and the procedures shared by all the experiments included in the present thesis.

3.1. Participants' characteristics

All the participants who took part in the present experiments showed right-handed dominance (Oldfield, 1971), and reported normal or corrected-to-normal vision. They gave informed written consent to participate in the studies. Participation was voluntary. The experimental procedures were approved by the Institutional Review Board at the Padua University and were in accordance with the declaration of Helsinki.

3.2. Apparatus and procedures

Participants were tested in a well-lit room. They were seated on a height adjustable chair, the thorax pressed gently against the front edge of the table (90 X 90 cm) and the feet were supported. A head-chin-rest was used to restrict head movements in order to maintain a constant viewing distance from the target. The target object was placed along the mid-sagittal plane of the participant, 55 cm away from the edge of the table and consisted of a steel ball (0.5 cm Ø), positioned on a wooden support (1 cm in width and 20 cm in length). The starting position implied the ulnar side of the hand placed upon a starting switch (11 X 8 cm) aligned to the participants' body midline and placed 22 cm away from the edge of the table. A container (11 X 8 cm), into which the steel ball had to be placed once grasped, was positioned 4 cm from the starting pad.

3.3. Recording techniques

Movements were recorded by using the 3D-optoelectronic SMART-D system (Bioengineering Technology & Systems, B|T|S|), equipped with six infrared cameras (sampling rate 140 Hz) placed in a semicircle at a distance of 1.2 m from the table. Three infrared reflective markers (0.25 mm Ø) were taped to the wrist (radial aspect of the distal styloid process of the radius), the thumb (ulnar side of the nail) and (3) the index finger (radial side of the nail). To optimize the tracking of all markers, the optoelectronic system was calibrated before data collection by adjusting the camera position, roll angle, zoom, focus, threshold and brightness. These procedures were followed by a static and dynamic calibration. For the static calibration, a three-axes frame of 5 markers at known distances from each other was placed in the middle of the table. For the dynamic calibration, a three markers wand was moved throughout the workspace of interest for 60 s. Coordinates of the markers were reconstructed with an accuracy of 0.3 mm over the field of view. The standard deviation of the reconstruction error was 0.2 mm for the x, y and z axes.

3.4. Data processing

The SMART-D Tracker software package (B|T|S|) was used to produce a 3-D reconstruction of the marker positions as a function of time. The data were filtered using a finite impulse response linear filter (transition band = 1 Hz, sharpening variable = 2, cut-off frequency = 10 Hz; D'Amico & Ferrigno, 1992). The wrist marker was used to measure the reaching component of the action, whereas the finger and the thumb markers were used to measure the grasping component of the action.

3.5. Data analysis

Post-error adjustments were computed using the robust method (**PEA**$_{robust}$) proposed by Dutilh and colleagues (2012). This method consists in comparing trials following (E + 1) and immediately preceding (E - 1) an error. Post-error analyses were restricted to sequences of trials comprising E - 1 and E + 1 correctly performed trials. Post-correct adjustments (**PCA**$_{robust}$) were computed in a similar manner, considering triplets of correct trials.

Linear mixed-effects (LME) models were used to compare post-error and post-correct adjustments for each kinematic component and for RTs. Error rates were analyzed using a Generalized mixed-effects (GLME) models with a binomial link function (Pinheiro & Bates, 2000) after coding error and correct responses with 1 and 0, respectively. Statistical analysis was performed using the computing environment R (R Core Team, 2012), and the packages lme4 (Bates, Maechler, Bolker, & Walker, 2015) and lmerTest (Kuznetsova, Brockhoff, & Christensen, 2013). For the LME and GLME models, random effects consisted of Participants and Blocks. The random structure was selected by means of AIC (Akaike's information criterion) weights (Wagenmakers & Farrell, 2004). Models were fitted using maximum likelihood (ML). p-values were estimated by likelihood ratio tests of the full model against the null model. For LME analyses Cohen's d indices as measure of effect sizes (Cohen, 1988) were reported, whereas for GLME analysis I reported the odds ratio statistic (OR, see Szumilas, 2010). Visual inspection of residual did not show any obvious deviation from normality and homoscedasticity. In order to evaluate the relative evidence for the alternative hypothesis (H1) compared to the null hypothesis (H0), I performed statistical comparisons using the Bayesian approach implemented in the BayesFactor package for

R (Morey, Rouder, & Jamil, 2014). The default Cauchy distribution centered on zero with rate [r] = 0.707 was used. The traditional interpretation of Bayes Factor (Jeffreys, 1961) suggests that a BF greater than 3 represents a sufficient support for the alternative hypothesis, whereas values less than 1 indicate that the data support the null-hypothesis.

4. EXPERIMENT 1: THE GRASPING SIDE OF POST-ERROR SLOWING

The interpretation of PES has been challenged on multiple fronts. Firstly, recent studies have proposed that errors may produce a decrease in performance accuracy and that PES may occur because of the detrimental effect that errors exert on subsequent information processing (see section 1.5.2). Secondly, previous research has been questioned because of the limited ecological validity of speeded RT tasks, commonly adopted to study the PES effect (see section 2.6).

In the present study, I examined error-reactivity in the context of goal-directed actions. In particular, I investigated the extent to which the PES effect influences the execution of the reach to grasp movements. For this purpose, besides considering RTs, I conducted a kinematical analysis in order to explore the kinematical reorganization of the grasping and reaching components after an error. Participants were asked to reach out and grasp a steel ball, without knocking the wooden support over. In order to correctly perform this task, participants had to carefully transport the hand in the proximity of the target and accurately close the fingers upon the steel ball.

I hypothesized that whether error reactivity effects extended to movement execution, I should find evidence of post-error adjustments also at kinematical level. Moreover, functional and non-functional accounts of error reactivity have a different prediction with respect to the post-error accuracy. Indeed, if errors lead to a more cautious movement execution, as suggested by functional accounts, I expected to find a post-error improvement of accuracy. Conversely, if error processing has a detrimental effect on subsequent information processing, as suggested by non-functional accounts, then I expect to find a decrease in post-error accuracy. A further aim of this experiment is to investigate whether erroneous movements have the ability to elicit the hysteresis effect. If errors produce an hysteresis effect then I expect to find a post-error decrease of accuracy, as the motor system would perseverate in implementing an erroneous kinematics on the following trial.

4.1. Method

4.1.1. Participants

As there were no previous studies investigating error reactivity in the context of goal directed actions upon which to refer for an a priori power analysis, I selected a target sample size of 15 subjects (8 females, 7 males) with a mean age 26 (SD = 3.5 yrs), which would give 82.1% power to detect a large effect ($f = 0.4$) at an α level of $p = .05$ (GPOWER 3.1; Erdfelder, Faul, & Buchner, 1996).

4.1.2. Procedure and design

The experimental setting is represented in Figure 9A. Before each trial the shutter goggles were closed and the participant held the ulnar side of the hand placed upon the starting switch with the tip of the index and the tip of the thumb in contact with each other (Figure 9B). As soon as the shutter goggles became transparent, participants were asked to reach out and grasp the steel ball by means of a precision grip (Figure 9B), lift it and deposit it into the container. The task required executing the movement as fast and accurately as possible, without knocking the wooden support over. If the wooden support was dropped the trial was considered as an error. During the inter-trial interval, when the shutter goggles were opaque, the experimenter put the steel ball (and the wooden support in the case of error) back in the correct position. The inter-trial interval varied randomly from 6500 to 8500 ms. Each session consisted of 3 blocks of 40 trials separated by a 3-min break. Participants underwent a practice session of 10 trials before the experimental session began. The experiment lasted approximately 50 minutes.

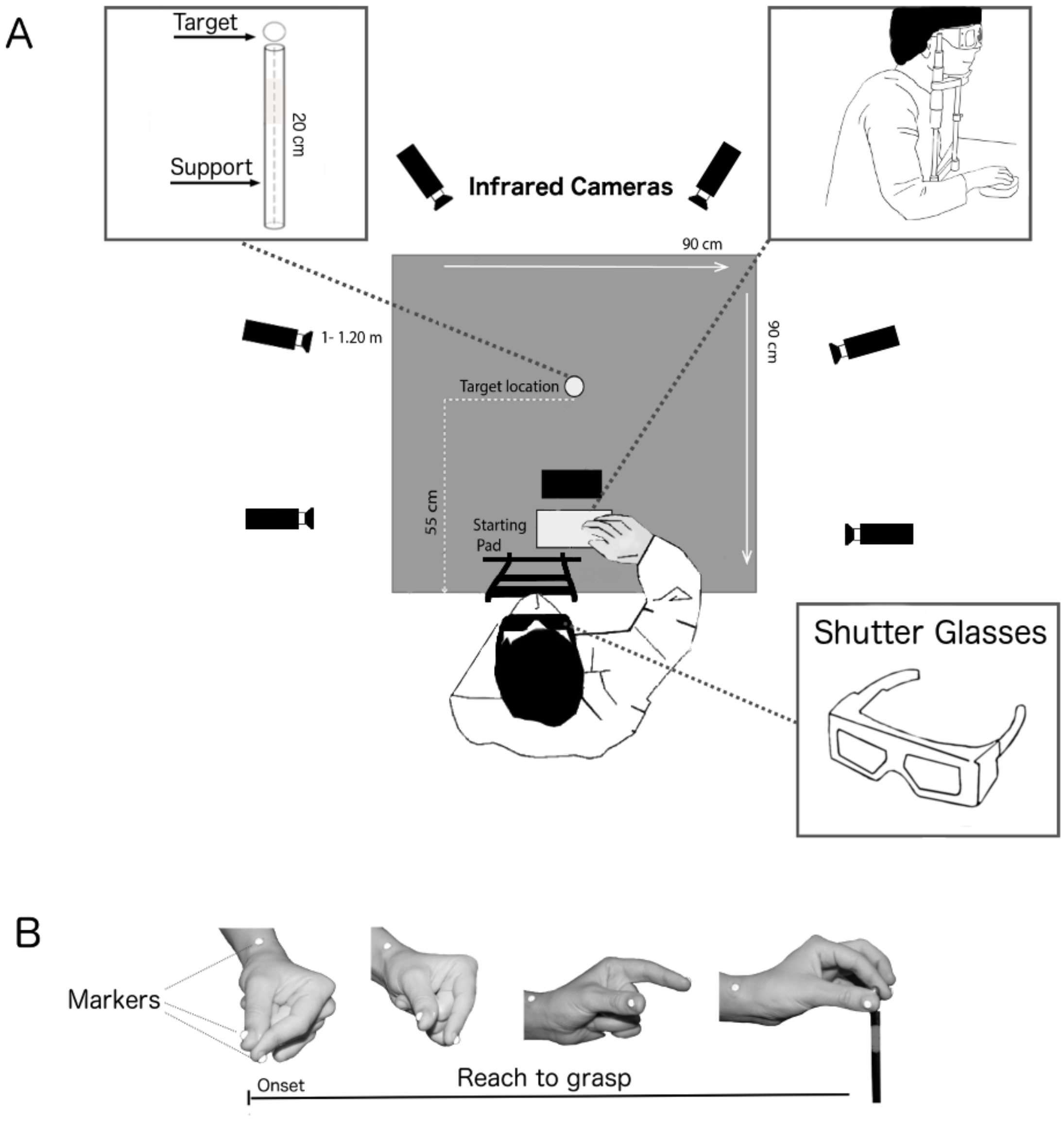

Figure 9. Graphical representation of the experimental set-up (A) and the designated start position for reach-to-grasp (B).

4.1.3. Dependent variables

RT was defined as the time between the opening of the shutter glasses and the release of the starting switch, which corresponded to the movement onset. Movement onset was calculated as the time at which the tangential velocity of the wrist marker crossed a threshold (5 mm/s) and remained above it for longer than 500 ms. End of movement was defined as the time at which the hand made contact with the target and quantified as the time at which the hand opening velocity crossed a threshold (5 mm/s) after reaching its minimum value and remained under it for longer than 500 ms. The time from the onset to the end of the movement was defined as total movement time. For the reaching component (see Figure 10A) I considered reaching time (the interval between the onset of the movement and the time at which the wrist velocity reached its minimum value after the peak of wrist velocity), the amplitude of maximum peak velocity (the maximum velocity of the wrist during the reaching phase), the amplitude of peak wrist deceleration (the amplitude of maximum deceleration of the wrist during the reaching phase), and the time of maximum peak wrist deceleration (the time at which the maximum wrist deceleration occurred). The grasping component was computed based on the relative distance between the markers located on the index finger and the thumb. For this component (see Figure 10B), grasping time (the time from the beginning of fingers opening up to their contact with the target), the amplitude of maximum opening velocity (the maximum velocity reached during the opening phase with reference to the distance between the thumb and the index finger), the amplitude of maximum closing velocity (the maximum velocity reached during the closing phase with reference to the distance between the thumb and the index finger), and the time of peak grip aperture (the point in time at which the index finger and the thumb reached the maximum distance) were considered.

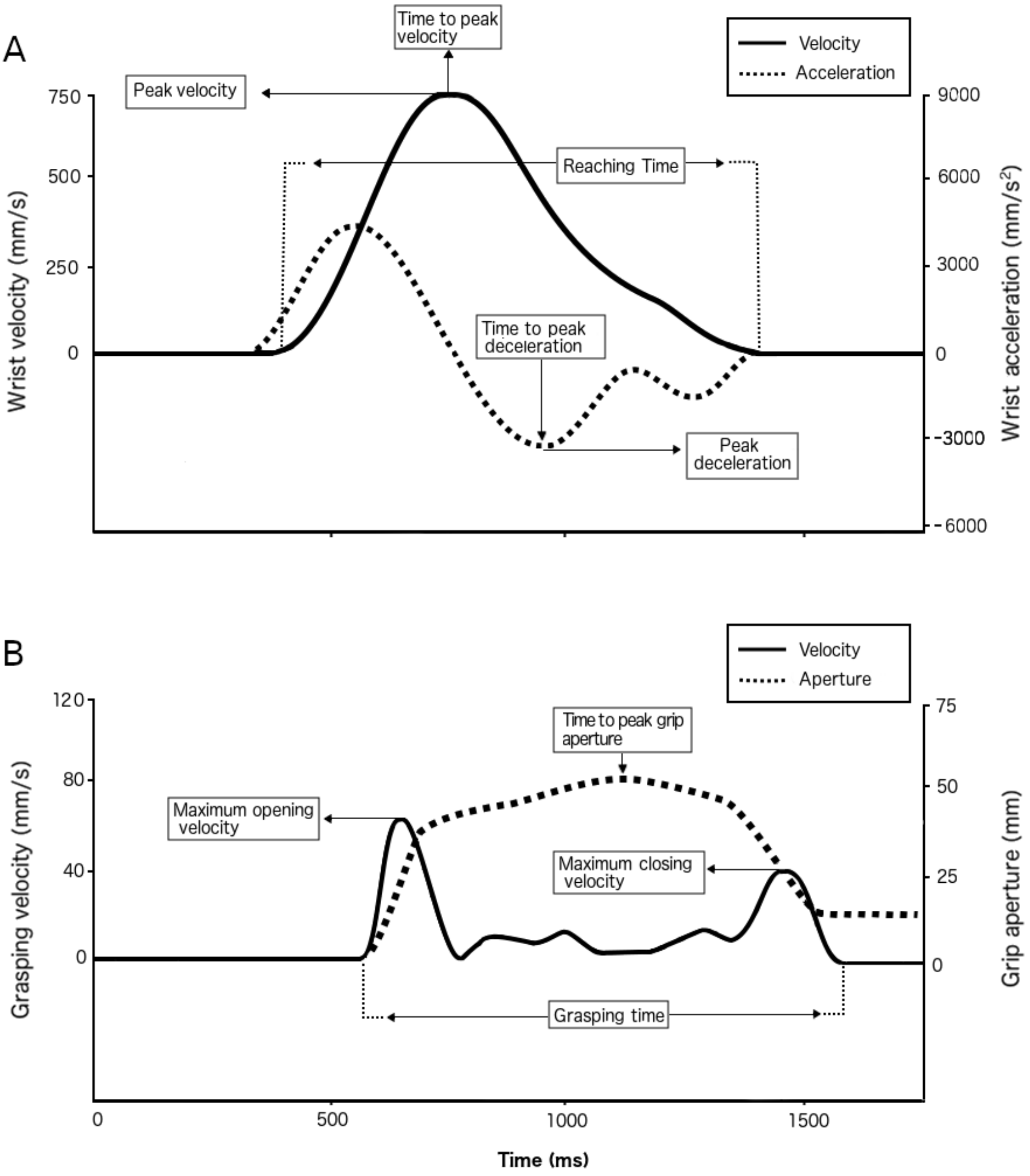

Figure 10. Graphical representation of the experimental variables. Panel A: plot of wrist velocity (*solid line*) and wrist acceleration (*dashed line*). Panel B: plot of grasping velocity (solid line) and grip aperture (dashed line).

4.1.4. Principal component analysis

A principal component analysis (PCA) with Oblimin rotation was applied to decompose the data into their underlying factors and reduce the number of temporal and kinematic variables so as to obtain a protection against the Type I error. RTs were not included in this analysis. On the basis of Kaiser's rule (Kaiser, 1960), I selected three components having eigenvalues above 1, which accounted for 82% of the variance (40%, 21%, 21%, respectively). Weights of the kinematic parameters for the first three components are reported in Table 1.

Table 1. Weights of the kinematic parameters for the first three components of the reach to grasp movement.

