Conflict In Unidimensional Task Settings

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List of Abbreviations

ABI	alternation based interference
ACC	anterior cingulate cortex
AFM	additive factors method
ANOVA	analysis of variance
cm	centimetre
DC	direct current
DLPFC	dorsolateral prefrontal cortex
EEG	electroencephalogram
EOG	electrooculogram
ERN	error related negativity
ERP	event related potential
fMRI	functional magnetic resonance imaging
FO	first-order
Hz	Hertz
LRP	lateralized readiness potential
M	mean
ms	millisecond
MSE	mean square error
MT	movement time
µV	microvolt
p	probability
PET	positron emission tomography
PFC	prefrontal cortex
PRP	psychological refractory period
R	response
RSI	response stimulus interval
RT	reaction time
S	stimulus
SAT	speed-accuracy trade-off
SO	second-order

1. Introduction

Making fast and accurate decisions is an essential part of everyday life. However, decision making is often hindered by the presence of ambiguous information and opponent response alternatives. For example, when driving on the motorway to an unfamiliar destination, we might be unsure about which exit to take. A fellow passenger tells us to leave at the next exit; however, we believe that the subsequent exit is the correct one. In such a situation we have to choose between conflicting alternatives: “staying on the motorway” versus “leaving the motorway”. A possible way of resolving such a discrepancy is to focus on the most relevant information, while ignoring irrelevant and distracting information. When we are confident that we are correct about which exit to take, it is easier to ignore the fellow passenger. However, in situations of high uncertainty, it is likely that we will choose the wrong exit or even worse, cause an accident. Managing conflict between response alternatives is assumed to be one of the central functions of cognitive control (Allport, 1980; Neumann, 1987; Norman & Shallice, 1986; Yeung, Botvinick, & Cohen, 2004).

Despite having negative effects on decision making and increasing the likelihood of an error, conflict has recently been re-evaluated as a key signal that can trigger compensatory adjustments which help to deal with such situations. The conflict monitoring theory specified this beneficiary influence of conflict in a conflict-control loop, where conflict signals the need for top-down attentional adjustments and subsequently triggers the up-regulation of cognitive control (Botvinick, Braver, Barch, Carter, & Cohen, 2001; van Veen, Cohen, Botvinick, Stenger, & Carter, 2001). Within this model, conflict is detected in the anterior cingulate cortex (ACC). After conflict detection, the ACC signals the need for control to other brain areas, namely, the prefrontal cortex (PFC). Subsequently, the PFC is responsible for resolving conflict and regulating behavioural adjustments (Carter & van Veen, 2007; Kerns, et al., 2004). Thus, following the detection of conflict, behavioural actions are implemented by increasing control levels. To return to the motorway scenario described above, the perceived conflict between “leaving the motorway” and “staying on the motorway” should trigger subsequent behavioural adjustments. For example, we may reduce speed in order to have sufficient time to read the traffic signs at the motorway exit.

The identification of neural substrates underlying conflict processing has substantially increased the interest in conflict research. However, despite an upsurge in the number of studies investigating conflict, some fundamental basics of the concept are still under debate. One key question that is central to ongoing research is the question concerning the locus of conflict origin within information processing. Another up to date question is how conflict eventually leads to beneficial consequences for subsequent

behaviour via control mechanisms. In this context, whether control processes are implemented strategically or automatically after conflict detection is of particular interest. Moreover, the nature of these control mechanisms (i.e. where and how they actually affect information processing) is still under debate. Conflict is traditionally studied in paradigms where a relevant and an irrelevant response dimension are present (e.g. Stroop, Flanker, Simon task). The relevant and irrelevant dimensions can interfere with each other and cause conflict. However, conflict and conflict adjustments were also shown to play a role in uni-dimensional task settings. Here conflict and conflict adjustments can explain post-error slowing effects (Botvinick, et al., 2001) and sequential effects, such as alternation-based interference (Jentzsch & Leuthold, 2005). This thesis will address questions concerning the origin of conflict during information processing in uni-dimensional task settings, the relationship between conflict and cognitive control, the nature of control mechanisms and possible limitations of the conflict monitoring approach.

1.1. Information Processing

Within cognitive psychology, cognition is considered computational in nature, with cognitive processes resembling an information processing procedure. From stimulus input (such as the fellow passenger saying “we should leave the motorway”) to the decision that is made and to the execution of the action, it is generally assumed that several phases of information processing have to be completed. For example, a common information processing architecture of a simple cognitive process may involve the following: first, the stimulus has to be perceived (perceptual stage), second, a decision has to be made (decision stage), and finally, a response will have to be executed (motor stage) (e.g. Donders, 1969). A key question that is central to the nature of information processing concerns whether information processing takes place in a serial or parallel fashion, and whether information transfer from one stage to another is continuous or discrete (Ratcliff, Van Zandt, & McKoon, 1999). In serial models, information processing takes place sequentially without temporal overlap between stages (e.g. Sternberg, 1969). In contrast, parallel processing models propose that information processing can take place simultaneously (e.g. McClelland, 1979). The difference between discrete and continuous models of information processing refers to assumptions regarding the way information is accumulated and transferred. In discrete models, information accrual occurs in distinct steps. In contrast, information accrual within continuous models is a gradual process that occurs over time.

Laming (1968) described decision making processes in simple reaction time (RT) tasks in the random-walk model. In this model the decision process, which equals an

information accumulation process, is required in order to distinguish noise from signal. It is assumed that information is abstracted from the signal in a stream of independent observations as long as a signal is present (Laming, 1968). These single observations are summed and the decision process is based on the principle that noise will average to zero (e.g. Sikström, 2004). Such a stochastic process can result in random fluctuations of information accumulation (see Figure 1). Information accumulation begins at a starting point and continues until the amount of accumulated information reaches one of the response boundaries. At this point, the response will be initiated. The reached threshold can be either the correct threshold (correct response initiated), or the opposite threshold (wrong response initiated). The drift rate of the information accumulation process determines the time needed to reach the boundary (i.e. response threshold) and thus determines the decision time, whereas the accumulated information determines which response will be executed. Importantly, RT measurements consist of sensory processing time (input time), decision time, and the time required to execute the response (movement time). In Laming's model, input time and movement time are independent of experimental manipulations, and thus are expressed by adding a constant amount to the decision time in order to determine RTs. Diffusion models (e.g. Ratcliff, et al., 1999) can be seen as an extension of the discrete version of the random-walk model first described by Laming.

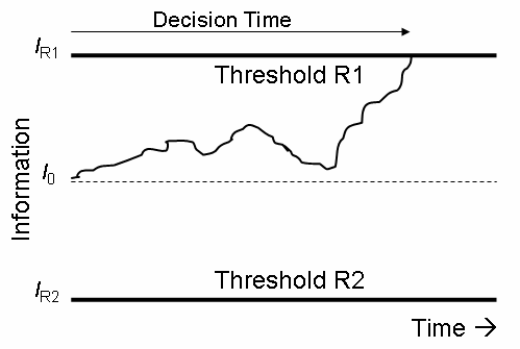

Figure 1. Information accumulation process in a random-walk model (adapted from Laming, 1968). Information (I) accumulates over time starting at I_0. Once either threshold R1 (I_{R1}) or R2 (I_{R2}) is reached, the response will be initiated. Errors occur if the wrong response threshold is reached.

Increasingly, these models are aimed to close the gap between the model's features, behavioural phenomena and the proposed underlying neural processes and brain anatomy. For example, the popular connectionist model (Rumelhart & McClelland, 1986) proposes that information is processed in a distributed fashion throughout the brain, and that processing in various processing units can take place in parallel. In this model

information accumulation for the decision process is described as the pattern of activity in a neural network. The different units are connected, and the connections can be essential for parameters like, how accessible the information is, and how much a certain process in a specific information processing unit will influence the overall information processing procedure.

Despite ongoing debates about the nature of human information processing mechanisms (e.g. Ratcliff, et al., 1999), there are several similarities between popular information processing models, such as the connectionist and the diffusion models. For example, information processing models generally explain response latencies and response accuracy on the basis of information or activity accumulation over time (e.g. Laming, 1968; McClelland, 1979; Ratcliff, et al., 1999). Performance is determined by the speed and quality of information accrual in a central decision process. Additionally, it is proposed that certain thresholds of information have to be reached before the according response (correct or incorrect) will be triggered. Therefore, response latencies depend on the level of response threshold and speed of information accrual. A higher threshold requires a higher level of information accrual before a response will be executed. Moreover, quicker information accumulation results in shorter response latencies. Lower thresholds should result in faster responses, but also increase the likelihood that the response will be erroneous.

Further assumptions are integrated within most information processing models. For example, it is assumed that available processing capacity is limited (e.g. Kahneman, 1973), and thus only a limited amount of capacity requiring processes can be active at a time point. Moreover, it is generally accepted that an instance of control is needed in human information processing theories, which guides and directs information processing consistent with task instructions, internal goals or plans.

1.2. Cognitive Control

The executive system, also referred to as cognitive control, forms part of our cognitive system that allows us to act flexibly and to adapt behaviour appropriately to novel situations. Cognitive control is essential to overcome predominant, reflexive and habitual response tendencies, to focus on relevant information, to ignore irrelevant stimuli, to direct information processing within a goal-relevant context, to set high level goals and planning of behaviour and direct other cognitive systems in accomplishing those goals (Carter, et al., 2000; Logan, 1985; E. K. Miller, 2000; Monsell & Driver, 2000; Shallice, 1988). The concept of controlled or directed information processing can be traced back to James (1890). James suggested that only selected items are processed to a higher level and build the basis of experience: “My experience is what I agree to attend to. Only those

items which I notice shape my mind – without selective interest, experience is an utter chaos" (James, 1890, p. 402). In summary, cognitive control elevates information processing beyond automated stimulus-response associations and mere replay of well-learned sets of behaviour and is essential to overcome processing conflicts.

A distinction between controlled and automated processing styles has been integrated in many information processing models developed in the 1970s. For example, the two-process information processing theory by Shiffrin and Schneider (1977) outlines in detail the differences between automated and controlled perceptual selection, and the benefits of a processing system that is able to switch between these processing styles. Automated processing is fast, can evolve in parallel and is not capacity dependent. In contrast, controlled processing requires attention, is capacity limited, and these limitations prevent several control processes occurring simultaneously. As controlling behaviour is an essential but also effortful process that requires time and cognitive capacities, it would be wasteful and inefficient to keep control levels high under all circumstances (Matsumoto & Tanaka, 2004; Shiffrin & Schneider, 1977). Indeed, in certain circumstances it might be useful to react automatically in order to save cognitive resources for other processes.

Although various information processing models emphasise a distinction between controlled and automated processing styles (Baddeley & Della Sala, 1996; Norman & Shallice, 1986; Shiffrin & Schneider, 1977), these models focus on how control influences and modifies processing, leaving open the question regarding how the need for control is dynamically detected and how control adjustments are regulated. One of the foremost researchers who discussed dynamic adjustments in the allocation of mental resources was Kahneman (1973). He proposed that tasks are evaluated for their demand on mental capacity. According to task demands and intentions, a central processor allocates cognitive resources. Some tasks can be performed relatively automatically and thus, no allocation of cognitive resources takes place. In contrast, attempting to perform a challenging task leads to the allocation of cognitive resources. This approach can explain an overall difference in control activation levels between tasks, but does not outline how such a mechanism functions dynamically on a trial-to-trial basis.

1.3. Conflict Monitoring and the Prefrontal Cortex

A major criticism of traditional information processing models is that they often assume that the cognitive system "somehow knows" when control is required and assign control recruitment to an underspecified homunculus-like agent (Newell, 1980), without specifying the dynamics of control regulations. The conflict monitoring theory (Botvinick, et al., 2001) provides an explanation of how control processes are dynamically

implemented within a broad range of experimental paradigms. The two key components for dynamic trial-to-trial control adjustment are the online monitoring of performance that evaluates for the need for control, and a system that subsequently implements control appropriately (Botvinick, et al., 2001; Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004). *Conflict* is the crucial variable in this model, notifying the cognitive system about the need for control. It is proposed that information processing is constantly monitored for processing conflicts. After conflict is detected, subsequent behavioural adjustments are triggered via the up-regulation of cognitive control.

The conflict monitoring model of Botvinick et al. (2001) outlines a distributed processing network within the brain, which involves both the detection of conflict and resulting processing adjustments (see Figure 2). Response conflict is evaluated within the ACC at the level of response decision. This produces subsequent response adjustments that are implemented by the PFC. In the succeeding sections the following questions central for this theory are addressed: How does conflict arise during information processing? Where is conflict detected and what are the underlying neural substrates? How are post-conflict control adjustments implemented?

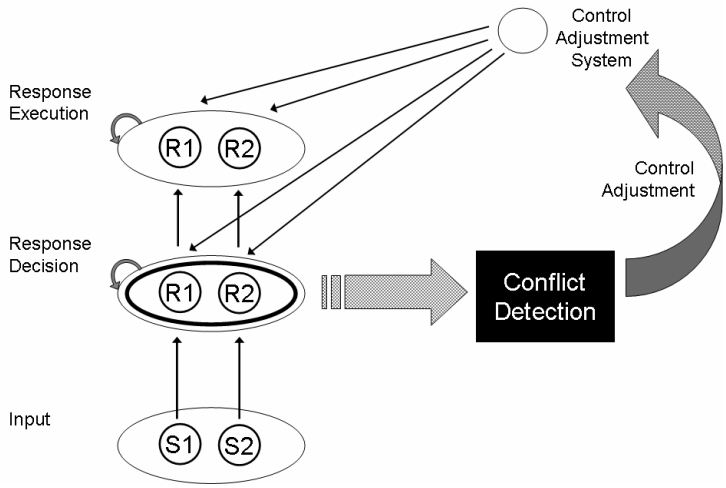

Figure 2. Schematic diagram of the conflict monitoring model architecture (adapted from: Jones, Cho, Nystrom, Cohen, & Braver, 2002). In the original model, the control adjustment system is referred to as a strategic priming unit. Conflict is measured at the response decision layer within the ACC, and subsequent control adjustments are triggered within the control adjustment system in the PFC.

1.3.1. Evaluative Aspect of Conflict Monitoring

The conflict monitoring system requires that conflict is detected and evaluated during information processing. In the following paragraphs the conceptualization of conflict in the

conflict monitoring theory (Botvinick et al., 2001) and the proposed neural system underlying conflict detection will be introduced.

1.3.1.1. Theoretical Aspects of Conflict

Conflict was defined by Yeung, Botvinick and Cohen (2004) as the "coactivation of mutually incompatible responses" and is assumed to arise because of constraints on appropriate response generation in trial N (Jones, et al., 2002). It is proposed that evidence for alternative responses accumulates in separate Stimulus (S) – Response (R) pathways, with activation in each pathway decaying over time if there is no further input. In choice RT tasks this can lead to time periods where incompatible response units are activated simultaneously, thus producing conflict. Although the conflict monitoring unit is proposed to measure conflict at a response level, crosstalk can occur at all levels of information processing, from stimulus identification, memory retrieving levels and response selection.

The conflict monitoring model is built on basic assumptions about information processing within a connectionist framework (e.g. McClelland, 1992; Rumelhart & McClelland, 1982). Connectionist models assume that information processing takes place in parallel, is distributed, interactive and continuous (Ratcliff, et al., 1999). The conflict monitoring model consists of identical processing units, which communicate with each other through inhibitory and excitatory connections, whereby representations of alternative responses are connected through inhibitory weights. Effectively, if the task demands two different choice responses, there are two such units, one for each response. Conflict is conceptualized as energy in a inhibitory network of alternative response units (Hopfield, 1982). Hopfield's energy is a combined measure over all activity units within the network of interest and with respect to their connections. High conflict, meaning high energy, results from simultaneous high activity in reciprocal inhibitory connected units (see Equation 1). Thus, conflict is not a set parameter in the model, but can be measured by the relative activity of alternative response units (Yeung, et al., 2004). Conflict is not present when only one unit is active and rises when both units are active. Conflict is particularly high, when both response units are similarly active and when the overall activity is high in both units (Botvinick, et al., 2001).

$$Conflict = E = -\sum_{i \neq j} \sum e_i e_j w_{ij}$$

Equation 1. Conflict is calculated as energy (E) within the response layer. The subscripts *i* and *j* represent the units of interest. *e* is the activity within a unit, *w* is the weight between a pair of units, representing the strength of the connection between the units (Hopfield, 1982).

1.3.1.2. Neural Correlates of Conflict Detection

In order to adjust the level of control to the actual conflict level detected, the regulating unit requires access to the current state of the cognitive system in order to assess if control is required. Therefore, the conflict monitoring theory proposes a conflict monitoring unit, which detects conflict as activation spreads throughout the network, and sends this conflict signal to brain areas responsible for regulative adjustments. This conflict monitoring process is proposed to be a function of the ACC, which is located adjacent to the corpus callosum on the medial surface of the frontal lobes (e.g. Harrison & Gittins, 2003).

It was proposed early on that the ACC plays an important role in controlling behaviour (e.g. LaBerge, 1990). For example, one study analyzed the functional anatomy of selected and divided attention in a visual discrimination task of shape colour and speed using PET (Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1991). In extra-visual brain areas, selective and divided attention showed different activation patterns, suggesting that attentional strategies are reflected in different brain networks. Only in the condition where participants had to divide their attention to more than one stimulus feature, was activation reported in the ACC and the PFC. LaBerge (1990) argued that selective attention to one stimulus feature can be automated quickly, while in contrast, dividing attention to more than one stimulus feature is harder to automate, which explains the ACC activation. Further studies showed ACC activation in language tasks, tasks involving memory and learning, perceptual target detection tasks and other tasks activating mental capacities (for a review, see Cabeza & Nyberg, 1997, 2000).

However, given the wide variety of tasks and experimental settings where ACC activity was reported, it remained unclear what its specific function is and if there is a common mechanism triggering ACC activation. Botvinick et al. (2001) reviewed ACC findings, and proposed that “ACC activation can be understood as a response to the occurrence of conflict during information processing”. Botvinick et al. (2001) proposed that tasks reporting ACC activity can be grouped into paradigms where participants have to overcome proponent or automated response alternatives, choose between undetermined responses, and in situations where errors occur.

1.3.2. Executive Aspect of Conflict Monitoring

Importantly, in contrast to previous models of cognitive control, which focus on how processing changes in controlled and automated styles, the conflict monitoring model specifies the dynamics of control adjustments in a trial-to-trial fashion. After conflict is detected in trial N, it is assumed that this information is passed to other brain systems, where control is recruited and according behavioural adjustments will be implemented in

trial N+1. In the following paragraphs the theoretical aspects of control adjustments and the neural systems involved in regulating these adjustments will be introduced.

1.3.2.1. Theoretical Model of Post-Conflict Adjustments

Computationally, control levels are calculated as described in Equation 2, taking into account that shifts in the level of control can take place gradually over a series of trials (Botvinick, et al., 2001). Therefore, the control level C in trial N+1 is not only determined by the conflict level in the directly preceding trial, but also by the conflict level in the preceding sequence of trials. Control in trial N+1 is calculated as the sum of the control level in trial N and the conflict energy in trial N. λ is limited to values between 0 and 1, reflecting that control is based on the exponentially weighted average of conflict levels in the sequence of preceding trials (Botvinick et al., 2001). If λ is close to 1, control shifts are gradual or non existent. In contrast, a λ value close to 0 expresses abrupt changes in control level. Another important aspect of this measure of control is that it can be shifted in both directions: After a high conflict trial control levels will be raised, while after a low conflict trial control will be lowered.

$$Control(N+1) = C(N+1) = \lambda C(N) + (1-\lambda)(\alpha E(N) + \beta)$$

Equation 2. Control (C) in trial N+1 is calculated as the sum of conflict (E) and the control level (C) in the previous trial N. α and β are scaling parameters. The value of λ ranges between 0 and 1 indicating that control shifts take place more (λ close to 1) or less (λ close to 0) gradually.

1.3.2.2. Neural Correlates of Post-Conflict Adjustments

An area thought to be highly involved in implementing control adjustments is the PFC (e.g. Cohen, Braver, & O'Reilly, 1996; Kerns, et al., 2004; E. K. Miller, 2000; Norman & Shallice, 1986). The PFC is a set of neocortical areas, that are highly interconnected and connected with sensory and motor neocortical areas, and additionally with many subcortical areas such as the hippocampus (e.g. Fuster, 2001; E. K. Miller, 2000). The high connectivity of the PFC makes it the ideal structure to integrate information and guide information processing as needed for complex behaviour (E. K. Miller, 2000). Miller (2000) reviewed findings showing the importance of the PFC for cognitive control. For example, people with PFC damages are often easily distracted and have difficulties ignoring irrelevant novel events (e.g. Kane & Engle, 2002). In addition to the underlying neural connections, Botvinick et al. (2001) suggested that indirect evidence for the conflict monitoring network between detection of conflict within the ACC and control

implementation within the PFC can be taken from studies often showing coactivation of the ACC and the PFC (e.g. Braver, et al., 1997; Raichle, et al., 1994).

1.4. Conflict Monitoring in Multi-dimensional Task Settings

The conflict monitoring model was built mainly upon findings from interference paradigms. Such paradigms are typically multi-dimensional in nature, where more than one response alternative can be activated within a trial, for example the Stroop, the Eriksen Flanker and the Simon paradigm. Importantly, the conflict monitoring model gives a unified account for explaining a wide range of studies reporting ACC activation and behavioural findings, which previously have been seen as rather isolated phenomena. Critically, the conflict monitoring model predicts both behavioural adjustments after conflict detection and changes in brain activation. Behavioural changes after conflict should be the result of an up-regulation of cognitive control. Changes of neural activity are twofold; first, changes related to conflict evaluation in the ACC and second, changes related to the implementation of control adjustments in the PFC. In the following paragraphs the findings regarding conflict monitoring in multi-dimensional tasks settings will be summarized.

1.4.1. Interference Paradigms

One of the most famous interference paradigms is the Stroop paradigm (Stroop, 1935). In the Stroop task, participants are asked to name the colour in which a word is printed and to ignore the semantic meaning of the word. Incongruent trials consist of a word such as green which is printed in a colour different from the colour expressed by the words semantic meaning (e.g. the word "green" printed in blue). Typically, incongruent trials result in a processing delay and less accurate performance. Traditionally it is argued that the automatic tendency to read the word interferes with the process of naming the font colour, resulting in processing interference (e.g. Posner & Snyder, 1975). According to the conflict monitoring model (Botvinick et al., 2001), conflict is high in incongruent trials as the two input units for “display colour” and “word unit” simultaneously activate opponent response alternatives. Interestingly, this interference effect is subject to global control levels. For example, Logan and Zbrodoff (1979) found reduced interference effects in a Stroop task when incongruent trials were presented more frequently than congruent trials. From this, it was proposed that with a high frequency of incongruent trials, participants keep their overall control levels high and therefore, succeed better in inhibiting automatic response tendencies.

Similar effects have been reported in the Simon task (Simon, 1990), where stimuli are mapped onto two response alternatives (left vs. right key) and are presented

at either left or right screen locations. Participants are asked to respond to the identity of the stimulus (shape, colour, etc.). Despite stimulus location being irrelevant, it cannot be ignored. RTs increase when stimulus location and response location are incongruent (stimulus on the left side, response right key) (for a review, see Hommel & Prinz, 1997). Importantly, the Simon effect also diminishes as the frequency of non-corresponding trials increases (e.g., Stürmer, Leuthold, Soetens, Schröter, & Sommer, 2002). Again, interpretations suggest that overall control levels are kept on a high level when the probability of incongruent trials is high. As a result, overall high control levels reduce the overall processing conflict in the cognitive system.

A third typical interference paradigm is the Eriksen Flanker task (Eriksen & Eriksen, 1974). In this task participants have to respond to a central stimulus which is flanked by irrelevant stimuli. These flanker stimuli are either congruent (e.g., << $\underline{\leq}$ <<), triggering the same response, or incongruent stimuli (e.g., << $\underline{\geq}$ <<), triggering an opponent response alternative. Analogous to the Stroop and the Simon task, participants respond faster and more accurately in congruent than in incongruent trials, which is explained by difficulties in ignoring the irrelevant flanker stimuli. In the conflict monitoring theory (Botvinick, et al., 2001), incongruent trials in the Flanker task are characterised by high conflict due to opposing stimuli in the relevant and irrelevant response dimension, which results in simultaneous activation of the correct and the incorrect response alternative. Again, in conditions with high frequency incompatible trials performance in these trials is better than in conditions with low frequency incompatible trials (Casey, et al., 2000), suggesting that overall control levels are kept high if incompatible trials are likely.

1.4.2. Conflict Adaptation Effect

The conflict monitoring model predicts behavioural changes after conflict detection, implemented through the up-regulation of cognitive control, as described above. Importantly, evidence for online trial-to-trial control adjustments has been first reported in multi-dimensional task settings. It has been shown that the size of the congruency effect varies not only upon the overall proportion of congruent and incongruent trials, but also upon the trial sequence. Behavioural evidence for online conflict monitoring has been drawn primarily from the *conflict adaptation effect* (Gratton, Coles, & Donchin, 1992), calculated as follows: (RTci – RTcc) – (RTii – RTic), where ci is an incongruent trial preceded by a congruent one, cc are two consecutive congruent trials, ii two consecutive incongruent trials, and ic a congruent trial preceded by an incongruent trial. In this study participants had to perform an Eriksen Flanker task (Eriksen & Eriksen, 1974). As expected, participants were slower in incongruent trials. Importantly, Gratton et al. showed that the Flanker effect was smaller after incongruent trials. Thus, the interference

effect was reduced if a trial was preceded by an incongruent trial (see Figure 3). Botvinick et al. (2001) explained these behavioural effects with reference to conflict monitoring theory. According to the conflict monitoring theory, incongruent trials are high in conflict. Thus, incongruent trials lead to an up-regulation of control reflected in activation of attentional resources for top-down processing adjustments. As a result, high control levels result in more focused attention towards the central target location in the following trial. This increased attentional focus towards the target location reduces the influence of distracting flanker information. On the other hand, upregulation of control leads to slower responses in congruent trials following incongruent trials, due to the reduced facilitating influence of the Flanker stimuli. The conflict adaptation effect has turned into a popular measurement for cognitive control recruitment and has also been reported in the Stroop (Kerns, et al., 2004) and the Simon tasks (Notebaert, Soetens, & Melis, 2001) with the congruency effects being reduced or absent after incongruent trials.

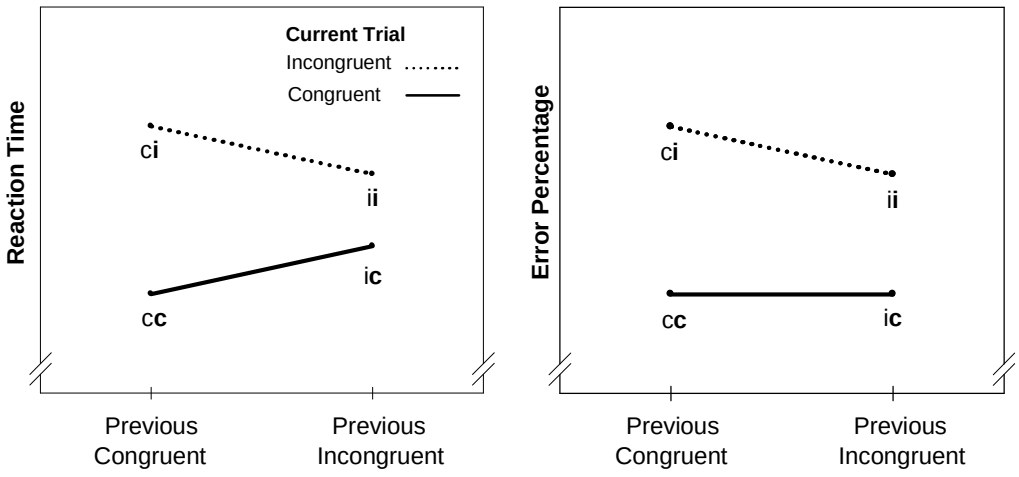

Figure 3. Conflict adaptation effect as reported by Gratton, Coles and Donchin (1992). Responses are faster and more accurate in current incongruent trials, if they are preceded by another incongruent trial rather than a congruent trial.

To sum up, there is widespread behavioural evidence in multi-dimensional task settings that support the conflict monitoring approach. However, doubts have been raised regarding whether proposed top-down control adjustments are needed to explain the conflict adaptation effect. Mayr, Awh and Laurey (2003) highlighted that 50% of the ii and cc trials are exact repetitions of both target and flanker stimuli. In contrast, none of the ic or ci trials are complete repetitions of the stimulus, and thus the conflict adaptation effect may be a confound related to bottom-up response priming. Mayr et al. investigated

whether the conflict adaptation effect also occurs if exact target and flanker repetitions from the ii and cc trials are excluded. If the conflict monitoring model holds true, the conflict adaptation effect should still be present after the exclusion of mere stimulus repetitions. In contrast, if stimulus specific response priming underlies the conflict adaptation effect, it should be absent after the exclusion of complete stimulus repetitions. The results showed that the conflict adaptation effect was absent. Thus, Mayr et al. concluded that stimulus-specific repetition priming can explain the conflict adaptation effect reported in the Flanker task. However, this absence of the conflict adaptation is not reported in all Flanker experiments (e.g. Ullsperger, Bylsma, & Botvinick, 2005). For example, Ullsperger et al., in the first of a series of Flanker experiments, found the conflict adaptation effect despite the exclusion of stimulus repetitions. In a second experiment, they increased the stimulus set by using the digit numbers 1-9, thus reducing the effect of trial-to-trial stimulus repetitions. Even after excluding all stimulus repetitions, they still found the conflict adaptation effect. The authors suggested that negative priming in sequences such as <<>>> followed by >><<>> might have obscured the conflict adaptation effect in the study of Mayr et al. In Ullsperger's et al. study, the use of longer inter-trial intervals and the rather short stimulus presentation might have reduced the influence of negative priming effects. Kerns et al. (2004) excluded stimulus repetition trials in the Stroop paradigm, and still found the conflict adaptation effect. Similarly, exact repetitions of stimuli and responses sequences are not sufficient to explain response adjustments in case of the Simon effect (Stürmer, Leuthold, Soetens, Schröter, & Sommer, 2002). In summary, although there is evidence that other factors, such as stimulus specific priming and sequential confounds concerning stimulus and response repetitions (see Mayr et al., 2003) influence response adjustments, such factors cannot explain the entire conflict adaptation effect. Thus, one can assume that control adjustments are an additional major factor in explaining the conflict adaptation effect.

1.4.3. Neural Correlates of Conflict Monitoring

In addition to the behavioural findings summarised in the previous paragraphs, neuroimaging studies provide further evidence supporting the idea of conflict monitoring and subsequent control adjustments. Pardo, Pardo, Janer and Raichle (1990) using PET, investigated brain areas responsible for conflict processing in the Stroop task. They found increased ACC activation during incongruent trials compared to congruent trials. It was concluded that the ACC plays an important role in overcoming interference due to a proponent tendency to read the word. Evidence for online trial-to-trial conflict monitoring was first drawn from fMRI studies showing ACC activation in a sequence of trials in the Flanker task (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999). Participants had to perform a typical Flanker task, whereby they had to respond to an arrow symbol flanked

by congruent or incongruent arrows (e.g., << ≤ << or << ≥ <<). ACC activity was larger in incongruent than in congruent trials, suggesting that conflict is processed within the ACC. Importantly, ACC activity was larger in c**i** trial sequences than in i**i** sequences. These findings are in line with the conflict monitoring model, as conflict is high in incongruent trials and leads to a subsequent up-regulation of control. Thus, high control levels in i**i** trials result in less conflict during information processing and thus reduced ACC activity. Botvinick et al. concluded that ACC activity most likely reflects conflict processing in the current trial, whereby the amount of conflict is determined by actual control levels.