	Component 1	**Component 2**	**Component 3**
Movement time (ms)	.972	.001	-.015
Reaching time (ms)	.918	.011	.082
Grasping time (ms)	.959	.004	-.002
Time of peak grip aperture (ms)	.536	-.011	-.441
Time of peak wrist deceleration (ms)	.650	-.097	-.040
Maximum opening velocity (mm/s)	.071	.012	.965
Maximum closing velocity (mm/s)	-.072	-.004	.780
Peak wrist velocity (mm/s)	.114	.998	-.052
Peak wrist deceleration d((mm/s^2)	-.181	.894	.083

The first component had positive weights for total movement time, reaching time, grasping time, the time of peak grip aperture and the time of peak wrist deceleration. This component can be interpreted as a global descriptor of the *reach-to-grasp timing*. The amplitude of peak wrist velocity and the amplitude of peak wrist deceleration showed a positive correlation with the second component. This component can be interpreted as a descriptor of *reaching kinematics.* Finally, the amplitude of maximum opening velocity and the amplitude of maximum closing velocity weighted substantially on the third component, suggesting that it can be interpreted as a *gripping velocity* component. The three components were positively correlated with each other, ranging from .20 to .44. All analyses were performed using the R package ‘psych’ (Revelle, 2012).

4.1.5. Data analysis

The data analysis followed the procedure outlined in paragraph 3.5. The $\mathbf{PES}_{\mathrm{robust}}$ was computed with respect to the three kinematical components and RT. Outliers were eliminated by refitting the model after removing data points with absolute standardized residuals exceeding 3 standard deviations (less than 2%). I discarded 67 of the total of 1.800 trials because of technical problems.

4.2. Results

4.2.1. Accuracy

The overall error percentage was 18%. Post-error changes in error-rate were evaluated by comparing post-error error rates with post-correct error rates. A GLME analysis, with random intercept for Participants and Blocks, showed that subjects were more accurate following an error (16.2% error-rate) than following a correct response (20% error-rate), $z = -2.23$, $p = .025$, $OR = 0.68$.

4.2.2. Reaction times

The mean post-correct and post-error RT adjustments are shown in Figure 11. A LME analysis, with random intercept for Participants (modeling both slopes and intercepts) and Blocks (modeling intercepts only), revealed a significant difference between post-correct and post-error adjustments $\chi^2 = 6.37$, $p = .011$, $BF > 100$, $d = 0.44$. Contrast analysis showed a significant post-error slowing (**PES**$_{robust}$ = 42.80ms ± 12.21), $t(193) = 3.50$, $p < .001$, $BF = 56.73$, $d = 0.25$. After a correct response, subjects showed a *post-correct speeding*, that is, a statistically significant decrease in RTs (-15.18ms ± 4.96) on the following trial, $t(810) = -3.06$, $p = .002$, $BF = 8.20$, $d = 0.10$.

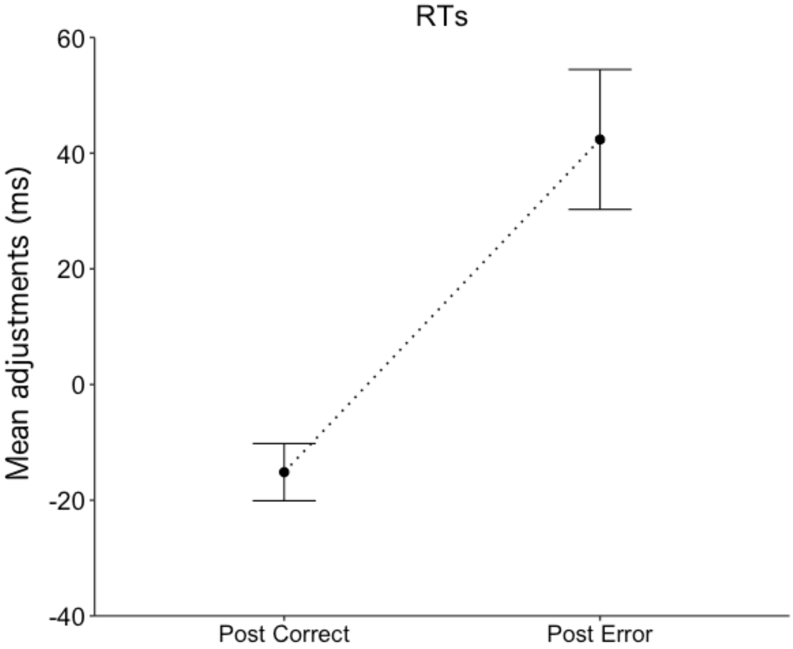

Figure 11. Mean post-correct and post-error adjustments in RTs. Vertical bars indicate standard error of the mean. Positive values indicate a slowing down.

4.2.3. Reach-to-grasp timing

The mean of post-correct and post-error adjustments of the Reach-to-grasp timing are shown in Figure 12A. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), revealed a significant difference between post-correct and post-error adjustments $\chi^2 = 27.85$, $p < .001$, $BF > 100$, $d = 0.21$.

After an error, subjects showed a significant post-error slowing (**PES**$_{\text{robust}}$ = 0.097 ± 0.032), $t(195) = 2.98$, $p = .003$, $BF = 11.80$, $d = 0.21$, whereas after a correct trial a post-correct speeding was present (-0.851 ± 0.015), $t(806) = -5.64$, $p < .001$, $BF > 100$, $d = 0.20$.

4.2.4. Reaching kinematics

The mean of post-correct and post-error adjustments of the Reaching Kinematics component are shown in Figure 12B. A LME analysis, with random intercept for Participants (modeling both slopes and intercepts) and Blocks (modeling intercepts only), revealed no significant difference between post-correct and post-error adjustments, $\chi^2 = 0.04$, $p = .829$, $BF = .09$, $d = 0.02$. Neither a significant post-error slowing (**PES**$_{\text{robust}}$ = 0.012 ± 0.010, $t(200) = 1.20$, $p = .228$, $BF = 0.03$, $d = 0.08$) nor a post-correct adjustment (0.008 ± 0.005, $t(815) = 1.72$, $p = .082$, $BF = .33$, $d = 0.06$) was found.

4.2.5. Gripping velocity

The mean of post-correct and post-error adjustments of the Gripping velocity are shown in Figure 12C. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), revealed a significant difference between post-correct and post-error adjustments, $\chi^2 = 10.88$, $p < .001$, $BF = 17.1$, $d = 0.26$. After an error, subjects showed a significant reduction of gripping velocity (**PES**$_{\text{robust}}$ = -0.101 ± 0.040), $t(199) = -2.53$, $p = .012$, $BF = 3.60$, $d = 0.18$. After a correct trial, a significant post correct speeding was found (0.040 ± 0.018), $t(804) = 2.13$, $p = .033$, $BF = 0.75$, $d = 0.07$.

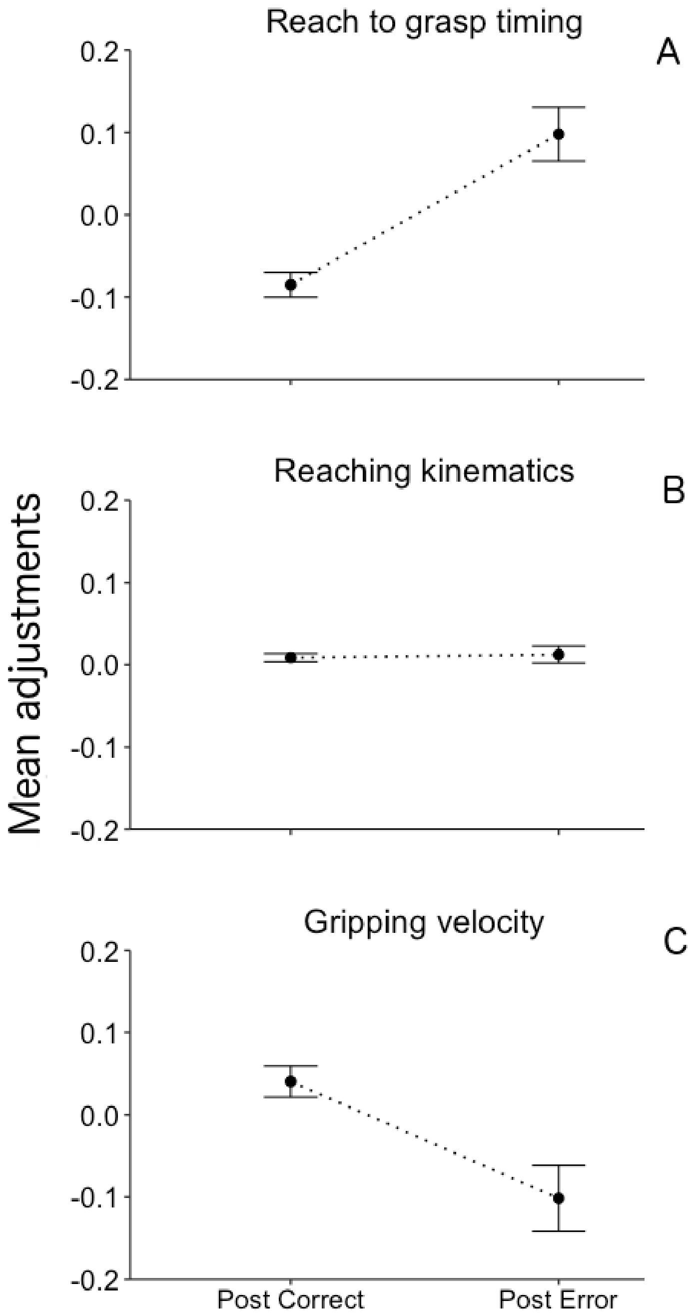

Figure 12. Mean post-correct and post-error adjustments of the reach to grasp timing (panel A), the reaching kinematics (panel B), and the gripping velocity (panel C) components. For the reach to grasp timing, positive values indicate a slowing down. For the reaching kinematics and the gripping velocity component, negative values indicate a slowing down. Vertical bars represent standard error of the mean.

4.3. Discussion

The present study had two aims. First, to investigate error-reactivity in the context of goal-directed actions, in order to examine whether PES effects impact on realistic and complex movements. Second, to verify if error reactivity influences the motor execution of the reach to grasp movement. As an aside, I wanted to test if the hysteresis effect was present after erroneous movements. Results showed that error reactivity effects are present during motor execution, but not in a generalized manner. Indeed, I found that when reach to grasp is the task, error reactivity effects influenced in a targeted manner only the grasping component kinematical parameterization, whereas reaching seems to be impermeable to PES effects. Moreover, the present data demonstrated that the hysteresis effect does not emerge after an error.

4.3.1. The motor preparation phase

At the level of motor preparation, in line with the previous literature, results showed that errors lead to a slowdown of RT. The PES occurred despite I adopted a ITI (from 6500 to 8500) longer than those used by previous studies to investigate error reactivity (from 200 to 5000; e.g., Hajcak, McDonald, & Simons, 2003; Marco-Pallares et al., 2008; King, Korb, von Cramon, & Ullsperger, 2010; Danielmeier et al., 2011; Caudek, Ceccarini, & Sica, 2015). Dutilh and colleagues (2012b), found that with ITIs longer than 1000 ms, PES can be attributed almost entirely to a strategic increase in response caution. Therefore, given that we used very long ITIs, our results seem to be driven by a strategic planning phase, in which the strategic influences of error reactivity are more evident. This idea is confirmed by the fact that performance accuracy tends to increase after an error, supporting the functional of theories of error-reactivity.

4.3.2. The motor execution phase

The present data showed that error reactivity effects also extend to motor execution, albeit in a selective manner. In fact, I found that PES effects are confined to the reach-to-grasp timing and to the parameterization of the grasping component, whereas no effects were found for the reaching component. Contrary to the grasping component, the reaching component seems to be immune to error reactivity effects. A possible explanation for these results may reside in the task we employed for this study, in which grasping kinematics were directly involved in error commission. In fact, in our task all errors occurred during the grip closure phase, when fingers made contact with the target. This might lead to the implementation of strategic post-error adjustments confined to the grasping component, and it may be aimed to improve the control of digit placement upon the target as to ensure successful completion of the task. In these terms, error-reactivity could be considered as a very efficient process, which, instead of inducing a general slowing of the movement, limits its effects in a targeted manner to the component which is directly involved in error commission. To test this idea, in Experiment 2 (Chapter 5) an ‘only reaching’ condition was included in order to verify whether the reaching component is impermeable to error-reactivity effects even when the reaching movement is not embedded in a prehension movement involving also the grasping component.

Another aspect that should be noted is that my results are inconsistent with the idea that the hysteresis effect is elicited by erroneous movements. In fact, if errors had produced a hysteresis effect, the motor system would have perseverated in implementing an erroneous movement kinematics on the following trial, increasing the probability of new errors. However, we did not find any evidence of a post error decrease of accuracy. This may suggest that error reactivity processes lead to an updating of action plan which overwrites any detrimental effect of preceding motor events.

5. EXPERIMENT 2: POST-ERROR SLOWING IN REACHING

The results of Experiment 1 (Chapter 4) left open the question of whether error-reactivity processes are confined at the level of the grasping component, or whether error-reactivity impacts on the component directly involved in error. To investigate this aspect in the present experiment participants performed two conditions, namely a reach-to-grasp and a reaching condition. The reach-to-grasp condition was the same as for Experiment 1. For the reaching condition, the task required the participants to reach out and touch the wooden support with their fist, without knocking it over. In this experiment, the accuracy of the reaching component was examined not only in terms of error-rates, but also in terms of submovements' organization and endpoint variability. With respect to submovements, previous studies have reported that reaching movements consist of two consecutive phases: a primary submovement, and a homing-in phase (e.g., Elliott, Helsen, & Chua, 2001; Khan & Franks, 2003; Meyer et al., 1988). The primary submovement is represented by a bell-shaped velocity profile, and it has been interpreted as a ballistic movement portion driven by the initial control plan. The inaccuracy of the initial control plan can be adjusted during the homing-in phase, at the time proprioceptive and visual feedback are used to reduce any spatial difference between hand and target positions. This is achieved by means of corrective adjustments, termed as *secondary submovements*. The production of secondary submovements is considered as one of the major mechanisms of movement accuracy regulation (e.g., Houk et al., 2007; Novak, Miller, & Houk, 2002). Endpoint variability of the reaching movement measures the variability of wrist position at the end of the reaching movement (Gordon, Ghilardi, Cooper, & Ghez, 1994; Eliasson, Rosblad, & Forssberrg, 2004). The range of the distribution of movement's endpoints reflects the precision of the reaching movement (e.g., Eliasson et al., 2004).

At the level of motor preparation, I expected to find a PES for both the reach to grasp and the reaching conditions as for Experiment 1. With respect to motor execution, if the influence of error-reactivity processes is only evident at the level of the grasping component, then the results for the reach-to-grasp condition should be the same as those obtained for Experiment 1. If error reactivity processes strategically slow down the component directly involved in error commission, then kinematic parameterization for the reaching condition should be modulated by PES effects. Otherwise, if reaching is immune to PES effects, then no changes at the level of kinematics (including the endpoint variability of the reaching movement and the number of submovements) and error-rates should be present.

5.1. Method

5.1.1. Participants

A total of 15 subjects participated in the experiment, eleven women and four men, with a mean age of 23.1 years (SD = 2.8). None of them had participated in Experiment 1.

5.1.2. Procedure, design, data processing

In the present experiment, two conditions were performed: a reach-to-grasp condition and a reaching condition. The reach-to-grasp condition was the same as for Experiment 1. Procedure and sequence of events for the reaching condition were the same as for the reach-to-grasp condition except that participants were asked to reach out and touch the wooden support with their fist, hitting with the metacarpophalangeal index joint a target area (1 *X* 3 cm) placed 3 cm from the top of the support (see Figure 13).

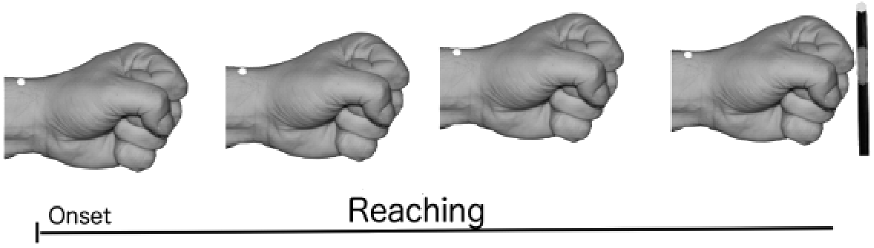

Figure 13. Schematic representation of the reach to-grasp movement and markers' positioning upon the anatomical landmark of interest.

Participants had to execute the movement as fast and accurately as possible, without knocking the wooden support over. Each subject performed a total of 240 trials. The two experimental conditions (reach-to-grasp and reaching) were presented in blocks of 40 trials. Order of blocks was counterbalanced across participants. The experimental session was preceded by 20 practice trials. The experiment lasted approximately 100 minutes. RTs and error-rates were computed as for Experiment 1.

For the determination of whether in post error and post correct trials the reaching component differed with regard to the total number of secondary movements, I performed a submovements analysis. The end of the primary submovement was identified by the first of any of the following secondary submovements (see Meyer et al., 1988, Figure 14): reversals in the trajectory (Type 1 submovement), defined as a zero-crossing from positive to negative value occurred in the velocity profile; reaccelerations towards the target (Type 2 submovement), defined as a zero-crossing from negative to positive value occurred in the acceleration profile; decreases in the rate of deceleration (Type 3 submovement), defined as a zero-crossing from positive to negative value appeared in the jerk profile. Only secondary movements emerging during the deceleration phase were considered, because corrective adjustments usually emerge in close proximity to the target (Fradet, Lee, & Dounskaia, 2008).