Importantly, conflict monitoring theory suggests a dissociation between the role of the ACC in conflict evaluation and the role of the PFC in regulating control adjustments. Dissociations between the ACC and the PFC activity have been reported in a task-switch version of the Stroop paradigm (MacDonald, Cohen, Stenger, & Carter, 2000). Participants were provided with an instruction before each trial indicating whether they should read the word (automated task) or name the colour (control demanding task). Using fMRI measurements, activity during the preparation period was compared to stimulus related processing after trial onset. Results showed that the dorsolateral prefrontal cortex (DLPFC) was highly activated in the preparatory period, especially for the colour naming task. Thus, it was concluded that the DLPFC is involved in top-down control implementation, through actively representing the demands of the less automated task. In line with this suggestion, largest DLPFC activation during the preparatory phase resulted in the smallest Stroop interference effect in this trial. In contrast, ACC activity was evident after stimulus onset only, and it was especially high in incongruent colour naming trials. From this it was concluded that the ACC is involved in conflict processing, whereas the DLPFC is involved in implementing control adjustments.

More than a mere dissociation, the conflict monitoring model predicts an increase in control related activation after conflict detection. More specifically, in this model the outcome of conflict evaluation within the ACC is signalled to the PFC, which subsequently implements post-conflict control adjustments. This interaction between the ACC and the PFC was investigated by Kerns et al. (2004) in the Stroop task. As expected, high conflict incongruent trials showed increased ACC activity, which is likely to reflect conflict processing. Trials following high conflict trials showed behavioural adjustments (i.e. conflict adaptation effects) in conjunction with increased PFC activation. In conclusion, these findings suggest the ACC to be involved in conflict detection and the PFC to be responsible for subsequent allocation of control. Similar activation patterns have been reported in a fMRI study in the Flanker task (Durston, et al., 2003). With an increasing number of compatible trials preceding an incompatible trial, RTs on the incompatible trial increased. In parallel the activity in the ACC, the DLPFC and the superior parietal cortex increased. Of particular interest was the timing and the duration

of activation in these brain parts. Both the ACC and the DLPFC were activated before the superior parietal cortex. ACC activity was restricted in time, whereas PFC activity remained high for a longer time. This is in line with the conflict monitoring theory, proposing that the preliminary function of the ACC is conflict detection. In contrast the PFC is involved in regulating subsequent post-conflict adjustments. It was proposed that these adjustments take place through top-down attentional biasing via the superior parietal cortex (Durston, et al., 2003).

1.5. Conflict in Unidimensional Task Settings

So far conflict processing in multidimensional task setting, where relevant and irrelevant information activate opposing response alternatives, has been introduced. However, it has recently been shown that conflict related response adjustments can also occur in tasks where only task relevant information is presented in each trial, and hence, no overlap between relevant and irrelevant response dimensions occur (e.g. Jentzsch & Leuthold, 2005). One example of conflict in uni-dimensional tasks are error trials. It is suggested that the activation of the executed erroneous response overlaps with the activation of the required correct response. This results in conflict and subsequent behavioural adjustments, for example, post-error slowing (e.g. Botvinick, et al., 2001; Brewer & Smith, 1984; Rabbitt, 1966). Besides error processing, it has been suggested that sequential effects in uni-dimensional task settings, traditionally explained by automated bottom-up facilitations (e.g., Kirby, 1976), can also be attributed to control adjustments under certain circumstances (e.g. Jentzsch & Leuthold, 2005). The following section will introduce in detail the above two phenomena of conflict in uni-dimensional task settings which are central for this thesis.

1.5.1. Alternation Based Interference and Conflict Monitoring

Jentzsch and Leuthold (2005) reported that responses are consistently slower when trial N follows a response change rather than a response repetition from trial N-2 to trial N-1 (see also: Jentzsch & Sommer, 2002; Melis, Soetens, & van der Molen, 2002; Soetens, Deboeck, & Hueting, 1984; Sommer, Leuthold, & Soetens, 1999; Vervaeck & Boer, 1980). This effect, termed alternation-based interference (ABI), was largely independent of the nature of the first-order S-R transition (relationship between trial N-1 and trial N). Jentzsch and Leuthold (2005) suggested that the ABI effect is a form of top-down control adjustment. More specifically, Jentzsch and Leuthold (2005) suggested that in the case of a two-choice uni-dimensional task, conflict arises from information processing within separate S-R pathways, defined by the alternative stimuli (e.g., A, Y) and responses (left, right). Information is conceived to accumulate within each processing pathway and is

subject to decay once no further input is received (see Figure 4). Importantly, activation within the response pathways is assumed to extend beyond response activation and to decay gradually over time. Evidence for sustained response activation was drawn from an electrophysiological study conducted by Jentzsch and Sommer (2002). Jentzsch and Sommer showed that amplitude developments of the lateralized readiness potential (LRP) depend on the event history and can be accounted for by accumulation of residual activation traces from preceding trial activation. Thus, in rapidly unfolding event sequences where the RSI interval is short, activation from previous events can interact with current activation. This will generate processing conflicts that vary as a function of the specific event history. In the case of a response alternation from trial N-2 to trial N-1, residual activation of the response to trial N-2 and activation of the alternative response in trial N-1 result in strong conflict, since conflict is assumed to be high when alternative response paths are simultaneously activated (e.g. Botvinick, et al., 2001; Jones, et al., 2002). The performance monitoring system detects this conflict and signals this to other parts of the cognitive system, which subsequently adjust information processing. In this case, control adjustments manifest as a RT slowing in trial N (ABI effect) (see Figure 4, lower panel).

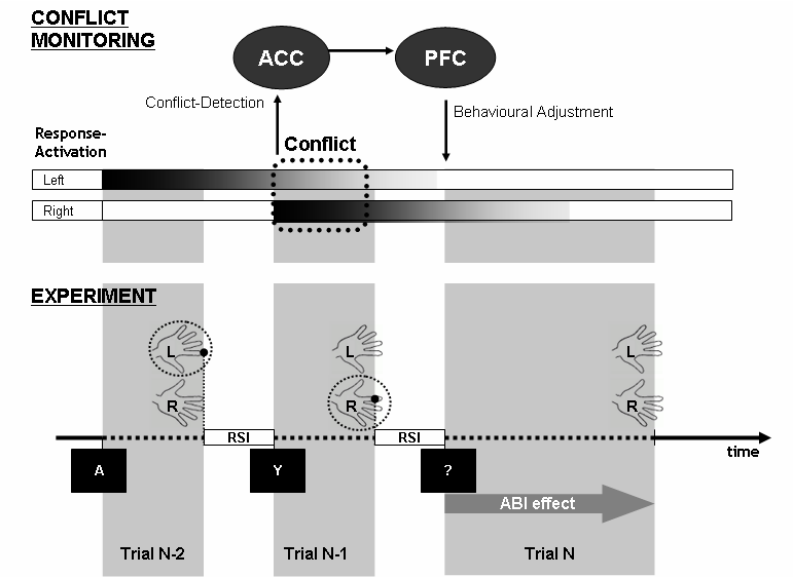

Figure 4. Upper panel: Conflict monitoring model - overlapping response activation from trial N-2 to trial N-1 results in conflict and triggers subsequent behavioural adjustments in trial N. Lower panel: Example of a response alternation trial from trial N-2 to trial N-1 resulting in the ABI effect in trial N. '?' stands as a placeholder for the stimulus in trial N, indicating that the ABI effect is idependent of the first-order transition. ACC = Anterior Cingulate Cortex; PFC = Prefrontal Cortex; RSI = Response Stimulus Interval.

In the original experiments it was not clear whether the interference results from stimulus or response changes. In order to differentiate between these two alternatives, Jentzsch and Leuthold (2005) used the information reduction paradigm (e.g. Bertelson, 1965), in which four stimuli are mapped to two response alternatives. Such a paradigm allows one to distinguish between independent influences of stimulus (S) sequence and response (R) sequence on information processing. To do so, trial transitions are split into three categories: identical transitions, where stimulus and response are repeated; equivalent transitions, where the stimulus changes but it is associated with the same response; and different transitions, where both stimulus and response change. Responses were found to be slower in trial N when the response alternated from trial N-2 to trial N-1 (different-X), as compared to when the response was repeated (identical-X and equivalent-X) (see Figure 5). Hereby X stands as placeholder for first-order trial transition from trial N-1 to trial N, which could be either, identical, equivalent or different. In a series of four experiments, Jentzsch and Leuthold (2005) provided strong evidence for the assumption that the ABI effect results from processing adjustments following response conflict in the preceding trial. That is, they consistently demonstrated that a response change rather than a mere stimulus change was necessary to generate the ABI effect. In addition, manipulations that increase conflict strength (e.g. S-R incompatibility) result in larger ABI effects. Based on this evidence, and in line with the cognitive control theory, the authors suggested that the ABI effect is caused by processing adjustments following conflicts between competing response activations.

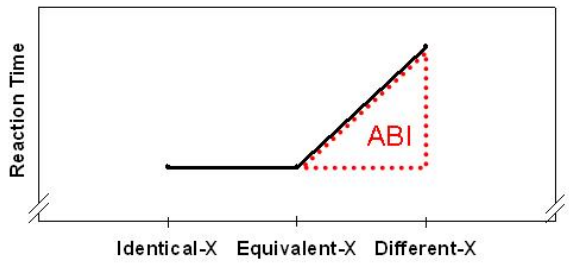

Figure 5. Alternation based interference effect (ABI) as reported by Jentzsch and Leuthold (2005). Responses are slower in trials following response alternations (different-X), than in trials following stimulus and the response repetitions (identical-X) and in trials following a stimulus change and a response repetition (equivalent-X).

The fact that the ABI effect is response related excludes alternative explanations such as the *noise* hypothesis (Laming, 1968; Soetens, et al., 1984). Laming originally proposed the idea that before a stimulus can be identified and an appropriate response executed, an internal representation of the stimulus has to be built. The harder it is to build an

internal representation of a stimulus, the longer it will take us to respond. If a stimulus is successively repeated, the internal representation of the stimulus is sharpened, thus easing stimulus identification. In contrast, in case of a response alternation the representation of both stimuli is weak. This is, as the previous stimulus looses it sharpness built up via a series of stimulus repetitions, the new stimulus has not yet built a sharp internal standard. Thus, after an alternation trial, the representation of both standards will be rather weak and stimulus identification will be difficult, possibly resulting in the ABI effect. Critically, such an approach predicts the source of the ABI effect to be stimulus-located, and also the behavioural delay in the subsequent trial to be due to a delay in stimulus processing.

Alternative explanations for the ABI effect, such as a post-execution bottleneck (e.g. Jentzsch, Leuthold, & Ulrich, 2007), can also be excluded. Kirby (1980) assumed that a response monitoring process of limited-capacity might consume more time after alternations than repetitions, thereby causing the sequence-based delay in RT, i.e. the ABI effect. This occurs via a postponement of response selection in the actual Trial N until response monitoring from Trial N-1 is finished. Such a post-execution bottleneck hypothesis predicts an underadditive effect between stimulus contrast and the ABI effect (for details, see Jentzsch & Leuthold, 2005). However, this alternative was ruled out as an explanation of the ABI effect by Jentzsch and Leuthold, due to the result of additivity between the ABI effect and a manipulation of stimulus contrast.

Taken together, the ABI effect is one example of a post-conflict adjustment occuring in a uni-dimensional task setting, whereby it is assumed that conflict arises through overlapping activation in response alternation trials.

1.5.2. Errors and Error Processing

Error processing is another example of conflict processing. The commission of an error can have severe consequences in terms of both money and human life. An improved understanding of the mental processes accompanying erroneous decisions can help to reduce error occurrence and minimise negative consequences. Although recent studies have often investigated error processing in multi-dimensionsal task settings, originally error processing was studied uni-dimensional tasks (e.g., Rabbitt, 1966).

Error occurrence has been comprehensively discussed within various information processing models (e.g. Laming, 1968; Ratcliff, 1978; Ratcliff & Rouder, 1998; Ratcliff, et al., 1999). Within the framework of the random walk model, Laming (1968) describes wrong expectations as a possible factor contributing to error occurrence. Wrong expectations shift the starting point of evidence accumulation towards the incorrect response threshold, and this can result in premature execution of the wrong response.

Similarly, McClelland (1979) explained error occurrence by a variability of activation within the response unit. More specifically, McClelland outlined that priming effects, expectations or anticipations may change the baseline activation in a response unit, which then increases the likelihood that the wrong response will be executed. Besides changes in baseline activation, errors are often explained by changes in response strategy. It is well know, that participants can follow different speed-accuracy instructions (e.g. Gehring, Goss, Coles, & Meyer, 1993). When speed is emphasized over accuracy, participants tend to respond fast but also commit more errors. Alternatively, when accuracy is emphasized over speed, participants respond rather slowly but errors are rare. These tradeoffs between speed and accuracy (SAT) are usually explained by strategic changes in the settings of the response thresholds (e.g. Usher, Olami, & McClelland, 2002). In the accuracy condition, response thresholds are raised, resulting in increased RTs and less errors. The opposite is the case in the speed condition with lower response thresholds and thus, faster and less accurate responses.

The specifics of the diffusion model in explaining errors were outlined by Ratcliff and Rouder (1998). As accumulation of information is not constant, but varies over time and from trial to trial, this variability can contribute to error occurrence. Variability in drift rates across trials gives rise to relatively slow errors. In contrast, variability in the starting point for evidence accumulation in each trial gives rise to relatively fast errors. In summary, error occurrence and RTs of error responses are determined by the variability of the starting point of evidence accumulation, the boundary positions and the drift rates.

Importantly, the idea that errors and information processing conflict are closely linked is a popular conception within cognitive psychology. Indeed, conflict is regarded as the major factor contributing to error occurrence (e.g. Carter, et al., 1998b). Error processing has also recently been discussed by Botvinick et al. (2001) in the context of conflict monitoring. In the following paragraphs behavioural effects accompanying error occurrence will be introduced. Following this, the role of error occurrence within traditional information processing models and in the conflict monitoring model will be discussed. Subsequently, neural correlates of error occurrence will be introduced.

1.5.2.1. Behavioural Correlates of Error Occurrence

Early investigations of erroneous decision processes focused on analyzing RTs in forced choice tasks. Rabbitt (1966) analyzed RTs in a 4-choice and a 10-choice task, where participants had to respond to a light signal by touching a corresponding position within a grid with the dominant index finger. As soon as the correct grid was touched, the next light was switched on after a 20 ms interval (response stimulus interval – RSI). These early experiments demonstrated that RTs in error trials have shorter latencies than the average correct trial. In contrast, responses following an error trial were significantly

slower than average correct RTs. This phenomenon is known as *post-error slowing*. Moreover, error correction responses were particularly fast and occurred within very short latencies (< 200 ms) after error execution.

A detailed investigation of error related RT changes was conducted by Laming (1968), using a series of line judgement experiments. He presented two standard lines at the beginning of the experimental block. During a block, single lines were presented in each trial and the participants had to judge which of the standard lines was presented by pressing one of two buttons. Throughout these experiments error trials were faster than the average RT of correct trials. In contrast, trials following an error (post-error trials) were much slower than the average RT of correct trials. This effect lasted for multiple trials after the erroneous response, before returning gradually back towards average RT. In addition, Laming reported that responses were more accurate after error trials.

Brewer and Smith (1984) studied the changes in response speed both before and after errors. Participants had to perform a 4-choice task and respond to one of four stimulus lights, aligned horizontally from left to right. Responses were compatible to stimulus location (i.e. response fingers were left-middle, left-index, right-index, right-middle finger). RSI duration was 1500 ms. Their results showed that response speed in trial N-6 before the error was already significantly faster that the overall correct RT, and that RT decreased further, being fastest on the trial directly preceding the error trial. In line with Rabbitt's (1966, 1979) and Laming's (1968) findings, RTs after errors were significantly slower than overall correct RTs.

To sum up, immediate trial-by-trial performance adjustments are happening in the context of errors. These include: (1) *Pre-error speeding* - response times in trials preceding errors being particularly fast (Brewer & Smith, 1984) (2) Error trials are particularly fast (Laming, 1968; Rabbitt, 1966) (3) *Post-error slowing* - trials following an error are particularly slow (Laming, 1968; Rabbitt, 1966).

1.5.2.2. Models of Error Processing

1.5.2.2.1. Error Detection and Conflict

As mentioned above, errors trials are a typical example of high conflict trials which can occur in uni-dimensional task settings. The conflict monitoring theory (Botvinick et al., 2001) assumes that conflict in erroneous decision is high, due to coactivation of the erroneous executed response and the unexecuted correct response. The idea that multiple response pathways can be simultaneously activated in erroneous decision processes was already suggested by Rabbitt and Rogers (1977). They introduced a parallel processing model that explained error detection through comparison of multiple response pathways. They suggested multiple independent processing pathways which accumulate evidence for the appropriate response. Each of these pathways analyses the

input signal and indicates either response R1 or response R2. Importantly, processing time varies between the pathways. If at the point in time when the response is initiated, there is more activation in the incorrect response channel, the incorrect response will be executed. Despite response initiation, other pathways will continue evaluating the input signal, acquiring additional evidence that either validates or disproves the executed response.

Evidence for the coactivation of the correct response pathway during the execution of an incorrect response, was taken from the observation that participants often perform error correction responses and the latencies of correction responses are extremely short. Often the interval between the error and the error correction response is less than 200ms, which is far below average RTs and makes it one of the fastest cognitive processes (Rabbitt, 1966; Rabbitt & Rogers, 1977; Rabbitt & Vyas, 1981; Rodríguez-Fornells, Kurzbuch, & Münte, 2002). This suggests that the correct response must already be activated in the cognitive system during the execution of the incorrect response (Rabbitt & Rogers, 1977; Rabbitt & Vyas, 1981). Moreover, participants are faster in performing the intended correct response after the detection of an error than indicating error detection via a neutral response (Rabbitt, 1968). Again this was taken as evidence that activation of the correct response takes place in parallel to erroneous response activation, and thus is quickly performed.

Rabbitt and Vyas (1981) tested the idea of ongoing stimulus evaluation in a simple RT experiment. If ongoing stimulus processing is responsible for the extremely fast error correction responses, the duration of stimulus presentation should influence the error correction rate. Indeed, the longer the stimulus was presented, the more likely that error correction takes place. Thus, the ability to detect and correct errors seems highly influenced by the opportunity to continuously process the stimulus input.

In summary, there is substantial evidence for conflict to be present in error trials via the overlapping activation of correct and erroneous response channels. This argument builds the basis for Botvinick et al.'s (2001) conflict monitoring unit to be active during error processing.

1.5.2.2.2. Response Tracking and Conflict Monitoring

In addition to the presence of conflict in erroneous trials, error trials are typically followed by particularly slow responses (e.g. Laming, 1968; Rabbitt, 1966). Rabbitt (1969) suggested that changes in response speed in erroneous trials and trials following errors are due to the ambiguous instructions often given to participants within experimental situations (e.g. "respond as fast and accurate as possible"). Later, Rabbitt proposed a trial-by-trial tracking model of this process (Rabbitt, 1979). He suggested that in order to satisfy the instructions, participants systematically speed-up responding in a trial-by-trial

fashion. This means that after each correct trial, response thresholds are gradually lowered, until becoming so low (and RT fast), that an error occurs. In trials following an error, participants return to a “safer” RT threshold in order to avoid subsequent errors. From here, the process of gradually lowering thresholds begins again, until the next error occurs. Today it is still a common assumption that shifts along a speed-accuracy tradeoff (SAT) function to less or more conservative response thresholds can explain both pre-error speedup and post-error slowing (e.g. Brewer & Smith, 1984; Jentzsch & Leuthold, 2006; Laming, 1968, 1979; Rabbitt, 1966, 1979, 1981; Vickers, 1980). However, although pre-error speedup and post-error slowing are assumed to be implemented by similar mechanisms, research has, apart from a few early studies (e.g., Brewer & Smith, 1984; Rabbitt, 1979, 1981), focused on the effects of post-error slowing.

These original ideas of a trial-to-trial tracking mechanism have been included in the conflict monitoring theory (Botvinick et al., 2001). First, errors elicit a strong conflict signal due to simultaneous activation of both representations for the executed erroneous response and the intended correct response (e.g. Gehring & Fencsik, 2004; Jones et al., 2002; Yeung, Botvinick, & Cohen, 2004). Such conflict subsequently triggers post-error adjustments, via the up-regulation of control mechanisms, to ensure more accurate future performance. Importantly, up-regulation of control is also assumed to take place through a shift along the SAT function. If control is high, response thresholds are raised, which should be expressed in slower and also more accurate responses. If conflict is low, for example, after a run of correctly performed trials, response thresholds are lowered, increasing the probability of an incorrect response. Thus, conflict monitoring could explain both why correct trials successively become faster, with pre-error trials being particularly fast, and why trials following errors are often slow. Several limitations of this SAT based explanations are outlined in the following section.

1.5.2.2.3. Limitations of the Conflict Monitoring Approach

As described above, conflict is likely to be present in error trials. Moreover, RT changes before and after errors are likely to reflect control adjustments through shifts along the SAT function. However, doubts have been raised that shifts along the SAT function is the only mechanism underlying RT changes after errors (cf. Rabbit & Rogers, 1977). For example, trials following an error are often delayed by 300 ms or more and “this seems far more than necessary to return RT to a ‘safe’ level after a fast error” (Rabbitt & Rogers, 1977).

Sanders (1998) pointed out that post-error slowing seems much larger in studies using short rather than long RSIs (e.g. Laming, 1979; Rabbitt & Rogers, 1977). Previous research has shown that strategic top-down influences on information processing, such as the formation of expectancies, are effective only for RSIs longer than 500 ms (e.g.

Sommer, Leuthold, & Soetens, 1999). If strategic influences dominate control adjustments, post-error slowing should become larger for longer RSIs. That is, since more time is available to adjust behaviour after error detection, strategic influences should become more effective. One possibility to accommodate the finding of an increase in post-error slowing with decreasing RSI in the framework of conflict monitoring would be to assume that control adjustments are not under strategic control, but instead are automatically triggered by the occurrence of an error. In addition, one would have to assume conflict signals are decaying over time. That is, the closer the interval between the error trial and the next trial, the stronger the adjustment signal is, thus resulting in increased post-error slowing. This could explain why post-error slowing is largest at short RSI conditions.

However, accuracy measurements in trials after error commission challenge this automatic version of SAT shifts in the case of short RSIs. If shifts along the SAT curve take place, this should not only result in slower responses but also more accurate performance after error commission. However, Rabbitt, who generally used rather short RSIs (20 - 220 ms), found large post-error slowing effects and reported that error rates often increased after error commission (Rabbitt, 1966; Rabbitt & Rogers, 1977). In contrast, Laming (1968), who used mostly long RSIs (> 1s), found less pronounced post-error slowing effects in conjunction with increased accuracy in post-error trials. In summary, short RSI conditions had a tendency to result in particularly large post-error slowing effects and increased error rates, a combination difficult to account for when arguing on the basis of SAT shifts. In this thesis alternative mechanisms that possibly underlie the changes in response speed in trials following and preceding errors will be investigated.

1.5.2.3. Neural Correlates of Error Detection

1.5.2.3.1. N_E/ ERN

In contrast to the evidence of neural activity of conflict processing in multi-dimensional task settings, which was mainly drawn from fMRI and PET research, the neural correlates of error processing have predominantly been investigated using EEG measurements. The error negativity (N_E) (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990) or error-related negativity (ERN) (Gehring, Coles, Meyer, & Donchin, 1990) is an event-related potential (ERP) component that peaks about 100-150 ms after response onset[1]. The ERN can be elicited in a range of experimental paradigms, such as incorrect responses in choice RT tasks (e.g. Bernstein, Scheffers, & Coles, 1995), uninhibited responses in the NoGo condition of the Go-NoGo task (Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996), by feedback (Miltner, Braun, & Coles, 1997) and also late

[1] To ease readability, the N_E /ERN component will be referred to as the ERN from now on.

responses in deadline RT tasks (e.g. Luu, Flaisch, & Tucker, 2000). The ERN is found regardless of the stimulus (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000) and response modality (Holroyd, Dien, & Coles, 1998). For a detailed tutorial account of the ERN, see Falkenstein et al. (2000).

Dipole modelling has located the source of the ERN in the medial frontal cortex, and therefore the ERN is likely to be generated within the ACC or the supplementary motor area (SMA) (e.g. Dehaene, Posner, & Tucker, 1994; Holroyd, et al., 1998). There is converging evidence that the ACC is the most likely source of the ERN activity. One reason is that EEG measurements represent synchronous postsynaptic activation in parallel aligned pyramidal cells. In contrast to the tangentially orientation of the SMA pyramidal cells, the perpendicular orientation of cells in the ACC could generate a fronto-central negativity such as the ERN (Holroyd & Coles, 2002; Yeung, et al., 2004). Moreover, fMRI studies show consistent evidence for ACC activity in conditions where errors occur or are likely to occur (e.g. Carter, et al., 1998a).

1.5.2.3.2. Relation between the ERN and Behavioural Adjustments

Whereas it is well accepted that the ERN is involved in the processing of errors or conflict, its relationship to subsequent behavioural adjustments is less clear. The results of studies investigating the relation between the ERN and the amount of post-error slowing are mixed. Some studies suggest that the brain structures involved in generating the ERN, such as the ACC, are not only involved in error detection but also in initiating subsequent behavioural adjustments. Evidence for the functional significance of the ERN (or the ACC) in initiating subsequent behavioural adjustments were mainly drawn from studies showing a correlation between the size of the ERN amplitude (or ACC activation) and the post-error RT (e.g. Debener, et al., 2005; Gehring, et al., 1993; Hester, Barre, Mattingley, Foxe, & Garavan, 2007; Kerns, et al., 2004; West & Travers, 2008). For example, Debener et al. (2005) investigated single-trial ERN amplitudes in a speeded version of the Eriksen Flanker task. Single-trial ERN amplitudes predicted the post-error RTs, with a larger ERN resulting in increased RTs in the subsequent trial. Thus, the authors concluded that post-error slowing is driven by post-response activity in the ACC.

Gehring et al. (1993) tested whether the ERN is related to an error compensation system. If the ERN reflects activity of an error compensation system, the size of the ERN amplitude should be related to compensatory behavioural adjustments. They investigated the ERN amplitude in an Eriksen Flanker task. There were three different response conditions. In the first condition, participants had to respond particularly fast and not worry too much about error occurrence (speed condition). In the second condition, participants had to respond accurately and not worry too much about response speed (accuracy condition). The third condition was neutral with neither speed

nor accuracy being emphasised over the other. The ERN was largest in the accuracy condition, suggesting that the importance of an error is reflected in the ERN amplitude. Moreover, the ERN amplitudes were correlated with compensatory mechanisms with a larger ERN producing a reduction in response force in the error trial, increased error correction responses, and increased post-error RT. Thus, it was concluded that the tendency to adopt a more conservative response strategy after an error is reflected in the size of the ERN amplitude. Similar relations between error-related neural activity and post-error behaviour have been reported in a Stroop paradigm (West & Travers, 2008), and a paradigm related to the Go/Nogo task, where participants respond to all letters with the same button press, but to respond to stimulus repetitions with another key (Hester, et al., 2007).

However, other studies have failed to demonstrate a relationship between the ERN and the subsequent compensatory adjustments. For example, Gehring & Fencsik (2001) adopted a four-choice RT task, and did not find a correlation between the size of the ERN and the amount of post-error RT. Another study investigated the ERN and post-error RT in children diagnosed with attention-deficit hyperactivity disorder (ADHD), and despite showing diminished differences in the ERN (between erroneous and correct trials), the amount of post-error slowing was unaffected (van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2007). Other studies comparing patients with damage to the ACC reported similar dissociations regarding the correlation of ERN amplitude and the amount of post-error slowing (e.g. Stemmer, Segalowitz, Witzke, & Schönle, 2004). Taken together, these studies have raised doubts regarding a direct link between the ERN and behavioural adjustments following errors. Thus, it remains unclear what mechanism is reflected in the ERN and whether the ERN amplitude directly relates to post-error adjustments.

1.5.2.3.3. Theoretical Models of the Process Reflected in the N_E/ ERN

The ERN was originally observed after the occurrence of an error. Thus, in early models regarding the functional significance of the ERN, it was seen as a neural correlate of an error signalling mechanism (e.g. Gehring, et al., 1990). With additional studies showing relations between the ERN and compensatory behavioural actions (e.g. post-error slowing), it was suggested that the ERN is also involved in initiating behavioural adjustments (Gehring, et al., 1993). The early onset of the ERN, almost simultaneous to response execution, suggests that sensory or proprioceptive feedback could not be involved in the generation of the ERN (e.g. Gehring, et al., 1993). In contrast, the ERN is more likely to be part of an internal, feedback independent system. Despite general agreement that peripheral feedback about the response is not needed to elicit an ERN, there is ongoing controversy about the nature of the internal monitoring system triggering

the ERN. The main controversy as to what process is reflected by the ERN is the following: On the one side, the ERN is proposed to reflect the error detection process itself (e.g. Falkenstein, et al., 2000), while on the other, the ERN is proposed to reflect conflict detection processes (e.g. Botvinick, et al., 2001; Carter, et al., 1998b; Yeung, et al., 2004).

Coles, Scheffers and Holroyd (2001) described in detail the original claim that the ERN reflects an error detection process (see Figure 6). The central component of this error detection model is a comparison processor, which compares the actual response which was executed with the intended correct response. The representation of the correct response results from ongoing stimulus processing after response decision (Rabbitt & Vyas, 1981). If this comparison process detects a mismatch between the two representations, an error will be detected and an error signal will be generated. As a result the ERN is generated and subsequent compensatory actions will take place (e.g. inhibition or correction responses). The central claim in this model is that the ERN is proposed to reflect error detection processes, and error detection emerges from a mismatch between the executed and the response which should have been executed.

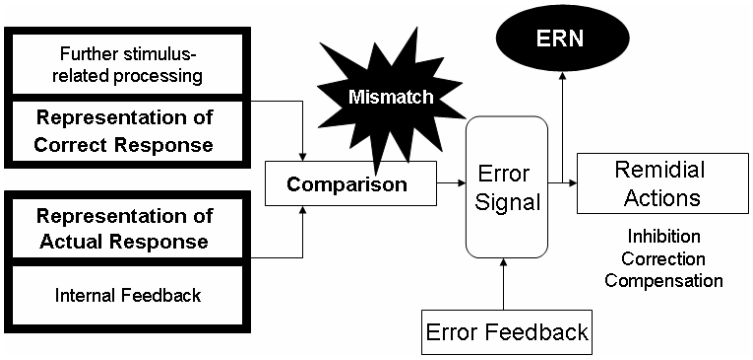

Figure 6. Error detection through a comparison mechanism detecting mismatch between the representation of the actual executed response and the representation of the required correct response (adapted from Coles, Scheffers & Holroyd, 2001).

However, doubts have been raised concerning whether the ERN reflects a pure error detection process. For example, an ERN has also been reported in correct trials (e.g. Falkenstein, et al., 2000; Luu, et al., 2000). Moreover, the proposal that the ERN reflects an internal comparison process between the executed incorrect response and the intended correct response cannot account for the exact timing of the ERN for the following reasons (see: Holroyd, Yeung, Coles, & Cohen, 2005; Yeung, et al., 2004): The error detection theory claims a comparison process between the response representation

and the executed response to take place. Thus, it remains unclear, why error execution cannot be withhold, as the outcome of the comparison process seems available with response execution, when the ERN onset is typically reported. On the other side, it is also not specified how this comparison process can be so quick starting instantly with response execution.

In contrast to the error detection model, the conflict monitoring theory proposes that the ERN reflects a measure of processing conflict (e.g. Botvinick, et al., 2001; Carter, et al., 1998b; Yeung, et al., 2004). Central to this argument is the proposal that the ERN is not the result of an incorrect response. Rather, incorrect trials often involve increased processing conflict and it is this increased processing conflict that triggers high ACC activation, which is measured in the ERN. Evidence for similarities in neural activation during erroneous and high conflict trials was provided by Carter et al. (1998b). They demonstrated via the use of fMRI that ACC activation in error trials resembles ACC activation in trials with high response competition. Botvinick et al. simulated conflict activation in correct and erroneous trial in an Eriksen Flanker task. Consistent with the error detection model, it is assumed that in erroneous trials, ongoing stimulus evaluation leads to coactivation of the correct and the erroneous response unit, resulting in conflict (Rabbitt & Vyas, 1981). More specifically, Botvinick et al.'s model predicted largest coactivation of competing response units after response execution, which fits well with empirical findings of ERN latencies. However, within this conflict detection model there should also be an ERN for incongruent but correct trials, which is usually not reported (e.g. Scheffers & Coles, 2000). In this context it is important to state that although the conflict monitoring model predicts similarities between the processing of erroneous and high-conflict correct trials, it also predicts certain differences. These differences specifically regard the timing of maximum coactivation (Yeung, et al., 2004). In erroneous trials, coactivation should be largest just after response execution. In contrast, in high-conflict correct trials, the model predicts this coactivation to occur before response execution, and thus no ERN should be measurable after response execution.

Computational models of Yeung et al., (2004) extend the work of Botvinick et al. (2001) relating the ERN to conflict detection. Yeung et al. also demonstrated that a conflict related explanation of ERN activation can explain some findings which, at first, seem to contradict the conflict monitoring approach. The model parameters for the ERP data were drawn from Botvinick et al.'s conflict monitoring model. Similarly, to simulate error occurrence, additional processing variability (noise) was added to each processing cycle of the model. The model predicted the exact timing of peak conflict strength in both erroneous and correct trials. Indeed, Yeung et al. showed that conflict in erroneous trials is largest at the point in time when the ERN reaches it peak, due to continued processing of the stimulus which results in maximum coactivation between the erroneous executed

response and the correct response directly after response execution. In contrast, in correct trials involving conflict, coactivation of the two response alternatives reaches it maximal overlap earlier. Yeung et al. suggested that conflict in correct trials is reflected in the amplitude of the N2. The N2 is traditionally reported in the go/nogo task, where participants are instructed to withhold responses in nogo trials (e.g. Eimer, 1993), but it has recently been shown to also occur in other paradigms involving conflict such as the Eriksen Flanker task (e.g. Kopp, Rist, & Mattler, 1996). The N2 can be measured over frontocentral electrodes, and is a negative component peaking approximately 250 to 350 ms after the presentation of the nogo stimulus (e.g. Eimer, 1993). Originally, the N2 was proposed to reflect top-down triggered inhibition mechanisms involved in suppressing response execution. Nieuwenhuis, Yeung, Van den Wildenberg and Ridderinkhof (2003) showed that the N2 and the ERN have similar neural sources within the ACC using a dipole modelling approach in a go/nogo task. Moreover, the N2 was enhanced for low frequency stimuli, independent of go/nogo condition. Thus, they suggested that the N2 is likely to reflect conflict. In summary, Yeung et al. concluded that due to the precise timing of peak conflict, ERN peak latency, N2 peak latency, and the consistent neural sources for both components suggests that both components reflect conflict processing.

Importantly, the conflict monitoring model as suggested by Yeung et al. (2004) can also explain several relationships between ERN size and behavioural results, which seemed to contradict the conflict hypothesis of the ERN. For example, it has been shown that the ERN is larger in congruent than in incongruent error trials (Scheffers & Coles, 2000). According to the conflict monitoring theory, conflict is high in incongruent trials. Thus, one would expect the opposite, a larger ERN for incorrect incongruent trials than incorrect congruent trials. Yeung et al.'s model suggests that in congruent trials, the evidence for the required correct response builds up faster and there is more activation for the required correct response than in an incongruent trial. This is due to the fact that in congruent trials the stimulus itself is non-ambiguous regarding which response it activates. Thus, the relative coactivation between the executed erroneous response and the required correct response should be higher, suggesting that conflict is larger in erroneous congruent trials than in erroneous incongruent trials. This is in line with the findings that the ERN is larger in congruent than in incongruent error trials.

Interestingly, it has been shown that there is a component similar to the ERN elicited by error feedback (Miltner, et al., 1997). Both the error detection and the conflict monitoring theory were criticized as they have difficulties in explaining ERN activity to external feedback indicating error occurrence, especially as feedback in these studies is often provided substantially later than the actual error occurrence, sometimes up to 600 ms (e.g. Miltner, et al., 1997). This is because both these theories are built on internal representations and that such competing internal representations have to be activated

simultaneously (Holroyd, et al., 2005). An alternative reinforcement learning model has been developed by Holroyd and Coles (2002). This model links the ERN directly to activity in the mesencephalic dopamine system. The dopamine system is claimed to be responsible for error detection, and is supposed to react to events that are worse than expected by a decrease of activation. In contrast positive events would result in an increase of activation. This activity level in the basal ganglia is subsequently signalled to the ACC. Moreover, this model proposes various distinct motor-control units representing different response modes, and can be reinforced in the case of successful trial performance. For example, one motor unit might be specialised for guessing responses, and another for highly controlled responses. If there is no error signal, the ACC will reinforce the motor processing unit used in the current trial. If the system signals an error, the ACC will not reinforce the current response strategy and thus, a change of response modes can be expected. Importantly, like the conflict monitoring model, this model is able to explain a wide range of the behavioural effects in relation to the ERN (Holroyd, et al., 2005). The strength of the reinforcement learning model is its ability to explain error detection and ERN in scenarios where the coactivation of the correct and the erroneous response do not occur simultaneously, such is the case with the feedback ERN. However, doubts have been raised about the assumption concerning whether the feedback ERN has the same functional significance as the response-locked ERN (van Veen & Carter, 2006). For example, recent fMRI studies have demonstrated that the feedback ERN did not elicit ACC activity as typically reported for the ERN (e.g. van Veen, Holroyd, Cohen, Stenger, & Carter, 2004).

In summary, there are three major hypotheses regarding the functional significance of the ERN. First, it was proposed that the ERN reflects error detection processes (e.g. Falkenstein, 2000). Second it was suggested, that the ERN reflects conflict rather than error detection processes, as conflict is typically high in erroneous trials (e.g. Botvinick et al., 2001). Third, it was proposed that the ERN is part of a reinforcement learning process (Holroyd & Coles, 2002). As described, the error detection and conflict monitoring approach of the ERN focus on the ERN occurring instantly after error commission, which will be closer investigated in this thesis.

1.6. Objectives

The previous sections introduced two types of conflict that occur in uni-dimensional task settings relevant for this thesis. First, it was shown that conflict can arise through overlap of opposing activation in response alternation trials, resulting in the ABI effect (Jentzsch & Leuthold, 2005). Second, conflict can arise in tasks only having one relevant response dimension when participants commit an error (e.g. Botvinick, et al., 2001; Yeung, et al.,

2004). Despite a plethora of studies investigating conflict during information processing and its role for subsequent adjustments, there are still open questions about the concept of conflict and the impact of it on information processing. The following key questions will be addressed in this thesis: First, where exactly does conflict originate throughout information processing? Second, what are the mechanisms underlying post-conflict adjustments, both in the case of error processing and the ABI effect? Third, how do neural correlates of error processing, specifically the ERN, relate to conflict and compensatory behavioural adjustments in a uni-dimensional task setting? In the following paragraphs the objectives regarding the role of conflict addressed in this thesis will be introduced in detail, separately for error processing and the ABI effect.

1.6.1. Conflict in Response Alternation Trials

Within the conflict monitoring model of Botvinick et al. (2001), it is assumed that conflict arises through opposing activation overlap at a response selection stage. However, it is also stated that conflict may arise during other stages of information processing. To date, it is still not clear whether conflict emerges from opposing activation overlap in perceptual stages, response selection stages or in motor programming stages. The ABI effect provides a suitable experimental setting to investigate in detail where conflict originates within information processing in uni-dimensional task settings. Jentzsch and Leuthold (2005) showed that response slowing occurs only after response alternations and not after stimulus alternations. This provides evidence that conflict in the case of the ABI effect is due to response related activation overlap and not competing stimulus related activation. However, in this study a response change always implied a change of response hand. Thus, in all response change trials, both the abstract response code (left vs. right) and the effector specific code (left hand vs. right hand) changed. Therefore, it remains unclear whether the conflict underlying the ABI effect arises at an abstract, an effector specific, or at both levels of response representation. In Experiment 1 and 2 of this thesis the locus of conflict origin in the case of the ABI effect will be investigated.

An additional question which is still under debate regards how exactly post-conflict adjustments are implemented. Current models of cognitive control are relatively vague in their specification of how exactly conflict-related control adjustments affect processing in trial N+1. Botvinick et al. (2001) suggested that response slowing after high conflict trials is implemented through shifts along the SAT function. More specifically, response thresholds are increased after conflict trials resulting in both slower and more accurate responses. However, in the experiments of Jentzsch and Leuthold (2005), response accuracy did not increase after high conflict trials. In contrast, response accuracy decreased in two out of four experiments. In order to investigate the mechanisms underlying response slowing in the case of the ABI effect, the locus of

response adjustments will be investigated in Experiment 3 of this thesis. Knowledge about the locus of response adjustments after high conflict trials is an important step for further understanding of the underlying mechanisms. The fact that Jentzsch and Leuthold (2005) found that conflict originates at response related stages in trial N, does not necessarily allow us to draw conclusions about the impact of conflict-triggered response adjustments on trial N+1 performance. One possibility would be that, as conflict originates at response related stages, only response related stages are slowed down after conflict. On the other hand, similarly to the hard bottleneck reported after task switch trials (Oriet & Jolicœur, 2003; but see: MacKenzie & Leuthold, 2005) , it is also possible that stimulus processing, response related processing and motor stages are delayed after conflict. In summary, in the first part of this thesis, both conflict origin and the mechanisms underlying post-conflict adjustment in the case of conflict evolving through response alternation trials will be investigated.

1.6.2. Conflict in Erroneous Trials

The second example of conflict within a uni-dimensional task setting which will be investigated in this thesis is error processing. Despite many recent studies investigating errors in multi-dimensional tasks (e.g. Debener, et al., 2005; Gehring & Fencsik, 2001), there is no specific motivation as to why errors should be investigated in multi-dimensional tasks. In contrast, early studies reporting error related phenomena implemented uni-dimensional tasks (e.g. Rabbitt, 1966, 1968; Rabbitt & Rogers, 1977). The use of multi-dimensional task settings evokes additional conflict through the two response dimensions which might interfere with the conflict elicited by errors, thus making interpretation of results more complicated.

The exact nature of error processing and the role it plays within executive function in general remains an important and highly debated topic within cognitive science. One of the major problems of traditional (e.g. Brewer & Smith, 1984; Rabbitt, 1979) and current theoretical models (Botvinick, et al., 2001) explaining error processing, is their limited scope in explaining RT changes before and after errors. More specifically, these models rely solely on shifts along a SAT function. However, trial processing after an error is both substantially prolonged and error prone if there is limited time available between error occurrence and the onset of the next trial (e.g. Rabbitt, 1966; Rabbitt & Rogers, 1977). Such impairment is difficult to explain by SAT shifts alone. Thus, in this thesis alternative mechanisms underlying post-error slowing will be investigated and the question regarding the exact nature of these mechanisms will be addressed in Experiment 6. Moreover, despite both pre-error speeding and post-error slowing being assumed to arise from similar mechanisms (i.e. shifts along the SAT function), research has mainly focused on the effects of post-error slowing, with the exception of a few early

studies (e.g. Brewer & Smith, 1984). Thus, Experiment 7 of this thesis will investigate the relationship between pre-error and post-error slowing, and the mechanisms underlying pre-error speeding and post-error slowing.

In addition to the debate regarding the mechanisms underlying behavioural changes before and after errors, the debate about the nature of the process reflected in the ERN in still ongoing and will be another key question in this thesis. As outlined above the major question concerns whether the ERN reflects error detection or conflict processing (e.g. Coles, et al., 2001; Yeung, et al., 2004). Interestingly, the predictions of the conflict monitoring and the error detection theory diverge when considering the size of the ERN to different error types (e.g. Bernstein, et al., 1995; Gehring & Fencsik, 2001). Conflict monitoring theory predicts larger conflict the more similar the opposing activation between the two response alternatives are, and thus, that the ERN should be the larger when the required correct response and the executed erroneous response are similar. In contrast, the error detection theory predicts that the ERN reflects the outcome of a comparison processes between the executed and the required response, and thus, the ERN should be the larger the more dissimilar the correct response is to the executed erroneous response. Experiment 4 and 5 in this thesis will contrast these two hypotheses regarding the ERN by investigating the effect of required and executed response similarity on the size of the ERN (see also: Bernstein, et al., 1995; Gehring & Fencsik, 2001). This context also provides an opportunity to investigate whether changes in ERN amplitude relate to the amount of subsequent behavioural adjustments. As described above, previous findings regarding the relationship between the size of the ERN and the amount of post-error slowing are ambiguous (e.g. Debener, et al., 2005; Gehring & Fencsik, 2001). To summarise, this thesis will investigate the processes reflected in the ERN, the relationship between the ERN and subsequent behavioural adjustments, and the mechanisms underlying behavioural changes before and after errors.

1.7. Chronophysiological Measurements

In order to localise effects of conflict adjustments during information processing, this thesis utilises the high temporal-resolution of EEG measurements. In the 1920's, Hans Berger developed an apparatus, the so called "electroencephalograph", which could measure electrical activity in the brain. EEG is the recording of small voltage changes on the scalp, which are produced by the electrical activity of neurons in the brain. Changes in the EEG signal represent the summation of synchronous activity of thousands of neurons that have a similar spatial orientation in the cortex. Event-related potentials (ERPs) represent voltage changes in the EEG signal that are time-locked with the presentation of sensory stimuli (e.g. visual, auditory) or motor events (e.g. key press

response). As ERPs are relatively small signals when compared to general background EEG noise, one common procedure to increase the signal-to-noise ratio is simply averaging individual trials time-locked to the event of interest (e.g. stimulus onset, response). The averaging procedure should make the ERP visible because it is assumed that event independent voltage changes (background noise) fluctuate randomly and are not time-locked to a specific event. Filtering is a second method to eliminate background noise. For example, line noise in surrounding electrical equipment (50Hz) can be removed via the use of a low pass filter. This method is helpful when signal and noise have different frequencies. For a detailed introduction to the ERP technique, see Luck (2005). Importantly, ERPs provide high-resolution measures of the timing of neural activity accompanying cognitive processes. Therefore, ERPs are ideal markers for localising a process within the information processing chain (see Figure 7). For example, if an experimental manipulation affects perceptual processing, ERP markers of perceptual processing should show a shift in their onset (or peak) latency. In the following sections the chronometric ERP markers utilised in the present thesis will be introduced.

1.7.1. Lateralized Readiness Potential

The Lateralized Readiness Potential (LRP) has proved an important ERP measure of the duration of different information processing stages. The LRP is based on the Bereitschaftspotential (readiness potential) that was first reported by Kornhuber and Deecke (1965). The readiness potential precedes volitional movements and develops approximately one second prior to movement onset. It is a negative, ramp-shaped potential, maximal over central scalp sites. The readiness potential is more negative over the motor cortex contralateral to the responding hand (Kutas & Donchin, 1974; Vaughan, Costa, & Ritter, 1968).

With the goal to isolate the lateralization of the readiness potential, the following procedure to calculate the LRP was suggested by Coles (1989). First, the potential recorded over the primary motor cortex ipsilateral to the correct response hand is subtracted from the potential over the contralateral scalp, separately for left and right hand responses. This step eliminates all activity that is symmetrically distributed over centrolateral recording sites. In order to eliminate all asymmetric activity not related to the response, these difference potentials are averaged over left and right hand responses, resulting in the LRP. Since the subtraction is performed with respect to the correct response hand, deviation of the LRP in the negative direction reflects correct response activation, while deviation in the positive direction indicates activation of the incorrect response. There is good evidence that the LRP relates to hand-specific activation, and therefore, it is used as a real-time measure of selective motor preparation (e.g J. Miller &

Hackley, 1992). After the onset of the LRP it is still possible to abort a voluntary response (e.g., Miller & Hackley, 1992), which indicates that the onset of the LRP indexes central rather than peripheral motor processes.

Like other psychophysiological measures, the LRP onset can be measured relative to the onset of the stimulus (S-locked; S-LRP) or the response (R-locked; LRP-R). The interval between stimulus and the onset of the S-LRP is used as estimation for the overall duration of processing stages prior to the onset of hand-specific response activation, from perceptual to response selection stages. The interval between LRP-R onset and the overt response is assumed to represent the duration of RT processes that occur after the onset of hand-specific response activation (see Figure 7). Thus, one can determine whether early or late processes are influenced by an experimental manipulation. If a manipulation affects early processes (i.e. processes before hand-specific response selection) then the S-LRP intervals should differ. In contrast, manipulations influencing late processes (i.e. after the hand-specific response selection) should affect the LRP-R interval (Leuthold, Sommer, & Ulrich, 1996; Osman & Moore, 1993; Osman, Moore, & Ulrich, 1995).

1.7.2. P1 and N1

P1 and N1 are visual evoked ERPs measured over the primary visual cortex, and are usually maximal at lateral posterior-occipital electrode locations PO7 (left hemisphere) and PO8 (right hemisphere). The P1/N1 complex is used as a marker for the timing of perceptual processes. The latency of these early ERP components have been shown to sensitively reveal effects of stimulus contrast manipulations (e.g. Jaskowski, Pruszewicz, & Swidzinski, 1990; Jentzsch, Leuthold, & Ulrich, 2007; Vaughan, Costa, & Gilden, 1966), with delayed peak amplitudes for low compared to high contrast stimuli. Dipole modelling of the P1 component has demonstrated that its scalp distribution is consistent with a neural generator source within lateral extrastriate cortex (Clark & Hillyard, 1996).

1.7.3. P300

The P300 is a positive-going component that occurs approximately 300 ms after a task relevant sensory stimulus, usually maximal in midline central electrode sites (e.g. Pz). It is known to vary systematically with the information provided by the stimulus and the expectancy of an event. Although it is one of the most widely investigated ERP components, there is still some debate as to the process(es) reflected by the P300 (see: Verleger, 1997). Nonetheless, its latency and amplitude are often used to assess whether a variable influences stimulus evaluation and categorization processes (e.g. Kutas, McCarthy, & Donchin, 1977; Müller-Gethmann, Ulrich, & Rinkenauer, 2003). The P300 seems to be relatively independent from late processes (e.g. J. Miller & Low, 2001),

and therefore is often used to determine whether early processes are influenced by an experimental manipulation.

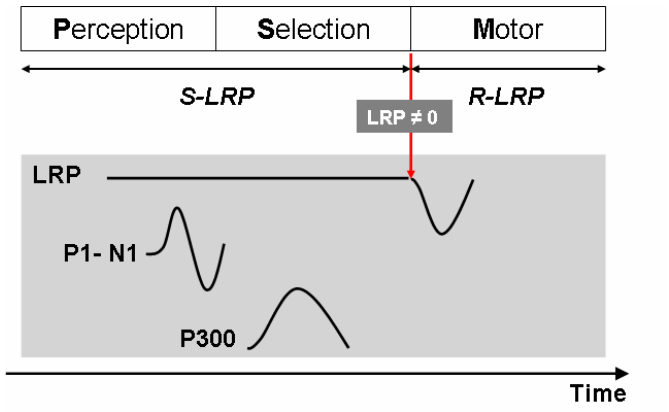

Figure 7. Information processing stages (upper panel) and corresponding chronometric electroencephalographic components (lower panel).

2. Experiments

The following chapter outlines the empirical studies conducted to investigate the role of conflict monitoring in uni-dimensional task settings. This experimental part can be divided into two main sections. The first experiments (Experiments 1-3) are dedicated to investigate more closely conflict origin and the locus of post-conflict adjustments after response alternations. The second experimental part (Experiments 4-7) investigates errors in uni-dimensional task settings and the role conflict monitoring plays in explaining error processing.

2.1. Experiment 1

Responses are slower in trials following a response alternation than in trials following a response repetition (e.g. Jentzsch & Leuthold, 2005; Melis, et al., 2002). This finding is termed the alternation based interference (ABI) effect and is interpreted as a processing adjustment following conflict (Jentzsch & Leuthold, 2005). Critically, response activation is assumed to extend beyond response execution and to gradually decay over time (e.g. Jentzsch & Sommer, 2002). This produces overlapping activation of opposing response alternatives between the previous and current trial when the response alternates, (i.e. conflict as conceptualized within the conflict monitoring theory) (Botvinick et al., 2001). This conflict subsequently triggers the response adjustments in the form of the ABI effect (Jentzsch & Leuthold, 2005).

The first experiment in this thesis was conducted to examine the locus of conflict origin in the case of the ABI effect. To distinguish between independent influences of stimulus (S) and response (R) transitions, Jentzsch and Leuthold (2005) used the information reduction paradigm (e.g. Bertelson, 1965), mapping four stimuli onto two response alternatives. In a series of four experiments, they found strong evidence that a response change was necessary to produce the ABI effect, whereas a stimulus change alone did not result in subsequent adjustments. Based on this evidence, and in line with conflict monitoring theory (Botvinick et al., 2001), they suggested that the ABI effect is caused by conflict between competing response activations. However, the paradigm used in this initial study required simple keypress responses where a response change always implied a change in response effector. Thus, conflict could either arise at an abstract level of response code representation overlap (“left” vs. “right”, *abstract conflict hypothesis*) or at an effector specific response level (left hand vs. right hand*, effector specific conflict hypothesis*).

To distinguish between the abstract and the effector specific conflict hypothesis, manual and vocal responses were implemented in the present experiment. When

participants respond manually the ABI effect is expected to occur similarly to the effect reported in the original study by Jentzsch and Leuthold (2005). The vocal response condition is crucial for testing the predictions of the effector specific and the abstract response code hypothesis. More specifically, if response conflict is caused by overlap of effector specific activation, conflict should not arise via vocal responses. This is because the effector mouth is involved in both response alternatives (saying “L” and “R”). Hence, no competing effector activation can arise and cause conflict (see Figure 8). As a result, no increase in response times should be found after response alternation trials (different-X transition, e.g. “L” followed by “R”) when compared to trials after identical (identical-X) or equivalent (equivalent-X) response transitions (e.g. “L” followed by “L”). However, if overlap of an abstract response code causes conflict, the ABI effect should still be present in the vocal condition. This is because the abstract response code of saying “L” and “R” should be as separate as the code for pressing the left or right response button.

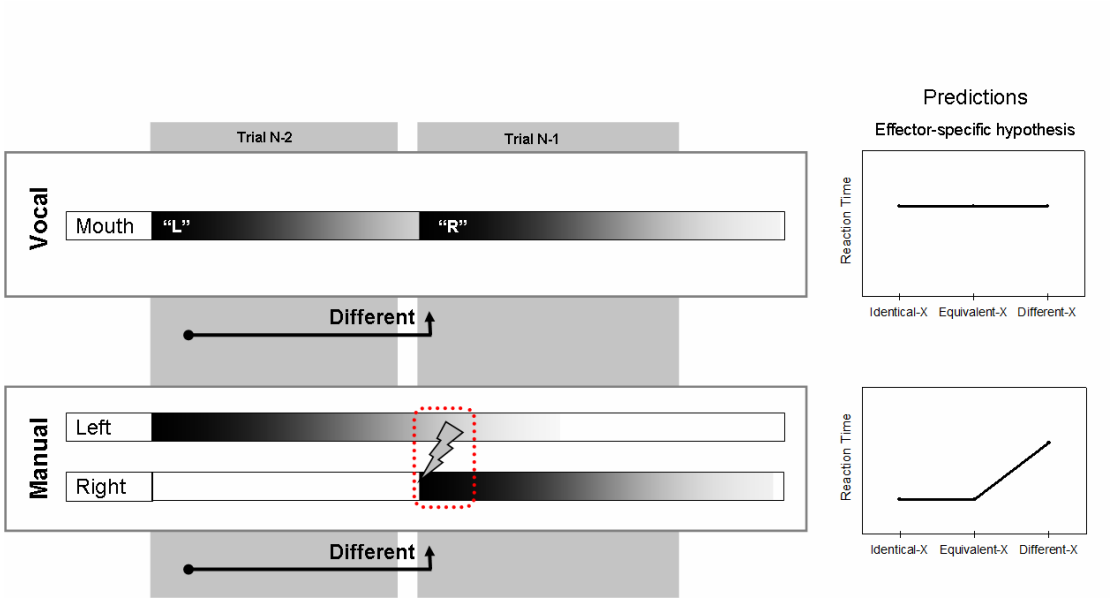

Figure 8. *Left panels*: Effector-specific activation in the different trial transition, for the vocal and the manual response condition. *Right panels*: Predictions of the effector-specific conflict hypothesis regarding the size of the ABI effect in vocal and manual response conditions. Importantly, in the vocal response condition only one response effector is involved and thus, no ABI effect should be found.

2.1.1. Method

Participants

24 participants (M_{age} = 23.96 years, SD_{age} = 6.60 years, 17 woman) with normal or corrected-to-normal vision were tested in a single session lasting approximately 45 min. All participants were naive about the experimental hypothesis and were paid £4.

Stimuli and Apparatus

The Stimuli were presented on an Envy 17-in. (43-cm) CRT monitor controlled by an IBM-compatible personal computer. Participants were seated in a dark room with a constant 75 cm viewing distance. The response keys were mounted 15 cm apart in the horizontal plane of the participant. Left and right keypress responses were made with the index fingers of the left and right hands, respectively. Vocal responses (“L” or “R”) were spoken in a headset microphone, which was connected to an ERTS voice key allowing RT measurements in the vocal response condition. As the voice key signals the onset of any auditory signal, the experimenter recorded response type (“L” versus “R”) manually, allowing the subsequent exclusion of incorrect vocal responses. The stimuli consisted of the letters D, M, T and X presented centrally in white on a black background. All stimuli measured approximately 5x5 mm.

Procedure

Participants were instructed to respond as fast as possible while maintaining a high level of accuracy. As the voice key was highly sensitive to all kind of noise, participants were instructed to be careful and to try to avoid any additional noise which was not related to the required responses (e.g. laughing, moving the chair, etc.). The experiment was divided into two parts, in which participants were asked to perform either a keypress response by pressing either the left or right key (with the left or right index finger), or a vocal response by saying “L” or “R”. The order of the response conditions was balanced across participants. Two letters were mapped onto each of the two response alternatives (e.g. D and T => left key or “L”, M and X => right key or “R”). The assignment of letters to response alternatives was balanced across participants, but for a given participant assignment of letter to either left or right was held constant for the keypress and the vocal condition. Stimuli were presented until response. If no response was recorded within 2000 ms after stimulus onset, the next trial was presented. Presentation of stimuli was randomized within a given block. A RSI of 250 ms was used which started with response onset.

For each of the two response conditions two practice blocks (40 trials each) were conducted, and ten experimental blocks consisting of 80 trials each. Due to the equal probability of the presentation of the four stimuli, identical and equivalent trials each constituted about 25% of all trials, whereas on the remaining 50% of the trials, both the stimulus and the response alternated (different trials). Blocks were separated by short rests, where participants were provided with written feedback about the mean RT from the completed block.

Data Reduction and Analysis

All trials with errors from trial N-2 to trial N were excluded from the RT analysis. Also, only trials with RTs longer than 100 ms and shorter than 2000 ms were considered correct. In the analysis of error rates, only choice errors were included. All trials with missing responses, too-slow responses and too fast responses were discarded from the analysis (< 1%).

For the analysis of trial sequence, data were split according to the relationship of trial N-2 to trial N-1, which resulted in three transition levels (identical-X, equivalent-X, and different-X). The label X is used to indicate that the mean RT for a given trial N is calculated while disregarding the first-order transition X. Dependent variables were subjected to repeated measures ANOVAs with within-subject variables of trial transition (identical-X, equivalent-X, and different-X) and response condition (keypress vs. vocal). Conservative Huynh-Feldt *F*-tests were used throughout and for post-hoc comparisons Bonferroni corrections were applied.

2.1.2. Results

Reaction Time

Figure 9 (upper panel) shows mean RT as a function of trial transition and response condition averaged across participants. The ANOVA showed a main effect of response condition, $F(1, 23) = 108.07$, $MSE = 7903.07$, $p < .001$, with faster RTs for keypress responses (500 ms) than for vocal responses (654 ms). The main effect of trial transition was also significant, $F(2, 46) = 33.63$, $MSE = 454.75$, $p < .001$, $\varepsilon = 1.02$, with faster RTs for identical-X (575 ms) and equivalent-X (560 ms) than for different-x (596 ms) trials. Post-hoc tests showed that the difference between equivalent-X and different-X trials was significant, representing a clear ABI effect, $F(1, 23) = 55.51$, $MSE = 545.90$, $p < .001$. Importantly, post-hoc tests looking at the response conditions separately showed the ABI effect to be present in both response conditions. In the vocal condition the ABI effect measured 29 ms, $F(1, 23) = 18.99$, $MSE = 523.24$, $p < .001$. In the manual response condition the ABI effect measured 42 ms, $F(1, 23) = 61.91$, $MSE = 346.71$, $p < .001$. Although the ABI effect is numerically smaller in the vocal condition than in the manual condition, the interaction between response condition and trial transition was not significant, $F(2, 46) = 2.30$, $p > .10$.

However, contrary to expectations, post-hoc tests revealed that identical-X and equivalent-X trials differed significantly, $F(1, 23) = 15.12$, $MSE = 348.01$, $p < .01$. This effect was due to a significant 16 ms difference in the vocal response condition between the identical-X and the equivalent-X response condition, $F(1, 23) = 8.01$, $MSE = 384.70$, $p < .05$, and a significant difference of 14 ms in the manual response condition, $F(1, 23) = 5.67$, $MSE = 391.24$, $p = .05$. Importantly, when calculating the ABI effect as the

difference between identical-X and different-X trials, this was still significant for both the vocal response condition, $F(1, 23) = 6.17$, $MSE = 316.36$, $p < .05$, and the manual response condition, $F(1, 23) = 17.75$, $MSE = 556.71$, $p < .001$.

Error Rates

Figure 9 (lower panel) shows mean error percentage as a function of trial transition and response condition averaged across participants. The main effect of response condition was significant, $F(1, 23) = 11.26$, $MSE = 13.41$, $p < .01$, with more errors made with vocal responses (6.02 %) compared to manual key-press responses (3.97 %). There was no effect of trial transition ($F(2,46) = 1.35$, $p = .27$), and no interaction ($F(2,46) = 1.50$, $p = .23$). Due to technical errors 11.52 % of the trials in the vocal response condition had to be excluded. However, these trials were also equally distributed over the three sequential conditions, identical-X (11.62 %), equivalent-X (11.40 %), and different-X (11.56 %).

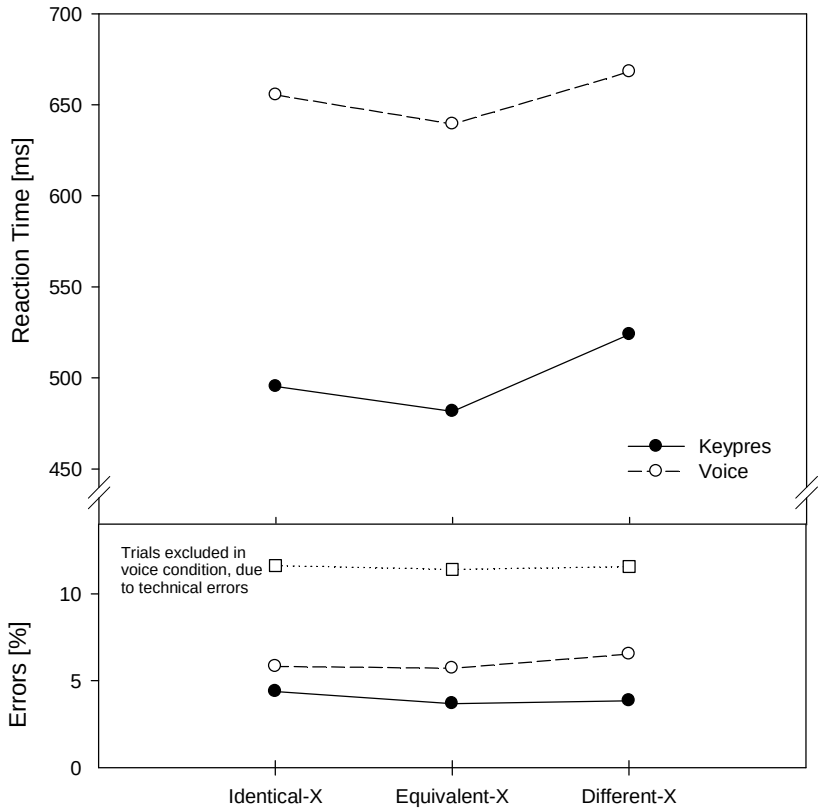

Figure 9. Mean RT and percentage of choice error as a function of sequence (identical-X, equivalent-X, different-X) and response condition (keypress vs. vocal).