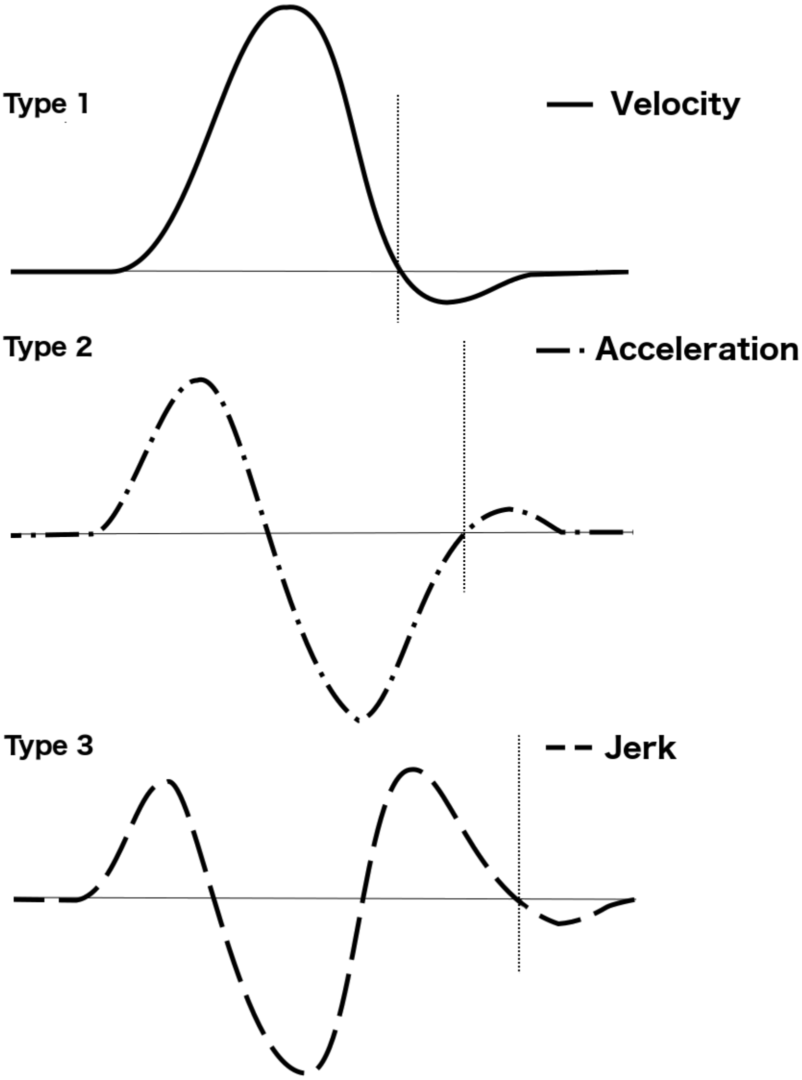

Figure 14. Examples of discrete movements with secondary submovements of type 1, 2, and 3. The vertical line represents the hypothetical end of the primary submovement.

The endpoint variability of the reaching movement was defined as the mean Euclidean distance between the endpoint of each trial and the overall average endpoint position of each participant (Darling & Gilchrist, 1991; Rossetti, 1994).

For the reach-to-grasp condition, I extracted the same dependent kinematic variables as for Experiment 1, whereas for the reaching condition, I considered the reaching time, the time and the amplitude of peak wrist velocity, the time and the amplitude of peak wrist deceleration. Furthermore, for both conditions, the reaching component was also investigated considering *the primary submovement length* (distance covered in the primary submovement, calculated as a percentage of the total movement length), and *the homing-in phase duration* (the time from the end of the primary submovement to the end of the entire movement).

5.1.3. Principal component analysis

In order to simplify data interpretation, I performed a PCA (with Oblimin rotation) for each condition. For the reach-to-grasp condition four components were selected (Table 2), namely a global descriptor of the reach-to-grasp timing (comprising the total movement time, the reaching time, the grasping time, the time of peak grip aperture and the time of peak wrist deceleration), a reaching kinematics component (the amplitude of peak wrist velocity and the amplitude of peak wrist deceleration showed a positive correlation with the second component), a grasping submovements component (including the primary submovement length and the homing-in phase duration), and a gripping kinematics component (including the amplitude of maximum opening velocity and the amplitude of maximum closing velocity). The four selected components accounted for 78% of the variance (33%, 17%, 14%, 14% respectively). The four components were correlated with each other, ranging from .04 to -.26.

Table 2. Weights of the kinematic parameters for the first four components of the reach to grasp condition.

	Component 1	Component 2	Component 3	Component 4
Movement time (ms)	.963	-.005	-.009	-.007
Grasping time (ms)	.823	.102	.086	-.119
Reaching time (ms)	.957	.006	.025	-.013
Time of peak grip aperture (ms)	.641	-.143	-.141	-.116
Time of peak wrist deceleration (ms)	.610	-.283	-.117	.083
Maximum opening velocity (mm/s)	.045	.043	.049	.893
Maximum closing velocity (mm/s)	-.166	-.036	-.041	.799
Peak wrist deceleration (mm/s^2)	.140	.959	-.083	.050
Peak wrist velocity (mm/s)	-.182	.900	.067	-.039
Homing-in phase duration (ms)	.227	-.008	.869	.092
Primary submovement length (%)	-.239	-.027	.845	-.070

For the reaching condition, three components were selected (Table 3), which accounted for 79% of variance (31%, 27%, 21%, respectively). The first component, comprising the reaching time, the time of maximum peak wrist deceleration, and the time of maximum peak wrist velocity, can be interpreted as a global descriptor of the *reaching timing.* The amplitude of peak wrist velocity and the amplitude of peak wrist deceleration showed a strong correlation with the second component, and can be considered as a *reaching kinematics* component. Finally, the primary submovement length and the homing-in phase duration weighted substantially on the third component, suggesting that it can be interpreted as a *reaching submovements* component. The three components were correlated with each other, ranged from -.02 to .21.

Table 3. Weights of the kinematic parameters for the first three components of the reaching condition.

	Component 1	Component 2	Component 3
Reaching time (ms)	.708	-.039	-.088
Time of maximum peak wrist velocity (ms)	.864	.033	.143
Time of maximum peak wrist deceleration (ms)	.805	-.067	-.100
Peak wrist deceleration (mm/s^2)	.063	.983	-.027
Peak wrist velocity (mm/s)	-.057	-.955	.019
Primary submovement length (%)	.320	-.029	.855
Homing-in phase duration (ms)	-.359	.021	.826

5.1.4. Data analysis

The same approach to data analysis and model comparison adopted for Experiment 1 was used. Outlier removal led to the discarding of ~1.75 % and ~0.95% of trials from the reach-to-grasp condition and reaching condition, respectively. Because of technical problem, we removed 22 trials from the reach-to-grasp condition and 9 trials from the reaching condition.

5.2. Results

5.2.1. Grasping condition

Accuracy. The overall error percentage was 21.3%. A GLME analysis, with random intercept for Participants and Blocks, showed that subjects were more accurate following an error (17.7% error-rate) than following a correct response (23.1% error-rate), $z = -3.54$, $p < .001$, $OR = 0.58$.

Submovements analysis. The average number of submovements was 2.35 ± 2.12. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), did not revealed any difference between post correct (2.32 ± 2.06) and post error (2.44 ± 2.30) trials in terms of total number of submovements ($\chi^2 = 1.79$, $p = .180$, $BF = 0.09$, $d = 0.05$).

Endpoint variability. The mean of the endpoint variability of the reaching movement was 8.03mm ± 4.52. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), did not show any difference between post correct (8.02mm ± 4.38) and post error (8.05mm ± 4.93) trials in terms of endpoint variability ($\chi^2 = 0.02$, $p = .885$, $BF = 0.09$, $d = 0.01$).

Reaction times. The mean post-correct and post-error RT adjustments are shown in Figure 15. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), revealed a significant difference between post-correct and post-error adjustments $\chi^2 = 37.16$, $p < .001$, $BF > 100$, $d = 0.45$. Contrast analysis showed that when an error was committed, subjects showed a significant post-error slowing ($\mathbf{PES}_{\text{robust}} = 103.55$ms ± 28.21), $t(246) = 3.67$, $p < .001$, $BF = 93.39$, $d = 0.23$. After a correct response, subjects showed a post-correct speeding (-32.93ms ± 8.7), $t(774) = -3.76$, $p < .001$, $BF = 89.18$, $d = 0.13$.

Reach-to-grasp timing. The mean of post-correct and post-error adjustments of the Reach-to-grasp timing are shown in Figure 16A. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), revealed a significant difference between post-correct and post-error adjustments, $\chi^2 = 41.18$, $p < .001$, $BF > 100$, $d = 0.48$. After an error, subjects showed a significant post-error slowing ($\mathbf{PES}_{\text{robust}} = 0.175 \pm 0.044$, $t(235) = 3.95$, $p < .001$, $BF = 259.44$, $d = 0.25$), whereas after a correct trial a post-correct speeding was present (-0.130 ± 0.022), *t* (769) = -5.80, $p < .001$, *BF* > 100, *d* = 0.20.

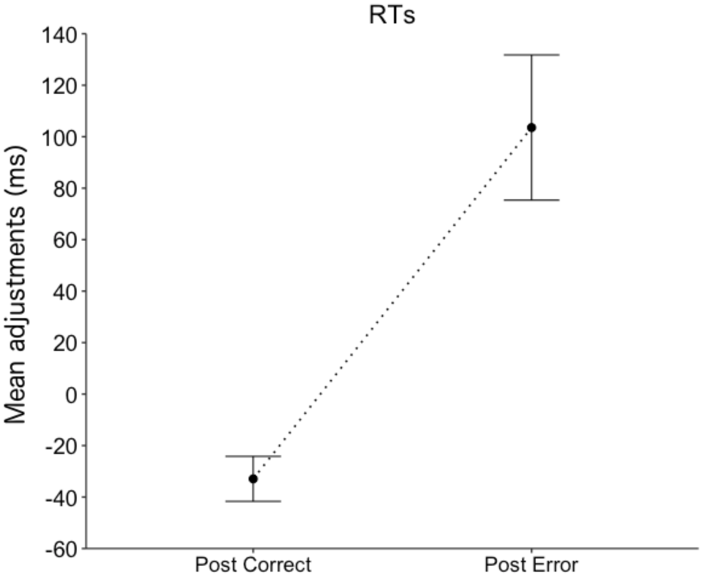

Figure 15. Mean post-correct and post-error adjustments in RTs. Vertical bars indicate standard error of the mean. Positive values indicate a slowing down.

Reaching kinematics. The mean of post-correct and post-error adjustments for the Reaching kinematics component are shown in Figure 16B. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only) did not show any difference between post-correct and post-error adjustments, $\chi^2 = 3.10$, $p = .077$, BF = 0.35, *d* = 0.12. Neither a significant post-error slowing ($\mathbf{PES}_{\text{robust}} = -0.023 \pm 0.024$, *t* (243) = -0.96, *p* = .334, *BF* = 0.03, *d* = 0.06) nor a post-correct adjustment (0.020 ± 0.011, *t* (775) = -1.76, *p* = .078, *BF* = 0.01, *d* = 0.06) were found.

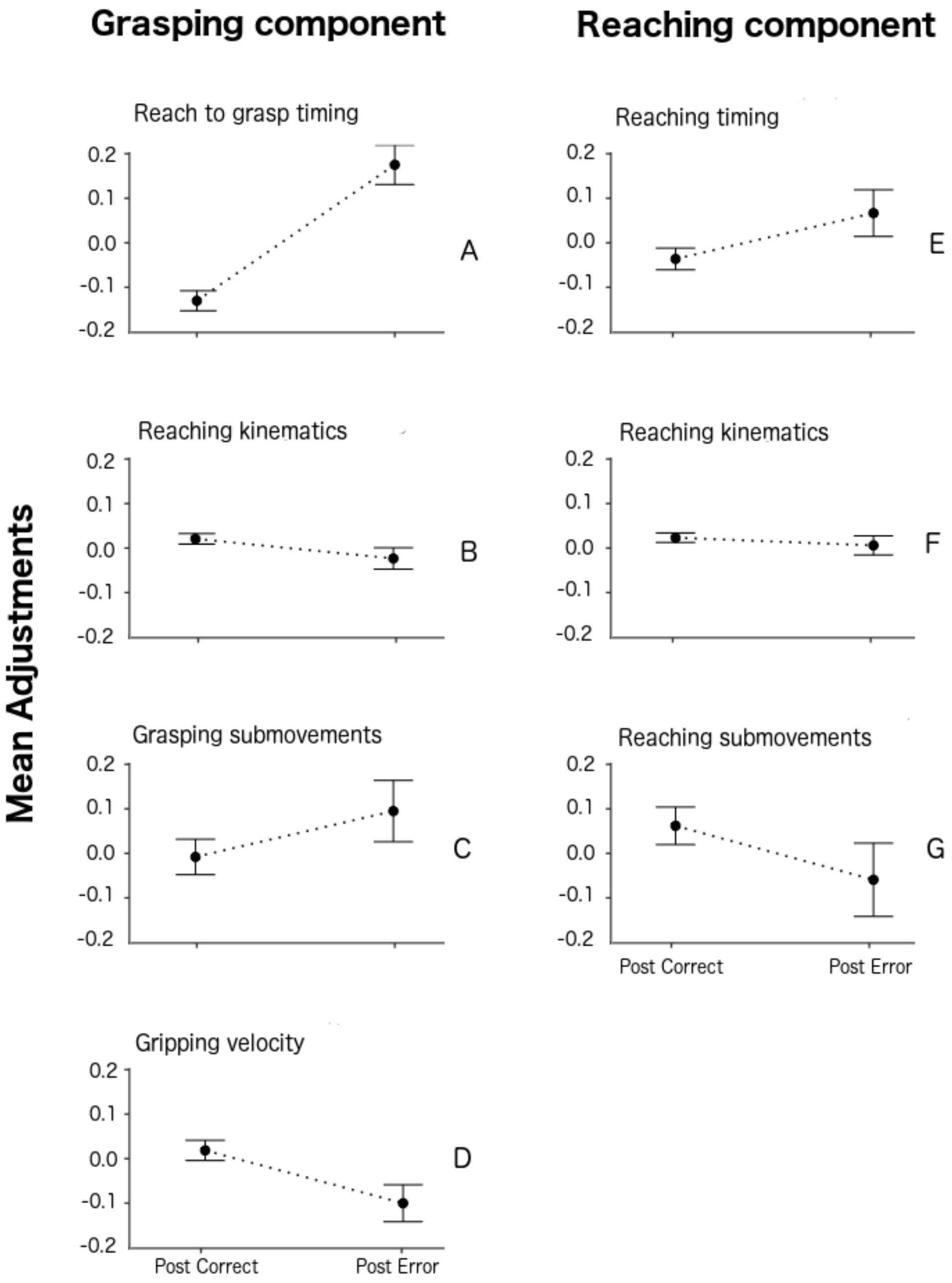

Figure 16. Mean post-correct and post-error adjustments for the kinematic components of the grasping (left panel) and the reaching (right panel) condition. Vertical bars represent standard error of the mean.

Grasping submovements. The mean of post-correct and post-error adjustments of the Grasping submovements component are shown in Figure 16C. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), did not show any difference between post-correct and post-error adjustments $\chi^2 = 1.63$, $p = .201$, $BF = 0.29$, $d = 0.09$. Contrast analysis did not show any evidence of post-error slowing (**PES**$_{robust}$ = 0.094 ± 0.068), $t(245) = 1.37$, $p = .169$, $BF = 0.03$, $d = 0.08$), or post-correct speeding (-0.007 ± 0.039), $t(769) = -0.19$, $p = .841$, $BF = 0.04$, $d = 0.007$).

Gripping velocity. The mean of post-correct and post-error adjustments of the Gripping velocity is shown in Figure 16D. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), revealed a significant difference between post-correct and post-error adjustments, $\chi^2 = 6.49$, $p = .010$, $BF = 3.12$, $d = 0.19$. After an error, subjects showed a significant reduction of gripping velocity (**PES**$_{robust}$ = -0.100 ± 0.041), $t(244) = -2.41$, $p = .016$, $BF = 2.44$, $d = 0.16$. No evidence of post-correct adjustments (0.018 ± 0.023) was found, $t(766) = 0.81$, $p = .413$, $BF = 0.02$, $d = 0.03$.

5.2.2. Reaching Condition

Accuracy. The overall error percentage was 21.5%. A GLME analysis, with random intercept for Participants and Blocks, showed no significant difference of post-error (21% error-rate) and post-correct (19.5% error-rate) accuracy, $z = -0.20$, $p = .840$, $OR = 0.97$.

Submovements analysis. The average number of submovements was 3.08 ± 3.02. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), did not revealed any difference between post correct (3.01 ± 2.80) and post error (2.97 ± 2.76) trials in terms of total number of submovements ($\chi^2 = 0.31$, $p = .572$, $BF = 0.04$, $d = 0.01$).

Endpoint variability. The mean of the endpoint variability of the reaching movement was 8.82mm ± 13.03. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), did not show any difference between post correct (10.75mm ± 6.01) and post error (10.10mm ± 6.64) trials in terms of the endpoint variability ($\chi^2 = 2.49$, $p = .114$, $BF = 0.36$, $d = 0.12$).

Reaction times. The mean post-correct and post-error RT adjustments are shown in Figure 17. A LME analysis, with random intercept for Participants (modeling both slopes and intercepts) and Blocks (modeling intercepts only), revealed a significant difference between post-correct and post-error adjustments, $\chi^2 = 6.61$, $p = .010$, $BF > 100$, $d = 0.41$. Contrast analysis showed a significant post-error slowing ($\mathbf{PES}_{\text{robust}} = 43.11$ms ± 11.88), $t(191) = 3.62$, $p < .001$, $BF = 85.46$, $d = 0.26$. After a correct response, a post-correct speeding (-13.72ms ± 4.60) was present, $t(838) = -2.95$, $p = .003$, $BF = 5.95$, $d = 0.10$.

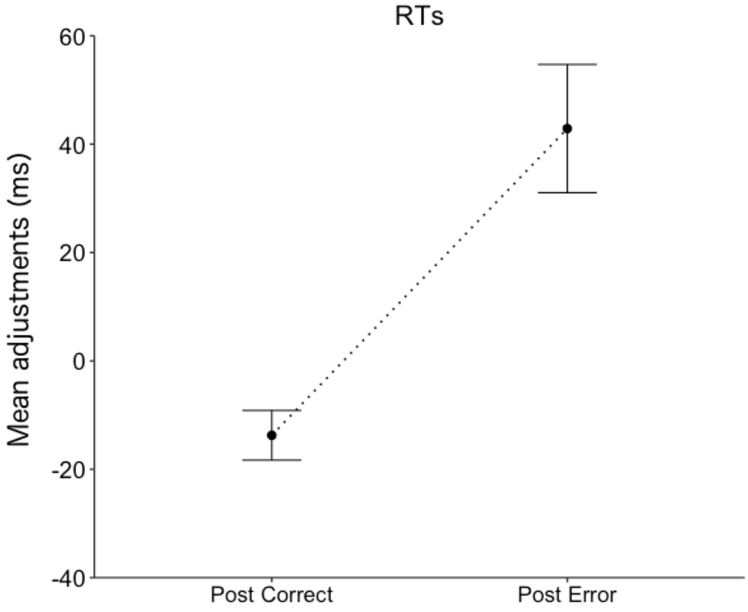

Figure 17. Mean post-correct and post-error adjustments in RTs. Vertical bars indicate standard error of the mean. Positive values indicate a slowing down.

Reaching timing. The mean of post-correct and post-error adjustments of the Reaching timing component are shown in Figure 16E. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), did not show any difference between post-correct and post-error adjustments, $\chi^2 = 3.34$, $p = .067$, $BF = 0.44$, $d = 0.14$. Contrast analysis did not show any evidence of post-error (**PES**$_{\text{robust}} = 0.066 \pm 0.052$, $t(195) = 1.27$, $p = .204$, $BF = 0.31$, $d = 0.09$) or post-correct adjustment (0.036 ± 0.024, $t(836) = -1.48$, $p = .136$, $BF = 0.21$, $d = 0.05$).

Reaching kinematics. The mean of post-correct and post-error adjustments of the Reaching kinematics component are shown in Figure 16F. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), did not show any difference between post-correct and post-error adjustments $\chi^2 = 0.50$, $p = .475$, $BF = 0.11$, $d = 0.05$. Contrast analysis did not show any evidence of post-error slowing

(**PES**$_{robust}$ = 0.006 ± 0.021), t(197) = 0.28, p = .778, BF = 0.10, d = 0.02), whereas a post-correct speeding was present (0.023 ± 0.010), t(842) = -2.19, p = .028, BF = 0.01, d = 0.07).

Reaching Submovements. The mean of post-correct and post-error adjustments of the Reaching submovements component are shown in Figure 16G. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), did not show any difference between post-correct and post-error adjustments $\chi^2 = 1.60$, p = .205, BF = 0.18, d = 0.10. Contrast analysis did not show any evidence of post-error slowing (**PES**$_{robust}$ = -0.059 ± 0.082), t(195) = -0.72, p = .472, BF = 0.15, d = 0.05), or post-correct speeding (0.061 ± 0.042), t(836) = 1.46, p = .142, BF = 0.21, d = 0.05).

5.3. Discussion

The core aim of the present study was to investigate whether the immunity of the reaching component from error reactivity effects is also present in an “only reaching” task. With respect to the reach to grasp condition the results mirrored those obtained for Experiment 1, in the sense that the failure to reach out and grasp an object slows down the temporal aspects of the subsequent action with specific reference to the grasping component, without affecting the reaching component. For the reaching condition, I found a PES effect at the level of RT, but however, the motor execution of reaching movement resulted impermeable to error-reactivity effects. This statement is supported by error rates, endpoint variability and submovements analyses. Such results strengthen the hypothesis that error-reactivity effects extend to motor execution, but seem to be limited to the grasping component.

5.3.1. The motor preparation phase

The results of the present study showed that, at the level of RTs, the PES effect is present in both the reach to grasp and the reaching condition. However, only the grasping component seems to benefit of the adaptive component of error reactivity, in terms of accuracy. Conversely, for the reaching component, no evidence of post error improvements of accuracy was found. In fact, I did not find any difference between post error and post correct trial with respect to error-rates, number of submovements and endpoint variability, which suggests that the reaching accuracy regulation is not influenced by error-reactivity effects.

For the reach to grasp condition of the present experiment, I found a larger PES in RT than in Experiment 1. This result may depend on the fact that Experiment 2 lasted much longer than Experiment 1, and besides, grasping conditions and reaching conditions were interleaved. A variety of studies have reported that PES is influenced by the control demands of the task (e.g., Hogan, Vargha-Khadem, Kirkham, & Baldeweg, 2005; Schroder, Moran, Infantolino, & Moser, 2013; Regev & Meiran, 2014). For instance, Schroder and colleagues (2013) manipulated cognitive demands in a flanker task by reversing stimulus-response mappings between blocks. They found a larger PES in the more-demanding S-R reversal blocks than in nonreversal blocks. Our results are in line with previous literature suggesting that task demands play an important role in PES emergence.

5.3.2. The motor execution phase

At the level of motor execution, for the reach to grasp condition, results mirrored those of Experiment 1. Coherently, no post error adjustments were found for the reaching condition. These results support the *Visual Channel Hypothesis* (VCH) by Jeannerod (1981). According to the VCH the visuomotor mechanisms responsible for prehension are structured in a modular way. The “reaching channel” extracts from the visual world information on the spatial location of the objects and activates those muscles which are relevant to carry the hand to the object location. The “grasping channel” extracts information concerning the size and shape of the object and transforms it in the activation of distal muscles relevant to grasp the object. The VCH has been challenged on multiple fronts (see section 2.4), however the very fact that I found error-reactivity effects only for the grasping component speaks in favor of a modular organization of the reach to grasp movement. These results will be discussed more fully in the General Discussion (Chapter 8).

6. EXPERIMENT 3: POST-ERROR SLOWING: A MULTIMODAL TECHNIQUES STUDY

The functional accounts of PES suggest that error commission leads to the implementation of strategic adjustments by inhibiting the activation of the motor system in order to buy more time and enable more controlled responding (Ridderinkhof et al., 2002). According to the inhibitory account, errors increase the strength of selective suppression on the post-error trial (Marco-Pallares et al., 2008) by activating a right hemispheric network including the pre-SMA, IFC and STN, associated with motor stopping and motor inhibition (Aron et al., 2007). Similarly, the CMT holds that an error-monitoring system, based on the activity of the ACC, triggers cognitive control processes by activating the rDLPFC, which decreases activity in the response priming unit (Botvinick et al., 2001).

Although functional accounts share the idea that the motor system is biased by error commission, this hypothesis has never been tested. To examine this aspect, I investigated whether the excitability of the cortical spinal tract (CST) is influenced by the correctness of the previous motor act by means of the Transcranial Magnetic Stimulation (TMS). TMS is a non-invasive method for the stimulation of the nervous system. Through electromagnetic induction delivered by a stimulation coil, TMS can produce a transient and non-invasive depolarization of neurons, with the electric current passing across the scalp and skull without physical contact. The stimulation of M1 by means of TMS generates motor-evoked potentials (MEPs) that can be recorded from peripheral contralateral muscles with electromyography (EMG). The amplitude of the elicited MEP has been considered as a marker of excitability of the CST (Priori et al., 1998). In the present experiment, I stimulated M1 with single Transcranial Magnetic Stimulation pulses (spTMS) and compared MEPs after correct and incorrect reach to grasp movements. I hypothesized that if the activity of the motor system during the preparation of the movement is decreased by error commission, then I should find evidence for a decrement of MEPs amplitude after an error. The spTMS was coupled with kinematical analysis in order to investigate both the added benefit of monitoring and occasionally adjusting motor programs in flight (Erlhagen & Schöner, 2002) and the potential correlation of the CST activity with the behavioral measures of the reach to grasp movement.

6.1. Method

6.1.1. Participants

A total of 20 subjects (mean age = 22.3 years, SD = 2.25 years, 12 males) were recruited from the student population of the University of Padua. None of them had participated in the previous experiments. All participants were screened for TMS exclusion criteria and for neurological, psychiatric, and medical problems (Wassermann, 1998; Rossi et al., 2009).

6.1.2. Procedure

Before and after the experimental session, the baseline CST excitability was assessed by acquiring 15 MEPs while participants passively stared a white fixation cross, positioned at the edge of the table. In order to check for any changes related to the TMS per se, I compared the two baseline period (30 MEPs in total). The average amplitude of the two series was then utilized to compute participant’s individual baseline for data normalization procedure.

The experimental setting is represented in Figure 18A. In the present experiment, the task was identical to that reported in Experiment 1 (paragraph 4.1.2) except for what follows. Before each trial participants were asked to close their eyes and to hold their hand, as relaxed as possible, upon the starting switch. The sound of the TMS pulse served as a “Go” signal, after which participants were asked to open their eyes, reach out and grasp the steel ball by means of a precision grip, lift it and deposit it into the container. The task required executing the movement as fast and accurately as possible, without knocking the wooden support over. The inter-trial interval varied randomly from 6500 to 8500 ms. Each experimental session consisted of 180 trials. Participants underwent a practice session of 10 trials before the experimental session began. The experiment lasted approximately 200 minutes.

6.1.3. Transcranial magnetic stimulation

Single-pulse TMS (pulse characteristics: 100 µs rise time, 1 ms duration) were delivered on the participant’s left M1, in correspondence with the right representation of the first dorsal interosseous muscle (FDI) (Figure 18C), by using a 70 mm figure-of-eight coil connected to a Magstim Bistim2 stimulator (Magstim Co., Whitland, United Kingdom). The coil was positioned on the head with a 45° angle with respect to the inter-hemispheric fissure (Figure 18B). The optimal scalp location (OSL), was selected by stimulating the presumed area of FDI until which the minimum TMS stimulation elicited the maximum MEP from the FDI muscle. During the experiment, the coil was held in the correct position by a mechanical arm. Moreover, the experimenters continuously checked the position of the coil with respect to the OSL. For each participant, I computed the resting motor threshold, that is the minimum stimulation intensity at which TMS generate MEPs of at least 50 µV peak- to-peak amplitude in a relaxed muscle in 5 out of 10 consecutive trials (Rossini et al., 1994). During the experimental session, the pulse intensity was set at 120% of resting motor threshold. TMS stimulation was controlled by means of E-Prime V2.0 software (Psychology Software Tools Inc., Pittsburgh, PA, United States).

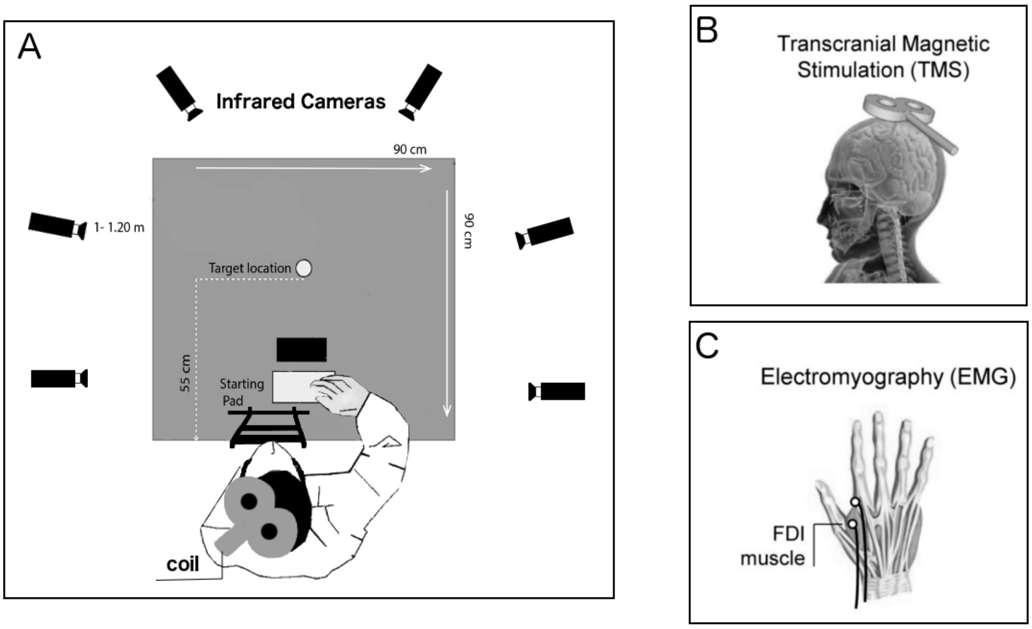

Figure 18. (A) Graphical representation of the experimental set-up. (B) The TMS coil was placed over the participant's left M1. (C) MEPs activity were measured from electrodes placed over the FDI muscles of the right hand.

6.2. Data analysis

6.2.1. MEP data

Individual peak-to-peak MEP amplitudes (mV) were analyzed off-line using Brain Vision Analyzer (Brain Products BmbH, Munich, Germany). The MEP peak-to-peak amplitude for FDI was used to measure participants' CS excitability. I discarded trials in which EMG activity was greater than 100 μV in the 100 ms window preceeding the TMS stimulation. To control for inter-individual variability, MEPs amplitude values obtained during the experimental session were divided by average MEPs amplitude of each participant recorded during the baseline blocks (MEP ratio = MEP obtained / average MEP baseline). MEP ratios were used as dependent variable.

6.2.2. Behavioral data

RT was defined as the time between the TMS Go signal and the release of the starting switch, which corresponds to the movement onset.

Movement onset was calculated as the time at which the tangential velocity of the wrist marker crossed a threshold (5 mm/s) and remained above it for longer than 500 ms. End of movement was defined as the time at which the hand made contact with the target and quantified as the time at which the hand opening velocity crossed a threshold (5 mm/s) after reaching its minimum value and remained under it for longer than 500 ms. The time from the onset to the end of the movement was defined as total movement time. For the grasping component, I considered the amplitude of maximum closing velocity, that is the maximum velocity reached during the closing phase with reference to the distance between the thumb and the index finger. For the reaching component, I considered the amplitude of peak wrist deceleration, that is the maximum velocity of the wrist during the movement time.

6.2.3. Data analysis

The data analysis followed the procedure outlined in paragraph 3.5. Trials outside the range of ± 2.5 standard deviations were discarded (less than 2.9%).

6.3. Results

6.3.1. Behavioral results

Accuracy. The overall error percentage was 15.4%. A GLME analysis, with random intercept for Participants and Blocks, revealed that participant's error rate did not significantly change after an error (21.1%) compared to a correct trial (14.1%), $z = 1.747$, $p = .081$, $OR = 1.24$.

Reaction times. The mean post-correct and post-error RT adjustments are shown in Figure 19A. A LME analysis, with random intercept for Participants (modeling intercepts only), revealed a significant difference between post-correct and post-error adjustments $\chi^2 = 10.115$, $p = .001$, $BF = 8.11$, $d = 0.20$. Contrast analysis showed that the post-error slowing was significantly different from zero ($\mathbf{PES}_{\text{robust}} = 10.61 \pm 4.34$), $t\ (288) = 2.44$, $p = .007$, $BF = 2.45$, $d = 0.15$, whereas, after a correct response, subjects showed a post-correct speeding (-5.58 ± 1.84), $t\,(1959) = -3.025$, $p = .001$, $BF = 4.85$, $d = 0.07$.

Maximum closing velocity. The mean of post-correct and post-error adjustments of the Maximum closing velocity are shown in Figure 19B. A LME analysis, with random intercept for Participants (modeling intercepts only), revealed a significant difference between post-correct and post-error adjustments, $\chi^2 = 11.92$, $p < .001$, $BF > 35.83$, $d = 0.21$. Contrast analysis showed that the post-error slowing was significantly different from zero ($\mathbf{PES}_{\text{robust}} = -24.51 \pm 10.60$), $t\,(324) = -2.313$, $p = .010$, $BF = 1.80$, $d = 0.14$. No evidence of post-correct speeding was found after a correct response (2.18 ± 2.56), $t\ (2091) = 0.852$, $p = .197$, $BF = 0.01$, $d = 0.01$.