2.1.3. Discussion

Jentzsch and Leuthold (2005) reported that in choice RT tasks participants respond slower after response alternation than after response repetition trials (see also: Soetens, et al., 1984; Vervaeck & Boer, 1980). This so-called ABI effect was explained with reference to overlapping response activity that is proposed to cause conflict and resulting post-conflict adjustments. However, the paradigm used in the initial study required simple keypress responses where a response change always implied a change in response hand. Thus, response conflict could arise either at an abstract (left, right) or an effector specific response code representation (left hand, right hand). The aim of the present experiment was to test the effector specific conflict hypothesis versus the abstract response code conflict hypothesis.

To do so, post-conflict adjustments were compared between vocal and manual response conditions. Overall, vocal responses were slower than manual responses, a finding which has been reported in other choice RT tasks implementing vocal and manual responses (e.g. Barch, et al., 2001). The ABI effect in the manual condition was predicted to occur as reported by Jentzsch and Leuthold (2005). Critical for distinguishing between the hypotheses were the predictions for the vocal response condition. In the case of the effector specific conflict hypothesis, the vocal condition should not show any ABI effect, as there is only one effector involved in responding. In contrast to these predictions, post-conflict adjustment took place in both the vocal condition (29 ms) and the manual condition (42 ms). Statistically the two response conditions did not differ. The fact that there was a clear ABI effect in the vocal condition clearly supports the abstract response conflict hypothesis over the effector specific response code hypothesis.

However, the data does not allow unambiguous interpretation. Numerically, the ABI effect is clearly smaller in the vocal condition. Moreover, identical-X and equivalent-X trials differed significantly in addition to differences between equivalent-X and different-X trials. This leads to a very small ABI effect when comparing identical-X and different-X, especially in the vocal response condition (13 ms). Moreover, one could argue that saying “L” versus saying “R” involves different low-level muscular activity, which potentially causes conflict in line with the effector specific conflict hypothesis. A second experiment was conducted to rule out these potential problems.

2.2. Experiment 2

The first experiment suggested that conflict is likely to originate at an abstract response level, but the results were not unambiguous. The aim of Experiment 2 was to further investigate the nature of response representations that lead to the ABI effect. In Experiment 2, participants were asked to perform sequential key press responses on each trial with either the same hand (1-hand condition: left-left vs. right-right) or with two different hands (2-hand condition: left-right vs. right-left). Importantly, using sequential keypresses and interpreting their activation patterns overcomes doubts regarding the similarity of the effector specific response activation, which was one of the main problems in the first experiment.

The effector specific conflict hypothesis claims that conflict arises at an effector specific activation level. In the current experiment the opposing effectors are the two response fingers, that is the left (E1) and the right (E2) index finger. Critically, the effector specific conflict hypothesis predicts different conflict levels in the 1-hand and 2-hand condition in response alternation trials. If conflict arises at the level of effector representation, in the 1-hand condition effector specific conflict should be strong after different trial transitions (e.g. $E_1\mathbf{E_1}$ followed by $\mathbf{E_2}E_2$, see Figure 10). However, after identical and equivalent trials (e.g. $E_1\mathbf{E_1}$ followed by $\mathbf{E_1}E_1$) conflict should be small. Critically, in the 2-hand response condition, both response hands are involved in each trial. Thus, competing response activations should also be present in identical and equivalent trials (e.g., $E_1\mathbf{E_2}$. followed by $\mathbf{E_1}E_2$). Strictly, the effector specific conflict hypothesis even predicts an inverse ABI effect, as in response alternation trials (e.g. $E_1\mathbf{E_2}$ followed by $\mathbf{E_2}E_1$, see Figure 10) the second keypress in trial N-2 equals the first keypress in trial N-1, and thus conflict should be smaller than in response repetition trials.

In contrast, if conflict arises at an abstract level of response coding, the ABI effect should have approximately the same size irrespective of the response condition (1-hand or 2-hand). This is due to the fact that the chain of abstract response code activation should be similar for 1-hand (“double left” vs. “double right”) and 2-hand responses (“right followed by left” vs. “left followed by right”).

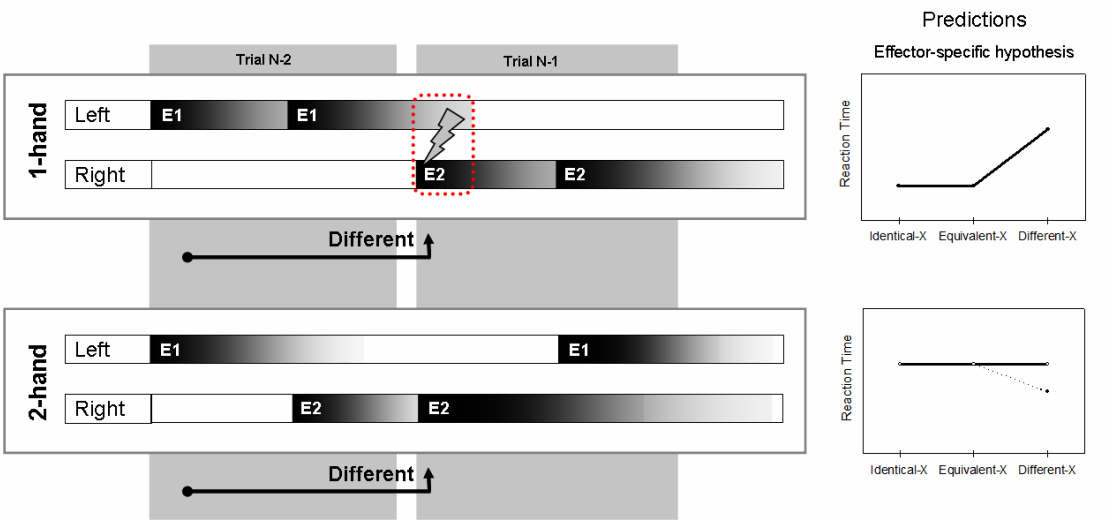

Figure 10. *Left panels*: Activation of effector specific response units (left hand [E_1] and right hand [E_2]) in the different trial transitions in the 1-hand and the 2-hand condition. *Right panels*: Predictions of the effector specific response conflict hypothesis for the 1-hand and the 2-hand response condition. Importantly, in the 2-hand response condition, the effector specific response conflict hypothesis would predict no ABI effect.

2.2.1. Methods

Participants

24 participants (M_{age} = 22.00 years, SD_{age} = 4.75 years, 19 woman) with normal or corrected-to-normal vision were tested in a single session lasting approximately 55 min. All participants were naive about the experimental hypothesis and were paid £5.

Stimuli and Apparatus

Stimuli and apparatus were identical to the first experiment, except that in this experiment sequential keypress responses were recorded.

Procedure

The procedure used was identical to Experiment 1 unless stated. The experiment was divided into two parts, in which participants were asked to perform either a sequential double key-press response with the index finger of the same hand (1-hand: left–left or right–right) or the index fingers of both hands (2-hand: left–right or right–left). The order of the response conditions was balanced across participants. Four letters were mapped to two response alternatives, for example, in the 1-hand condition: D and T → left-left, M and X → right-right; in the 2-hand condition: D and T → left-right, M and X → right-left. The assignment of letters to response alternatives was balanced across participants but

for a given participant, the assignment of the first key press to a given stimulus was held constant across the two response conditions.

Two RSIs of 50 and 150 ms were used. RSI was defined as the interval between the second keypress and the subsequent stimulus onset. If no response was recorded within 2000 ms following stimulus onset, the next trial started. The two RSI conditions alternated between blocks. The order of the RSI condition was balanced across participants.

Each experimental part consisted of 8 blocks (160 trials each). For each of the two response conditions, two practice blocks (30 trials each) were conducted, one for each RSI level. Again blocks were separated by short rests, where participants were provided with written feedback about mean RT and mean error rate in the previous block.

Data Reduction and Analysis

RTs were defined as the interval between stimulus onset and onset of the first response. Movement times (MTs) represent the interval between the onset of the first and the second keypress response. MT analysis is important in studies using double keypress responses as changes in the MTs could potentially influence the amount of response activation overlap.

Only trials with RTs between 100 ms and 1500 ms were considered correct. All trials with missing responses or too-slow responses (RTs > 1500 ms) and too fast responses (RT < 100 ms) in trial N-2, N-1 or trial N were discarded from the analysis (< 1%). Data were split according to the relationship of trial N-2 to trial N-1, which resulted in three trial transition levels (identical-X, equivalent-X, and different-X). In the analysis of error rates, only choice errors were included.

The dependent variables (RT, choice error rate and MT) were subjected to repeated measures ANOVAs with within-subject variables of trial transition (identical-X vs. equivalent-X vs. different-X), RSI (50 vs. 150 ms) and response condition (1-hand vs. 2-hand). Conservative Huynh-Feldt *F*-tests were used throughout and for post-hoc comparisons, Bonferroni corrections were applied.

2.2.2. Results

Reaction Time

Figure 11 (upper panel) shows mean RT for the first keypress as a function of trial transition, response condition and RSI averaged across participants. The ANOVA revealed a main effect of trial transition, $F(2, 46) = 44.63$, $MSE = 1315.44$, $p < .001$, $\varepsilon = 0.70$, due to the following ordering: different-X (576 ms) > equivalent-X (530 ms) = identical-X (536 ms). Post-hoc tests showed that only equivalent-X and different-X trials

differed significantly, $F(1, 23) = 68.22$, $MSE = 1455.60$, $p < .001$. Post-hoc comparisons between identical-X and equivalent-X trials did not show a significant difference, $p > .10$.

RTs were faster for 1-hand trials (532 ms) than for 2-hand trials (563 ms), resulting in a main effect of response condition, $F(1, 23) = 12.37$, $MSE = 5392.31$, $p < .01$. Moreover, the main effect of RSI was significant, $F(1, 23) = 130.44$, $MSE = 796.84$, $p < .001$, with RTs being faster for the long (528 ms) than for the short RSI (566 ms).

Trial transition interacted with response condition, $F(2, 46) = 35.95$, $MSE = 522.84$, $p < .001$, $\varepsilon = 1.00$. Post-hoc comparisons revealed that the ABI effect (different-X minus equivalent-X) was larger for the 2-hand (63 ms) than for 1-hand condition (28 ms), $F(1, 23) = 44.92$, $MSE = 333.07$, $p < .001$. No difference between response conditions was observed when comparing identical with equivalent trials, $F < 1$.

The interaction of trial transition and RSI was significant, $F(2, 46) = 3.57$, $MSE = 189.63$, $p < .05$, $\varepsilon = 1.04$, due to slightly faster responses for equivalent-X than identical-X trials for long (510 vs. 520 ms, respectively), $F(1, 23) = 11.46$, $MSE = 214.27$, $p < .01$, but not short RSIs (551 vs. 552 ms, respectively), $F < 1$. RSI did not affect the size of the ABI effect, $F < 1$.

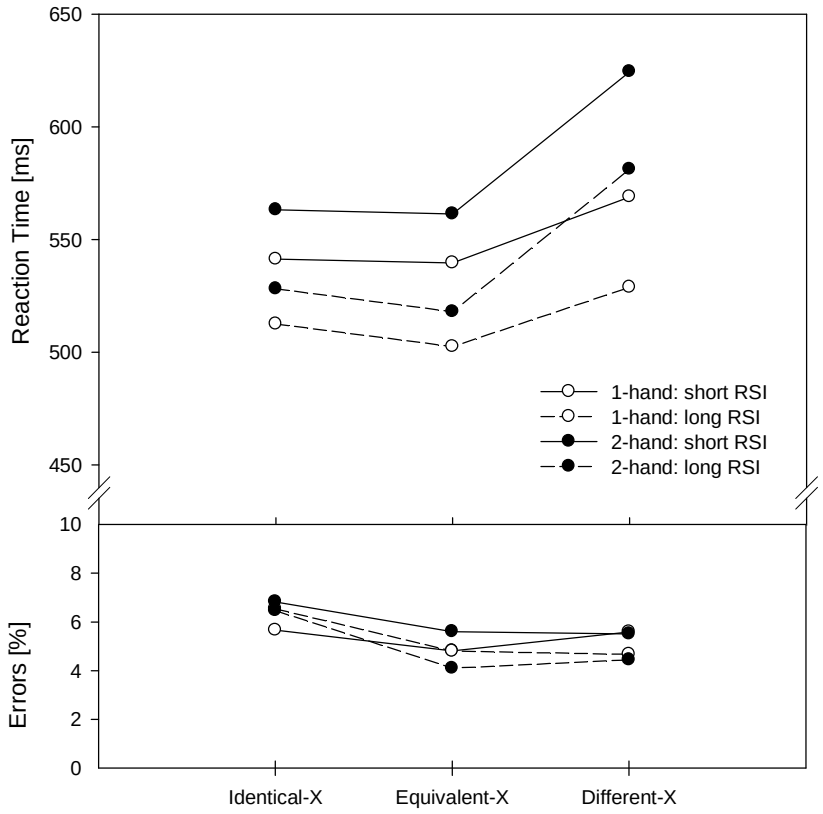

Figure 11. Mean RT (top panel) and percentage choice error (lower panel) as a function of RSI, response condition and second-order trial transition.

Error Rate

The overall error rate was 5.4 %. Figure 11 (lower panel) shows mean error percentage as a function of trial sequence, response condition and RSI averaged across participants. There was a main effect of trial transition, $F(2, 46) = 12.75$, $MSE = 5.25$, $p < .001$, $\varepsilon = 1.09$, due to the following ordering: identical-X (6.4 %) > equivalent-X (4.8 %) = different-X trials (5.1 %). Neither the main effect of response condition ($F < 1$) nor the effect of RSI was significant ($F < 1$). The interaction of trial transition and RSI was significant, $F(2, 46) = 3.68$, $MSE = 2.86$, $p < .05$, $\varepsilon = 0.96$. Post-hoc tests showed that is interaction is due to the difference between identical-X and equivalent-X trials being significant in the long RSI condition only, $F(1,23) = 18.82$, $MSE = 5.36$, $p < .001$.

Movement Time

Figure 12 shows movement time. The main effect of trial transition was significant, $F(2, 46) = 14.41$, $MSE = 48.70$, $p < .001$, $\varepsilon = 1.03$. Post-hoc tests showed that this effect is due to the following ordering: identical-X trials (213 ms) = equivalent-X trials (212 ms) > different-X trials (208 ms). MTs in the 1-hand condition had a tendency to take longer (223 ms) than in the 2-hand condition (199 ms), $F(1, 23) = 3.31$, $MSE = 12283.28$, $p = 0.08$. The interaction of trial transition, response condition and RSI was significant, $F(2,46) = 4.19$, $MSE = 41.08$, $p < 0.05$, $\varepsilon = 0.91$.

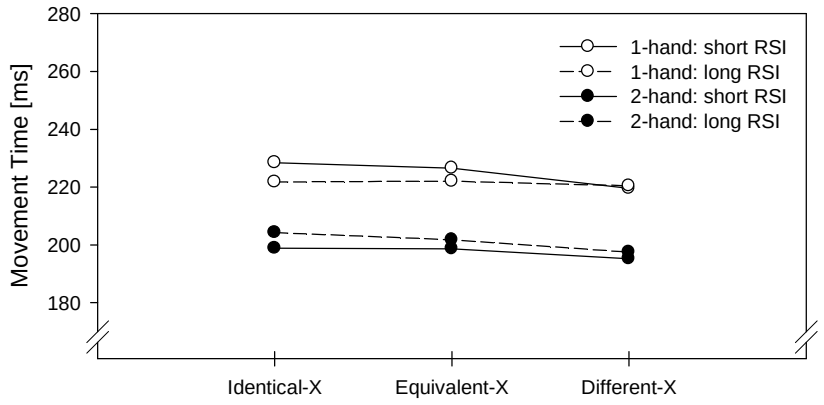

Figure 12. Movement time as a function of RSI, response condition and second-order trial transition.

2.2.3. Discussion

Experiment 1 did not allow an unambiguous interpretation of ABI effects in terms of an effector versus abstract response code level conflict origin. The present experiment aimed to investigate this more closely in an experimental setting that adopted sequential keypress responses. In the 1-hand condition, participants had to react with left or right index finger double keypresses. In the 2-hand condition, participants had to react with a left-right index or a right-left index finger keypress sequence. If overlap of pure effector specific activation leads to conflict, response adjustments in different-X trials should be absent in the 2-hand condition. This is because each response in the 2-hand condition involves both effectors and thus, effector specific activation overlap should be similar in all three trial transitions. However, the ABI effect was larger in the 2-hand than the 1-hand response condition. This finding contradicts the predictions of the effector specific conflict hypothesis, and suggests that conflict is likely to originate at an abstract level of response activation.

The analysis of the MTs showed that MTs ($RT_{second} – RT_{first}$) were in average 24 ms shorter in the 2-hand condition compared to the 1-hand response condition. Critically, in studies implementing double keypress responses, MT differences could influence the amount of activation overlap between trials. However, in the present study the RSI started with the second keypress. Thus, if the MTs are particularly long, this would enlarge the interval between the first keypress in a given trial and the subsequent trial. Importantly, however, the predictions of the effector specific response hypothesis are based on the activation of keypress responses directly following each other (e.g. the second keypress in trial N-2 and the first keypress in trial N-1). Thus, changes in MTs should not affect the size of effector specific response conflict and the MT differences cannot account for the presence of the ABI effect in the 2-hand condition. Given that the results suggest that conflict most likely arises at a level of abstract response activation, this MT shortening in the 2-hand condition could possibly account for the larger ABI effect in the 2-hand condition compared to the 1-hand condition (63 vs. 28 ms, respectively). This is, if one assumes that abstract response activation is highest at response initiation with the first keypress. However, the fact that the 100 ms RSI difference (50 vs. 150ms) did not affect the size of the ABI effect, speaks against such an impact of the 24 ms MT difference. Thus, it remains questionable why the ABI effect is larger in the 2-hand response condition.

A possible explanation for the larger ABI effect in the 2-hand condition is the increase in response complexity, since two fingers as well as two hands are involved in each response. One could assume that response complexity enlarges the amount of abstract response code activation, which would explain the increase in the size of the ABI effect. In line with this suggestion are the findings from a fMRI study investigating the

effects of response complexity by comparing repetitive finger movements with sequential movements (Wexler, et al., 1997). They reported that with increasing task demands and response complexity, brain activity increased and spread further in the cortex. This fits well with the suggestion that response code activation increases with increasing response complexity, resulting in increased activation overlap and larger conflict (Botvinick et al., 2001).

In summary, the presence of the ABI effect in the vocal response condition (Experiment 1) and in the 2-hand response condition (Experiment 2) provides converging evidence that conflict in the case of the ABI effect arises at an abstract rather than effector specific response code levels.

2.3. Experiment 3

The following experiment is published in Psychophysiology 45 (5), 751-758, Locus of response slowing resulting from alternation based interference, Dudschig and Jentzsch (2008).

Jentzsch and Leuthold's (2005) study suggested that the ABI effect is a processing adjustment resulting from conflict in a response-related stage. In addition, the first two experiments in this thesis revealed that abstract response code overlap is most likely to cause this conflict. However, information about the origin of the conflict does not necessarily allow us to draw conclusions about the impact of post-conflict response adjustments on performance. In other words: How does information processing change after conflict detection? In addition, which stage within the information processing is actually slowed down after high conflict trials? These questions will be addressed in Experiment 3. Current models of cognitive control are relatively vague in their specification of how exactly conflict-related control adjustments affect subsequent processing. A number of possibilities can be proposed. These possibilities are introduced in the following paragraphs.

The first possibility to explain response slowing after response alternation trials is that participants choose a more conservative point on the SAT function, as suggested by Botvinick et al. (2001). After conflict detection participants raise the response threshold and thus, more evidence has to be accumulated before the next response will be initiated, resulting in response slowing. If shifts on the SAT function explain response slowing after conflict detection in the case of the ABI effect, such an increase of the response threshold should always be expressed by both slower and more accurate performance. Yet, Jentzsch and Leuthold (2005) did not find an increase in response accuracy after response alternation trials (i.e. high conflict trials). In contrast, response accuracy was slightly reduced in two of their four experiments. Similarly, in the first and second experiment in this thesis, response accuracy did not increase after a response alternation. Thus, changes in SAT settings after conflict detection cannot fully account for the ABI effect.

An alternative explanation of response slowing after conflict could be that the rate of information accumulation changes (cf. Jones et al., 2002; Laming, 1968). In contrast to changes in the response threshold, changes in the information accumulation rate can account for response slowing effects which are not accompanied by a decrease in error rates. In fact, Laming suggested that in a sequence of alternating trials, information accumulation might be impaired in addition to being slower. This may result in a defective decision process because "the mean rate of extraction of information from the signal presented is less after an alternating sequence of signals" (p. 110). This means that after

alternation trials, more time may be required to accumulate information for initiating the response in the following trial. Moreover, this suggests that stimulus-response (S-R) processing becomes more difficult, possibly resulting in a loss of information. Importantly, changes in information accrual rate can not only affect overall information processing but potentially can affect selective information processing stages. For example, early perceptual stimulus processing could be unaffected by previous conflict detection and require the usual time, but the information accumulation rate might be reduced in central response selection stages, resulting in slowing of this specific information processing stage only. In summary, a change in the information accumulation rate could possibly account for the behavioural adjustments after response alternation trials. Such a mechanism of a selective influence on specific stages of information processing will be referred to in the following paragraphs as the *selective slowing hypothesis*.

A third possible approach to account for post-conflict adjustments might be that after conflict detection total subsequent trial processing is postponed, including even very early visual processing stages. In a recent study it was shown that in an alternating runs task-switching paradigm, switches in task-set can delay very early perceptual processing stages (Oriet & Jolicœur, 2003). Oriet and Jolicoeur utilised locus of slack logic (see Pashler & Johnston, 1989) while manipulating RSI and stimulus contrast. They assumed that the process of task-set reconfiguration occupies central resources and that this stage is specific to task-switch trials. They predicted that early perceptual stages would take place in parallel with task-set reconfiguration and thus, the effect of contrast at short RSIs would be absorbed into the slack period created by the reconfiguration process (i.e. an underadditive interaction). However, the effect of contrast was additive across RSI levels. This led the authors to conclude that the process of task-set reconfiguration acts as a hard bottleneck delaying even early perceptual processing. One could assume that after a response alternation trial, all trial processing might be postponed in a similar way due to reconfiguration of S-R mapping, resulting in the ABI effect. Such a mechanism will be referred to as the *hard bottleneck hypothesis* in the following paragraphs.

In order to distinguish between the *selective slowing* and the *hard bottleneck* hypothesis, the onset of the stimulus and response synchronized lateralized readiness potential (LRP), the peak latency of early visual components (P1 & N1) and the P300 will be analyzed. As described in the introduction, the LRP is an electrophysiological correlate indicating selective response activation (Coles, Gratton, & Donchin, 1988; De Jong, Wierda, Mulder, & Mulder, 1988). The interval from stimulus to the onset of the stimulus-synchronized LRP (S-LRP interval) indicates the amount of premotoric processing time, whereas the interval between the onset of the response-synchronized LRP and the response (LRP-R interval) is viewed as a relative measure for the duration

of motor processing time. Therefore, the LRP can be used to identify whether response slowing after a response alternation delays premotoric or motoric processing stages.

Importantly, the two remaining hypotheses for explaining alternation-based control adjustments predict distinct patterns of delays in the ERP components. On the one hand, if a *hard bottleneck* (e.g. Oriet & Jolicoeur, 2003) causes the response slowing observed in the ABI effect, even very early stimulus processing would be delayed. Thus, this hypothesis predicts that the P1, N1 and also P300 latencies would be delayed after a response alternation trial. On the other hand, if slower RTs in trials following a response alternation are due to *specific slowing* of processing in central or motoric stages, this hypothesis predicts that the S-LRP or LRP-R onset latencies should be affected, respectively. Critically, no effects in the visual ERP components should be observed.

2.3.1. Methods

Participants

24 participants (M_{age} = 21.9 years, SD_{age} = 3.0 years, 16 woman) with normal or corrected-to-normal vision were tested in a single session lasting approximately 70 min. All participants were naive about the experimental hypothesis, gave informed consent and were paid £8.

Stimuli and Apparatus

Stimuli and apparatus are the same as those reported in the first experiments. The stimuli consisted of the letters D, M, T and X presented centrally on the screen. The contrast of these letter stimuli was manipulated. Stimuli were presented on white background either in light grey (52 cd/m2, low stimulus contrast) or dark grey (1 cd/m2, high stimulus contrast). In addition, a black fixation cross (3 x 3 mm) was presented in the centre of the screen during the RSI periods.

EEG recording

EEG activity was recorded using a BIOSEMI Active-Two amplifier system with 72 Ag/AgCl electrodes. Two additional electrodes (Common Mode Sense (CMS) active electrode and Driven Right Leg (DRL) passive electrode) were used as reference and ground electrodes, respectively. EEG and EOG recordings were sampled at 256 Hz. Off-line, all EEG channels were recalculated to average reference. Horizontal EOG (hEOG) waveforms were calculated as follows: hEOG(t) = F9(t) minus F10(t). Trials containing blinks were corrected using a dipole approach (BESA Version 5.1.6; Berg & Scherg, 1994).

Procedure

Participants were asked to perform simple key-press responses with their index fingers. Two letters were mapped to the left index finger and two letters to the right index finger (e.g., D and M => left key, X and T => right key). The assignment of letters to response alternatives was balanced across participants. The experiment consisted of two parts, using either a short RSI (100 ms) or a long RSI (1000 ms). Half of the participants started with the short and the other half with the long RSI condition. Stimulus contrast alternated between blocks. Half of the participants started with high contrast and the other half with the low contrast stimuli. For each RSI condition two practice blocks (80 trials each) and ten experimental blocks (160 trials each) were presented.

Data Reduction and Analysis

Behavioural data was analysed in the same fashion as Experiment 1 and Experiment 2. Dependent variables (RT and error rate) were subjected to repeated measures ANOVAs with within-subject variables of second-order transition (identical-X, equivalent-X, different-X), RSI (100 vs. 1000 ms) and stimulus contrast (high vs. low). Again, conservative Huynh-Feldt *F*-tests were used throughout and for post-hoc comparisons, Bonferroni corrections were applied.

The EEG recordings were examined for artifacts (amplifier blocking, scalp muscular activity, and slow linear drift) for each participant and trial. Only trials with correct responses in trial N-2, N-1 and trial N, and without EEG artifacts were included in EEG data analysis. All signals were 10 Hz filtered and averaged separately for each experimental condition. The epoch for the S-locked data started 200 ms prior to stimulus onset and lasted for a total duration of 1200 ms. For R-locked analyses epochs started 1000 ms before the response and lasted for 1200 ms. Peak latencies of the P1, N1 and P300 were determined using an automatic peak detection algorithm. For the P1 and N1 components, peak latency was analysed over PO7 (left) and PO8 (right) electrode sites using two separate ANOVAs. P300 was analysed over the Pz electrode.

The LRP was calculated for each participant and each experimental condition. The ERP over the primary motor cortex ipsilateral to the response hand was subtracted from the ERP at contralateral recording sites, using the electrodes C3 and C4. The resulting difference waveform was averaged across hands to eliminate any ERP activity unrelated to hand-specific motor activation (cf. Coles, 1989). LRP onsets were measured and analysed by applying the jackknife-based procedure (J. Miller, Patterson, & Ulrich, 1998; Ulrich & Miller, 2001). 24 different grand average LRPs for each of the experimental conditions were computed by omitting from each grand average the ERP data of another participant. LRP onsets were determined in the waveform of each grand average. S-LRP onsets were measured relative to a 100 ms pre-stimulus baseline at the

point in time when LRP amplitude reached 50 % of maximal LRP amplitude in that specific condition. Effects in the LRP-R interval were obtained using the same relative LRP onset criteria with waveforms referred to a 100-ms baseline starting 600 ms before the actual response. As shown in previous work (e.g. Jentzsch & Sommer, 2002), LRP activation strongly accumulates over trials. If the RSI is very short, the LRP amplitude relative to a prestimulus baseline will therefore be extremely small. In the present experiment the S-LRP amplitude did not reach 0.5 µV in the short RSI condition. Therefore only ERP data for the long RSI was analysed.

LRP onsets and peak latencies of the P1, N1 and P300 components for the long RSI condition were submitted to separate ANOVAs with the independent variables contrast and second-order sequence. For jackknifed data, the *F*-values were corrected as follows: $F_c = F/(n-1)^2$, where F_c denotes the corrected *F*-value and n the number of participants (Ulrich & Miller, 2001). Conservative Huynh-Feldt *F*-tests were used throughout and for post-hoc comparisons Bonferroni corrections applied.

2.3.2. Behavioural Results

Reaction Times

Figure 13 (upper panel) shows mean RT as a function of second-order transition, stimulus contrast and RSI. The ANOVA revealed all three main effects to be significant. RTs were faster for high contrast (510 ms) than for low contrast stimuli (535 ms), $F(1, 23) = 64.09$, $MSE = 736.34$, $p < .001$. RTs were faster for long RSIs (498 ms) than for short RSIs (547 ms), $F(1, 23) = 24.89$, $MSE = 6955.89$, $p < .001$. Finally, effect of second-order transition was significant, $F(2, 46) = 77.02$, $MSE = 479.88$, $p < .001$, $\varepsilon = 0.90$, due to the following ordering of RTs showing a clear ABI effect: identical-X (514 ms) = equivalent-X (509 ms) < different-X trials (545 ms). Post-hoc tests comparing equivalent and different trials showed that these conditions differed significantly, $F(1, 23) = 142.03$, $MSE = 442.89$, $p < .001$. Post-hoc tests comparing identical-X and equivalent-X trials showed no difference, $F(1, 23) = 3.72$, $p = .13$.

There was also a significant interaction between RSI and trial transition, $F(2, 46) = 10.81$, $MSE = 349.19$, $p < .01$, $\varepsilon = 0.75$. This interaction was due to an increased ABI effect (difference between equivalent-X and different-X trials) for the short compared to the long RSI condition, $F(1, 23) = 28.51$, $MSE = 255.89$, $p < .001$. Importantly, although the ABI effect was numerically reduced in the long RSI condition, it was still significant, $F(1, 23) = 51.54$, $MSE = 265.40$, $p < .001$. No RSI dependent change was found when comparing identical-X and equivalent-X trials, $F(1, 23) = 3.77$, $p = .13$. No other effects reached significance, all $Fs < 1.5$, $ps > .20$.

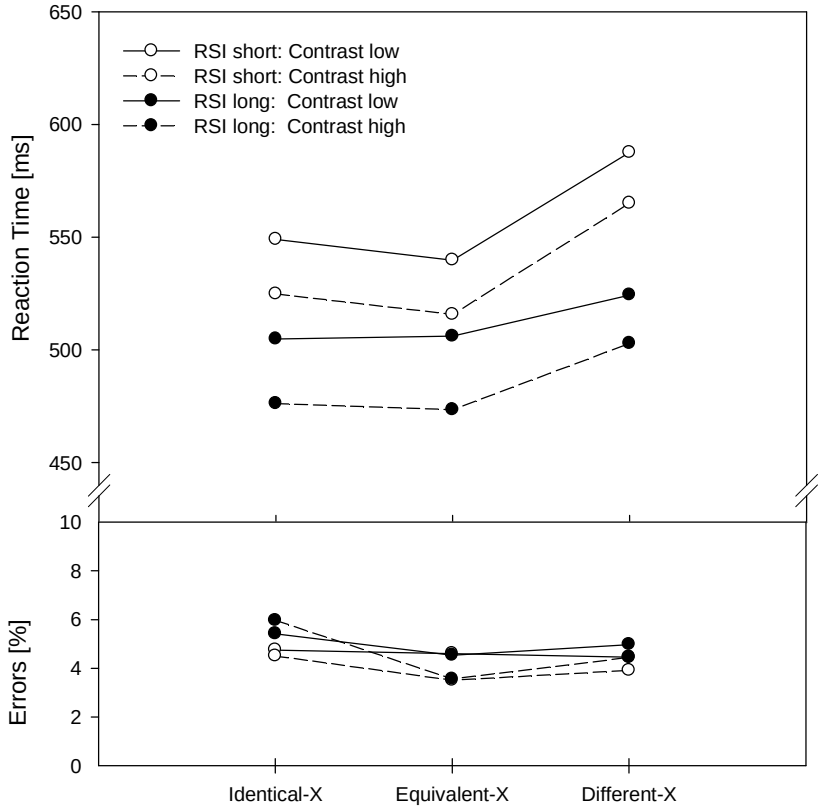

Figure 13. Reaction times and percentage of errors as a function of second-order transition, RSI and stimulus contrast.