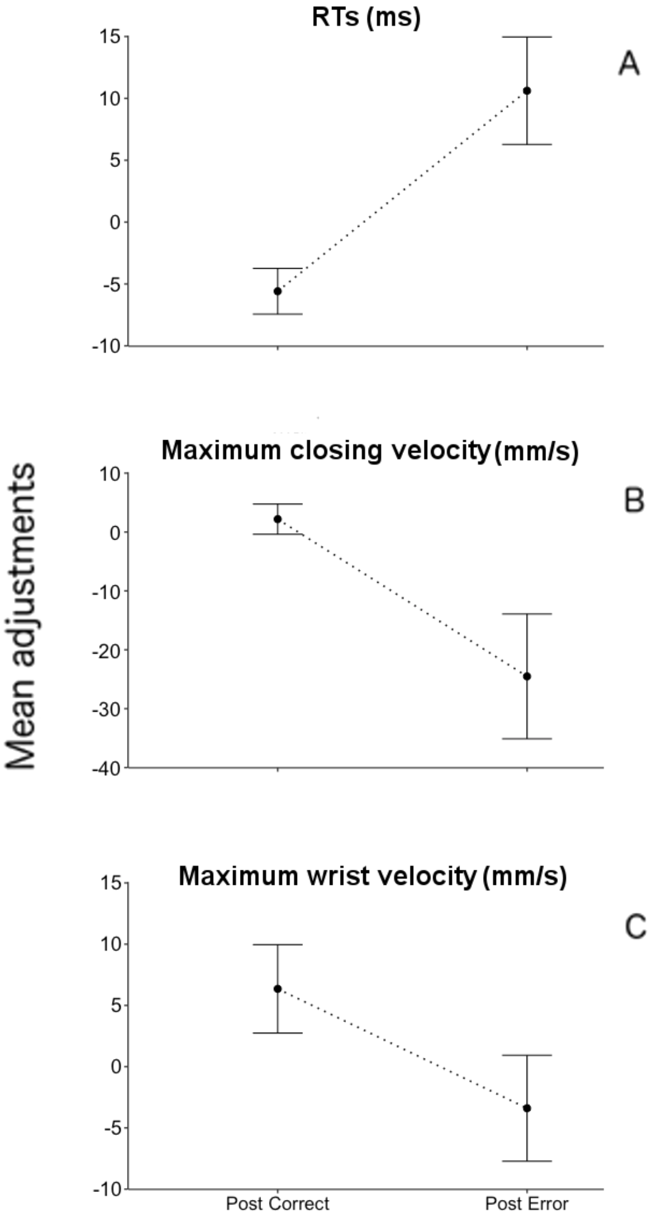

Figure 19. Mean post-correct and post-error adjustments of RTs (panel A), the Maximum closing velocity (panel B), and the Maximum wrist velocity (panel C). Vertical bars represent standard error of the mean.

Maximum wrist velocity. The mean of post-correct and post-error adjustments of the Maximum wrist velocity are shown in Figure 19C. A LME analysis, with random intercept for Participants (modeling intercepts only), revealed no significant difference between post-correct and post-error adjustments, $\chi^2 = 1.10$, $p = .292$, $BF = 0.14$, $d = 0.06$. Contrast analysis showed that the post-error slowing was not significantly different from zero (**PES**$_{\text{robust}}$ = -3.39 ± 4.32, $t(330) = -0.785$, $p = .783$, $BF = 0.31$, $d = 0.04$), whereas a significant post-correct speeding (6.35 ± 3.60, $t(2102) = 1.759$, $p = .039$, $BF = 0.22$, $d = 0.03$) was present after a correct response.

6.3.2. MEPs amplitude

The raw MEP amplitude recorded for each participant at the beginning and at the end of the experimental blocks was not significantly different, $t(503.8) = -0.321$, $p = .747$).

The mean of post-correct and post-error adjustments of the normalized MEPs amplitudes are shown in Figure 20. A LME analysis, with random intercept for Participants (modeling intercepts only), revealed a significant difference between post-correct and post-error adjustments $\chi^2 = 7.39$, $p = .006$, $BF = 2.54$, $d = 0.18$. Contrast analysis showed that the post-error slowing was significantly different from zero (**PES**$_{\text{robust}}$ = -0.178 ± 0.05), $t(261) = -3.212$, $p < .001$, $BF = 18.9$, $d = 0.14$. No evidence of post-correct speeding was found after a correct response (-0.02 ± 0.2), $t(1658) = -0.902$, $p = .183$, $BF = 0.09$, $d = 0.02$.

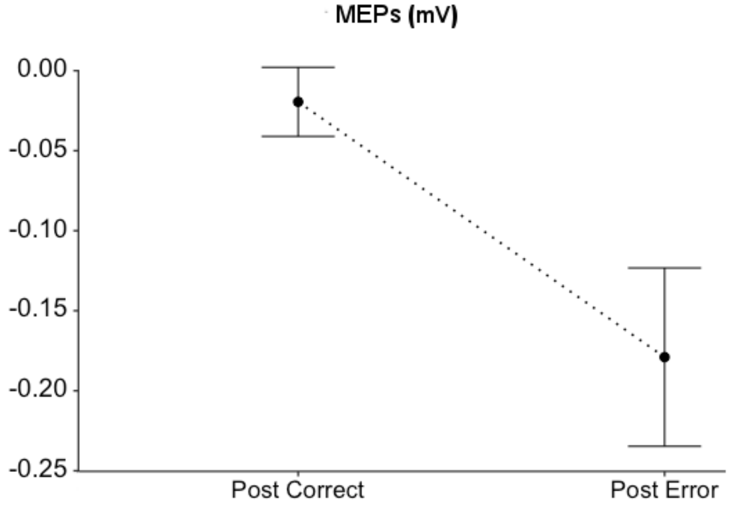

Figure 20. Mean post-correct and post-error adjustments of MEPs amplitudes (normalized). Vertical bars indicate standard error of the mean. Negative values indicate a reduction of MEPs amplitude.

6.3.3. Correlations between MEPs amplitude and kinematics

No correlations emerged when correlating post-error MEP amplitudes with post error RTs ($r = -.10$, $p = .114$), Maximum closing velocity ($r = -.06$, $p = .316$) and Maximum wrist velocity ($r = .001$, $p = .315$).

6.4. Discussion

The goal of the present study was to investigate if the of excitability of the CST is modulated by performance accuracy of the previous trial. Results extended previous literature by demonstrating that the CST activity is reduced after an error, supporting the “functional” idea that the processing of an error leads to an inhibition of the motor system.

In line with Experiments 1 and 2, behavioral results showed that error reactivity effects are not confined to the motor preparation of the reach to grasp movement, but they also extend to the grasping component. Indeed, I found that errors not only lead to slowdown of the RTs, but they also impact on the Maximum Closing Velocity, whereas the Maximum wrist velocity seems to be impermeable to error after-effects.

As concerns the CST activity related to FDI muscle, results showed a decrease of MEPs amplitude after an error, which suggests that the inhibitory circuits in M1 may contribute to the implementation of post error adjustments. Indeed, according to the inhibitory account (Ridderinkhof, 2002), the error monitoring system implements post-error adjustments by inhibiting the primary motor cortex (M1). As a result, motor activation will reach response threshold later, leading to longer RTs after an error. Similarly, the CMT (Botvinick et al., 2001), suggests that the inhibition of the CST activity is trigger by a conflict monitoring system involving the DLPFC and the ACC. Previous literature supports the hypothesis that the PES effect depends on a decrease of motor system activity (see section 1.5.1). For instance, Marco-Pallares and colleagues (2008) examined oscillatory electrical brain activity in a flanker task, showing that the slowdown of RTs after an error is correlated with an increased beta band power, traditionally considered a marker of motor inhibition and motor stopping (Kuhn et al., 2004; Marco-Pallares et al., 2008). Spruit, Wilderjans and van Steenbergen (2018) investigated how the cardiac effort (CE), that is the product of heart rate and systolic blood pressure, changes after an error. Results showed that the CE tends to increase after an error and correlates with the PES amplitude. Since the CE is thought to reflect inhibitory processes that contribute to suppress irrelevant information, the result of this study seems to support the idea that inhibition processes play a key-role in the implementation of the adaptive changes following errors.

Crucially, in the present study I did not find a PIA. A potential explanation of this result may reside in the fact that the experiment duration was longer than in the previous experiments and this may have affected the motivation of participants. The influence of motivation on error reactivity processes is well documented. Some studies found that the ERN increases when errors imply a monetary loss (Maruo, Schacht, Sommer, & Masaki, 2016; Stürmer et al., 2011). Maruo and colleagues (2011) investigated the effect of motivation on error-reactivity using a spatial Stroop task. A fixation cross was presented in the center of a screen and an arrow, pointing down or up, was shown above or below it. Participants were asked to indicate the pointing direction of the arrow, ignoring its location. In the reward condition, correct responses were rewarded with a small amount of money, whereas in the neutral condition participants' performance was not incentivized. Results showed an increased ERN amplitude in the reward condition respect to the neutral condition, indicating that motivational aspects play an important role in error-reactivity. Using a similar task, Stürmer and colleagues (2011) found that PES was stronger in the monetary reward condition than in the control condition. The present experiment lasted about 3 hours and this may have questioned the full implementation of post-error adjustments. This hypothesis is also supported by the lack of correlation between the amplitude of MEPs and post-error adjustments. Moreover, it is noteworthy that the results of the present experiment are weaker than those found in Experiments 1 and 2, in terms of effect size. Therefore, further research is needed to better clarify how the post-error reduction of MEPs contributes to the implementation of post-error adjustments.

7. EXPERIMENT 4: YOUR ERROR IN MY HAND: AN INVESTIGATION OF OBSERVATIONAL POST-ERROR SLOWING

For human beings, monitoring others’ errors is essential for efficient goal-directed behavior. Indeed, the mere observation of other individuals' errors provides a rich source of information that can be used to avoid potential errors and improve our performance without direct experience. Recently, some evoked related potential (ERP) studies have shown that when participants monitor errors made by other individuals a negative-going ERP component, similar to the ERN, can be recorded in the observer (oERN; Bates, Patel, & Liddle, 2005; van Schie et al., 2004). The neural generator of the oERN is supposed to be the anterior cingulate cortex (ACC) which also gives rise to the ERN (van Schie et al., 2004). In addition to electrophysiological studies, some behavioral investigations (Schuch & Tipper, 2007) pointed out that the PES effect is also present after error observation (observational PES; oPES). These results support the hypothesis about the existence of a *‘mirror’ error-monitor system*, which may be a key mechanism for the acquisition of learned behaviors on the basis of the actions of others (Schuch & Tipper, 2007). Recent studies, however, have questioned the idea that the oPES is the consequence of simulation of the other person’s error processing (Schuch & Tipper, 2007). For instance, Castellar and colleagues (2011) showed that the oPES is also present when participants were asked to monitor a computer’s performance, demonstrating that the slowing after an observed error occurs even when neither visible behavior to imitate nor high-level processes can be simulated.

To date, the oPES has been exclusively investigated by means of speeded reaction time (RT) tasks, measuring arbitrary button-press responses. With this in mind, the aim of the present study is twofold. To examine the oPES in the context of realistic goal-directed movements and to explore, for the first time, kinematical reorganization following the observation of an erroneous movement. The task required that participants and a confederate alternated in reaching out and grasping a steel sphere positioned on a wooden support. They were asked to perform the movement as naturally and accurately as possible, without knocking the wooden support over (i.e., the error). If oPES is not only evident at pre-movement level, but also during action execution, then I expected changes in kinematic parameterization as to reduce the probability of making an error in subsequent movements.

7.1. Methods

7.1.1. Participants

Fourteen participants (mean age = 22.8 years, SD = 3.05 years, 8 males) were recruited from the student population of the University of Padua. I did not perform a power analysis to determine the sample size, because I employed an original task which, for the first time, allows to determine the influence of error observation on kinematics. Thus, running a power analysis is not recommended because it may lead to arbitrary decisions concerning sample size.

7.1.2. Data processing

RT was defined as the time between the opening of the shutter glasses and the release of the starting switch). Movement onset was defined from the tangential speed of the wrist marker using a threshold of 5 mm/s. Movement offset was defined as the time at which the hand made contact with the target and it was quantified as the time at which the hand opening velocity crossed a threshold (5 mm/s) after reaching its minimum value and remained above it for longer than 500 ms.

As dependent variable, in addition to RTs, I considered the maximum height of the wrist trajectory from the working surface. As previously demonstrated, this measure shows consistent differences between situations in which two agents act within the same working space and situations in which agents act in isolation (Georgiou, Becchio, Glover, & Castiello, 2006; Becchio, Sartori, Bulgheroni, & Castiello, 2008; Quesque, Lewkowicz, Delevoye-Turrell, & Coello, 2013). For instance, wrist elevation is modulated according to the type of social interaction occurring between participants and by the level of involvement (e.g., action observation, cooperation, competition). And, of relevance for the present study, higher arm trajectories are evident when people share a reachable space independently from whether the aim to interact is made explicit or not (e.g., Quesque et al., 2013). Furthermore, it should be noted that the modulation of wrist elevation might represent a specific strategy aimed at improving the subsequent performance. An increase in hand elevation may ensure that fingers do not collide with the target or the support – as in our circumstances – during movement execution (Smeets & Brenner, 1999), and may lengthen the time window within which contact points can be established more precisely and firmly (Glover, 2004).

7.1.3. Procedure and Design

The experimental setting is represented in Figure 21. For each experimental session, two participants came into the lab and were introduced to each other. After receiving the instructions, they sat at a table in front of each other.

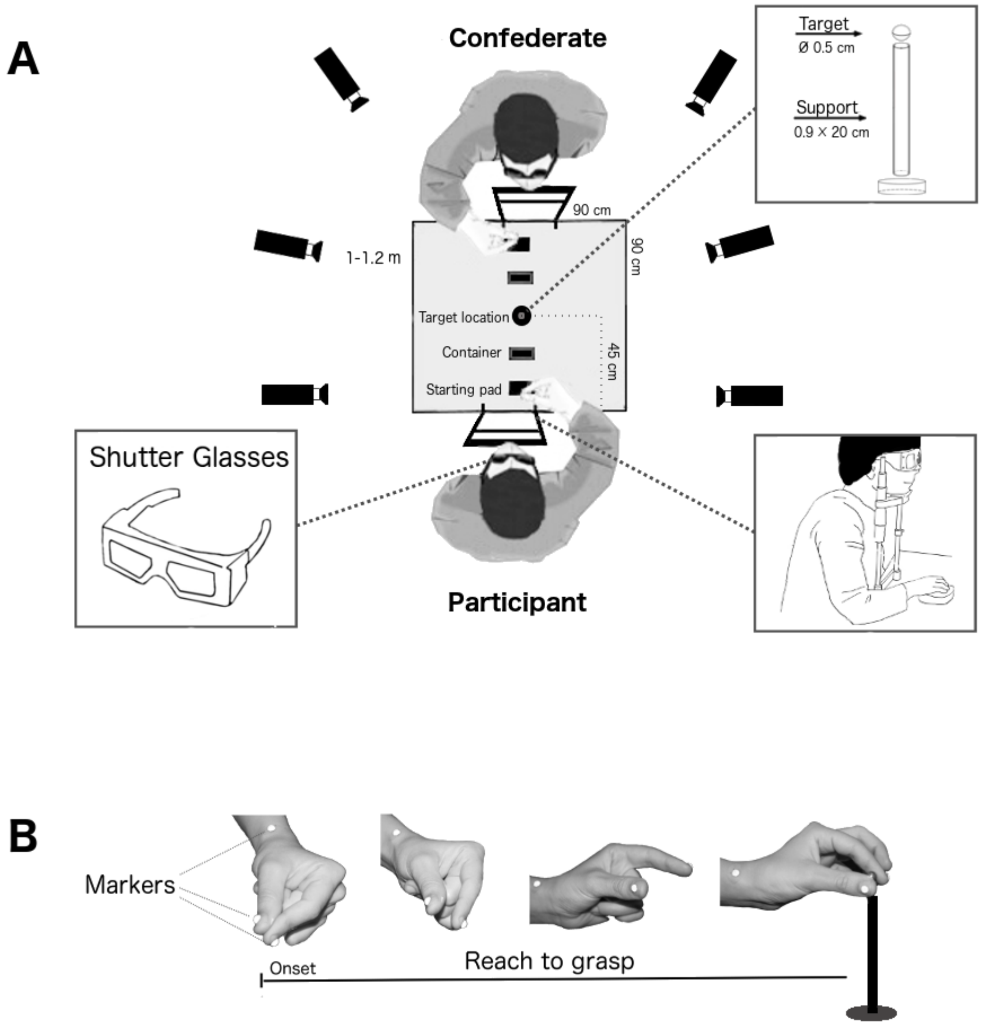

Figure 21. (A) Graphical representation of the experimental set-up. (B) Schematic representation of the reach to-grasp movement and markers' positioning upon the anatomical landmark of interest.

Unbeknown to the real participant, the other participant was a confederate. Spontaneous reports provided in post-experimental interviews indicated that participants were completely unaware that the partner was a confederate. Prior to each trial, the shutter goggles wore by the participants and the confederate were set in their opaque state. Both held the ulnar side of the hand upon a starting switch, with the thumb and index finger pinched together (Figure 21B). As soon as the shutter goggles opened, one of the two agents were asked to observe the other reaching out and grasping the steel sphere by means of a precision grip (PG; Figure 21B) and deposit it into the container. Then, on the following trial, the agent who previously observed the movement performed the action (i.e., execution trials). The 'observation' and the 'execution' trials were administered in an alternated fashion. The request for the 'execution' trials was to perform the movement as naturally and accurately as possible, to avoid dropping the wooden support. If the wooden support fell down the trial was considered incorrect. An experimenter put the steel ball (and the wooden support in the case of error) back in the correct position during the inter-trial interval, which varied randomly from 6500 to 8500 ms. On the basis of a predetermined random sequence the confederate made an error rate of about 15% of the total number of trials. The confederate was covertly signaled by the experimenter when to make an error. Each session consisted of 3 blocks of 80 trials (40 execution trials and 40 observation trials) separated by a 3-min break. Participants underwent a practice session of 20 trials before the experimental session began. The experiment lasted approximately 90 minutes.

7.1.4. Data Analysis

The data analysis followed the procedure outlined in paragraph 3.5. Given the nature of the experimental manipulation, kinematic data were recorded and analyzed only for the 'real' participant. Post-error adjustments were computed using the robust method ($\mathbf{PEA}_{\text{robust}}$) proposed by Dutilh and colleagues (2012), that is by comparing the participants' performance on trials following (E + 1) and immediately preceding (E – 1) a confederate's error. For comparison purpose, in a similar manner to $\mathbf{PEA}_{\text{robust}}$, I also computed post-correct adjustments ($\mathbf{PCA}_{\text{robust}}$) considering triplets of correct trials.

7.2. Results

7.2.1. Post-error adjustments after observing an error

Accuracy. A GLME analysis, with random intercept for Participants and Blocks, revealed that participant's error rate did not significantly change after observing an error (15.7%) compared to after observing a correct trial (12.5%), $z = 1.604$, $p = .109$, $OR = 1.32$.

Reaction Times. The mean post-correct and post-error RT adjustments are shown in Figure 22A. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), revealed a significant difference between post-correct ($\mathbf{PCA}_{\text{robust}} = -9.94$ ms ± 5.62) and post-error ($\mathbf{PEA}_{\text{robust}} = 36.85$ ms ± 13.85) adjustments, $\chi^2 = 10.56$, $p = .001$, BF = 14.8, $d = 0.29$. Contrast analysis showed that $\mathbf{PEA}_{\text{robust}}$ was significantly different from zero, $t\ (956) = 2.79$, $p = 0.005$, $BF = 3.80$, $d = 0.21$.

Maximum Height of The Trajectory. The mean of post-correct and post-error adjustments of the maximum height of trajectory are shown in Figure 22B. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), revealed a significance difference between post-correct ($\mathbf{PCA}_{\text{robust}} = -0.64$ mm ± 0.27) and post-error adjustments ($\mathbf{PEA}_{\text{robust}} = 2.01$ mm ± 0.61), $\chi^2 = 14.503$, $p < .001$, $BF = 97.87$, $d = 0.33$. Contrast analysis showed that $\mathbf{PEA}_{\text{robust}}$ was significantly different from zero, $t\ (982) = 3.16$, $p = 0.001$, $BF = 10.34$, $d = 0.26$.

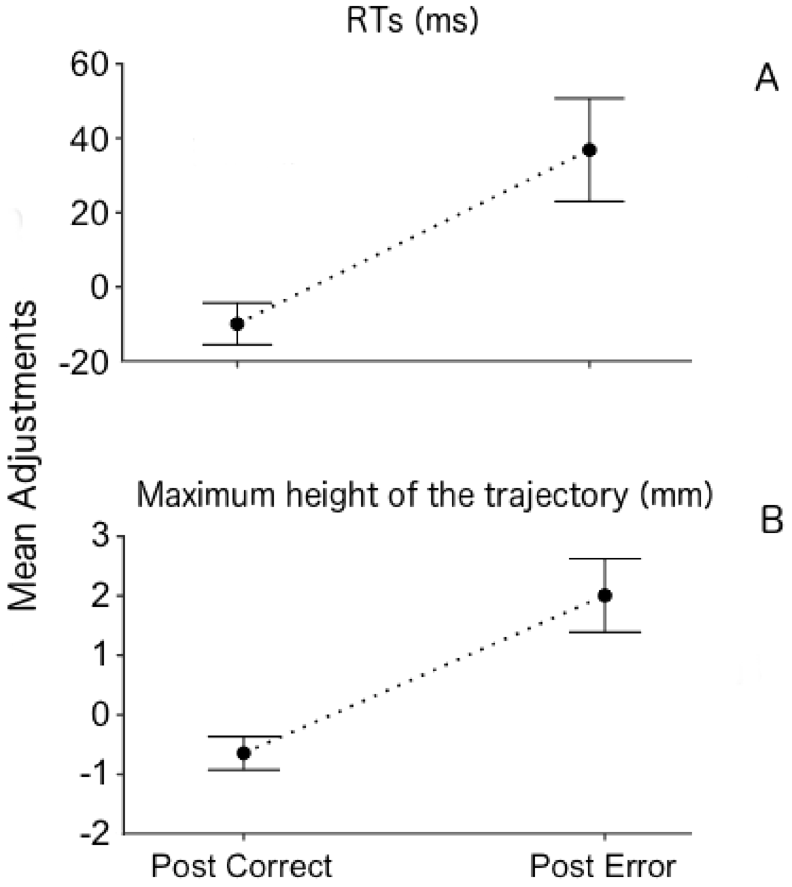

Figure 22. Mean post-correct and post-error adjustments in RTs (panel A) and the maximum height of the trajectory (panel B). For RTs, positive values indicate a post-error slowing. For the maximum height of the trajectory, positive values indicate a post-error increase of the amplitude of maximum height reached by the wrist. Vertical bars indicate standard error of the mean.

7.2.2. Post-error adjustments after own errors

Accuracy. The overall error percentage was 20.3%. A GLME analysis, with random intercept for Participants and Blocks, revealed that participant's error rate did not significantly change after an error (24.2%) compared to a correct trial (19.3%), $z = 0.583$, $p = .560$, $OR = 1.09$.

Reaction Times. The mean post-correct and post-error RT adjustments are shown in Figure 23A. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), did not show any significant difference between post-correct (**PCA**$_{robust}$ = -5.69 ± 5.59) and post-error (**PEA**$_{robust}$ = 5.78 ± 10.85) adjustments, χ^2 = 0.79, p = .373, BF = 0.14, d = 0.07.

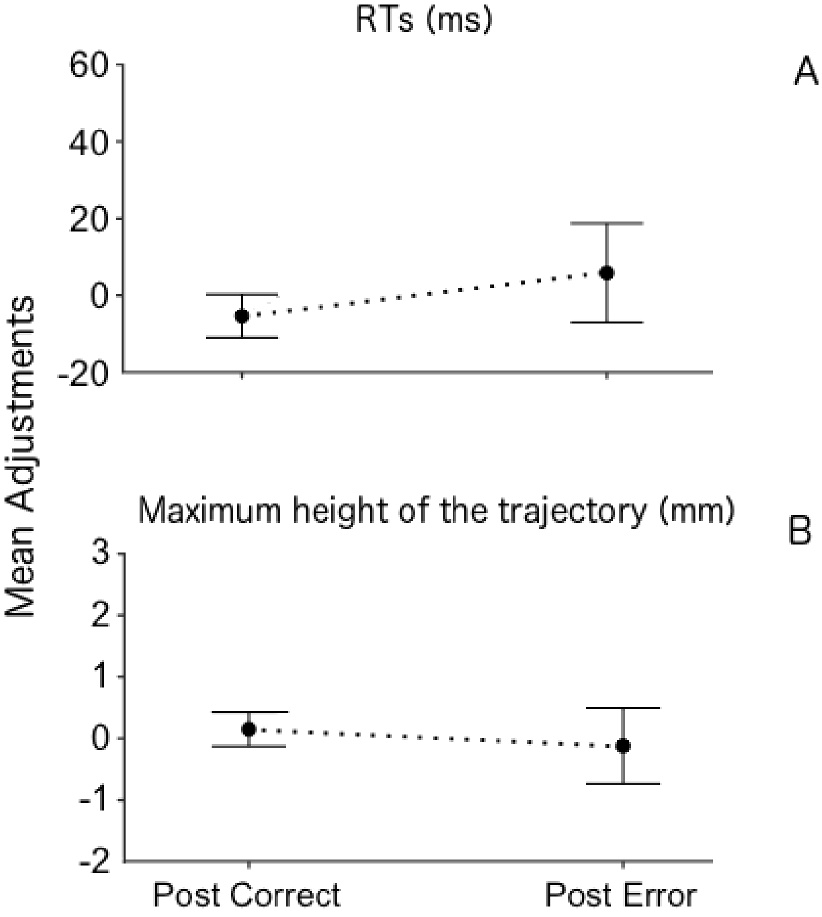

Figure 23. Mean post-correct and post-error adjustments in RTs (panel A) and the maximum height of the trajectory (panel B). For RTs, positive values indicate a post-error slowing. For the maximum height of the trajectory, positive values indicate a post-error increase of the amplitude of maximum height reached by the wrist. Vertical bars represent standard error of the mean.

Maximum Height of The Trajectory. The mean of post-correct and post-error adjustments of the maximum height of trajectory are shown in Figure 23B. A LME analysis, with random intercept for Participants and Blocks (modeling intercepts only), did not revealed any significance difference between post-correct ($\mathbf{PCA}_{\text{robust}} = 0.16 \pm 0.30$) and post-error adjustments ($\mathbf{PEA}_{\text{robust}} = -0.05 \pm 0.68$), $\chi^2 = 0.09$, $p = .759$, $BF = 0.09$, $d = 0.02$.

7.3. Discussion

The goal of the present study was twofold. To investigate whether the oPES is present when a more realistic, ecologically valid task is employed and whether error observation impacts on motor execution.

In line with the previous literature, RTs analysis showed that error observation led to a slowdown during the motor preparation phase (Schuch & Tipper, 2007; Castellar et al., 2011). This finding demonstrates that the oPES is a strong empirical evidence, which can also be observed using more realistic and ecologically valid tasks. Interestingly, no evidence of post-error improvement of accuracy (PIA) was found in this study. This result argues against the strict association between oPES and post-error accuracy and supports the hypothesis that the oPES and PIA are dissociated and sensitive to different aspects of the task (Castellar et al., 2001). With regard to this issue, Castellar and colleagues (2001) pointed out that the oPES is modulated by social context (cooperation vs. competition), while post-error accuracy is sensitive to the nature of the agent involved in the interaction (human vs. computer). The relation between PES and PIA has been a topic discussed at length even in the context of individual task performance, and several studies demonstrated that the PES and PIA do not always occur together and might represent different processes (for a review see Danielmeier & Ullsperger, 2011).

After making an error, participants did not show any evidence of post-error adjustments on the following execution trial. Recent investigations indicate that PES tends to decay over time (Danielmeier & Ullsperger, 2011; Jentzsch & Dudschig, 2009). In particular, short inter-trial intervals (ITIs) (< 500 ms) are usually associated with a larger PES, whereas long ITIs (> 8000 ms; Williams, Heathcote, Nesbitt, & Eidels, 2016) tend to elicit a decrease of PES. In our experiment participants were asked to observe the confederate undertaking the task before they again executed the movement. In these circumstances, the PES might not have survived because of the long interval between execution trials (~16 s). These results are consistent with the previous literature on oPES, suggesting that the aftereffects of the participant's performance tend to weaken when the task requires that the execution of the action is alternated with the observation of another person's action (e.g., Winkel et al., 2009).

Crucially, the results of the present study demonstrated that the observation of another person's error does not only influence the observer's overt behavior in terms of RTs, but impacts on the unfolding of the movement. In particular, I found a post-error increase in wrist elevation, which may be interpreted as a compensatory strategy aimed at grasping the target with a larger safety margin (Smeets & Brenner, 1999). Indeed, an increase in hand elevation, ensured that the index finger and thumb did not collide with the target during movement execution. Moreover, increasing the height of wrist elevation may serve to improve the quality of visual information by means of which contact points are established. In fact, it should be noted that in the present study the target was positioned along the mid-sagittal plane of the participants, and thus the vision of the target was partially covered by fingers during the gripping phase. An increase in wrist elevation may improve the visibility of the target during the honing phase as to adjust contact points and possibly the applied forces with the aim to avoid the falling of the support. In this sense, the oPES may be considered as a strategic adjustment, triggered by the mere observation of an error, aimed at improving the subsequent performance and avoid future errors.

8. GENERAL DISCUSSION

The experimental work included in this thesis provides a new way to examine the cognitive control mechanisms underlying post-error adjustments. In particular, for the first time, I examined the impact of error-reactivity effects on the execution of a realistic goal-directed action, that is the reach to grasp movement.

The principal finding stemming from Experiments 1 and 2 is that error-reactivity effects extend to movement execution, but they are confined to the grasping component, whereas the reaching component resulted impermeable to post-error effects. When the task involves grasping, a slowing in movement duration and a reduction in gripping velocity arise after the occurrence of an error. This might reflect advanced planning, which strategically prolongs the duration of the movement so that reach-to-grasp kinematics is scaled within this allotted time. Indeed, the motor control system relies on visual and proprioceptive feedback loops, and thus, the more time in which these loops can operate, the greater the influence control will exert (Glover, 2004). This type of programming keeps the timing of the commands independent from the spatial parameters of the movement. In other words, selection of the motor plan needing to be activated to carry out a given task keeping into account previous errors can be modified within a centrally generated temporal template that determines the co-ordination of a given action. This might be the easiest and most readily chosen organizational option of the neural system to compensate for the error. In this connection, the reduction of gripping velocity may serve to lengthen the time window within which contact points can be established more precisely and firmly. Several studies have investigated the effect of object properties and context on the planning of digit placement upon an object

(Fu, Zhang, & Santello, 2010; Lukos, Ansuini, & Santello, 2007; Zhang, Gordon, Fu, & Santello, 2010; Sartori, Straulino, & Castiello, 2011). In these studies, subjects grasped and lifted an object with the aim to prevent object tilt. For example, to do so when an object's center of mass is shifted to the right (resulting in a clockwise tilt if uncompensated), a compensatory counter-clockwise moment was required at object lift onset. Subjects created a compensatory moment by placing the thumb lower than the index finger (Lukos et al., 2007). In this example, digit placement is modulated to ensure successful completion of the task. Our finding of a post error reduction in grip velocity might signify that after an error it is necessary to apply changes related to finger positioning in order to avoid the falling of the target. To do this, participants need a slower closing of the fingers during the honing phase as to adjust contact points and possibly the applied forces. The finding of adaptation of fingers' closing velocity following an error extends to error coding the evidence that subjects' grasping is anticipatory. Anticipatory planning implies that objects are grasped in such a way that allows for movement flexibility. In our task, such flexibility allows to keep into account errors when performing the subsequent movement. Thus, fingers' movement trajectory was dependent on the outcome of the previous trial. In other words, the slowing of the reach-to-grasp timing after an error seems to reflect advanced planning related to the need to increase the control and avoid future errors. However, only the grasping components seems to benefit of the adaptive components of error-reactivity. Indeed, after a reaching error, I did not observe neither changes in terms of accuracy, nor a parametrical reorganization of the reaching movement. These results support the modular description of the reach to grasp movements provided by the VCH (Jeannerod, 1981). A point worth noting is that the strict dichotomy between reaching and grasping components has been occasionally questioned. Indeed, it has been revealed that the component controlling proximal movements can access information about stimulus size and accordingly can modify peak velocity and acceleration (e.g., Gentilucci et al. 1991; Jakobson & Goodale, 1991; Smeets & Brenner, 1999). This weakened the proposal of two independent channels, one responsible for the computation of the stimulus location and arm transport, and the other responsible for the computation of stimulus intrinsic properties and the organization of manipulation. However, the very fact that in our study changes in the organization of the reach-to-grasp movements due to error commission are only evident at the level of the grasping component speaks in favor of the possibility that the two components can be dissociated. In particular, our findings suggest that the impermeability of the reaching component to errors might be strategic, simplifying the processes underlying the reorganization of the movement sequence following an error. This proposal is corroborated by studies indicating that the hand closure event initiates during the deceleration phase, but the precise moment of the closure did not have any specific counterpart in the velocity profiles of the reaching component (Gentilucci et al., 1991; Jakobson & Goodale, 1991). However, it must be noted that this result may be related to the particular demands of the tasks we employed, and thus, further research with realistic and ecological tasks is needed to better clarify this issue.

In Experiment 3, I tested the hypothesis according to which the processing of an error leads to an inhibition of the motor system. To examine this aspect, I compared the CST excitability after erroneous and after correct movements, by means of MEPs technique. The principal finding emerging from this experiment is that the amplitude of MEPs tends to decrease after an error, indicating that the inhibitory circuits in M1 may contribute to the implementation of post error adjustments. However, an open question concerns to the brain networked areas that might cause the changes of the excitability of the CST here observed. A possible explanation is that the inhibition of M1 is triggered by a right hemispheric network including the pre-SMA, the IFC and the STN, whose activity has been found to correlate with PES (Ridderinkhof, 2002; Danielmeier & Ullsperger, 2011). An alternative hypothesis is that reduction of CST excitability is triggered by an error-monitoring system including the ACC and the DLPC, as suggested by the CMT (Botvinick et al., 2001). However, it should be noted that other non-frontal areas may bias the activity of M1. For instance, Gréa and colleagues (2002) found that a lesion of the posterior parietal cortex (PPC), and in particular of the intra-parietal sulcus, disrupts on-line adjustments during aiming movements. Coherently, the PPC may have a key role in error-reactivity, projecting to the DLPFC and to M1 (Amengual at al., 2013).

In Experiment 4, I found that the observation of another person's error does not only influence the observer's behavior in terms of RTs, but impacts on the unfolding of the reach to grasp movement. In particular, the wrist elevation tends to increase after an error. This evidence may be interpreted as a compensatory strategy aimed at both increase safety margins between the target and fingers and to improve the visibility of the target during the honing phase as to adjust contact points and possibly the applied forces with the aim in order to avoid the falling of the support. The results of this experiment suggest that if the observed action is marred by an error, then onlookers internalize the observed “error” as to put in place error avoidance strategies when the observed action is subsequently performed. In this view, the activated motor representation does not merely reflect an automatic resonance mechanism of motor structures paralleling observed movements. If this were the case error observation would inevitably determine an error during execution. Rather, it might reflect the interplay between an initial emulative process, which allows the observer to experience what is being observed, and a subsequent one which considers the consequences/outcome of the initially observed actions.

The new insights emerging from this thesis highlight the importance of using ecologically valid paradigms to obtain a more detailed and generalizable picture of the cognitive control mechanisms underlying post-error adjustments. In particular, the analysis of movement execution by means kinematical analysis should be the reference standard of future studies. Moreover, more innovative approaches should be developed to explore error-reactivity in natural contexts.