Error Rates

Figure 13 (lower panel) shows mean error rate as a function of second-order transition, stimulus contrast and RSI. The ANOVA revealed a significant effect of stimulus contrast, $F(1, 23) = 6.01$, $MSE = 2.73$, $p < .05$, due to a higher error rate for low contrast (4.79 %) than high contrast trials (4.31 %). The effect of second-order trial transition was significant, $F(2, 46) = 8.35$, $MSE = 3.62$, $p < .001$, $\varepsilon = 1.0$, due to the following ordering: identical-X trials (5.16 %) > equivalent-X (4.05 %) = different-X (4.45 %).

The interaction of stimulus contrast and trial transition was significant, $F(2, 46) = 3.80$, $MSE = 2.24$, $p < .05$, $\varepsilon = 0.74$, due the presence of a contrast effect for equivalent-X trials, $F(1, 23) = 7.20$, $MSE = 3.59$, $p < .05$, but not for the other two sequence types, ps > .1. Additionally, there was an interaction between RSI and trial transition, $F(2, 46) = 3.71$, $MSE = 1.86$, $p < .05$, $\varepsilon = 1.0$.

2.3.3. Electrophysiological Findings

LRP Onset

The ANOVA of the S-LRP onset showed a main effect of contrast, F_c (1, 23) = 5.95, *MSE* = 4.67, $p < .05$. S-LRP onset (see Figure 14, top panels) occurred earlier in the high contrast (302 ms) than the low contrast condition (334 ms). The main effect of second-order trial transition was significant, F_c (2, 46) = 3.93, *MSE* = 4.91, $p < .05$, $\varepsilon = 1.0$, due to the following ordering: identical-X (320 ms) = equivalent-X (307 ms) < different-X trials (327 ms). A post-hoc test comparing identical and equivalent trials only, showed no effect of sequence, $F_c < 1$. However the post-hoc test comparing equivalent and different trials only, showed a significant effect of sequence, F_c (1, 23) = 9.03, *MSE* = 3.78, $p < .05$. There was no significant interaction between stimulus contrast and sequence, $F_c < 1$.

The LRP-R onset (see Figure 14, bottom panels) was not significantly different in high and low contrast trials, $F_c < 1$. Also, there was no effect of trial transition on the LRP-R onset, $F_c < 1$, nor was there an interaction between contrast and sequence, $F_c < 1$.

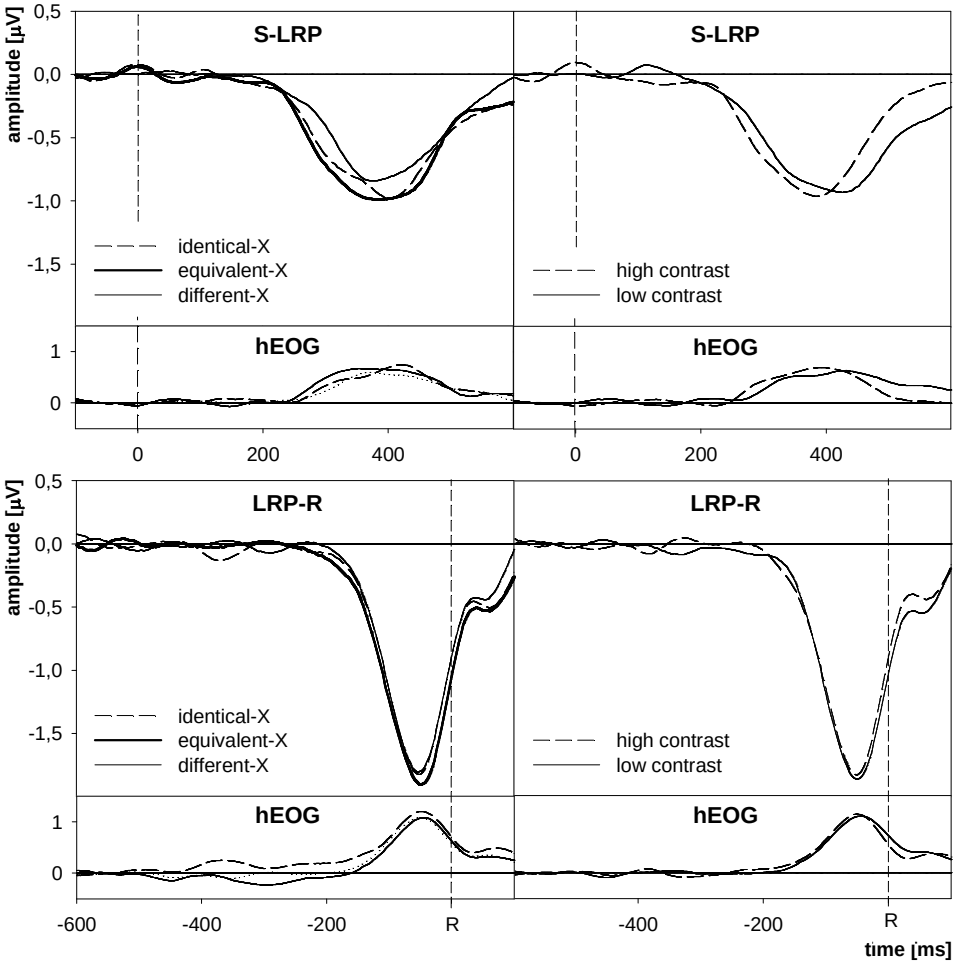

Figure 14. Stimulus-locked LRP and hEOG waveforms (top) and response-locked LRP and hEOG waveforms (bottom) in the long RSI condition as a function of second-order transition (left panels) and stimulus contrast (right panels).

P1 and N1 peak latency

P1 peak latency was earlier for high contrast than low contrast stimuli (see Figure 15), both over the left hemisphere, $F(1, 23) = 21.07$, $MSE = 2.03$, $p < .001$, (115 ms vs. 138 ms), and the right hemisphere, $F(1, 23) = 21.65$, $MSE = 2.20$, $p < .001$, (118 ms vs. 138 ms). There was no effect of trial transition and no interaction between stimulus contrast and trial transition, $F < 1$.

Analysis of N1 peak latency showed a significant main effect of stimulus contrast in both the left hemisphere, $F(1, 23) = 55.63$, $MSE = 1.15$, $p < .001$, (high contrast: 188 ms; low contrast: 220 ms) and right hemisphere, $F(1, 23) = 27.68$, $MSE = 1.97$, $p < .001$, (high contrast: 192 ms; low contrast: 216 ms). Again, there was no main effect of trial transition, and no interaction between stimulus contrast and trial transition in either hemisphere, $F < 1$.

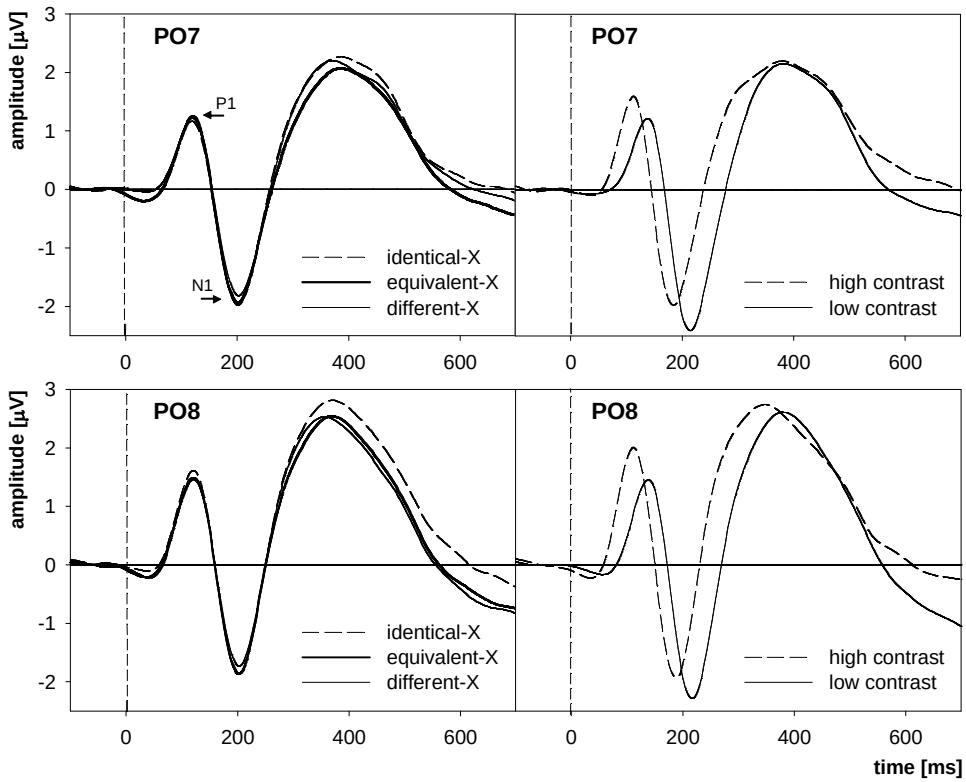

Figure 15. Stimulus-locked event-related brain potentials over the left hemisphere (PO7 electrode, top) and the right hemisphere (PO8 electrode, bottom) in the long RSI condition as a function of second-order transition (left panels) and stimulus contrast (right panels).

P300 peak latency

P300 peak latency (see Figure 16) showed a main effect of stimulus contrast, $F(1, 23) = 4.62$, $MSE = 13.11$, $p < .05$, peaking earlier for high contrast (480 ms) than low contrast (517 ms) stimuli. There was no main effect of trial transition and no interaction between stimulus contrast and trial transition, $F < 1$.

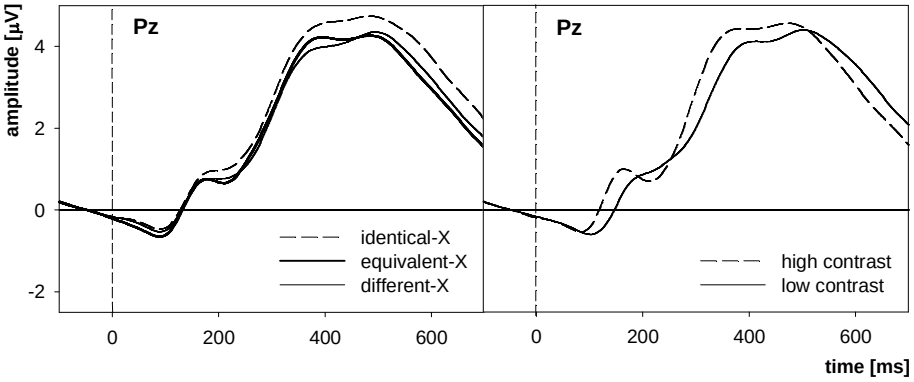

Figure 16. Stimulus-locked event-related brain potentials at the Pz electrode in the long RSI condition as a function of second-order transition (left panel) and stimulus contrast (right panel).

2.3.4. Discussion

The aim of Experiment 3 was to investigate how information processing is affected by post-conflict adjustment after response alternations. Knowledge about the locus of conflict adjustments in the case of the ABI effect can help further theoretical understanding of the mechanisms underlying post-conflict adjustment. Two hypotheses regarding the mechanisms underlying the slowing after response alternations were contrasted. First, similarly to the proposed task-set reconfiguration bottleneck in task switch paradigms (Oriet & Jolicœur, 2003), after response alternations all resources might be used for reconfiguring S-R mapping, and thus delay subsequent processing until reconfiguration has been completed. Importantly, such a *hard-bottleneck* mechanism would cause a postponement of all information processing in the following trial, including very early perceptual processing stages. The alternative *selective prolonging* hypothesis claims that RT adjustments after high-conflict trials are due to changes of the information accrual rate (cf. Jones et al., 2002; Laming, 1968). Importantly, compared to the hard-bottleneck hypothesis which claims a complete postponement of information processing, the change of information accrual rate can take place selectively at specific processing stages at any point of the information processing chain.

In line with the original study by Jentzsch and Leuthold (2005) and the first two experiments in this thesis, responses that followed response alternation trials were significantly slower. Importantly, this ABI effect was present in both the long and the short RSI condition, albeit the ABI effect was clearly reduced in the long RSI condition. The decrease in the size of the ABI effect with increasing RSI is in line with one of the basic

assumptions regarding the origin of the ABI effect. More specifically, response activation is assumed to extend beyond response execution and to decay over time. Thus, activation overlap (i.e. conflict), decreases with increasing RSI as there is more time for the response activation to decay before the next trial starts.

To investigate the locus of the ABI effect within the information processing chain, ERP measurements were made. As LRP activation strongly accumulates over trials (Jentzsch & Summer, 2002), ERP analysis was only conducted for the long RSI condition. The analysis of the LRP revealed that the ABI effect observed in RTs is reflected in a delay of S-LRP onset latency. In contrast, LRP-R onset latencies were not affected by previous response alternations. These results suggest that information processing in premotoric processing stages is affected by previous response alternations, whereas motor processing seems largely unaffected. P1 and N1 peak latencies, known to reflect early visual processes, were not affected by previous response alternations. In addition, the P300 peak latency, a component known to reflect stimulus evaluation processes, did not show any delays in accordance with the ABI effect. Taken together, these findings provide strong evidence for selective changes in premotoric but postperceptual information processing stages after the detection of conflict resulting from response alternations in previous trials. This suggests that central, response selection stages are the most likely locus of response adjustments in the case of the ABI effect. Importantly, the findings regarding the locus of response slowing after response alternation trials allows one to discriminate between the two hypotheses regarding the underlying mechanisms of the behavioural adjustments. The hard bottleneck hypothesis induced through stimulus-response reconfiguration can be excluded (cf. Oriet & Jolicoeur, 2003), as early perceptual components were not affected by previous response alternations. In contrast, the selective locus in central, response selection stages suggests that a change in the speed of information accumulation at central levels is likely to underlie post-conflict adjustments after response alternations (e.g. Jones et al., 2002).

However, alternative mechanisms which may account for these findings need to be considered. First, the delayed onset of hand-specific response activation after response alternations might result from an additional motor suppression process that is not the result of slower information accrual rates. More specifically, response alternations might result in generally increased activation in the motor system. Thus, in order for subsequent responses to reach a sufficient level of lateralization, both a suppression of the unwanted response alternative in addition to activation of the intended alternative would be required. This could potentially explain the delayed onset of the S-LRP after response alternations. Critically, such an explanation would predict an interaction between first- and second-order sequences with larger suppression needed for first-order

alternations than repetitions. Specifically, if the response changes from trial N-1 to trial N more suppression would be needed than if the response of trial N is a repetition of the previous response. However, as described in the introduction, the ABI effect is remarkably independent of first-order transitions, thus speaking against such a suppression mechanism.

Problematically, ERP data could not be analyzed in short RSI trials due to accumulation of the LRP activation over trials (see Jentzsch & Sommer, 2002). Although the ABI effect is reduced at the long RSIs, the effect is still present, which is crucial for the valid interpretation of the S-LRP onset delay. However, previous studies have reported a complete absense of the ABI effect for RSIs equal or longer than 500 ms (e.g. Soetens, Boer, & Hueting, 1985; Soetens & Notebaert, 2005). However, task-dependent differences in the temporal dynamics of the ABI effect might explain the discrepancy between these two previous studies and the present study. For example, weighting factors in response nodes as well as decay parameters might be different depending on the stimulus–response mapping, thus leading to a different amount of response activation, conflict and subsequent control adjustment. In the present study, a four-to-two S-R mapping was used, whereas in the studies of Soetens et al., a two-to-two S-R mapping was used. In addition, most other relevant studies have used spatial rather than symbolic S-R mappings, which is likely to influence the timing of response activation.

In conclusion, the present study suggests that the origin of processing conflict and subsequent conflcit-induced processing changes might not have the same locus. That is, conflict induced by a response alternation from Trial N-2 to Trial N-1 seems to influence current Trial N performance by selectively prolonging response selection stages, resulting in the ABI effect. These results emphasise the importance of a clear distinction between the locus of conflict origin and the locus of subsequent changes in information processing. However, these results might be specific to conflict in the case of the ABI effect.

2.4. Experiment 4

In addition to conflict that occurs via activation overlap in response alternation trials, errors are another example of conflict which can typically occur within uni-dimensional task settings. The following experiments focus on investigating the role conflict plays in erroneous trials, how conflict monitoring is activated during errors and how this can account for behavioural adjustments such as post-error slowing and pre-error speeding. Error processing was considered within the framework of conflict processing based on three major factors. First, conflict as conceptualized within the conflict monitoring theory is very likely to be present in error trials, as the correct and the erroneous response are likely to be coactivated (Rabbitt & Rogers, 1977). Second, behavioural adjustments after errors (i.e. post-error slowing) (e.g. Brewer & Smith, 1984; Laming, 1968; Rabbitt, 1966), resemble post-conflict adjustments triggered through a dynamic up-regulation of cognitive control (Botvinick, et al., 2001). Finally, brain correlates of error processing, especially the ERN, and correlates of conflict processing have both been localised to the ACC (e.g. Dehaene, et al., 1994), suggesting that similar neural processes underlie error processing and conflict processing.

With the identification of neural correlates related to error processing (Falkenstein, et al., 1990; Gehring, et al., 1990), the interest in the mechanisms underlying error processing increased. However, despite increased research investigating error processing, the functional significance of the ERN and its relationship to subsequent behavioural adjustments remains unclear. In Experiment 4 and 5, the two major hypotheses (error detection vs. conflict processing) regarding the underlying mechanisms of the ERN will be contrasted and its relationship to post-error slowing will be investigated.

The *error-detection* or *mismatch* theory proposes that the ERN reflects the error detection process itself (e.g. Coles, et al., 2001; Falkenstein, et al., 2000; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). In this case it is assumed that the ERN is generated on the basis of a comparison between the executed and the required response, with the ERN being elicited when this comparison process results in a mismatch. Therefore, the amplitude of the ERN should depend on the amount of mismatch between the two responses. More specifically, the ERN should increase with increasing mismatch between the correct and the erroneous response.

The *conflict-processing* theory proposes that ERN amplitude reflects conflict size (e.g. Botvinick, et al., 2001; Carter, et al., 1998b; Yeung, et al., 2004). Conflict is defined as competing activations within response channels, and is assumed to be the larger the more similar overlapping response alternatives (Botvinick, et al., 2001). It is assumed that an error can elicit a strong conflict signal due to simultaneous activation of the representation for the executed erroneous response and the representation of the

intended correct response (e.g. Jones, et al., 2002; Yeung, et al., 2004). Thus, if the ERN reflects conflict, it should be larger the more similar the erroneous response is to the required correct response.

In order to test these hypotheses, two 4-choice tasks will be implemented. The similarity between the required correct and the executed erroneous response can be defined as the degree of overlap between movement parameters (Rosenbaum & Kornblum, 1982). In 4-choice tasks response similarity varies between the three possible error types (e.g. Bernstein, et al., 1995; Gehring & Fencsik, 2001). For example, in a 4-choice task where responses have to be performed with the index and middle fingers of both hands, participants can commit the following types of errors: First, participants may respond with the correct side but incorrect effector (e.g. left index instead of left middle, CS-WE); second, participants may respond with the incorrect side but the correct effector (e.g. left index instead of right index, WS-CE); and finally, participants may respond with the wrong hand and the wrong effector (e.g. left index instead of right middle, WS-WE). Whereas the first and second error types share either response side (left vs. right) or effector (index vs. middle) with the correct response, the third error type does not share any response feature.

Previous findings regarding the size of the ERN in different errortypes within 4-choice tasks have been ambiguous (Bernstein, et al., 1995; Falkenstein, Hohnsbein, & Hoormann, 1996; Gehring & Fencsik, 2001). Bernstein et al. (1995) used four fingers as response effectors and found the smallest error rate and largest ERN for WS-WE error types (i.e. errors not sharing any feature with the correct response), thus supporting the error detection hypothesis. In contrast, Gehring and Fencsik (2001) used index fingers and toes as response effectors and found the ERN amplitude to decrease with an increasing mismatch between the executed erroneous response and the intended correct response, supporting the idea that conflict detection is reflected in the ERN. Gehring and Fencsik (2001) argued that the results of Bernstein et al. (1995) are questionable, as adjacent fingers interact on muscular, biomechanical and neural activation levels. Problematically, Gehring and Fencsik (2001) used a Stroop task in their experiment, which might lead to additional conflict (i.e. from compatible and incompatible stimulus dimensions) that may overlap with error processing. This may account for the differences in the ERN patterns compared to the findings of Bernstein et al. (1995). In order to overcome these problems, two analogue experiments were conducted. The first (Experiment 4) implemented finger responses only, whereas the second (Experiment 5) implemented finger and foot responses. If the ERN reflects an error detection process, it is predicted to be largest in the WS-WE error condition, which is most dissimilar to the required correct response. In contrast, if the ERN reflects conflict processing, it is predicted to be smallest in the WS-WE error condition.

2.4.1. Method

Participants

20 participants (M_{age} = 22.05 years, SD_{age} = 2.56 years, 16 woman) were included in this experiment. Participants had normal or corrected-to-normal vision and were tested in a single session. All participants were naive about the experimental hypothesis, gave informed consent and were paid £10.

Apparatus and Stimuli

Apparatus and stimuli were identical to the previous experiments if not stated differently. The four response keys were mounted in the horizontal plane in front of the participant. The four fingers were placed on the buttons in the following order starting with the left middle, followed by the left index, right index and right middle finger. The hands were approximately 15cm apart, and the fingers rested naturally on the keys. The stimuli consisted of the letters D, M, T and X presented centrally in black on a white background. In addition, a black fixation cross (3 x 3 mm) was presented in the centre of the screen during the RSI periods.

EEG recording

EEG recording was identical to the previous experiments.

Procedure and design

Participants were instructed to respond fast and accurately, and to keep the error rate below approximately 10 %. They were asked to perform simple key-press responses. Responses were performed with the index and middle fingers of the left and right hand. Each letter was mapped to one of the four response alternatives (e.g. D → left index, M → left middle, X → right index and T → right middle). The assignment of letters to response alternatives was balanced between participants, using a Latin square design. The interval between response execution and the following stimulus (RSI) was 1000 ms. The experiment consisted of 15 experimental blocks (160 trials each) and two practice blocks (40 trials each).

Data Analysis

Behavioural Data

Both reaction time (RT) and response accuracy were measured in each trial. Error trials were classified as one of three types, varying in the amount of response parameters shared with the required correct response (CS-WE, WS-CE, WS-WE).

Only trials with an RT between 200 and 1500 ms in were considered as correct. All trials with missing or too slow responses (< 1%) and too fast responses (< 1%) in

trials N-1 and/or trial N and/or trial N+1 were discarded from the analysis. For all experimental conditions a minimum of 18 trials per analysis cell were applied. As a result, 8 out of 28 tested participants had to be excluded from the data analysis.

ERP data

The EEG recordings were examined for artifacts (amplifier blocking, scalp muscular activity, and slow linear drift) for each participant and trial. Only trials without EEG artifacts were included in the EEG data analysis. All signals were averaged separately for each experimental condition. The response-synchronized epochs (time-locked to onset of keypress response) started 200 ms prior to response onset and lasted for a total duration of 1000 ms. All averages were baseline corrected, using a 100-ms baseline starting 150 ms before the onset of the response and were low-pass filtered at 15 Hz. The ERN was subjected to measurement of peak amplitude (time window for automatic peak detection was 0 to 100 ms after response onset) using the FCz electrode.

2.4.2. Behavioural Results

Error Reaction Time

The RTs for the different error types are shown in Figure 17. Error RTs varied with the similarity between the executed error response and the required correct response. This finding was supported by a main effect of Errortype, $F(2,38) = 8.39$, $MSE = 505.91$, $p < .001$, $\varepsilon = 1.08$. Post-hoc comparisons showed that there was no difference between the CS-WE (496 ms) and the WS-CE (503 ms) errors, each sharing one response feature with the correct response, $F < 1$. However, both WS-CE and CS-WE were significantly slower than the WS-WE errors (475 ms), $F(1,19) = 17.11$, $MSE = 451.19$, $p < .001$ and $F(1,19) = 7.81$, $MSE = 591.78$, $p < .05$, respectively.

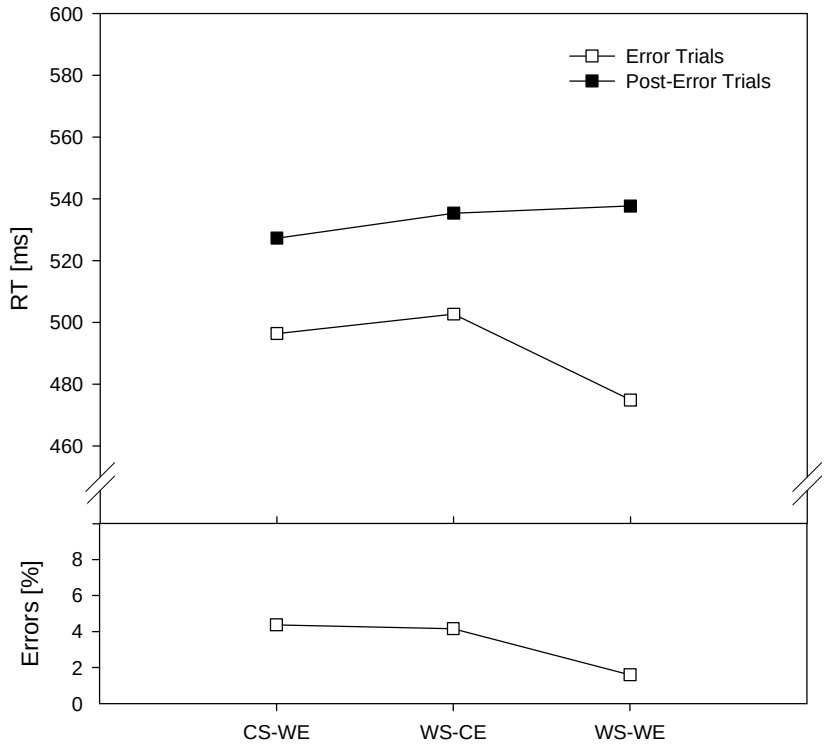

Figure 17. Mean RTs and error rate. Top plot: RTs in Error and Post-Error trials as a function of Errortype. Bottom plot: Error rate of the three Errortypes. CS-WE = Correct Side–Wrong Effector, WS-CE = Wrong Side–Correct Effector, WS–WE = Wrong Side–Wrong Effector.

Error Rates

The relative frequency of each error type varied, showing a clear effect of response similarity (see Figure 17, lower panel). This observation is supported by the main effect of Errortype, $F(2,38) = 21.35$, $MSE = 2.22$, $p < .001$, $\varepsilon = .67$. Post-hoc comparisons showed that there was no difference between CS-WE (4.4 %) and WS-CE (4.2 %) error rates, $F < 1$. However both, WS-CE and CS-WE occurred more frequently than WS-WE (1.6 %) errors, $F(1,19) = 37.90$, $MSE = 1.72$, $p < .001$ and $F(1,19) = 68.56$, $MSE = 1.12$, $p < .001$, respectively. Therefore, both response hand and response finger seem to contribute to the similarity of responses, as errors occur at a similar frequency. Errors are less frequent in the WS-WE condition, supporting the idea that these error types are the least similar in terms of shared features with the required correct response.

Post-Error Reaction Times

Figure 17 (upper panel) shows the post-error RTs as a function of Errortype. Post-error slowing was clearly present. An ANOVA including the post-correct responses (496 ms) shows this effect, $F(3,57) = 13.92$, $MSE = 533.63$, $p < .001$, $\varepsilon = 1.02$. However, there is no effect of Errortype on post-error RTs, $F < 1.56$, $p > .22$.

2.4.3. Electrophysiological Findings

ERP Data

Figure 18 shows the effects of Errortype on the ERN amplitude, with the ERN being largest in the WS-WE condition. The ANOVA showed a significant main effect Errortype on the peak amplitude, $F(2,38) = 7.77$, $MSE = 1.23$, $p < .01$, $\varepsilon = .81$. Post-hoc comparisons showed that the peak amplitude for CS-WE and WS-CE errors did not differ, $F < 1$. However, both the CS-WE and the WS-CE error amplitude differed significantly from the WS-WE error amplitude, $F(1,19) = 8.25$, $MSE = 1.84$, $p < .01$, and $F(1,19) = 11.07$, $MSE = 1.22$, $p < .01$, respectively. Thus, the ERN amplitude increases with increasing mismatch between the erroneous and the correct response.

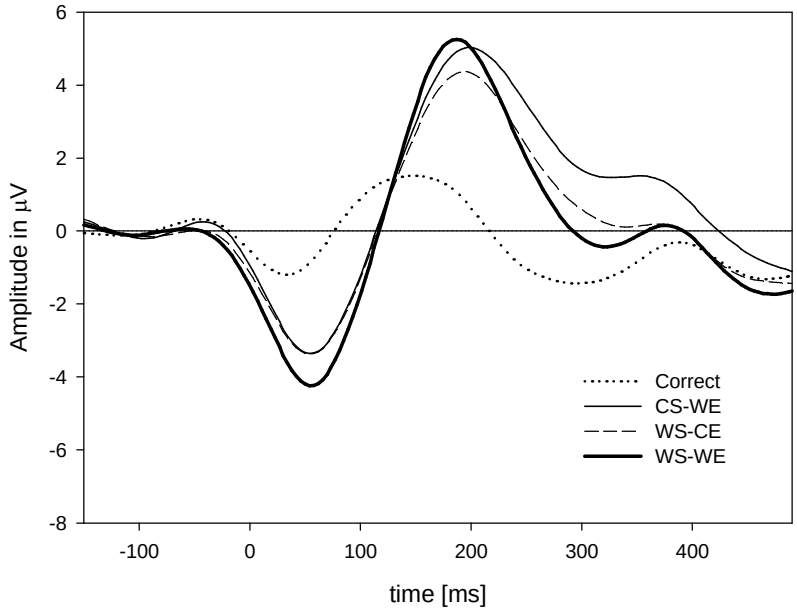

Figure 18. Event-related potential activity over FCz for correct responses and for the three Errortypes, time locked to the response. CS-WE = Correct Side–Wrong Effector, WS-CE = Wrong Side–Correct Effector, WS–WE = Wrong Side–Wrong Effector.

2.4.4. Discussion

The aim of the present experiment was to investigate the mechanisms underlying the ERN. The error detection hypothesis and the conflict detection hypothesis were contrasted. The error detection hypothesis predicts the ERN to increase with increasing mismatch between the executed erroneous response and the required correct response (e.g. Coles, et al., 2001). In contrast, the conflict detection hypothesis predicts the ERN to increase with increasing similarity between the executed erroneous and the required correct response (e.g. Botvinick, et al., 2001). In order to manipulate the similarity

between the erroneous and the correct response a 4-choice task was conducted (see also: Bernstein, et al., 1995; Gehring & Fencsik, 2001). In such an experimental setting, the three possible errors vary in the amount of shared response parameters, and thus, their similarity with the correct response (Rosenbaum & Kornblum, 1982). As expected, the WS-WE error was the least frequent error, supporting the idea that the less response parameters shared with the correct response, the less similar it is to the correct response, and thus, the less likely it will occur.

Importantly, the results showed that the ERN was largest for the WS-WE error, where the mismatch between the erroneous and the correct response is the largest. This clearly supports the error detection hypothesis, and speaks against the idea that the ERN reflects conflict size. Moreover, there was no relation between the size of the ERN and the subsequent amount of post-error slowing. This strengthens the idea that the ERN is related to error processing itself, rather than being the trigger for subsequent compensatory adjustments.

Critically, one could argue the reduced number of errors in the WS-WE condition, and not the decreased similarity between the error and the correct response cause the increase in the ERN. Previous studies (e.g. Gehring, et al., 1993) showed that the ERN increases with decreasing error frequency. However, in these early studies speed-accuracy instructions were manipulated. Thus, in the accuracy condition the error may have had more importance to the participant, with participants adopting more conservative positions along the SAT function. In the present experiment, WS-WE errors were less likely to occur. However, they were also particularly fast errors, thus excluding explanations based on SAT shifts that result in amplitude changes in the ERN, as proposed by Gehring et al.

One of the major criticisms raised by Gehring and Fencsik (2001) at experiments implementing responses with index and middle fingers is that adjacent fingers interfere at low-level motor activation levels, thus making interpretation of results regarding error similarity difficult (e.g. Häger-Ross & Schieber, 2000). Indeed, such low-level interaction could explain the differences in the size of the ERN between CS-WE error (adjacent finger response to the required correct response) and the WS-WE error. However, this does not explain the differences in the ERN amplitude between the WS-CE and the WS-WE condition in the present experiment (see also, Bernstein et al., 1995). In both of these errortypes the wrong response side is involved, and therefore no finger is adjacent to the correct response finger, yet the WS-WE error showed a larger ERN amplitude than the WS-CE error. This supports the idea that increasing mismatch, rather than low-level muscular interactions is responsible for the increase in the ERN amplitude. In summary, the present experiment suggests that the ERN reflects error detection processes, rather than conflict processing.

2.5. Experiment 5

Experiment 4 suggested, in line with the findings by Bernstein, Scheffers and Coles (1995) that the ERN increases with increasing mismatch between the executed and the required response, and thus reflects an error detection process. However, Gehring and Fencsik (2001) conducted a 4-choice experiment implementing finger and foot responses and found the opposite result. More specifically, the ERN was smallest in the most dissimilar errors (WS-WE). From this the authors concluded that the ERN is likely to reflect conflict processing. However, the study of Gehring and Fencsik involved a Stroop colour naming task, thus introducing additional conflict through relevant and irrelevant stimulus dimensions. In order to exclude the possibility of such additional conflict combining with error processing as well as potential overlap between the activation of adjacent fingers (e.g. Häger-Ross & Schieber, 2000), a uni-dimensional 4-choice task was performed in Experiment 5. This task was identical to Experiment 4, except that responses had to be executed with the left and the right hand and the left and the right foot. Identical to Experiment 4, the error detection theory predicts the ERN to be largest in the WS-WE error (e.g. Coles, et al., 2001), whereas the conflict detection hypothesis predicts the ERN to be smallest in the WS-WE error (e.g. Botvinick, et al., 2001).

2.5.1. Method

Participants

20 participants (M_{age} = 22.35 years, SD_{age} = 3.10 years, 17 woman) were included in this experiment. Participants did not participate in Experiment 4. All participants were naive about the experimental hypothesis, gave informed consent and were paid £10.

Apparatus and Stimuli

Apparatus and stimuli were equivalent to Experiment 4 if not stated differently. In this experiment only the two response keys for the left and right index finger were used, with two additional response keys placed approximately 30 cm apart on the floor in a position comfortable for participants to reach with their big toes of the left and right foot.

Procedure

Procedure and design were identical to Experiment 4, expect the fact that participants now responded with the index fingers of the left and right hand and the big toes of the left and right foot.

Data Analysis

Both behavioural and ERP data were analysed analogue to Experiment 4.

2.5.2. Behavioural Results

Error Reaction Times

RTs for error trials are displayed in Figure 19. The ANOVA showed that there is a main effect of Errortype, $F(2,38) = 20.59$, $MSE = 1346.03$, $p < .001$, $\varepsilon = 0.98$. Post-hoc comparisons showed that WS-WE errors were the fastest (464 ms), differing significantly from CS-WE errors (502 ms), $F(1,19) = 14.44$, $MSE = 1008.55$, $p < .01$, and WS-CE errors (538 ms), $F(1,19) = 30.80$, $MSE = 1799.79$, $p < .001$. In addition, WS-CE errors were slower than CS-WE errors, $F(1,19) = 10.71$, $MSE = 1229.74$, $p < .01$.

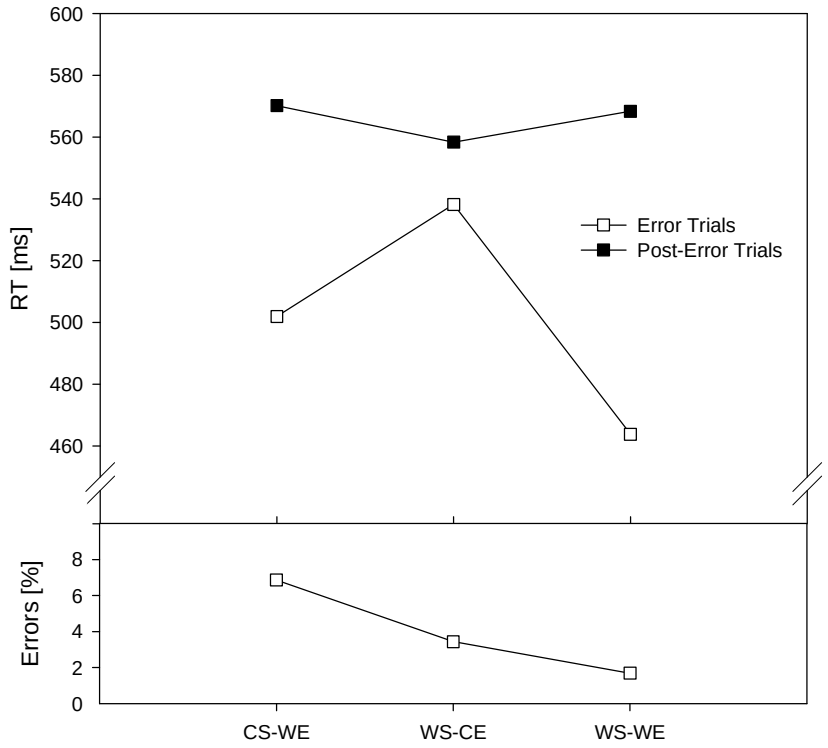

Figure 19. Mean RT and error rate. Top plot: RT in error and post-error trials as a function of Errortype. Bottom plot: Error rate of the three Errortypes. CS-WE = Correct Side–Wrong Effector, WS-CE = Wrong Side–Correct Effector, WS–WE = Wrong Side–Wrong Effector.

Error Rates

The relative frequency of the three Errortypes is shown in Figure 19 (bottom panel). There was a main effect of Errortype, $F(2,38) = 40.94$, $MSE = 3.37$, $p < .001$, $\varepsilon = .67$. This was due to a decrease of errors from CS-WE (6.86%) > WS-CE (3.42%) > WS-WE (1.69%). Again, least errors occurred in the Errortype most dissimilar to the required correct response (WS-WE), which differed significantly from both the CS-WE, $F(1,19) = 78.72$, $MSE = 3.39$, $p < .001$ and the WS-CE error, $F(1,19) = 25.55$, $MSE = 1.19$, $p <$

.001. However, the error rate for CS-WE and WS-CE error types also differed significantly in this experiment, $F(1,19) = 21.13$, $MSE = 5.54$, $p < .001$.

Post-Error Reaction Times

Figure 19 (upper panel) shows the post-error RTs as a function of Errortype. There was no effect of Errortype on the amount of post-error slowing, $F = 1.15$, $p > 0.10$, even though overall post-error slowing was clearly present when compared to responses following a correct trial (532 ms), $F(3,57) = 9.72$, $MSE = 640.98$, $p < .001$, $\varepsilon = .81$.

2.5.3. Electrophysiological Findings

Figure 20 shows the ERP waveforms separately for the three Errortypes and the correct responses time-locked to the keypress response. The ANOVA showed that there was a main effect of Errortype, $F(2, 38) = 3.62$, $MSE = 2.51$, $p < .05$. Post-hoc tests revealed that this was due to larger ERN peak amplitudes in the CS-WE and the WS-WE condition, than in the WS-CE condition, $F(1, 19) = 4.30$, $MSE = 2.60$, $p = .05$ and $F(1,19) = 5.46$, $MSE = 2.85$, $p < .05$, respectively. CS-WE and WS-WE errors did not differ in the size of the ERN amplitude, $F < 1$. Thus, in contrast to Experiment 4, errors sharing no parameters (WS-WE) with the correct response and also errors occurring on the correct response side (CS-WE) elicited particularly large ERNs.

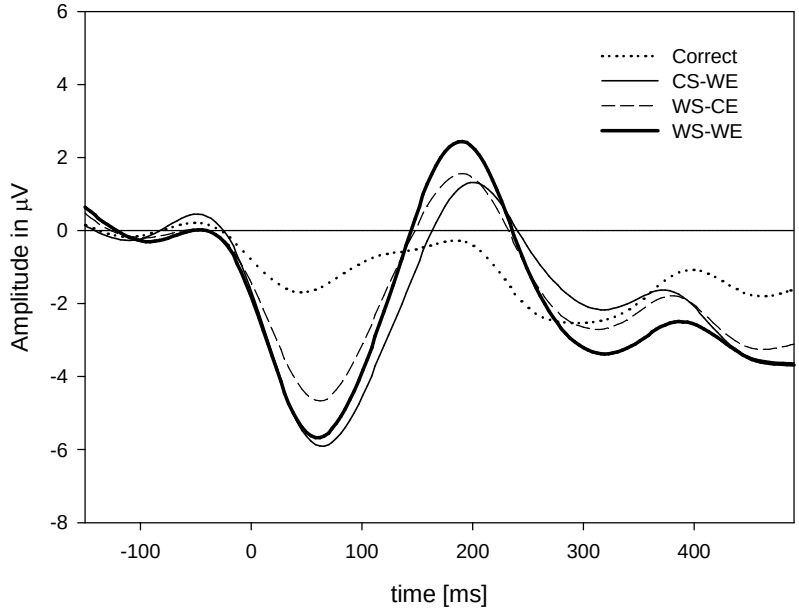

Figure 20. Event-related potential activity over FCz for correct keypresses and for the three Errortypes, time locked to the response. CS-WE = Correct Side – Wrong Effector, WS-CE = Wrong Side – Correct Effector, WS – WE = Wrong Side – Wrong Effector.

2.5.4. Discussion

The aim of the current experiment was to investigate the mechanisms underlying the ERN. To do so, the error detection and the conflict processing hypotheses were contrasted. The error detection hypothesis predicts the ERN to be largest the more dissimilar the error is to the required correct response (e.g. Coles, et al., 2001). In contrast, the conflict detection hypothesis predicts the ERN to be larger the more similar the error is to the required response (e.g. Botvinick, et al., 2001). The previous experiment using finger responses only showed that the ERN was largest for errors that were most dissimilar to the correct response, supporting the error detection hypothesis (see also: Bernstein, et al., 1995). In a 4-choice version of the Stroop task that implemented finger and foot responses, Gehring and Fencsik (2001) found the opposite result, with the ERN being smallest in the condition where the error was most dissimilar to the correct response. The present experiment implemented an identical 4-choice task as in Experiment 4, with the exception that participants now responded with their left and right hands and their left and right foot (see Gehring and Fencsik, 2001). Thus, the current experiment excludes potential overlap between activation of adjacent fingers (e.g. Häger-Ross & Schieber, 2000), and also excludes additional interference via irrelevant task dimensions like is possible in the Stroop task paradigm used by Gehring and Fencsik.

In line with previous studies (e.g. Gehring & Fencsik, 2001), the results showed that WS-WE errors were the least likely to occur, suggesting that they are most dissimilar to the required correct response. Analysis of RTs also showed similar patterns to the experiment of Gehring and Fencsik; specifically, WS-WE errors had the shortest latencies, followed by the CS-WE and the WS-CE error types. However, in contrast to the study conducted by Gehring and Fencsik, the most dissimilar error (WS-WE) was not accompanied by a smaller ERN than the two other errortypes that are more similar to the correct response. This finding speaks against the conflict detection hypothesis, which suggests that the ERN should be reduced the more dissimilar the error is to the required correct response. In contrast, in the present experiment the contralateral WS-CE errortype elicited the smallest ERN while both the CS-WE and the WS-WE errortypes resulted in larger ERNs. In line with Gehring and Fencsik's findings and the previous experiment, these changes in the ERN amplitude did not relate to changes in the amount of post-error slowing.

Despite these results speaking against the conflict detection hypothesis, they do not clearly support the error detection hypothesis. The error detection hypothesis predicts the ERN to be largest in for the error type most dissimilar to the required response (i.e. WS-WE). Thus, it remains questionable why the ERN is specifically small in the WS-CE error type, sharing the response effector with the required response, and not in the CS-

WE error, despite the CS-WE error sharing the same response side with the required response. One possible explanation is that in the current experiment, the shared response side in the CS-WE error type did not result in a reduction of the ERN amplitude because the effector changed between foot and hand, and thus the shared response side did not have any further advantage on error processing. In contrast, in the previous experiment (implementing finger responses only) in the CS-WE condition, both the executed erroneous and the required correct response involved different fingers (middle vs. index) but the same hand for responding, possibly resulting in the reduction of the ERN amplitude. In other words, it is not the same response side in the previous experiment but a “higher-ranking” effector response hand being shared in the CS-WE condition that possibly results in the reduction of the ERN amplitude.

However, alternative suggestions have to be considered. RT analysis showed that the WS-CE errors were particularly slow compared to the CS-WE and the WS-WE errors in the current experiment. One possible explanation as to why the WS-CE errors show particularly small ERNs concerns potential SAT shifts, which are known to infulence the ERN amplitude (e.g. Gehring, et al., 1993). Critically, Gehring et al. suggested that the importance of an error is reflected in the ERN amplitude. More specifically, he showed that when response accuracy is emphasized over response speed, this results not only in slower response and less errors but also in increased ERN amplitudes. However, in the current experiment the slowest responses (WS-CE) were showing the smallest ERN amplitudes, ruling out SAT shifts as the mechanism responsible for changes in ERN amplitude, as suggested by Gehring et al. It could still be possible that the overall changes in the RTs had effects on the ERN. However, RTs and response accuracy also varied between the CS-WE and the WS-WE errors and both errors showed similar ERN amplitudes, making such an impact of RTs on the ERN component amplitude unlikely.

In conclusion, the findings of Experiment 5 contradict the findings of Gehring and Fencsik (2001) in a multi-dimensional 4-choice Stroop task, also implementing finger and foot responses. In the present study the ERN is largest in the errors which do not share a response effector with the required response. This suggests that the ERN reflects error detection rather than conflict processing. Similar to Experiment 4, the ERN amplitude increased with a larger mismatch between the correct and the erroneous response (see also: Bernstein, et al., 1995), supporting the hypothesis that the ERN amplitude reflects error rather than conflict detection processes.

2.6. Experiment 6

The following experiment is published in the Quarterly Journal of Experimental Psychology, 62 (2), 209-218, Why do we slow down after an error? Mechanisms underlying the effects of post-error slowing, Jentzsch and Dudschig (2009).

In addition to open questions regarding the functional mechanisms underlying the ERN, the mechanisms underlying behavioural changes before and after errors remain unclear. The aim of the following experiment is to investigate the mechanisms underlying error processing and behavioural changes after error commission (i.e. post-error slowing). Critically, both traditional explanations (e.g. Laming, 1968) and the conflict monitoring theory (Botvinick et al., 2001) assume shifts along the speed-accuracy tradeoff (SAT) function to underlie post-error slowing effects. It is assumed that response thresholds are raised after error or conflict detection, resulting in slower responses. Importantly, if shifts on the SAT function can account for behavioural adjustments following errors, trials following errors should also be more accurate in addition to being slower. However, it was found that when there is little time to process the error (i.e. short RSI conditions), post-error slowing is large but also more error prone (e.g. Laming, 1979; Rabbitt & Rogers, 1977). The fact that post-error slowing is largest at shortest RSIs could be accounted for by the assumption that shits along the SAT function take place fast and automatically, rather than in a strategic and time consuming manner (e.g. Sommer, et al., 1999). However, increasing post-error slowing in combination with reduced response accuracy cannot be accounted for by automated shift in the response thresholds either. Thus, these findings challenge explanations of post-error slowing effects that rely solely on shifts on the SAT function. In the present experiment alternative mechanisms, which possibly can account for the effects of post-error slowing and post-error accuracy will be investigated.

The fact that trial performance after errors is substantially impaired when there is not enough time to “recover” from an error, suggests that there is a difficulty in adequately processing information rather than a processing advantage via an up-regulation of cognitive control. Welford (1959, 1980) suggested that central processing outlasts response execution and may interfere with subsequent processing if there is insufficient time between trials. More specifically, Welford (1980) suggested that response delays in the case of short RSIs can be due to response monitoring that requires access to central resources and extends for several hundred milliseconds after response execution. Recent evidence from a dual-task study (Jentzsch, Leuthold, & Ulrich, 2007) points to the existence of such a bottleneck that outlasts response execution. Crucially, monitoring an error trial involves dealing with both representations of the executed incorrect and the appropriate response, which should have been executed.

Thus, monitoring processes might be active for a longer time after incorrect than after correct trials (e.g. Laming, 1968, 1979; Rabbitt & Rogers, 1977). More specifically, more time might be needed to detect this conflict, to decide that an incorrect response has been performed, and to identify what exactly went wrong, resulting in longer response monitoring processes after erroneous responses. This delays access to central processing resources for subsequent trials and thus, possibly causes post-error slowing (see also Rabbitt, 2002; Rabbitt & Vyas, 1981). Importantly, since the monitoring process might substantially interfere with current trial processing, this process may also explain the lower response accuracy in post-error trials in short RSI conditions.

To test the existence of such a post-error monitoring bottleneck, stimulus contrast was manipulated in the present experiment (see Figure 21). Stimulus contrast manipulations are known to influence the duration of perceptual processing stages, whereby perceptual processing is prolonged for low compared to high contrast stimuli. Importantly, perceptual processing does not require central resources, and thus can be carried out in parallel to error monitoring processes (e.g. Jentzsch, et al., 2007). If post-error slowing is due to a postponement of central processing stages because of ongoing error monitoring, the effect of stimulus contrast should be absorbed into the 'postponement slack' (cf. Pashler & Johnston. 1989). Thus, results should show an underadditive effect of stimulus contrast with post-error slowing. Moreover, if such a bottleneck solely explains post-error slowing, post-error slowing should be particularly large if there is not enough time available to complete error monitoring before the next stimulus is presented (i.e. short RSIs).

In contrast, the traditional criterion adjustment hypothesis suggests that after error occurrence, response thresholds are raised, resulting in slower and more accurate responses. Critically, such a criterion adjustment does not assume a bottleneck stage. Therefore, both post-error trials and post-correct trials should be affected similarly by the stimulus contrast manipulation.

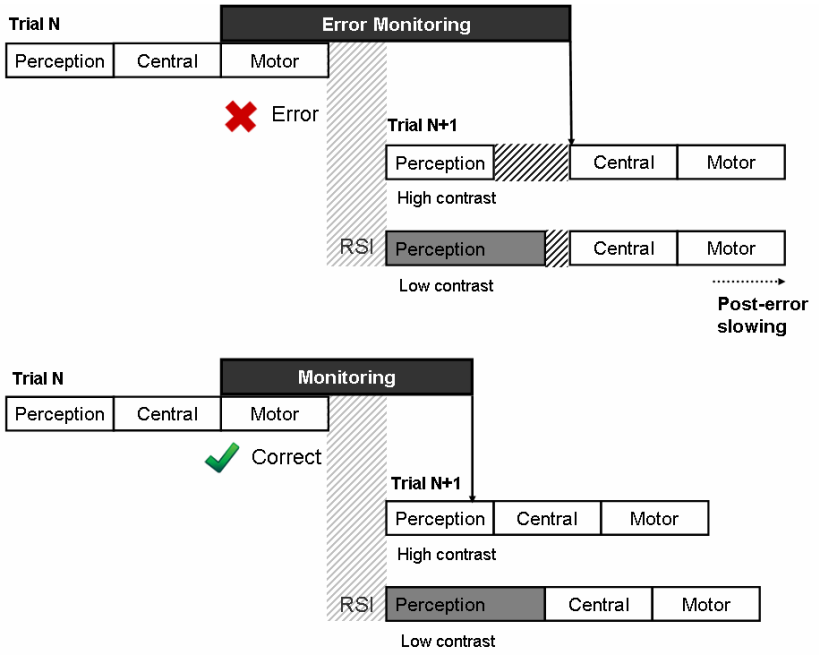

Figure 21. Extended response monitoring in error trials (upper diagram) compared to correct trials (lower diagram). Importantly, the effects of a stimulus contrast manipulation are absorbed if post-error slowing is due to postponement of central processing stages. In contrast, in correct trials where response monitoring finishes before the next trial starts, the effect of stimulus contrast is fully expressed in the RT measurements.

2.6.1. Method

Participants

A total of 20 participants (M_{age} = 24.9 years; SD_{age} = 3.10 years, 14 females) were tested in a single session lasting approximately 60 minutes. All participants gave informed consent and received payment (£5).

Stimuli and Apparatus

Stimuli and apparatus are identical to the previous experiments unless stated. Participants made single left and right key press responses with the index fingers of the left and right hand, respectively. The stimuli were letters D, T, M and X presented on a white background either in light grey (52 cd/m^2, low stimulus contrast) or dark grey (1 cd/m^2, high stimulus contrast).

Procedure

Procedure was identical to previous experiments unless stated. The experiment was split into two parts. In one half the RSI was 100 ms, and in the other half the RSI was 1000 ms. Order of RSI was balanced across participant. Four letters were mapped to two response alternatives, for example, T and M → left key, D and X → right key. A total of

two practice blocks (80 trials each) and 10 experimental blocks (160 trials each) of alternating high and low stimulus contrast blocks were presented for each RSI condition.

Data Reduction and Analysis

Only trials with an RT between 150 and 1500 ms in trials N-1 and N were considered as correct. All trials with missing or too slow responses (< 1%) and too fast responses (< 1%) in trials N-1 or N were discarded from the analysis. Two different trial types were analyzed: (1) trials where a correct response in trial N-1 is followed by correct responses in trial N (all-correct trial, C-C), (2) trials where an error response in trial N-1 is followed by correct responses in trial N (post-error trial, E-C). For all experimental conditions a minimum of 20 trials per analysis cell applied. Error rates were calculated for post-correct trials (E(post-correct) = CE/(CC+CE) x 100) and post-error trials (E(post-error) = EE/(EC+EE) x 100).

The dependent variables (RT and error rate) were subjected to repeated measures ANOVAs with within-subject variables of stimulus contrast (high vs. low), RSI (100 vs. 1000 ms) and Trialtype (all-correct vs. post-error). Conservative Huynh-Feldt *F*-tests were used throughout and for post-hoc comparisons Bonferroni corrections were applied.

2.6.2. Results

Reaction Times

Mean RTs are shown in Figure 22 (upper panel). Participants were slower under short RSI (625 ms) than under long RSI conditions (488 ms), $F(1,19) = 67.36$, $MSE = 11022.28$, $p < .001$. Responses were faster for high-contrast (549 ms) than low-contrast stimuli (564 ms), $F(1, 19) = 8.58$, $MSE = 1102.58$, $p < .01$. The main effect of Trialtype, $F(1, 19) = 84.96$, $MSE = 6219.50$, $p < .001$, was due to faster responses for all-correct trials (499 ms) than for post-error trials (614 ms). Trialtype interacted with RSI, $F(1, 19) = 61.83$, $MSE = 3906.88$, $p < .001$, due to a larger effect of post-error slowing for short RSIs (193 ms) than for long RSIs (37 ms). Post-error slowing was present for both the short RSI, $F(1, 19) = 115.05$, $MSE = 6451.45$, $p < .001$, and the long RSI, $F(1, 19) = 7.54$, $MSE = 3674.93$, $p < .05$.

Importantly, there was a three-way interaction between trial type, RSI, and contrast, $F(1,19) = 10.10$, $MSE = 846.41$, $p < .01$. For shorts RSIs the contrast effect was absent for post-error trials, $F(1,19) = 1.51$, $MSE = 3291.46$, $p > .10$, and present for all-correct trials, $F(1, 19) = 70.57$, $MSE = 76.52$, $p < .01$, resulting in a significant two-way interaction between Trialtype and stimulus contrast, $F(1, 19) = 6.02$, $MSE = 1722.64$, $p < .05$. For long RSIs the contrast effect was significant for both post-error trials, $F(1, 19) =$

26.09, *MSE* = 517.90, $p < .001$, and all-correct trials, $F(1, 19) = 34.18$, *MSE* = 166.11, $p < .01$, and not different in size for both trial types, $F(1,19) = 4.05$, $p > .10$.

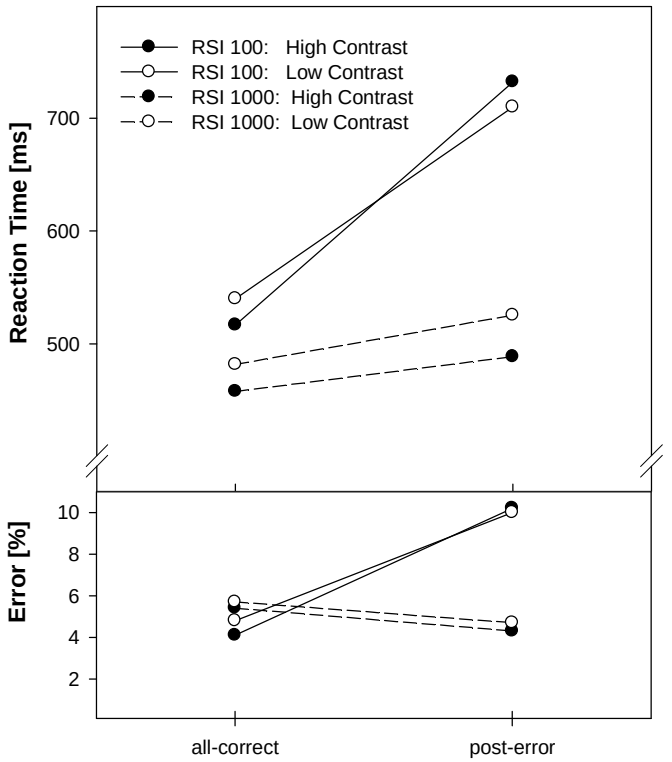

Figure 22. Reaction times (upper panel) and error percentages (lower panel) as a function of trial sequence (all-correct, post-error), RSI, and stimulus contrast.

Error Rates

Mean error rates are shown in Figure 22 (lower panel). Statistical analyses revealed a main effect of RSI, $F(1, 19) = 12.57$, *MSE* = 16.49, $p < .01$, due to a higher error rate for the short RSI (7.3 %) than for the long RSI (5.0 %). There was also a main effect of Trialtype, $F(1, 19) = 10.49$, *MSE* = 19.93, $p < .01$, due to higher error rates for post-error (7.3 %) than for post-correct trials (5.0 %). Importantly, trial type and RSI interacted, $F(1, 19) = 30.33$, *MSE* = 14.70, $p < .01$. For short RSIs error rates were significantly higher in post-error (10.1 %) than in post-correct trials (4.5 %), $F(1, 19) = 25.01$, *MSE* = 25.30, $p < .001$. In contrast, for long RSIs error rates were not significantly different in post-error trials (4.5 %) and post-correct trials (5.5 %), $F(1, 19) = 2.37$, $p = .14$.

2.6.3. Discussion

The aim of the present experiment was to investigate the mechanisms underlying post-error slowing. Traditionally, post-error slowing is explained via criterion adjustments on the SAT function (e.g. Brewer & Smith, 1984; Laming, 1968). After an error response thresholds are raised, resulting in slower and more accurate responses. This idea has been integrated within the conflict monitoring theory (Botvinick et al., 2001). In contrast, the response monitoring hypothesis postulates that responses are checked for response accuracy after response execution, a process that demands central capacity (e.g. Welford, 1980). Critically, the monitoring process following an error trial is longer lasting because it has to deal with the activation of the correct and the erroneous response. Such a monitoring bottleneck predicts that the effects of a stimulus contrast manipulation will be absorbed in the waiting period until monitoring has finished and cleared central resources (Pashler & Johnston, 1989). Importantly, the criterion adjustment hypothesis does not propose a central processing bottleneck, and therefore the effects of stimulus contrast manipulations should be present in both post-error and post-correct trials.

In the present experiment post-error trials were substantially slower than all-correct trials. Although post-error slowing was reduced in the long RSI condition, post-error trials were still significantly slower than all-correct trials (see also, Rabbitt & Rogers, 1977; Sanders, 1998). Importantly, the stimulus contrast effects and measurements of response accuracy allow one to distinguish between the error monitoring and the criterion adjustment hypothesis. In short RSI conditions, the effect of stimulus contrast was underadditive with post-error slowing, supporting the error monitoring hypothesis. Moreover, response accuracy was lowest in the short RSI condition, when post-error slowing was largest. This finding speaks against the criterion adjustment hypothesis. The response monitoring hypothesis can account for both increased post-error slowing and reduced accuracy rate, as ongoing error monitoring postpones processing and possibly interferes with current trial processing[2]. Interestingly, the effect of post-error slowing was still present at the long RSI condition and the effects of stimulus contrast were additive with post-error slowing effects. Moreover, response accuracy slightly increased in post-error trials in the long RSI conditions. The error monitoring hypothesis cannot account for these findings. However, these results do fit with the criterion adjustment hypothesis. Taken together, this suggests that when there is enough time to process the error,

[2] Problematically, a reduced number of trials contributed to RT averages for post-error trials and RTs are generally very high in post-error trials when RSI is short. An additional experiment was conduceted (not reported in this thesis) where categorization difficulty was manipulated. If increased error variance explains the absence of the contrast effect in the short RSI condition, one would expect other effects to disappear as well. However, the effects of categorization difficulty were significantly present for all RSI and trial type combinations, which makes it unlikely that the absence of the contrast effect for post-error trials under short RSIs is the result of problems such as an increased error variance.

criterion adjustments may take place, resulting in slower and more accurate performance in post-error trials. Taken together, this shows that post-error slowing cannot always be considered as the outcome of a beneficial adjustment process of response strategies (see also: Notebaert, et al., 2009).

However, alternative explanations have to be considered. For example, it is possible that after having made an error, participants might choose to strategically postpone responding until error processing is completed. Such a strategic postponement could account for increased post-error slowing in short RSI conditions but would have difficulties explaining the underadditive effects between post-error slowing and stimulus contrast. One would need to additionally assume that participants process perceptual information in parallel to ongoing error processing while strategically delaying central response selection. Such an idea of selective strategic postponement of response selection has previously been brought forward by Meyer and Kieras (1997). Importantly, such an account would be very similar to the response monitoring process except that it includes a strategic element for the occurrence of the bottleneck.

In summary, the findings of Experiment 6 suggest that contributions to post-error slowing are most likely two-fold. First, at short RSI conditions, it seems likely that postponement of trial processing due to ongoing response monitoring causes post-error slowing effects. Second, at long RSI conditions, it seems likely that response monitoring has finished and cognitive control adjustments via response criterion shifts can take place. Importantly, despite these results suggesting a central error monitoring bottleneck underlying the post-error slowing at short RSIs, it remains unclear what is the function of this monitoring process. As this monitoring process results in slower and less accurate performance, it is questionable what it is useful for. One possibility is that an error detection process extends beyond error execution. Not much is known about how error detection takes place, despite the process being assumed to be a crucial step antecedent to post-error adjustments being triggered (e.g. Botvinick et al., 2001). However, future studies are required to clarify the actual process resulting in the central processing bottleneck after errors.

2.7. Experiment 7

The following experiment is accepted for publication in Brain Research, Pre-error speeding and post-error slowing: Is it all strategy? Dudschig and Jentzsch (in press).