REFERENCES

Allain, S., Burle, B., Hasbroucq, T., & Vidal, F. (2009). Sequential adjustments before and after partial errors. *Psychonomic Bulletin & Review*, *16*, 356-362.

Amengual, J. L., Marco-Pallares, J., Richter, L., Oung, S., Schweikard, A., Mohammadi, B., ... & Münte, T. F. (2013). Tracking post-error adaptation in the motor system by transcranial magnetic stimulation. *Neuroscience*, *250*, 342-351.

Andersen, R. A., & Cui, H. (2009). Intention, action planning, and decision making in parietal-frontal circuits. *Neuron*, *63*, 568-583.

Anguera, J. A., Seidler, R. D., & Gehring, W. J. (2009). Changes in performance monitoring during sensorimotor adaptation. *Journal of Neurophysiology*, *102*, 1868-1879.

Aron, A. R., Behrens, T. E., Smith, S., Frank, M. J., & Poldrack, R. A. (2007). Triangulating a cognitive control network using diffusion-weighted magnetic resonance imaging (MRI) and functional MRI. *Journal of Neuroscience*, *27*, 3743-3752.

Bates, A. T., Patel, T. P., & Liddle, P. F. (2005). External behavior monitoring mirrors internal behavior monitoring: error-related negativity for observed errors. *Journal of Psychophysiology*, *19*, 281-288.

Bates, D., Maechler, M., Bolker, B., & Walker S. (2015). *lme4: Linear mixed-effects models using Eigen and S4* (R package Version 1.1 -8). Retrieved from http://CRAN.R-project.org/package=lme4

Battaglini, P. P., Muzur, A., Galletti, C., Skrap, M., Brovelli, A., & Fattori, P. (2002). Effects of lesions to area V6A in monkeys. *Experimental Brain Research, 144*, 419-422.

Baumann, M. A., Fluet, M. C., & Scherberger, H. (2009). Context-specific grasp movement representation in the macaque anterior intraparietal area. *Journal of Neuroscience, 29*, 6436–6448.

Becchio, C., Sartori, L., Bulgheroni, M., & Castiello, U. (2008). Both your intention and mine are reflected in the kinematics of my reach-to-grasp movement. *Cognition*, *106*, 894-912.

Begliomini, C., M. B. Wall, A. T. Smith, and U. Castiello. 2007. Differential cortical activity for precision and whole- hand visually guided grasping in humans. *European Journal of Neuroscience, 25*, 1245–1252.

Binkofski, F., Dohle, C., Posse, S., Stephan, K. M., Hefter, H., Seitz, R. J., & Freund, H. J. (1998). Human anterior intraparietal area subserves prehension A combined lesion and functional MRI activation study. *Neurology*, *50*, 1253-1259.

Bootsma, R.J., Marteniuk, R.G., Mackenzie, C.L. & Zaal, F.T.J. (1994). The speed-accuracy trade off in manual prehension: effects of movement amplitude, object size and object width on kinematic characteristics. *Experimental Brain Research, 98*, 535-541.

Bosco, A., Breveglieri, R., Chinellato, E., Galletti, C. & Fattori, P. (2010). Reaching activity in the medial posterior parietal cortex of monkeys is modulated by visual feedback. *Journal of Neuroscience, 30*, 14773-14785.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, *108*, 624.

Botvinick, M., Nystrom, L. E., Fissell, K., Carter, C. S., & Cohen, J. D. (1999). Conflict monitoring versus selection-for-action in anterior cingulate cortex. *Nature*, *402*, 179.

Burle, B., Possamaï, C. A., Vidal, F., Bonnet, M., & Hasbroucq, T. (2002). Executive control in the Simon effect: an electromyographic and distributional analysis. *Psychological Research*, *66*, 324-336.

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences*, *4*, 215-222.

Caminiti, R., Johnson, P. B., Galli, C., Ferraina, S., & Burnod, Y. (1991). Making arm movements within different parts of space: the premotor and motor cortical representation of a coordinate system for reaching to visual targets. *Journal of Neuroscience*, *11*, 1182-1197.

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*, 747-749.

Castiello, U. (2005). The neuroscience of grasping. *Nature Neuroscience Reviews, 6,* 726-736.

Castiello, U., Bennett, K. M. B., & Stelmach, G. E. (1993). Reach to grasp: the natural response to perturbation of object size. *Experimental Brain Research*, *94*, 163-178.

Caudek, C., Ceccarini, F., & Sica, C. (2015). Posterror speeding after threat-detection failure. *Journal of Experimental Psychology: Human Perception and Performance*, *41*, 324.

Chevrier, A., & Schachar, R. J. (2010). Error detection in the stop signal task. *Neuroimage*, *53*, 664-673.

Chieffi, S., & Gentilucci, M. (1993). Coordination between the transport and the grasp components during prehension movements. *Experimental Brain Research*, *94*, 471-477.

Chieffi, S., Fogassi, L., Gallese, V., & Gentilucci, M. (1992). Prehension movements directed to approaching objects: influence of stimulus velocity on the transport and the grasp components. *Neuropsychologia*, *30*, 877-897.

Cohen, J. (1988). The effect size. Statistical power analysis for the behavioral sciences. Hillsdale, NJ: Lawrence Erlbaum.

Cohen, M. X., Van Gaal, S., Ridderinkhof, K. R., & Lamme, V. (2009). Unconscious errors enhance prefrontal-occipital oscillatory synchrony. *Frontiers in Human Neuroscience*, *3*, 54.

Cohen, Y. E., & Andersen, R. A. (2002). A common reference frame for movement plans in the posterior parietal cortex. *Nature Reviews Neuroscience*, *3*, 553.

Critchley, H. D., Tang, J., Glaser, D., Butterworth, B., & Dolan, R. J. (2005). Anterior cingulate activity during error and autonomic response. *Neuroimage*, *27*, 885-895.

D'Amico, M., & Ferrigno, G. (1992). Comparison between the more recent techniques for smoothing and derivative assessment in biomechanics. *Medical and Biological Engineering and Computing*, *30*, 193-204.

Danev, S. G., & Winter, C. R. (1971). Heart rate deceleration after erroneous responses. *Psychological Research*, *35*(1), 27-34.

Danielmeier, C., & Ullsperger, M. (2011). Post-error adjustments. *Frontiers in Psychology*, *2*, 233.

Danielmeier, C., Eichele, T., Forstmann, B. U., Tittgemeyer, M., & Ullsperger, M. (2011). Posterior medial frontal cortex activity predicts post-error adaptations in task-related visual and motor areas. *Journal of Neuroscience*, *31*, 1780-1789.

Darling, W. G., & Gilchrist, L. (1991). Is there a preferred coordinate system for perception of hand orientation in three-dimensional space? *Experimental Brain Research*, *85*, 405-416.

Davare, M., Andres, M., Cosnard, G., Thonnard, J. L., & Olivier, E. (2006). Dissociating the role of ventral and dorsal premotor cortex in precision grasping. *The Journal of Neuroscience, 26*, 2260-2268.

Davare, M., Rothwell, J. C., & Lemon, R. N. (2010). Causal connectivity between the human anterior intraparietal area and premotor cortex during grasp. *Current Biology*, *20*, 176-181.

Davare, M., Zénon, A., Desmurget, M., & Olivier, E. (2015). Dissociable contribution of the parietal and frontal cortex to coding movement direction and amplitude. *Frontiers in Human Neuroscience*, *9*, 241.

De Bruijn, E. R., Hulstijn, W., Verkes, R. J., Ruigt, G. S., & Sabbe, B. G. (2004). Drug-induced stimulation and suppression of action monitoring in healthy volunteers. *Psychopharmacology, 177*, 151-160.

Desmet, C., Imbo, I., De Brauwer, J., Brass, M., Fias, W., & Notebaert, W. (2012). Error adaptation in mental arithmetic. *Quarterly Journal of Experimental Psychology*, *65*, 1059-1067.

Di Pellegrino, G., Ciaramelli, E., & Làdavas, E. (2007). The regulation of cognitive control following rostral anterior cingulate cortex lesion in humans. *Journal of Cognitive Neuroscience, 19*, 275-286.

Dixon, P., & Glover, S. (2009). Perseveration and contrast effects in grasping. *Neuropsychologia*, *47*, 1578-1584.

Dixon, P., McAnsh, S., & Read, L. (2012). Repetition effects in grasping. *Canadian Journal of Experimental Psychology/Revue Canadienne de Psychologie Expérimentale*, *66*, 1.

Dudschig, C., & Jentzsch, I. (2009). Speeding before and slowing after errors: Is it all just strategy? *Brain Research, 1296*, 56-62.

Dutilh, G., van Ravenzwaaij, D., Nieuwenhuis, S., van der Maas, H. L., Forstmann, B. U., & Wagenmakers, E. J. (2012). How to measure post-error slowing: a confound and a simple solution. *Journal of Mathematical Psychology*, *56*, 208-216.

Dutilh, G., Vandekerckhove, J., Forstmann, B. U., Keuleers, E., Brysbaert, M., & Wagenmakers, E. J. (2012). Testing theories of post-error slowing. *Attention, Perception, & Psychophysics*, *74*, 454-465.

Egner, T., & Hirsch, J. (2005). Cognitive control mechanisms resolve conflict through cortical amplification of task-relevant information. *Nature Neuroscience*, *8*, 1784.

Eliasson, A. C., Rösblad, B., & Forssberg, H. (2004). Disturbances in programming goal-directed arm movements in children with ADHD. *Developmental Medicine and Child Neurology*, *46*, 19-27.

Elliot, A. J., & Maier, M. A. (2007). Color and psychological functioning. *Current Directions in Psychological Science*, *16*, 250-254.

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

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*, 1714-1720.

Erdfelder, E., Faul, F., & Buchner, A. (1996). GPOWER: A general power analysis program. *Behavior Research Methods, Instruments, & Computers*, *28*, 1-11.

Erlhagen, W., & Schöner, G. (2002). Dynamic field theory of movement preparation. *Psychological Review*, *109*, 545.

Etkin, A., Egner, T., & Kalisch, R. (2011). Emotional processing in anterior cingulate and medial prefrontal cortex. *Trends in Cognitive Sciences*, *15*, 85-93.

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*, 447-455.

Fattori, P., Raos, V., Breveglieri, R., Bosco, A., Marzocchi, N., & Galletti, C. (2010). The dorsomedial pathway is not just for reaching: grasping neurons in the medial parieto-occipital cortex of the macaque monkey. *The Journal of Neuroscience, 30*, 342-349.

Fiehler, K., Ullsperger, M., & Von Cramon, D. Y. (2005). Electrophysiological correlates of error correction. *Psychophysiology*, *42*, 72-82.

Fluet, M. C., Baumann, M. A., and Scherberger, H. (2010). Context-specific grasp movement representation in macaque ventral premotor cortex. *The Journal of Neuroscience*, *30*, 15175–15184.

Fogassi, L., Gallese, V., Buccino, G., Craighero, L., Fadiga, L., and Rizzolatti, G. (2001). Cortical mechanism for the visual guidance of hand grasping move- ments in the monkey: a reversible inactivation study. *Brain, 124*, 571–586.

Fradet, L., Lee, G., & Dounskaia, N. (2008). Origins of submovements in movements of elderly adults. *Journal of Neuroengineering and Rehabilitation*, *5*, 28.

Friedman, D., Cycowicz, Y. M., & Gaeta, H. (2001). The novelty P3: an event-related brain potential (ERP) sign of the brain's evaluation of novelty. *Neuroscience & Biobehavioral Reviews, 25*, 355-373.

Fu, Q., Zhang, W., & Santello, M. (2010). Anticipatory planning and control of grasp positions and forces for dexterous two-digit manipulation. *Journal of Neuroscience*, *30*, 9117-9126.

Gallese, V., Murata, A., Kaseda, M., Nikim N. & Sakata, H. (1994). Deficit of hand preshaping after muscimol injection in monkey parietal cortex. *Neuroreport, 5*, 1525-1529.

Gallivan, J. P., Cavina-Pratesi, C., & Culham, J. C. (2009). Is that within reach? fMRI reveals that the human superior parieto-occipital cortex encodes objects reachable by the hand. *Journal of Neuroscience*, *29*, 4381-4391.

Gallivan, J. P., McLean, D. A., Valyear, K. F., Pettypiece, C. E., & Culham, J. C. (2011). Decoding action intentions from preparatory brain activity in human parieto-frontal networks. *The Journal of Neuroscience, 31*, 9599-9610.

Garavan, H., Ross, T. J., Murphy, K., Roche, R. A. P., & Stein, E. A. (2002). Dissociable executive functions in the dynamic control of behavior: inhibition, error detection, and correction. *Neuroimage*, *17*, 1820-1829.

Gehring, W. J., Liu, Y., Orr, J. M., Carp, J. (2011). The error-related negativity (ERN/Ne). In *The Oxford Handbook of Event-Related Potential Components* (pp. 231–291), S.L. Luck & E.S. Kappenman. Oxford, UK: University Press.

Gentilucci, M., Castiello, U., Corradini, M. L., Scarpa, M., Umilta, C., & Rizzolatti, G. (1991). Influence of different types of grasping on the transport component of prehension movements. *Neuropsychologia*, *29*, 361-378.

Georgiou, I., Becchio, C., Glover, S., & Castiello, U. (2007). Different action patterns for cooperative and competitive behaviour. *Cognition*, *102*, 415-433.

Glover, S. (2004). Separate visual representations in the planning and control of action. *Behavioral and Brain Sciences, 27*, 3-78.

Godschalk, M., Lemon, R. N., Nijs, H. G. T., & Kuypers, H. G. J. M. (1981). Behaviour of neurons in monkey peri-arcuate and precentral cortex before and during visually guided arm and hand movements. *Experimental Brain Research*, *44*, 113-116.

Gordon, A. M., Forssberg, H., Johansson, R. S., & Westling, G. (1991). Visual size cues in the programming of manipulative forces during precision grip. *Experimental Brain Research*, *83*, 477-482.

Gordon, J., Ghilardi, M. F., Cooper, S. E., & Ghez, C. (1994). Accuracy of planar reaching movements. *Experimental Brain Research*, *99*, 112-130.

Gratton, G., Coles, M. G., & Donchin, E. (1992). Optimizing the use of information: strategic control of activation of responses. *Journal of Experimental Psychology: General*, *121*, 480.

Gréa, H., Pisella, L., Rossetti, Y., Desmurget, M., Tilikete, C., Grafton, S., ... & Vighetto, A. (2002). A lesion of the posterior parietal cortex disrupts on-line adjustments during aiming movements. *Neuropsychologia*, *40*, 2471-2480.

Haggard, P., & Wing, A. M. (1991). Remote responses to perturbation in human prehension. *Neuroscience letters*, *122*, 103-108.

Haith, A. M., Pakpoor, J., & Krakauer, J. W. (2016). Independence of movement preparation and movement initiation. *Journal of Neuroscience*, *36*, 3007-3015.

Hajcak, G., McDonald, N., & Simons, R. F. (2003). To err is autonomic: Error-related brain potentials, ANS activity, and post-error compensatory behavior. *Psychophysiology*, *40*, 895-903.

Hare, T. A., Camerer, C. F., & Rangel, A. (2009). Self-control in decision-making involves modulation of the vmPFC valuation system. *Science*, *324*, 646-648.

Harty, S., Robertson, I. H., Miniussi, C., Sheehy, O. C., Devine, C. A., McCreery, S., & O'Connell, R. G. (2014). Transcranial direct current stimulation over right dorsolateral prefrontal cortex enhances error awareness in older age. *Journal of Neuroscience*, *34*, 3646-3652.

Hogan, A. M., Vargha-Khadem, F., Kirkham, F. J., & Baldeweg, T. (2005). Maturation of action monitoring from adolescence to adulthood: an ERP study. *Developmental Science*, *8*, 525-534.

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*, 679.

Holroyd, C. B., Dien, J., & Coles, M. G. (1998). Error-related scalp potentials elicited by hand and foot movements: evidence for an output-independent error-processing system in humans. *Neuroscience Letters*, *242*, 65-68.

Houk, J. C., Bastianen, C., Fansler, D., Fishbach, A., Fraser, D., Reber, P. J., ... & Simo, L. S. (2007). Action selection and refinement in subcortical loops through basal ganglia and cerebellum. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *362*, 1573-1583.

Houtman, F., & Notebaert, W. (2013). Blinded by an error. *Cognition*, *128*, 228-236.

Ito, S., Stuphorn, V., Brown, J. W., & Schall, J. D. (2003). Performance monitoring by the anterior cingulate cortex during saccade countermanding. *Science*, *302*, 120-122.

Jackson, S. R., Jackson, G. M., & Rosicky, J. (1995). Are non-relevant objects represented in working memory? The effect of non-target objects on reach and grasp kinematics. *Experimental Brain Research*, *102*, 519-530.

Jakobson, L.S. & Goodale, M.A. (1991). Factors affecting higher-order movement planning: a kinematic analysis on human prehension. *Experimental Brain Research, 86*, 199-208.

Jax, S. A., & Rosenbaum, D. A. (2009). Hand path priming in manual obstacle avoidance: Rapid decay of dorsal stream information. *Neuropsychologia*, *47*, 1573-1577.

Jeannerod M (1981). Intersegmental coordination during reaching at natural visual objects. In: Long J, Baddley A, editors. *Attention and Performances IX*. Hillsdale, NJ: Erlbaum; pp153-168.

Jeannerod, M. (1984). The timing of natural prehension movements. *Journal of Motor Behaviour, 16*, 235-254.

Jeannerod, M. (1986). Mechanisms of visuomotor coordination: a Study in normal and brain-damaged subjects. *Neuropsychologia, 24*, 41–78.

Jeannerod, M., Decety, J., & Michel, F. (1994). Impairment of grasping movements following a bilateral posterior parietal lesion. *Neuropsychologia*, *32*(4), 369-380.

Jeffreys, H. (1961). Theory of Probability. Oxford: UK: Oxford University Press.

Jentzsch, I., & Dudschig, C. (2009). Why do we slow down after an error? Mechanisms underlying the effects of post-error slowing. *The Quarterly Journal of Experimental Psychology*, *62*, 209-218.

Johnson, P.B., Ferraina, S., Bianchi, L. & Caminiti, R. (1996). Cortical networks for visual reaching: physiological and anatomical organization of frontal and parietal lobe arm regions. *Cerebral Cortex, 6*, 102-119.

Kaiser, H. F. (1960). The application of electronic computers to factor analysis. *Educational and Psychological Measurement*, *20*, 141-151.

Kanwisher, N., McDermott, J., & Chun, M. M. (1997). The fusiform face area: a module in human extrastriate cortex specialized for face perception. *Journal of Neuroscience*, *17*, 4302-4311.

Kelso, J. A. S., Buchanan, J. J., & Murata, T. (1994). Multifunctionality and switching in the coordination dynamics of reaching and grasping. *Human Movement Science*, *13*, 63-94.

Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science*, *303*, 1023-1026.

Khan, M. A., & Franks, I. M. (2003). Online versus offline processing of visual feedback in the production of component submovements. *Journal of Motor Behavior*, *35*, 285-295.

Kiehl, K. A., Liddle, P. F., & Hopfinger, J. B. (2000). Error processing and the rostral anterior cingulate: An event-related fMRI study. *Psychophysiology*, *37*, 216-223.

King, J. A., Korb, F. M., von Cramon, D. Y., & Ullsperger, M. (2010). Post-error behavioral adjustments are facilitated by activation and suppression of task-relevant and task-irrelevant information processing. *Journal of Neuroscience*, *30*, 12759-12769.

Klein, T. A., Endrass, T., Kathmann, N., Neumann, J., von Cramon, D. Y., & Ullsperger, M. (2007). Neural correlates of error awareness. *Neuroimage*, *34*, 1774-1781.

Krigolson, O. E., & Holroyd, C. B. (2007). Evidence for hierarchical error processing in the human brain. *Neuroscience*, *137*, 13-17.

Kudoh, N., Hattori, M., Numata, N., & Maruyama, K. (1997). An analysis of spatiotemporal variability during prehension movements: effects of object size and distance. *Experimental Brain Research*, *117*, 457-464.

Kühn, A. A., Williams, D., Kupsch, A., Limousin, P., Hariz, M., Schneider, G. H., ... & Brown, P. (2004). Event-related beta desynchronization in human subthalamic nucleus correlates with motor performance. *Brain*, *127*, 735-746.

Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. B. (2013). lmerTest: Tests for random and fixed effects for linear mixed effect models (R package Version, 2.0-25). Retrieved from http://cran.r-project.org/web/packages/lmerTest/

Laming, D.R.J. (1968). Information theory of choice-reaction times. Oxford, England: Academic Press.

Luck, S. J. (2014). *An introduction to the event-related potential technique*. Cambridge, MA, MIT press.

Lukos, J., Ansuini, C., & Santello, M. (2007). Choice of contact points during multidigit grasping: effect of predictability of object center of mass location. *Journal of Neuroscience*, *27*, 3894-3903.

Luppino, G., Murata, A., Covoni, P. & Matelli, M. (1999). Largely segregated parietofrontal connections linking rostral intraparietal cortex (areas AIP and VIP) and the ventral premotor cortex (areas F5 and F4). *Experimental Brain Research,128*, 181–187.

MacDonald, A. W., Cohen, J. D., Stenger, V. A., & Carter, C. S. (2000). Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science*, *288*, 1835-1838.

Maier, M. E., Yeung, N., & Steinhauser, M. (2011). Error-related brain activity and adjustments of selective attention following errors. *Neuroimage*, *56*, 2339-2347.

Marco-Pallarés, J., Camara, E., Münte, T. F., & Rodríguez-Fornells, A. (2008). Neural mechanisms underlying adaptive actions after slips. *Journal of Cognitive Neuroscience*, *20*, 1595-1610.

Marteniuk, R.G., Leavitt, J.L., Mackenzie & C.L., Athenes, S. (1990). Functional relationship between grasp and transport components in prehension task. *Human Movement Science, 9*, 149-176.

Maruo, Y., Schacht, A., Sommer, W., & Masaki, H. (2016). Impacts of motivational valence on the error-related negativity elicited by full and partial errors. *Biological Psychology*, *114*, 108-116.

Masina, F., Vallesi, A., Di Rosa, E., Semenzato, L., & Mapelli, D. (2018). Possible role of dorsolateral prefrontal cortex in error awareness: single-pulse TMS evidence. *Frontiers in Neuroscience*, *12*, 179.

Menon, V., Adleman, N. E., White, C. D., Glover, G. H., & Reiss, A. L. (2001). Error-related brain activation during a Go/NoGo response inhibition task. *Human Brain Mapping*, *12*, 131-143.

Metzler-Baddeley, C., Jones, D. K., Steventon, J., Westacott, L., Aggleton, J. P., & O'Sullivan, M. J. (2012). Cingulum microstructure predicts cognitive control in older age and mild cognitive impairment. *Journal of Neuroscience*, *32*, 17612-17619.

Meyer, D. E., Abrams, R. A., Kornblum, S., Wright, C. E., & Keith Smith, J. E. (1988). Optimality in human motor performance: ideal control of rapid aimed movements. *Psychological Review*, *95*, 340.

Morey, D., Rouder, J. N., & Jamil, T. (2014). *Bayes factor: Computation of Bayes factors for common designs* (R package version 0.9.8). Retrieved from http://CRAN.R-project.org/package=BayesFactor

Murata, A., Fadiga, L., Fogassi, L., Gallese, V., Raos, V. & Rizzolatti G. (1997). Object representation in the ventral premotor cortex (area F5) of the monkey. *Journal of Neurophysiology, 78*, 2226–2230.

Mushiake, H., Saito, N., Sakamoto, K., Itoyama, Y., & Tanji, J. (2006). Activity in the lateral prefrontal cortex reflects multiple steps of future events in action plans. *Neuron*, *50*, 631-641.

Napier, J. R. (1960). Studies of the hands of living primates. *Proceedings of the Zoological Society of London, 134*, 647-657.

Notebaert, W., & Verguts, T. (2011). Conflict and error adaptation in the Simon task. *Acta Psychologica*, *136*, 212-216.

Notebaert, W., Houtman, F., Van Opstal, F., Gevers, W., Fias, W., & Verguts, T. (2009). Post-error slowing: an orienting account. *Cognition*, *111*, 275-279.

Novak, K. E., Miller, L. E., & Houk, J. C. (2002). The use of overlapping submovements in the control of rapid hand movements. *Experimental Brain Research*, *144*, 351-364.

núňez Castellar, E., Kühn, S., Fias, W., & Notebaert, W. (2010). Outcome expectancy and not accuracy determines posterror slowing: ERP support. *Cognitive, Affective, & Behavioral Neuroscience*, *10*, 270-278.

núñez Castellar, E., Notebaert, W., Van den Bossche, L., & Fias, W. (2011). How monitoring other's actions influences one's own performance: post-error adjustments are influenced by the nature of the social interaction. *Experimental Psychology*, *58*, 499-508.

O'connell, R. G., Dockree, P. M., Bellgrove, M. A., Kelly, S. P., Hester, R., Garavan, H., ... & Foxe, J. J. (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*, 2571-2579.

Paulignan, Y., & Jeannerod, M. (1996). Prehension movements. The visuomotor channels hypoyhesis revisited. In A. M. Wing, P. Haggard, & R. Flanagan (Eds.), Hand and Brain: The Neurophysiology and Psychology of Hand movements. (pp. 265-282). San Diego: Academic Press.

Paulignan, Y., MacKenzie, C., Marteniuk, R., & Jeannerod, M. (1990). The coupling of arm and finger movements during prehension. *Experimental Brain Research*, *79*, 431-435.

Paulignan, Y., MacKenzie, C., Marteniuk, R., & Jeannerod, M. (1991). Selective perturbation of visual input during prehension movements. *Experimental Brain Research*, *83*, 502-512.

Peterson, N. N., Schroeder, C. E., & Arezzo, J. C. (1995). Neural generators of early cortical somatosensory evoked potentials in the awake monkey.

Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section, 96, 248-260.

Pinheiro, J. C., & Bates, D. M. (2000). Mixed-effects models in S and S-PLUS. In J. Chambers, W. Eddy, W. Hardle, S. Sheather, & L. Tierney, *Statistics and Computing* (pp. 57-96). New York, NY: Springer-Verlag.

Priori, A., Berardelli, A., Rona, S., Accornero, N., & Manfredi, M. (1998). Polarization of the human motor cortex through the scalp. *Neuroreport*, *9*, 2257-2260.

Quesque, F., Lewkowicz, D., Delevoye-Turrell, Y. N., & Coello, Y. (2013). Effects of social intention on movement kinematics in cooperative actions. *Frontiers in Neurorobotics*, 7.

R Core Team. (2014). R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from http://www.R-project.org/

Rabbitt, P. M. (1966). Errors and error correction in choice-response tasks. *Journal of Experimental Psychology*, *71*, 264.

Rabbitt, P., & Rodgers, B. (1977). What does a man do after he makes an error? An analysis of response programming. *Quarterly Journal of Experimental Psychology*, *29*, 727-743.

Raos, V., Umiltá, M. A., Gallese, V., & Fogassi, L. (2004). Functional properties of grasping-related neurons in the dorsal premotor area F2 of the macaque monkey. *Journal of Neurophysiology*, *92*, 1990-2002.

Regev, S., & Meiran, N. (2014). Post-error slowing is influenced by cognitive control demand. *Acta Psychologica*, *152*, 10-18.

Ridderinkhof, R. K. (2002). Micro-and macro-adjustments of task set: activation and suppression in conflict tasks. *Psychological Research*, *66*, 312-323.

Rosenbaum, D. A., & Jorgensen, M. J. (1992). Planning macroscopic aspects of manual control. *Human Movement Science, 11*, 61-69.

Rossetti, Y. (1994). Implicit short-lived motor representations of space in brain damaged and healthy subjects. *Consciousness and Cognition*, *7*, 520-558.

Rossi, S., Hallett, M., Rossini, P. M., Pascual-Leone, A., & Safety of TMS Consensus Group. (2009). Safety, ethical considerations, and application guidelines for the use of transcranial magnetic stimulation in clinical practice and research. *Clinical Neurophysiology*, *120*, 2008-2039.

Rossini, P. M., Barker, A. T., Berardelli, A., Caramia, M. D., Caruso, G., Cracco, R. Q., ... & De Noordhout, A. M. (1994). Non-invasive electrical and magnetic stimulation of the brain, spinal cord and roots: basic principles and procedures for routine clinical application. Report of an IFCN committee. *Electroencephalography and Clinical Neurophysiology*, *91*, 79-92.

Rushworth, M. F. S., Nixon, P. D., & Passingham, R. E. (1997). Parietal cortex and movement I. Movement selection and reaching. *Experimental Brain Research*, *117*, 292-310.

Sakata, H., & Taira, M. (1994). Parietal control of hand action. *Current Opinion in Neurobiology*, *4*, 847-856.

Sartori, L., Straulino, E., & Castiello, U. (2011). How objects are grasped: the interplay between affordances and end-goals. *PloS one*, *6*, e25203.

Savelsbergh, G.J.P., Steenbergen, B. & van der Kamp, J. (1996). The role of fragility in the guidance for precision grip. *Human Movement Science, 15*, 115-127.

Schroder, H. S., Moran, T. P., Infantolino, Z. P., & Moser, J. S. (2013). The relationship between depressive symptoms and error monitoring during response switching. *Cognitive, Affective, & Behavioral Neuroscience*, *13*, 790-802.

Schuch, S., & Tipper, S. P. (2007). On observing another person's actions: Influences of observed inhibition and errors. *Attention, Perception, & Psychophysics*, *69*, 828-837.

Smeets, J. B., & Brenner, E. (1999). A new view on grasping. *Motor Control*, *3*, 237-271.

Spruit, I. M., Wilderjans, T. F., & van Steenbergen, H. (2018). Heart work after errors: Behavioral adjustment following error commission involves cardiac effort. *Cognitive, Affective, & Behavioral Neuroscience*, 1-14.

Stark, E., Asher, I., & Abeles, M. (2007). Encoding of reach and grasp by single neurons in premotor cortex is independent of recording site. *Journal of Neurophysiology*, *97*, 3351-3364.

Steinborn, M. B., Flehmig, H. C., Bratzke, D., & Schröter, H. (2012). Error reactivity in self-paced performance: Highly-accurate individuals exhibit largest post-error slowing. *Quarterly Journal of Experimental Psychology*, *65*, 624-631.

Stemmer, B., Segalowitz, S. J., Dywan, J., Panisset, M., & Melmed, C. (2007). The error negativity in nonmedicated and medicated patients with Parkinson's disease. *Clinical Neurophysiology*, *118*, 1223-1229.

Stürmer, B. (2011). Reward and punishment effects on error processing and conflict control. *Frontiers in Psychology*, *2*, 335.

Sutton, R. S., & Barto, A. G. (1998). *Introduction to reinforcement learning* (Vol. 135). Cambridge: MIT press.

Szumilas, M. (2010). Explaining odds ratios. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, *19*, 227.

Torres, E. B., Quian Quiroga, R., Cui, H., & Buneo, C. (2013). Neural correlates of learning and trajectory planning in the posterior parietal cortex. *Frontiers in Integrative Neuroscience*, *7*, 39.

Turella, L., & Lingnau, A. (2014). Neural correlates of grasping. *Frontiers in Human Neuroscience*, *8*, 686.

Ullsperger, M., & Szymanowski, F. (2004). ERP correlates of error relevance. In Errors, Conflicts, and the Brain. Current Opinions on Performance Monitoring (pp. 171-177). Leipzig: Max Planck Institute for Human Cognitive and Brain Sciences.

Ullsperger, M., Danielmeier, C., & Jocham, G. (2014). Neurophysiology of performance monitoring and adaptive behavior. *Physiological Reviews*, *94*, 35-79.

Van Der Borght, L., Schevernels, H., Burle, B., & Notebaert, W. (2016). Errors disrupt subsequent early attentional processes. *PloS one*, *11*, e0151843.

van Schie, H. T., Mars, R. B., Coles, M. G., & Bekkering, H. (2004). Modulation of activity in medial frontal and motor cortices during error observation. *Nature Neuroscience*, *7*, 549.

Vocat, R., Pourtois, G., & Vuilleumier, P. (2011). Parametric modulation of error-related ERP components by the magnitude of visuo-motor mismatch. *Neuropsychologia*, *49*, 360-367.

Wagenmakers, E. J., & Farrell, S. (2004). AIC model selection using Akaike weights. *Psychonomic Bulletin & Review*, *11*, 192-196.

Wallace, S. A., Weeks, D. L., & Kelso, J. S. (1990). Temporal constraints in reaching and grasping behavior. *Human Movement Science*, *9*, 69-93.

Wardle, M. C., Yang, A., & de Wit, H. (2012). Effect of d-amphetamine on post-error slowing in healthy volunteers. *Psychopharmacology*, *220*, 109-115.

Wassermann, E. M. (1998). Risk and safety of repetitive transcranial magnetic stimulation: report and suggested guidelines from the International Workshop on the Safety of Repetitive Transcranial Magnetic Stimulation, June 5–7, 1996. *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section*, *108*, 1-16.

Weir, P.L., Mackenzie, C.L., Marteniuk, R.G. & Cargoe, S.L. (1991a). Is object texture constraint on human prehension? *Journal of Motor Behavior, 23*, 205-210.

Wessel, J. R., Danielmeier, C., Morton, J. B., & Ullsperger, M. (2012). Surprise and error: common neuronal architecture for the processing of errors and novelty. *Journal of Neuroscience*, *32*, 7528-7537.

West, R., & Travers, S. (2007). Tracking the temporal dynamics of updating cognitive control: An examination of error processing. *Cerebral Cortex*, *18*, 1112-1124.

Williams, P., Heathcote, A., Nesbitt, K., & Eidels, A. (2016). Post-error recklessness and the hot hand. *Judgment and Decision Making*, *11*, 174.

Winkel, J., Wijnen, J. G., Ridderinkhof, K. R., Groen, I. I., Derrfuss, J., Danielmeier, C., & Forstmann, B. U. (2009). Your conflict matters to me! Behavioral and neural manifestations of control adjustment after self-experienced and observed decision-conflict. *Frontiers in Human Neuroscience*, *3*, 57.

Wong, A. L., Haith, A. M., & Krakauer, J. W. (2015). Motor planning. *The Neuroscientist*, *21*, 385-398.

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: conflict monitoring and the error-related negativity. *Psychological Review*, *111*, 931.

Zhang, W., Gordon, A. M., Fu, Q., & Santello, M. (2010). Manipulation after object rotation reveals independent sensorimotor memory representations of digit positions and forces. *Journal of Neurophysiology*, *103*, 2953-2964.

PUBLICATIONS ASSOCIATED WITH THE THESIS

Ceccarini, F., & Castiello, U. (2018). The grasping side of post-error slowing. *Cognition*, *179*, 1-13.

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