In addition to response slowing after errors, it is well known that responses preceding an error are often faster than responses preceding a correct response. Traditionally, it is assumed that shifts along the SAT function to less or more conservative response thresholds can explain both pre-error speedup and post-error slowing (e.g. Brewer & Smith, 1984; Jentzsch & Leuthold, 2006; Laming, 1968, 1979; Rabbitt, 1966; Rabbitt & Rogers, 1977; Rabbitt & Vyas, 1981). Also the conflict monitoring theory (Botvinick et al., 2001) allows control regulations in both directions. More specifically, after error trials (i.e. high-conflict trials) control levels are raised, while in contrast, after a successive run of correct trials (i.e. low-conflict trials) control levels are down-regulated. However, although pre-error speedup and post-error slowing are assumed to arise from similar mechanisms, research has, apart from a few studies (e.g. Brewer & Smith 1984; Brewer & Smith, 1989; Rabbitt, 1966), almost exclusively focused on the effects of post-error slowing. The present experiment aims to extend the findings from Experiment 6 and to systematically investigate the relationship between pre-error speed-up and post error slowing.

Experiment 6 demonstrated that post-error slowing cannot be fully accounted for by explanations based on an up-regulation of cognitive control. Neither strategic nor automated regulations of cognitive control can account comprehensively for the effects of post-error slowing. Thus, the first aim of the current experiment is to investigate the effectiveness of strategic or automatic cognitive control regulation explanations for pre-error speeding effects. If down-regulation of control after a series of correct trials is a time consuming strategic process that demands central resources (e.g. Ratcliff & McCoon, 1981), pre-error speeding should not occur when the RSI is short. Pre-error speeding would only be expected to occur in long RSI conditions, where sufficient time for the implementation of top-down control processes is available. However, if response thresholds are automatically lowered when no conflict is experienced or alternatively, if random fluctuations in response thresholds are responsible for pre-error speeding (e.g. Brewer & Smith, 1984), the effect should be present for both short and long RSIs. The results of the previous experiment suggested that post-error slowing in short RSI conditions is most likely due to a postponement of current trial processing until error evaluation processes from the previous trial have finished. If post-error slowing in the current experiment can be explained by such a post-error monitoring bottleneck, post-error slowing should systematically increase with decreasing RSI. Moreover, as ongoing error evaluation processes may interfere with current trial processing, reduced post-error accuracy is expected in conditions with increased post-error slowing. In contrast, if

regulations of cognitive control implemented through SAT adjustments explain post-error slowing effects, post-error accuracy should increase with increasing amounts of post-error slowing.

The second aim of the present study is to investigate the relationship between the ERN (Falkenstein, et al., 1990; Gehring, et al., 1990) and post-error slowing. Findings regarding the relationship between the size of the ERN and the amount of post-error slowing are ambiguous. A number of researchers have reported positive correlations between post-error slowing and the size of the ERN or the amount of activity within the ACC, a possible neural source of the ERN (e.g. West & Travers, 2008; Debener et al., 2005; Gehring et al., 1993; Kerns et al., 2004). However, others have not found such a relationship between the ERN and post-error slowing (e.g. Gehring & Fencsik, 2001; van Meel et al., 2007), nor did Experiment 4 and 5 show such a relationship. The current experimental task provides an ideal setting to investigate this relationship as the RSI manipulation is expected to evoke different effects on post-error slowing between the RSI conditions, with post-error slowing effects expected to increase substantially with decreasing RSI. If one assumes that the ERN and post-error slowing are positively correlated, ERN amplitude should increase with decreasing RSI. However, if large post-error slowing effects at short RSIs results from a processing interference due to incomplete error processing and if one assumes that the ERN is a manifestation of error processing, one could predict that the ERN will decrease with decreasing RSI.

2.7.1. Method

Participants

24 participants (M_{age} = 22 years, SD_{age} = 3.18 years, 16 woman) took part in this experiment. Participants had normal or corrected-to-normal vision and were tested in a single session. All participants were naive about the experimental hypothesis, gave informed consent and were paid £8.

Apparatus and Stimuli

Apparatus and stimuli are identical to the previous experiments. The stimuli consisted of the letters D, M, T and X presented centrally in black on a white background. In addition, a black fixation cross (3 x 3 mm) was presented in the centre of the screen during the RSI periods.

EEG recording

EEG recording was identical to the previous experiments.

Procedure and design

Participants were instructed to respond fast and to keep the error rate between 5 and 10 %. They were asked to perform simple key-press responses. Two letters were mapped to the left index finger and two letters to the right index finger (e.g. D and M => left key, X and T => right key). The assignment of letters to response alternatives was balanced across participants. The experiment used four RSI conditions (100, 250, 500, or 1000 ms). The order of the RSI conditions was fully balanced across participants. For each RSI condition two practice blocks (80 trials each) and five experimental blocks (160 trials each) were presented.

Behavioural Data Analysis

Only trials with an RT between 200 and 1500 ms in trials N-1, N and N+1 were considered as correct. All trials with missing or too slow responses (< 1%) and too fast responses (< 1%) in trials N-1, N and N+1 were discarded from the analysis. Four different trial types were analysed: (1) trials where a correct response in trial N-1 is followed by correct responses in both trial N and trial N+1 (all-correct trial, C-C-C), (2) trials where a correct response in trial N-1 is followed by a correct response in trial N but an incorrect response in trial N+1 (pre-error trial, C-C-E), (3) trials where a correct response in trial N-1 is followed by an incorrect response in trial N but a correct response in trial N+1 (error trial, C-E-C), and (4) trials where an error response in trial N-1 is followed by correct responses in trial N and trial N+1 (post-error trial, E-C-C).

For all experimental conditions a minimum of 20 trials per analysis cell applied. Error rates were calculated for post-correct trials (E(post-correct) = CEC/(CCC+CEC) x 100) and post-error trials (E(post-error) = EEC/(ECC+EEC) x 100).

The dependent variables (RT and error rate) were subjected to repeated measures ANOVAs with within-subject variables of RSI (100, 250, 500 and 1000 ms) and Trialtype. The factor Trialtype consisted of 4 levels for the RT analysis (all-correct, pre-error, error, and post-error) and of 2 levels for the analysis of error rates (post-correct, post-error). Conservative Huynh-Feldt *F*-tests were used throughout and for post-hoc comparisons Bonferroni corrections were applied.

ERP Data Analysis

The EEG recordings were examined for artifacts (amplifier blocking, scalp muscular activity, and slow linear drift) for each participant and trial. Only trials without EEG artifacts were included in the EEG data analysis. Trials containing blinks were corrected using a dipole approach (BESA Version 5.1.6; Berg & Scherg, 1994). All signals were averaged separately for each experimental condition. The response-synchronized epochs (time-locked to onset of the keypress response) started 200 ms prior to response

onset and lasted for a total duration of 800 ms. All averages were baseline corrected, using a 100-ms baseline starting 150 ms before the onset of the response and were low-pass filtered at 15 Hz.

Difference waves (incorrect minus correct response) were calculated for each of the four RSI conditions and were subjected to peak amplitude measurement of the ERN. The time window for automatic peak detection was 0 to 100 ms after response onset using the FCz electrode.

2.7.2. Behavioural Results

Reaction Time

Mean RT (see Figure 23) significantly decreased with increasing RSI, resulting in a main effect of RSI, $F(3, 69) = 64.50$, $MSE = 2424.56$, $p < .001$, $\varepsilon = .93$, due to the following ordering of RTs: RSI 100 (524 ms) > RSI 250 (488 ms) > RSI 500 (444 ms) = RSI 1000 (438ms). RTs were affected by Trialtype, $F(3, 69) = 80.19$, $MSE = 5278.56$, $p < .001$, $\varepsilon = .47$, due to the following ordering: post-error (570 ms) > all-correct (462 ms) > pre-error (436 ms) = error (426 ms).

Importantly, the interaction between Trialtype and RSI was significant, $F(9, 207) = 42.76$, $MSE = 474.72$, $p < .001$, $\varepsilon = .54$. As can be seen in Figure 23, this interaction is due to larger RSI dependent slowing in post-error trials compared to all other Trialtypes, all $F_s(3, 69) > 49.21$, all $p_s < .001$. Post-error slowing, calculated as the difference in RT between post-error and all-correct trials, increased with decreasing RSI: RSI 1000 (48 ms) < RSI 500 (78 ms) < RSI 250 (132 ms) < RSI 100 (179 ms). Interestingly, when excluding post-error trials from the analysis, no interaction between Trialtype (all-correct, pre-error, and error) and RSI was observed, $F(6, 138) = 0.31$, $\varepsilon = .97$. Thus, whereas post-error slowing interacts with RSI, pre-error speed-up is not affected by RSI.

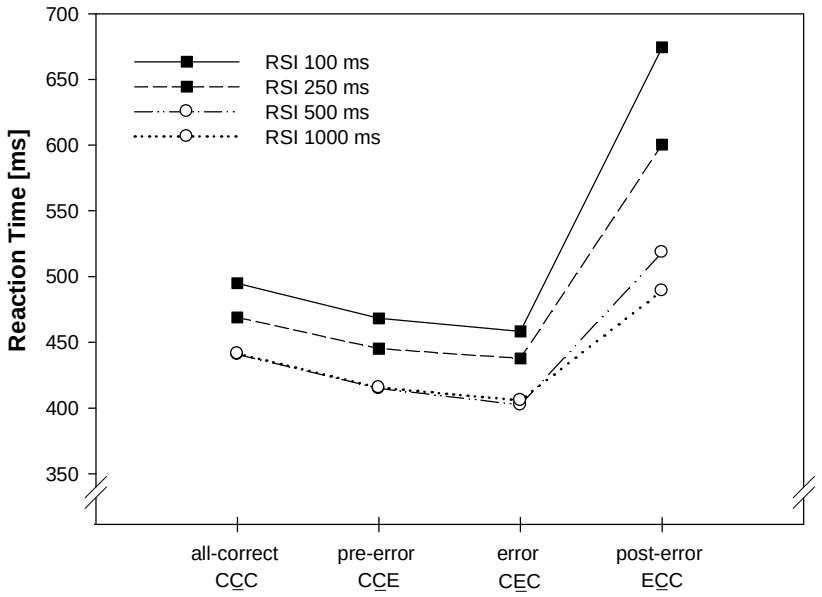

Figure 23. RT as a function of RSI and Trialtype (all-correct, pre-error, error and post-error).

Error Rates

Error Rates (see Figure 24) were not affected by Trialtype, $F < 1$. The effect of RSI was significant, $F(3, 69) = 2.95$, $MSE = 9.25$, $p < .05$, $\varepsilon = 0.98$, due to higher error rates in the 250 ms RSI (9.63 %) compared to the 100 ms RSI 100 (7.96 %) condition. The interaction of RSI and Trialtype was significant, $F(3, 69) = 6.21$, $MSE = 9.29$, $p < .01$, $\varepsilon = 1.0$, due to higher error rates for post-error trials (100 RSI: 9.2 %; 250 RSI: 10.7 %) compared to post-correct trials (100 RSI: 6.7 %; 250 RSI: 8.5 %) for the shorter RSIs: RSI 100, $F(1, 23) = 5.42$, $MSE = 14.76$, $p < .05$, RSI 250: $F(1, 23) = 3.20$, $MSE = 18.26$, $p = .09$; and slightly but non-significantly lower error rates for post-error trials (500 RSI: 7.9 %; 1000 RSI: 7.7 %) compared to post-correct trials (500 RSI: 9.7 %, 1000 RSI: 8.6 %) for the longer RSIs: RSI 500: $F(1, 23) = 4.08$, $MSE = 8.92$, $p = .06$, RSI 1000: $F(1,23) = 0.69$, $MSE = 16.16$, $p = .42$.

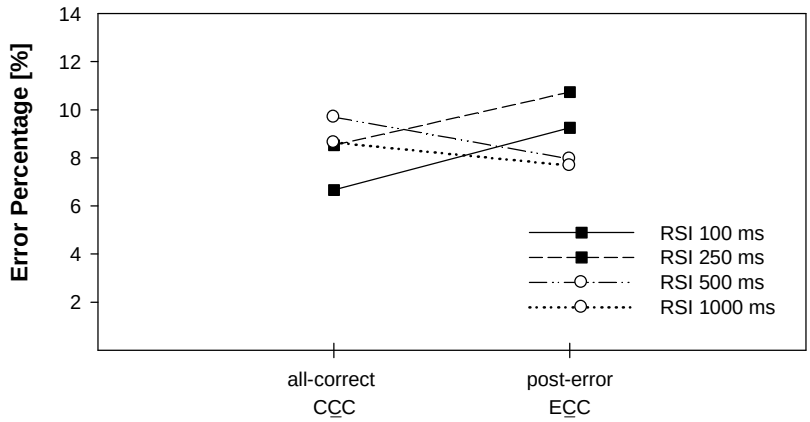

Figure 24. Error Rates as a function of RSI and Trialtype (all-correct and post-error).

2.7.3. Electrophysiological Findings

ERN amplitude

The peak amplitude of the ERN (difference wave: incorrect minus correct trials) was subjected to a one-way ANOVA with the factor RSI. There was a main effect of RSI, $F(3, 69) = 3.70$, $p < .05$. Post hoc analyses showed that this effect was due to smaller ERN amplitudes in the two short RSI conditions (100, 250 ms) compared to the two longer conditions (500, 1000 ms), $F(1, 23) = 8.20$, $MSE = 2.85$, $p < .01$ (see Figure 25, right panel). It should be mentioned that no significant difference in the ERN amplitude was observed when comparing the two shortest RSI conditions, $F(1, 23) = 0.01$, $p > .93$; and the two longest RSI conditions, $F(1, 23) = 0.57$, $p > .45$. This is important, because this rules out a simple component overlap account for explaining the observed ERN amplitude effects. In other words, increasing overlap of ERP activity with decreasing RSI resulting from two subsequent events is unlikely to account for the ERN findings[3].

[3] Two additional amplitude analyses of the ERN using only error trials were conducted in order to confirm the findings derived from the difference waves. First, the peak amplitude of the ERN in error trials was measured using a 100-ms baseline starting 150 ms before the onset of the response. Second, a peak-to-peak amplitude measurement was applied using the amplitude difference between the positive peak immediately preceding the ERN and the ERN. Both amplitude measures were subjected to a one-way ANOVA with the factor RSI. Both measures showed a main effect of RSI, $F(3, 69) = 3.78$ and 4.49, $p < .05$, respectively. Post-hoc analyses showed that this effect was due to slightly smaller ERN amplitudes in the two short RSI conditions (100, 250 ms) as compared to the two longer conditions (500, 1000 ms), $Fs(1, 23) = 8.12$ and 12.40 , $p < .01$, respectively.

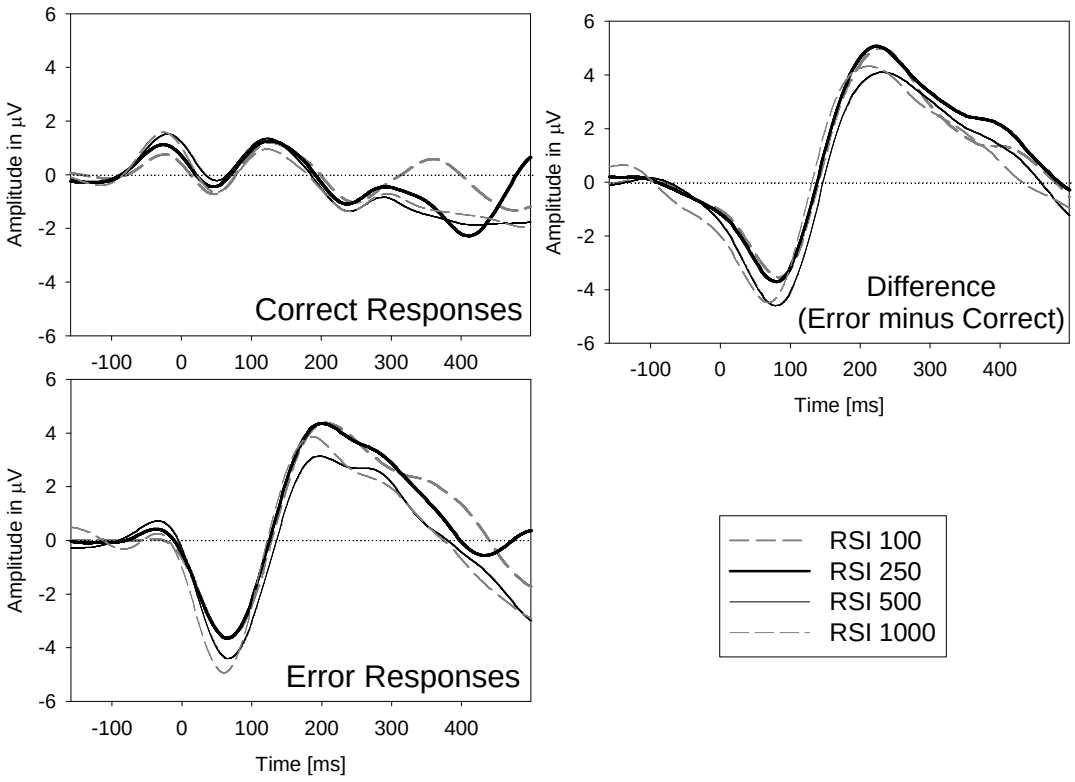

Figure 25. Response-locked ERP averages at the FCz Electrode, following a correct response (top-left) or an error response (bottom-left) as a function of RSI. The difference waves (error minus correct) are shown in the right panel.

2.7.4. Discussion

The aim of the present study was to investigate the mechanism underlying the effects of post-error slowing and pre-error speeding. Both effects have traditionally been explained by adjustments of response criteria to more or less conservative levels. However, despite pre-error speeding and post-error slowing being assumed to arise from the same mechanisms, research has mainly focused on investigating post-error slowing only (although see: Brewer & Smith, 1984). Moreover, empirical results do not always fit with this simple SAT shift explanation. Specifically, post-error slowing is particularly large and post-error accuracy reduced when insufficient time is available between the error and the subsequent trial (e.g. Experiment 6). These findings cannot be accounted for by automated nor by strategic adjustments of the response threshold. Thus, in Experiment 6 it was proposed that error evaluation processes demand central resources and can interfere with subsequent event processing when there is insufficient time for error evaluation to finish before the next trial starts. Only when sufficient time is available can strategic changes of response thresholds take place, resulting in more conservative (slower and more accurate) behaviour.

In order to investigate the mechanisms underlying both pre-error and post-error adjustments, RSI was systematically manipulated in the present experiment. If response criteria are lowered strategically after a correct trial, then one would expect pre-error speeding to be particularly large for longer RSI conditions. In contrast, criteria could also be changed automatically or even to fluctuate randomly (e.g. Brewer and Smith, 1984). In this case we would expect pre-error speeding to be unaffected by the RSI manipulation. The results showed that pre-error speeding was of similar size (20 ms) in all four RSI conditions. Thus, strategic and time-consuming processes seem to play a minor role in explaining this adjustment. It is more likely that response thresholds are automatically lowered after correct trials or alternatively, fluctuate randomly (e.g. Brewer and Smith, 1984) increasing the likelihood of an error when the threshold becomes too low. Post-error slowing systematically decreased with increasing RSI. In addition, post-error accuracy was reduced when RSI was short and numerically increased in long RSI conditions. These findings provide further support for the idea that an error monitoring bottleneck postpones and interferes with current trial processing in short RSI conditions. Only where there is sufficient time for error processing to finish before the next trial starts, can possible up-regulations in cognitive control account for post-error slowing effects.

Given the substantial variations in the amount of post-error slowing across different RSI conditions, this experimental setting seemed to provide an ideal opportunity to investigate the functional significance of the ERN with respect to its impact on subsequent performance adjustments. The results did not show a positive relationship between ERN amplitude and the amount of subsequent post-error slowing. Indeed, the opposite was observed with the ERN being slightly reduced for the shorter RSI conditions where the largest effects of post-error slowing were observed. This clearly speaks against the idea that the ERN directly triggers subsequent compensatory behaviour and thus, determines the amount of subsequent post-error slowing (see also: Yeung et al., 2004). In contrast, the small reduction in ERN amplitude in the short RSI conditions might reflect unfinished error processing. However, this effect needs to be interpreted with caution. Manipulation of RSI can affect ERP data quite substantially as brain activity from previous trial processing overlaps strongly with activity from current processing at short RSIs (e.g. Jentzsch & Sommer, 2002). However, given that there were no differences in ERN amplitude when comparing the 100 with the 250 ms RSI condition or the 500 with the 1000 ms condition, the observed differences in ERN amplitude are unlikely to be the result of overlapping activity.

In summary, the present experiment supports the idea that pre-error adjustments are regulated through automatic adjustments of response criteria, or even through random fluctuations of response thresholds (e.g. Brewer and Smith, 1984). In contrast, error processing can produce substantial interference that results in impaired subsequent

performance when insufficient time is available to process the error adequately. Only when there is enough time between the error and the subsequent trial, can possible advantageous strategic adjustments of response criteria underlie the effects of post-error slowing. Moreover, the amplitude of the ERN did not relate to the amount of post-error slowing and therefore seems not to be directly involved in triggering performance adjustments.

3. General Discussion

The aim of this thesis was to investigate the role of conflict during information processing. Current research has predominantly investigated conflict within multi-dimensional task settings, for example, the Stroop, Flanker and Simon tasks (e.g. Gehring & Fencsik, 2001; Kerns, et al., 2004; Matsumoto & Tanaka, 2004; Stürmer, et al., 2002). Within such multi-dimensional tasks, relevant and irrelevant response dimensions activate opposing response alternatives. This results in conflict and post-conflict adjustments in subsequent trials (e.g. Botvinick, et al., 2001; Gratton, et al., 1992). However, conflict can also occur in uni-dimensional task settings, where only one response dimension is present in each trial. For example, in the case of error trials it is assumed that conflict originates via coactivation of the executed erroneous response and required correct response (e.g. Bernstein, et al., 1995; Botvinick, et al., 2001). Additionally, conflict can result from overlapping response activations across successive correct trials (Jentzsch & Leuthold, 2005). Investigations of conflict in uni-dimensional tasks can provide important additional insights regarding the mechanisms underlying conflict processing.

Recently, the role of conflict during information processing has been re-evaluated. Traditionally, conflict has been regarded as a challenge to efficient information processing. If two opposing response alternatives compete for execution (i.e. high conflict), it is more difficult to choose the correct response alternative. Thus, responses in situations with high conflict are often slower (e.g. Gratton, et al., 1992). Additionally, conflict is regarded as a major factor contributing to error occurrence (e.g. Carter, et al., 1998b). However, recent theories suggest that conflict and conflict detection might serve as the driving force in regulating top-down control influences over behaviour (e.g. Botvinick et al., 2001). In other words, it has been suggested that conflict can have beneficial consequences on behaviour by triggering changes in processing styles towards a processing style which is most adequate to the current situation. That is, if conflict is detected during information processing, this signals the need for control and subsequently an up-regulation of cognitive control takes place. More specifically, it is suggested that conflict detection takes place in the ACC which sends the conflict signal to the PFC from where subsequent adjustments are implemented. The conflict monitoring theory has proved extremely influential as it provides one of the first theoretical perspectives regarding how control adjustments are dynamically implemented and regulated. Although traditional information processing theories acknowledged the distinction between automated and controlled processing styles (e.g. Baddeley & Della Sala, 1996; Norman & Shallice, 1986; Shiffrin & Schneider, 1977), they did not explicitly specify the mechanism(s) involved in the transition between processing styles, or indeed, how the need for a change in processing style is detected. Botvinick et al. addressed this

problem by introducing the conflict-control processing loop model, whereby the detection of conflict leads to an increase in control level. In contrast, if no conflict is detected during information processing, there is no need to increase control levels. Indeed, when no conflict is detected, control levels can be down-regulated, resulting in rather automated processing. To summarise, conflict monitoring theory proposes that conflict detection regulates control levels and thus, triggers changes in information processing style, which result in behavioural adjustments (Botvinick, et al., 2001).

The conflict-control loop model consists of several distinguishable processing units which are necessary to make such a mechanism work (Botvinick, et al., 2001). First, conflict has to originate within the information processing chain. Second, conflict has to be detected. Third, the conflict signal has to trigger subsequent adjustments. Finally, subsequent adjustments have to be implemented. Despite numerous studies investigating conflict and related control adjustments, several key questions regarding the concept of conflict monitoring are still under debate. For example, there is no agreement regarding where conflict originates during information processing. Botvinick et al. (2001) assumed that conflict originates at a response-related level. However, conflict can potentially occur in all processing stages between stimulus perception and response execution. Thus, the first question addressed in this thesis concerned the locus of conflict origin within uni-dimensional task settings. Specifically, where does activation overlap between opposing response alternatives during information processing occur in order for subsequent post-conflict adjustments to take place? Moreover, little is known about how behavioural adjustments after conflict detection are actually implemented, whether adjustments are regulated in an automatic or strategic manner and which processing stages are subsequently influenced. For example, response slowings after high conflict trials are often attributed to changes in speed-accuracy settings (Botvinick, et al., 2001). However, responses after high conflict trials have been reported to be both slower and more error prone, thus excluding SAT shifts as the sole mechanism underlying post-conflict adjustments (e.g. Jentzsch & Leuthold, 2005; Rabbitt & Rogers, 1977). Thus, alternative mechanisms underlying post-conflict adjustments and possible limitations of the conflict monitoring approach were investigated in this thesis. Finally, this thesis investigated the neural correlates underlying error processing, more specifically the ERN (Falkenstein, et al., 1990; Gehring, et al., 1990). Within the conflict monitoring framework, it was suggested that the ERN reflects the high processing conflict in erroneous trials (e.g. Botvinick, et al., 2001; Gehring & Fencsik, 2001; Yeung, et al., 2004). However, such a proposal is at odds with the original suggestion that the ERN reflects a pure error detection process (e.g. Coles, et al., 2001; Falkenstein, et al., 2000). Indeed, recent evidence regarding the mechanisms underlying the ERN is ambiguous (e.g. Gehring & Fencsik, 2001; Bernstein et al., 1995).

In summary, this thesis investigated two types of conflict which typically occur in uni-dimensional task settings; more specifically, conflict in the case of errors and conflict in the case of response alternations (Botvinick, et al., 2001; Jentzsch & Leuthold, 2005). Before the findings for each of the conflict type are discussed, a brief summary of the ABI effect and why it was suggested to resemble conflict processing will follow in the next paragraphs.

I Alternation Based Interference and Conflict Monitoring

Responses following a response alternation are slower than responses following a response repetition, especially in short RSI conditions. This effect on trial N increases as the number of previous response alternations increases (e.g. Jentzsch & Sommer, 2002; Melis, et al., 2002; Soetens, et al., 1984). Jentzsch and Leuthold (2005) showed that this higher-order effect, termed ABI effect, of response sequence on response times is mainly driven by the second-order response transition (from trial N-2 to trial N-1) and occurs independently of the first-order transition (from trial N-1 to trial N). Jentzsch and Leuthold suggested that the ABI effect is due to control adjustments, similar to the adjustments proposed in the conflict monitoring theory (Botvinick et al., 2001). Other mechanisms such as a response monitoring process that creates a bottleneck that postpones processing after response alternation trials (e.g. Kirby, 1980; Pashler & Johnston, 1989) or the noise hypothesis (Laming, 1968) were excluded. For example, Kirby assumed that after response execution, S-R retrieval during the response selection stage is more time consuming in alternation compared to repetition trials, resulting in a central processing bottleneck. Critically, a central bottleneck after response alternations trials would produce underadditive effects of stimulus contrast, as perceptual processing is known to be carried out in parallel to central S-R retrieval stages (e.g. Pashler & Johnston, 1989). However, Jentzsch and Leuthold showed that stimulus contrast manipulations were not absorbed after response alternation trials. Moreover, the noise hypothesis (Laming, 1968; Soetens, et al., 1984) proposes that the representation of an internal standard is weaker in the case of response alternations. Thus, in trials following alternation trials all stimuli would be rather weakly represented, as none of the stimuli were repeated in a row of trials. Critically, the noise hypothesis clearly suggests a stimulus related locus of conflict and conflict adjustments. However, stimulus conflict alone did not result in subsequent control adjustments (Jentzsch & Leuthold, 2005), thus ruling out the noise hypothesis as an explanation of the ABI effect.

In contrast to traditional explanations and additional bottom-up explanations concerning the facilitative influence of response repetitions, Jentzsch and Leuthold

(2005) showed that top-down control adjustments triggered by conflict detection are likely to play an important role in explaining higher-order sequential effects, specifically the ABI effect. Crucially, in the case of the ABI effect it was proposed that conflict originates via overlapping activation of opposing responses in subsequent trials. For example, if response N-2 was a left hand response and response N-1 was a right hand response, overlapping activation of the two response alternatives creates conflict in trial N-1 (see introduction for details). After conflict is detected, control adjustments take place that result in slower responses in trial N (i.e. the ABI effect).

Evidence for Abstract Response Code Conflict

Despite the conflict monitoring model defining conflict as "coactivation of mutually incompatible responses" (Yeung, et al., 2004), with the assumption that conflict arises at the level of response representation, it is also possible that conflict arises at other levels during the S-R processing sequence (Botvinick, et al., 2001). Thus, it remains unclear whether stimulus, response or motor activation overlap is the source of conflict which triggers a subsequent up-regulation of control and related adjustments.

Jentzsch and Leuthold (2005) suggested that conflict in the case of the ABI effect is due to opposing response activation. More specifically, they used an information reduction paradigm (Bertelson, 1965) and showed that stimulus alternations alone do not trigger adjustments in the subsequent trial. Only response alternations from trial N-2 to trial N-1 resulted in slower responses in trial N. However, in the experiments implemented by Jentzsch and Leuthold, response alternation trials always involved a change of the response effector ('left hand' vs. 'right hand') and abstract response code ('left vs. 'right'). Thus, it remained unclear whether conflict arises at the level of abstract response representation or effector specific activation overlap. This formed the rationale of Experiment 1 and Experiment 2 of this thesis. In Experiment 1 participants had to respond vocally, thus, the same effector (mouth) is involved in each response eliminating effector-specific conflict. In contrast, conflict could occur at the level of abstract response code representation ('left' vs. 'right'). The results showed the typical ABI effect when participants responded vocally. With no effector-specific conflict to be present, this result suggests that conflict arises at an abstract response code level. However, one can argue that conflict resulted from opposing activation of low level muscular activity which is different for saying 'L' and saying 'R'. Experiment 2 implemented sequential keypress responses within each trial. Each stimulus required two keypresses, either double keypresses of the same response ('left-left' vs. 'right-right'), or alternating keypresses ('left-right' vs. 'right-left'). Effector-specific conflict is equally present within all trials when

opposing response effectors are involved in each response (‘left-right’ or ‘right-left’), independent of the trial transition. Thus, the effector-specific conflict hypothesis predicts response slowings in all three trial transitions (identical-X, equivalent-X and different-X). Actually, one could even expect a reverse ABI effect in the alternating keypress condition, as in different-X transitions a ‘left-right’ response is followed by a ‘right-left’ response, involving a repetition of the right response effector. However, the typical ABI effect was found whereby response slowing again only occurred in trials following response alternation trials (i.e. different-X transitions). Indeed, there was a larger ABI effect in the alternating response condition than in trials where double keypresses were requested. As effector-specific conflict did not predict specific response slowings after response alternation trials, these results suggest that conflict arises at the level of abstract response activation. The finding that the ABI effect was even larger in the alternating response condition could be due to the fact that higher response complexity involves more overall response activation and thus possibly produces more conflict (e.g. Wexler, et al., 1997). Taken together, Experiment 2 supports the findings from Experiment 1, suggesting that conflict arises at an abstract level of response representation.

In summary, both experiments provide evidence that conflict after response alternations originates at the level of abstract response code activation overlap rather than effector specific activation overlap. It is important to mention that these findings might be specific to the origin of conflict within response alternation trials. Conflict from other sources, for example, response errors may involve a different locus of conflict origin. Moreover, knowing where conflict originates does not have any implications regarding where post-conflict adjustments take place. Findings regarding the locus of post-conflict adjustments will be discussed in the following section.

Evidence for Central Processing Slowing after Response Alternation Trials

Behavioural changes following high conflict trials are proposed to be the outcome of an effective conflict monitoring system (e.g. Botvinick, et al., 2001; Gratton, et al., 1992). More specifically, the conflict monitoring model suggests that these changes take place through an up-regulation of cognitive control, whereby top-down attentional pathways are strengthened (Botvinick, et al., 2001). However, little is known regarding the actual processes affected in information processing after high conflict trials. Indeed, adjustments are likely to take place in a very task specific manner. Botvinick et al. (2001) suggested that control related response slowings after high conflict trials take place through adjustments on the SAT function, via a shift of the response criterion resulting in

more or less conservative response settings. Critically, changes in response criterion after conflict should not only result in slower but also more accurate performance. However, in the studies of Jentzsch and Leuthold (2005), as well as in Experiment 1, 2 and 3 of the present thesis, response accuracy did not increase after response alternation trials. Jentzsch and Leuthold suggested that one could argue that response accuracy was very high overall and thus, changes in response thresholds were only reflected in response times. However, in two of the four experiments of Jentzsch and Leuthold, response accuracy decreased after conflict detection. Thus, it is possible that in contrast to changes in response threshold, changes in the speed of information accrual may account for the ABI effect. Interestingly, Laming (1968) suggested that after response alternations trials, impairments in speed and response accuracy might be due to "the mean rate of extraction of information from the signal presented is less after an alternating sequence of signals" (p. 110). Thus, control adjustments after conflict may be due to changes in the rate of information accrual, which makes responses not only slower but also results in lower accuracy rates due to information loss resulting in harder S-R processing.

As described earlier, Jentzsch and Leuthold excluded the noise (Laming, 1968) and the response monitoring hypothesis (Kirby, 1980) as explanations for the ABI effect. In the present thesis, two alternative hypotheses regarding the mechanisms underlying the ABI effect were contrasted. First, as described above, changes in the speed of information accrual might take place. Importantly, changes in the information accrual speed may be specific to one information processing stage, for example the response selection stage (cf. Jones, et al., 2002; Laming, 1968). On the other hand, the ABI effect could be due to an overall postponement of trial processing following response alternation trials (e.g. Oriet & Jolicœur, 2003). Oriet and Jolicœur suggested such a hard bottleneck mechanism to be active in task switch trials, due to a process of task-set reconfiguration. If one assumes that a form of reconfiguration process is also involved in response alternation trials, one would expect all subsequent trial processing to be postponed in a similar manner.

Thus, the hard bottleneck hypothesis and the hypothesis of the change in information accrual speed would differ in their prediction regarding the locus of the post-conflict adjustments. If a hard bottleneck causes response slowing, all information processing stages including early visual processing should be delayed after response alternation trials (Oriet & Jolicœur, 2003). In contrast, if response slowing is due to slower information accrual, this can occur in specific processing stages after early visual processing (cf. Jones, et al., 2002; Laming, 1968). Experiment 3 investigated the locus of the ABI effect. Experiment 1 and 2 suggested that conflict originates at a level of abstract response code representation. However, this does not rule out the possibility that post-

conflict adjustments take place at a different level of information processing. Using chronometric electrophysiological measurements, Experiment 3 showed that the S-locked LRP onset was delayed in trials following response alternations. In contrast, components reflecting perceptual processing, such as the P1/N1 and the P300, were not affected by previous response alternations. Also, the LRP-R onset was not affected by trial transition, suggesting that the motor processing time was not influenced by response transition. Thus, these findings suggest that response slowing after response alternations is due to specific prolonging of central processing stages. Importantly, knowledge about the functional locus of post-conflict adjustments in the case of the ABI effect allows us to draw conclusions regarding the mechanisms underlying this slowing. Specifically, as central processes are affected, a hard processing bottleneck delaying all information processing stages can be excluded (e.g. Oriet & Jolicœur, 2003). In contrast, it is plausible that the information accrual rates in central processing stages are slowed down in trials following response alternations, resulting in the ABI effect (cf. Jones, et al., 2002; Laming, 1968).

However, alternative explanations and potential problems have to be addressed. All experiments investigating the ABI effect reported in this thesis average trials across first-order sequences (see also: Jentzsch & Leuthold, 2005). However, such an averaging approach ignores potential contributions from a nonsymmetrical, one-sided mechanism to the second-order ABI effect. For example, one-sided effects might produce a processing cost in the current trial after second-order response alternations, but only in first-order alternations and no costs or benefits in first-order response repetitions. However, previous studies showed that such a mechanism is not present at short RSIs (e.g. Jentzsch & Leuthold, 2005; Jentzsch & Sommer, 2002). Moreover, Jentzsch and Leuthold showed that interactions of first-order and second-order effects are due to first-order stimulus repetitions being faster and more accurate than first-order stimulus alternations, a finding explained by stimulus-trace mechanisms (cf. Treisman & Williams, 1984). The size of the ABI effect was only modulated by first-order transitions when participants respond to response location in incompatible S-R mapping conditions (the ABI effect increased more for first-order response alternations in the location condition). Importantly, this was not observed when participants responded to a property other than stimulus location. Thus, the increase of the ABI effect in the incompatible response condition is potentially modulated by one-sided effects, such as negative priming (cf. Shiu & Kornblum, 1996). Crucially, this explanation cannot account for the increase of the ABI effect in none location based conditions, for example, when participants respond to arrow symbols or letters. Thus, even if the size of the ABI effect is partly due to one-sided mechanisms under certain circumstances, response conflict also determines the size of the ABI effect, ruling out such an automatic and one-sided

mechanism as a sole contributing factor to the ABI effect. If such a biased mechanism is rather automated and not particularly time consuming, there is no reason to assume such a mechanism causes the ABI effect in long RSI conditions in the present thesis.

In summary, Jentzsch and Leuthold (2005) showed that the ABI effect originates at a response related level of information processing. In addition, Experiments 1 and 2 in this thesis showed that conflict originates at an abstract response code level. However, conflict induced by a response alternation from trial N-2 to trial N-1 influenced current trial N performance by selectively prolonging response selection stages (cf. Jones, et al., 2002; Laming, 1968). These results show that it is important to clearly differentiate between the locus of conflict origin and the locus of the effect of conflict in the following trial. Importantly, control adjustments might be different depending on the exact nature of processing conflict. Thus, the present results might be specific to the ABI effect. Further studies need to clarify the generality of the proposed mechanism to a wider range of conflict situations. In the following sections the findings of this thesis regarding conflict and post-conflict adjustments in erroneous trials will be discussed.

II Errors and Conflict Monitoring

In most studies investigating human information processing, error trials are excluded from the analysis despite errors and error occurrence being an important part of human cognition. Errors were of interest for this thesis as error trials are another example of a conflict situation within a uni-dimensional task setting. In this context, conflict is assumed to arise from the simultaneous activation of the executed erroneous and the required correct response (e.g. Botvinick, et al., 2001; Rabbitt & Vyas, 1981). Moreover, post-error slowing is seen as an indicator of successful response adjustment after errors, due to a change in response strategy towards a more conservative response mode (Botvinick, et al., 2001). Besides the behavioural findings, the ERN was suggested to be a signal of ongoing conflict processing and conflict detection processes (Botvinick, et al., 2001).

Evidence for the ERN Reflecting Error Detection Processes

Investigations of error processing have flourished with the discovery of the ERN (Falkenstein, et al., 1990; Gehring, et al., 1990). Many studies of error processing are driven by an interest in identifying the exact mechanisms underlying the ERN. Despite this increasing interest in error processing, the underlying mechanisms and the relationship between the ERN and subsequent compensatory behaviour, such as post-error slowing (e.g. Brewer & Smith, 1984; Laming, 1968; Rabbitt, 1966, 1968), are still

under debate. Originally, it was suggested that the ERN reflects an error detection process (e.g. Falkenstein, et al., 1990; Falkenstein, et al., 2000). The error detection process involves a comparison between the representation of the executed response, and the representation of the required response (Coles et al., 2001). It is assumed that the representation of the required response results from ongoing processing of the stimulus input beyond error response initiation (Rabbitt & Vyas, 1981). An alterative explanation of the ERN posits a conflict detection process (e.g. Botvinick, et al., 2001; Yeung, et al., 2004). Here, the ERN results from error trials being high in conflict and not the error per se.

Despite similarities between the error detection and the conflict detection hypotheses, their predictions differ regarding the size of the ERN. Specifically, if the ERN reflects an error detection process, the ERN should be larger the more dissimilar the erroneous response is to the required correct response (e.g. Gehring & Fencsik, 2001), as the ERN is elicited by a mismatch in the comparison process (e.g. Coles, et al., 2001). In contrast, if the ERN reflects a conflict detection process, it should be larger when the erroneous and the required response are similar, as conflict is greatest in this situation (Botvinick, et al., 2001). Experiment 4 and 5 of this thesis contrasted the predictions of the error detection and the conflict detection hypotheses regarding the ERN within a 4-choice task. In 4-choice tasks three error possibilities exist, each differing in their similarity to the correct response. In Experiment 4 participants responded with their index and middle fingers of the left and the right hand. The results showed that the ERN was largest when the error and required response were most dissimilar (e.g. left index instead of right middle finger), suggesting that the ERN reflects an error detection process. Experiment 5 utilised finger and foot responses and showed that the ERN was largest when participants responded with the wrong effector (i.e. left hand instead of left foot, or left hand instead of right foot). Thus, both experiments clearly speak against the conflict detection hypothesis, as the ERN was largest in the erroneous trials which were rather dissimilar to the required response. The only finding challenging the error detection hypothesis was that in Experiment 5, the ERN was smallest for errors made with the correct effector only (i.e. left hand instead of right hand) but not for error on the correct response side (i.e. left hand instead of left foot). Thus, it seems that the shared response side alone did not have any advantage on error processing. This means that if participants respond with the left hand instead of the left foot, the fact that both responses involved the left body side does not have any advantage on error processing. In contrast, in Experiment 4 the correct response side was confounded with the correct response hand. If participants responded with the left index instead of the left middle finger, they still chose the correct response hand. Thus, it is possible that the shared effector “hand” resulted in the reduction of the ERN amplitude, rather than the shared

response side in Experiment 4. In summary, Experiment 4 and 5 support the error detection over the conflict detection hypothesis of the ERN.

In conclusion, these results suggest that the ERN increases with easier error detection through a larger mismatch signal in the comparison process. However, there was also a clear ERN in correct trials (see also: Vidal, et al., 2000). As an alternative explanation Falkenstein et al. (2000) suggested that the ERN reflects the comparison process between the executed and the required response, rather than the outcome of this process (see also: Vidal, et al., 2000). The present findings suggest that the ERN increases with the mismatch between the required and the executed response but do not exclude the suggestion that the ERN reflects the comparison process itself. Further studies are needed to address this question.

Evidence for Dissociation Between the ERN Amplitude and Post-error Slowing

In addition to the question regarding the mechanism underlying the generation of the ERN, it is still under debate whether the ERN relates to subsequent behavioural adjustments, such as post-error slowing (e.g. Brewer & Smith, 1984; Laming, 1968; Rabbitt, 1966, 1968). The findings of studies investigating the relationship between the ERN amplitude and subsequent behavioural adjustments, specifically post-error slowing, have been ambiguous. A number of studies suggested that the brain structures generating the ERN, especially the ACC (e.g. Dehaene et al., 1994), are involved in both detecting errors and also initiating subsequent behavioural control adjustments. More specifically, in multi-dimensional task settings such as the Eriksen Flanker or the Stroop task, positive correlations between the ERN amplitude (or the activity of the ACC) and RTs in post-error trials have been reported (e.g. Debener et al., 2005; Gehring et al., 1993; Kerns et al., 2004; West and Travers, 2008). However, some other studies did not report such a correlation between post-error slowing and the ERN amplitude (e.g. Gehring and Fencsik, 2001; van Meel et al., 2007) and therefore challenge the idea of a direct link between the amount of the ACC activity and subsequent behavioural control adjustments.

Experiments 4 and 5 investigated the relationship between ERN amplitude and subsequent behavioural adjustments in a uni-dimensional 4-choice task. Despite a clear impact of the mismatch between the erroneous and the required response on the ERN amplitude, these differences in the ERN amplitude were not reflected in the post-error slowing. In other words, the amount of subsequent post-error slowing was not related to the size of the ERN. Additionally, in Experiment 7 four different RSI conditions were implemented, resulting in large differences in the amount of post-error slowing, whereby

post-error slowing was largest in short RSI conditions. Thus, if the ERN predicts the amount of post-error slowing, larger ERN amplitudes should be observed in the short RSI conditions. However, the ERN amplitude did not vary between the four RSIs. Analysis of difference waves suggested that the ERN was slightly reduced in the two shortest RSIs. One can suggest that if the ERN reflects error processing, the ERN in the case of the short RSIs was reduced due to unfinished error processing. As differences in RSI potentially affect the ERP measurements, these results have to be interpreted with care. Importantly, for the two short and the two long RSIs, the ERN did not differ. This speaks against a pure RSI effect on the ERN. Still, further studies are required to analyse the functional significance of the ERN and to confirm such a suggestion.

Evidence Against Strategic Control Adjustments Underlying Pre- and Post-error Effects

The finding that trials following errors are often slower than trials following correct responses is a well known phenomenon (e.g. Brewer & Smith, 1984; Laming, 1968; Rabbitt, 1966; Rabbitt & Rogers, 1977). In addition to the responses being slower after error commission, trials preceding errors are typically faster than average response times (e.g. Brewer & Smith, 1984). It is generally assumed that pre-error speeding and post-error slowing result from the same mechanism. Rabbitt (1979) proposed a trial-by-trial tracking model that assumes a systematic speed-up of responses until error occurrence, whereby responses subsequently slow before systematic speed-up returns. Critically, such traditional models assume that behavioural adjustments take place through shifts along the SAT function. More specifically, it is suggested that after error commission response thresholds are raised in order to avoid future errors (e.g. Brewer & Smith, 1984; Rabbitt, 1966, 1979). In contrast, after a sequence of correct trials, response thresholds are lowered, in order to speed up responses. In line with such a trial-by-trial tracking mechanism, conflict monitoring theory suggests bi-directional control regulations (Botvinick, et al., 2001). Specifically, after an error (high conflict trial), control is up-regulated while after a correct trial (low conflict trial), control will be down-regulated. Importantly, Botvinick et al. suggested that changes in response speed are due to adjustments of the response thresholds along the SAT function. Taken together, both traditional models and the conflict monitoring model suggest that both pre-error speeding and post-error slowing are directly linked via the same mechanism, the regulation of response threshold or level of control.

However, several arguments have been raised against such a SAT adjustment taking place after errors. Rabbitt and Rogers (1977) pointed out that post-error slowing effects which can be as large as 300 ms seem larger than needed in order to return to safe response thresholds. Moreover, it is not clear whether these regulations of response thresholds along the SAT function take place in an automated or strategic and time consuming manner (e.g. Sommer, et al., 1999). If strategic adjustments are involved, one would expect the behavioural changes to be particularly pronounced when there is sufficient time for the strategic process to be implemented. Previous research has shown that strategic top-down influences on information processing are only effective for RSIs longer than 500 ms (e.g. Sommer, et al., 1999). However, Sanders (1998) pointed out that studies using particularly short RSIs often reported larger post-error slowing effects than studies implementing longer RSIs (e.g. Laming, 1979; Rabbitt & Rogers, 1977). These findings suggest that changes in response thresholds must be caused by automated rather than by strategic and time consuming mechanisms. Problematically for all SAT based explanations, many studies that have implemented short RSIs, not only show pronounced post-error slowing effects, but also increased error rates (e.g. Rabbitt, 1966; Rabbitt & Rogers, 1977). SAT explanations assume increased post-error slowing effects are due to increased response thresholds. However, increased response thresholds should result in slower but importantly also more accurate performance.

Experiment 6 of this thesis investigated an alternative mechanism for post-error slowing effects. More specifically, it has been suggested that after errors a response monitoring bottleneck engages central resources (e.g. Welford, 1959, 1980), which interferes with subsequent trial processing. Thus, in trials following errors central processing would be delayed until error monitoring from the previous trial has finished and cleared central resources (see also: Gehring & Fencsik, 2001; Hochman & Meiran, 2005; Laming, 1979). To test such a central bottleneck hypothesis after error occurrence, stimulus contrast was manipulated. If postponement of central processing causes post-error slowing, manipulations of stimulus contrast should be absorbed in this postponement slack (Pashler & Johnston, 1989). In contrast, if post-error slowing is due to changes in response threshold, there is no postponement slack period in which contrast can be absorbed, and effects of stimulus contrast should be additive to post-error slowing effects.

In line with the error monitoring hypothesis (e.g. Welford, 1959, 1980), the results of Experiment 6 showed that stimulus contrast manipulations are absorbed in post-error trials, resulting in an underadditive effect for short RSIs. Moreover, post-error slowing was particularly large and more error prone in short RSI conditions compared to long RSIs. Interestingly, in long RSI conditions post-error slowing was still present and the effect was additive with stimulus contrast manipulations. Thus, at long RSI conditions

error monitoring cannot account for the effects of post-error slowing, as the effects of stimulus contrast are not absorbed any longer, suggesting that any error monitoring process will be completed during the long RSI interval. Moreover, in long RSIs post-error slowing was both smaller and error rates numerically decreased. Taken together, these findings suggest that the mechanisms underlying post-error slowing are two-fold. First, for short RSIs ongoing error processing creates a central bottleneck resulting in a postponement of response selection stages in the subsequent post-error trial, which results in pronounced post-error slowing. Second, when error processing has been completed (i.e. at long RSIs) strategic adjustments of response thresholds are likely to take place, resulting in smaller post-error slowing effects, together with an increase of response accuracy.

Importantly, despite these results demonstrating a response monitoring bottleneck that occupies central resources after errors and postpones subsequent processing, the role of this monitoring process remain unclear. If response monitoring leads to subsequent performance impairments (i.e. slower and more error prone responses), it does not seem beneficial for information processing to monitor responses. One possibility is that response monitoring resembles an error detection process (e.g. Rabbitt & Vyas, 1981; see also Rabbitt, 2002). In both traditional error processing theories and the conflict monitoring theory (Botvinick, et al., 2001), it is assumed that error detection is crucial for post-error adjustments to take place. Despite the importance of error detection, not much is known about how exactly and when error detection takes place. If error detection takes place during actual trial processing, why cannot the actual error be suppressed? It seems possible that error detection extends beyond response execution and occupies central resources, and causes a central bottleneck. In line with this suggestion, neurophysiological measurements such as the ERN peak after error execution and have been suggested to reflect error detection processes (e.g. Coles, et al., 2001; Falkenstein, et al., 2000). Thus, it seems likely that such an error detection mechanisms causes the central bottleneck, however further studies are needed to confirm this suggestion.

As described above, it has been shown that neither automated nor strategic changes in response thresholds fully account for the effect of post-error slowing, especially in short RSI conditions. More specifically, before strategically influenced control adjustments can influence performance beneficially, sufficient time is required in order to process the error completely. This questions the direct link between pre-error speeding and post-error slowing effects, and questions the mechanisms underlying pre-error speeding. In Experiment 7 pre-error speeding and its relationship to subsequent post-error slowing effects were investigated. If pre-error speeding is due to strategic adjustments in response styles through a down-regulation of response thresholds after a

correct trial, one could expect it to be particularly pronounced if there is enough time to implement such strategic adjustments (e.g. Sommer, et al., 1999). In contrast, if pre-error speeding is due to an automated down-regulation of response thresholds, or even random fluctuations of response thresholds, one would expect pre-error speeding to be independent of the time available after a correct trial (e.g. Brewer & Smith, 1984). The results of Experiment 7 showed that pre-error speeding was present in similar amounts (~ 20 ms) across different RSI levels. Thus, it is most likely that pre-error adjustments are due to either automatic adjustments or random fluctuations in response thresholds after correct responses. In contrast, both Experiment 6 and Experiment 7 demonstrated RSI dependent post-error slowing effects, being largest at short RSI conditions and systematically decreasing with increasing RSI.

Taken together, these results suggest that error processing can produce substantial interference when there is not enough time to fully process the error before the next trial starts. Beneficial adjustments of response thresholds resulting in slower and more accurate performance can explain post-error slowing effects, but only if there is enough time to process the error completely. In contrast, pre-error speeding effects seem to be due to automatic changes or random fluctuations in response threshold. These results clearly suggest that one cannot assume identical mechanisms to cause pre-error speeding and post-error slowing effects. Moreover, interpretations of post-error slowing as a measurement of beneficial adjustments in cognitive control have to be treated with care as a significant amount of post-error slowing was due to interference processes.

III Conclusions

The ABI effect and post-error slowing are the two main phenomena which occur in uni-dimensional tasks and have been explained in the framework of conflict monitoring (e.g. Botvinick, et al., 2001; Jentzsch & Leuthold, 2005). Response slowings following high conflict trials are traditionally explained by shifts along the SAT function. However, both response alternation trials and error trials are typically followed by slower and often less accurate responses, clearly speaking against SAT shifts as the underlying mechanisms. In this thesis it was shown that post-conflict slowing in case of the ABI effect was due to prolonging of central processing stages, which can be explained by a change in the information accumulation rate (cf. Jones, et al., 2002; Laming, 1968). Moreover, the experiments showed that conflict in case of the ABI effect occurs at the level of abstract response code representations. In the case of error occurrence it was shown that especially if there is little time available to recover from an error until the next trial starts, post-error slowing is particularly large and error prone. This performance impairment was explained by a central error monitoring bottleneck that extends beyond error occurrence and delays subsequent processing. In contrast, if there is enough time for completing error processing before the next trial starts, beneficial shifts in response strategies could take place, resulting in slower and more accurate performance. In contrast, pre-error speeding occurred in similar amounts independent of the time available between trials. Thus, it was suggested that automatic down-regulation of response thresholds or random fluctuations of response thresholds are most likely to account for the effects of pre-error speeding. Regarding neurophysiological correlates of error processing, it was shown that the ERN increases with increasing mismatch between the required correct and the executed erroneous response, suggesting that the ERN increases with the ease of error detection and thus, reflects error rather than conflict detection processes. Interestingly, post-error slowing did not reflect these changes in the ERN amplitude.

In summary, both the ABI effect and post-error slowing effects cannot be accounted for fully by beneficial adjustments of response strategies. In contrast, interference processes between ongoing conflict processing and the subsequent trial seem to play an important role in explaining very pronounced post-error slowing effects. Beneficial cognitive control adjustment did take place, but only when there was sufficient time to process the error. Also, conflict in the case of response alternations does not trigger beneficial adjustments along the SAT function, but rather results from slowing in central evidence accumulation. Thus, interpretations of response slowings after high conflict trials as a marker of beneficial top-down control adjustments have to be regarded with care.

4. Abstract

People have the ability to decide quickly between multiple response alternatives and to deal with conflicting information. Traditionally, it was assumed that conflict has mainly disadvantages for information processing and results in slower and less accurate responses. However, recently it was suggested that conflict plays an important regulatory role in implementing cognitive control adjustments (Botvinick et al., 2001). In the present thesis the causes and outcomes of conflict in unidimensional task settings were investigated. More specifically, conflict was analysed in response alternations and in erroneous trials. Importantly, after both response alternation trials and erroneous trials, responses are slowed down in the subsequent trial. In the conflict monitoring model such response slowing after high conflict trials are often explained by up-regulations of response thresholds, which should result in slower but also more accurate performance. In contrast, it has been shown that after an error or after response alternations, responses are often both slower but also less accurate. In this thesis, is was suggested that after erroneous trials an error monitoring bottleneck occupies central processing resources, resulting in postponement of subsequent processing. This was supported with the finding that stimulus contrast manipulations, known to influence perceptual processing, were absorbed in this postponement slack (see: Pashler & Johnston, 1989). Only if there was sufficient time to completely process the error before the next trial started could beneficial control adjustments take place. In contrast, trials before an error were faster independent of the time available between the trials. This suggests that the down-regulation of response thresholds after a correct trial takes place automatically, or is due to random fluctuations of response thresholds (e.g. Brewer & Smith, 1984). Additionally, the ERN was largest if the erroneous and the required response did not share any response parameter. As conflict is larger the more similar two response alternatives are, this finding suggests that the ERN reflects error detection rather than conflict detection processes. Moreover, it was shown that the response slowing after response alternations was reflected in a delay of the S-LRP onset. Neither LRP-R nor visual components (P1/N1 and P300) were affected after response alternations. This shows that alternation based interference is caused by slowing in central information processing stages, likely due to slower information accrual (cf. Jones et al., 2002; Laming, 1968). Additionally, it was shown that conflict originates through the overlap of abstract response activation (‘left’ vs. ‘right’) rather than effector specific activation overlap (left vs. right hand). In conclusion, these findings suggest that response slowing after high conflict trials is not always the result of beneficial adjustments in cognitive control settings. In contrast, response slowing after high conflict trials can be also caused by postponement or a slow down of central information processing stages.

5. Zusammenfassung

Um schnell und richtig zu handeln, müssen wir in der Lage sein, zwischen verschiedenen Alternativen zu entscheiden und in Konflikt stehende Information zu verarbeiten. In der Kognitionspsychologie wurde ursprünglich angenommen, dass Konflikt sich in erster Linie nachteilig auf die Informationsverarbeitung auswirkt. Denn besteht Konflikt zwischen mehreren Reaktionsalternativen, reagieren wir oft langsamer und es ist wahrscheinlicher, dass uns ein Fehler unterläuft. Jedoch wurde neuerdings auch vorgeschlagen, dass Konflikt eine wichtige regulatorische Aufgabe zukommt (Botvinick et al., 2001). Hierbei wird angenommen, dass Konflikt den Einfluss von kognitiver Kontrolle auf die Informationsverarbeitung verstärkt. In der vorliegenden Arbeit wurden die Ursachen und die Auswirkungen von Konflikt in eindimensionalen Aufgaben untersucht. Das heißt, es wurde Konflikt in Reaktionswechsel und Konflikt in fehlerhaften Durchgängen analysiert. Hierbei ist besonders, dass in beiden Fällen, d.h. nach einem Fehler und nach einem Reaktionswechsel, die Reaktionen im folgenden Durchgang verlangsamt sind. Solche Verlangsamungen werden in der Konflikt-Monitoring-Theorie mit einer Erhöhung des Reaktionskriteriums erklärt, was in langsameren aber auch genaueren Reaktionen resultieren sollte (Botvinick et al., 2001). Im Gegensatz dazu wurde gezeigt, dass Reaktionen nach einem Fehler oder nach einem Reaktionswechsel oft langsamer aber auch fehlerhaft sind. In der vorliegenden Arbeit wurde vorgeschlagen, dass nach einem Fehler, wenn wenig Zeit besteht diesen vollständig zu verarbeiten, zentrale Informationsverarbeitungskapazitäten besetzt sind, und die zentrale Verarbeitung des folgenden Durchgangs verschoben werden muss. Dies wurde dadurch bestätigt, dass Manipulationen des Stimuluskontrastes, die die Dauer der visuellen Verarbeitung beeinflussen, in diesem Verarbeitungsflaschenhals absorbiert wurden (vgl. Pashler & Johnston, 1989). Nur wenn genug Zeit besteht, den Fehler vollständig zu verarbeiten bevor der nächste Durchgang beginnt, konnten vorteilhafte Anpassungen des Verarbeitungsstils stattfinden. Im Gegensatz dazu waren Reaktionen vor einem Fehler generell schneller, unabhängig davon wieviel Zeit zwischen den Durchgängen bestand. Dies kann dadurch erklärt werden, dass die Herab-Regulation des Reaktionskriteriums nach einem korrekten Durchgang automatisch stattfindet, oder sogar zufällige Veränderungen des Reaktionskriteriums für diese schnelleren Reaktionszeiten vor fehlerhaften Durchgängen verantwortlich sind (z.B. Brewer & Smith, 1984). Die Untersuchung der elektrophysiologischen Korrelaten von Fehlern zeigte, dass die Amplitude der ERN größer ist, wenn die fehlerhafte und die erforderte korrekte Reaktion keine Reaktionsparameter teilen und somit sehr unterschiedlich sind. Da ein Konflikt am größten ist je ähnlicher sich zwei Reaktionsalternativen sind, zeigt dieses Ergebnis, dass die ERN eher Prozesse der Fehlerentdeckung und nicht der Konfliktentdeckung

widerspiegelt. Elektrophysiologische Ergebnisse zeigten auch, dass die Reaktionsverlangsamung nach einem Reaktionswechsel, sich in einer Verschiebung des S-LRP widerspiegelten. Weder die motorische (LRP-R), noch die visuelle Verarbeitungsdauer (P1/N1, P300) waren beeinflusst nach Durchgängen mit einem Reaktionswechsel. Dies bedeutet, dass nach einem Reaktionswechsel zentrale Verarbeitungsstufen mehr Zeit beanspruchen, was wahrscheinlich auf eine Verlangsamung der Informationsakkumulation zurückgeht (vgl. Jones et al., 2002; Laming, 1968). Darüberhinaus wurde gezeigt, dass ein Konflikt in Durchgängen mit einem Reaktionswechsel auf einen Konflikt zwischen den abtrakten Reaktionscodes zurückgeht (d.h. ‚links' vs. ‚rechts'), und nicht auf Konflikt zwischen den Effektoren (z.B. linke vs. rechte Hand).

Schlussfolgernd lässt sich festhalten, dass Reaktionsverlangsamungen nach fehlerhaften Durchgängen oder Durchgängen mit einem Reaktionswechsel, nicht unbedingt eine strategische und vorteilhafte Reaktionsanspassung darstellen. Dies ist besonders von Bedeutung, da bisher in Patientenstudien Reaktionsverlangsamungen nach Durchgängen mit hohem Konflikt oft als Hinweis auf ein funktionierendes kognitives Kontroll-System interpretiert wurden (siehe auch: Notebaert et al., 2009). Im Gegensatz dazu zeigten die vorliegenden Ergebnisse vielmehr, dass ein großer Teil der Reaktionsverlangsamungen nach Fehlern und nach Reaktionswechsel aufgrund einer Verschiebung oder Verlangsamung der Informationsverarbeitung ohne einen direkten vorteilhaften Einfluss, zustande kommt.

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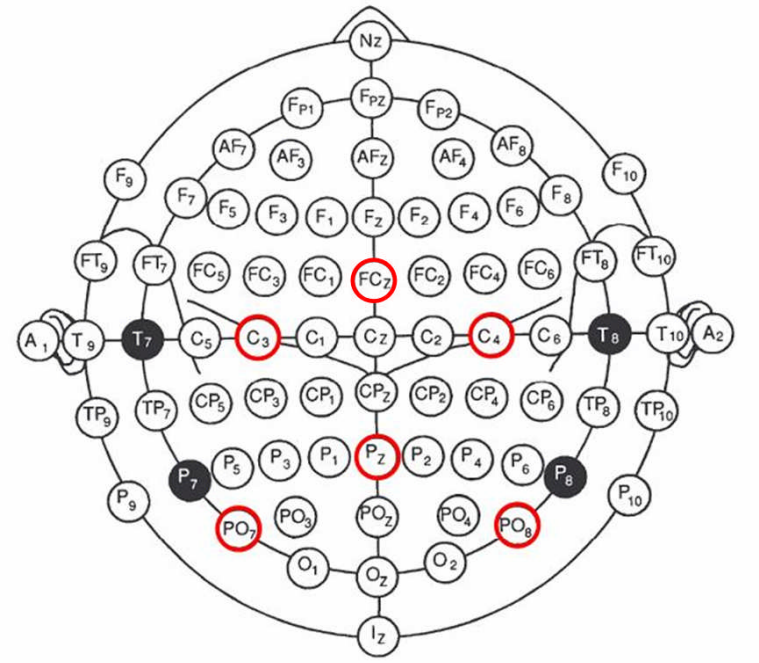
Appendix

Extension of International 10-20 System of Electrode Placement



(Picton, Lins, & Scherg, 1995)

Label	Position
FP	fronto-polar
AF	antero-frontal
F	frontal
FC	fronto-central
C	central
T	temporal
CP	centro-parietal
TP	tempo-parietal
M	mastroid
P	parietal
PO	pareto-occipital
O	occipital
I	inion
Z	Midline

Numbering
Even numbers: right hemisphere
Odd numbers: left hemisphere

High-lightening
C3 and C4
= Electrode sides for LRP calculation
Pz
= P300 Electrode
PO7 and PO8
= P1 and N1 Electrodes
Fcz
= ERN Electrode