The Effects of Conflict Strength and Ageing on Cognitive Control

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Abstract

In this thesis, I investigated effects of conflict strength and ageing on cognitive control. Conflict strength was manipulated in the Eriksen flanker task using two different approaches: 1. independent variation of flanker and target contrast; 2. manipulation of stimulus onset asynchrony (SOA). Reducing flanker contrast relative to target contrast decreased conflict strength, as shown by a reduction in compatibility effects, when contrast conditions were presented in a randomized fashion but not when they were presented block-wise. An SOA of 100 ms did lead to increased compatibility effects compared to SOAs of 0 ms and 200 ms. Effects of conflict appear to be reflected in the N2 component of the ERP. Although priming played a crucial role in the emergence of the sequential adjustment effect, conflict strength also influenced this effect to a certain degree, supporting the claim that sequential adjustments represent an adaptation of cognitive control. Post-error slowing and error-related ERP components, on the other hand, were not affected by the conflict manipulations, suggesting that errors cannot be explained in terms of conflict processing. Effects of ageing on cognitive control were investigated in a group of middle-aged participants. Although physiological indicators of conflict and error processing were compromised in this age group and overall response times were increased, compatibility, sequential adjustment, and post-error slowing effects were of comparable size as in young adults. These findings suggest that participants could successfully compensate for age-related physiological changes at this early stage of ageing. In conclusion, the research presented in this thesis provided important information to extend our knowledge of factors influencing cognitive control processes.

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List of Abbreviations

ACC	anterior cingulate cortex
c	compatible
CMS	common mode sense
CNV	contingent negative variation
DLPFC	dorsolateral prefrontal cortex
DRL	driven right leg
EEG	electroencephalography
EMG	electromyography
EOG	electrooculography
ERP	event-related potential
fMRI	functional magnetic resonance imaging
FRN	feedback-related negativity
ic	incompatible
Nc/CRN	correct negativity/correct-related negativity
Ne/ERN	error negativity/error-related negativity
Pe	error positivity
POP	preparing to overcome prepotency
RSI	response-stimulus interval
SMA	supplementary motor area
SOA	stimulus onset asynchrony

1 Literature Review

1.1 What is Cognitive Control?

In everyday life people frequently encounter situations in which they must selectively focus their attention on particular aspects of the surroundings, while ignoring irrelevant information. For example, consider driving a car. The driver has to focus his/her attention on the road whilst avoiding distractions from task-irrelevant stimuli and events, such as pedestrians or flashing lights of advertisements. At the same time, the driver has to pay enough attention to apparently unimportant stimuli to react on time should a given stimulus become relevant (e.g., when a pedestrian suddenly crosses the road). The set of skills that are needed to selectively regulate attention and focus on goal-relevant while ignoring irrelevant information is called cognitive control. Other tasks that require high levels of control are planning and monitoring of actions, inhibition of inappropriate response tendencies, task set maintenance, and goal setting. The common characteristic of these tasks is that they cannot be conducted automatically and require some level of top-down control.

1.2 The Conflict Monitoring Theory

Many of the hypotheses tested in this thesis are derived from the conflict monitoring account of cognitive control by Botvinick and colleagues (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Botvinick, Cohen, & Carter, 2004). The authors distinguish between a regulative and an evaluative dimension of control. The regulative dimension is the actual implementation of control, i.e. the top-down influence of a control system on information processing. The evaluative dimension of control describes how people establish the need for control adjustments. In particular, the authors postulate the existence of a conflict monitoring system that monitors the level of conflict in information processing and feeds this information back to the system that is responsible for the implementation of control. This system adjusts the control level accordingly to prevent the occurrence of further conflicts. Control will be increased after the experience of conflict and it will be relaxed over time if no new conflict occurs.

The basic ideas of this theory are shown in *Figure 1*, using a model of a simple choice task by Usher and McClelland (1995) as an example. The input layer represents the two possible stimuli S1 and S2. The conflict monitoring system,

depicted in grey, monitors for conflict between the representations of responses R1 and R2 (e.g., button presses) in the response layer and passes this information on to the response-priming layer, which regulates how much each of the two responses is preactivated. The inhibition arrow at the response layer depicts the reciprocal inhibitory connection between the two responses, representing the fact that these responses are incompatible with each other.

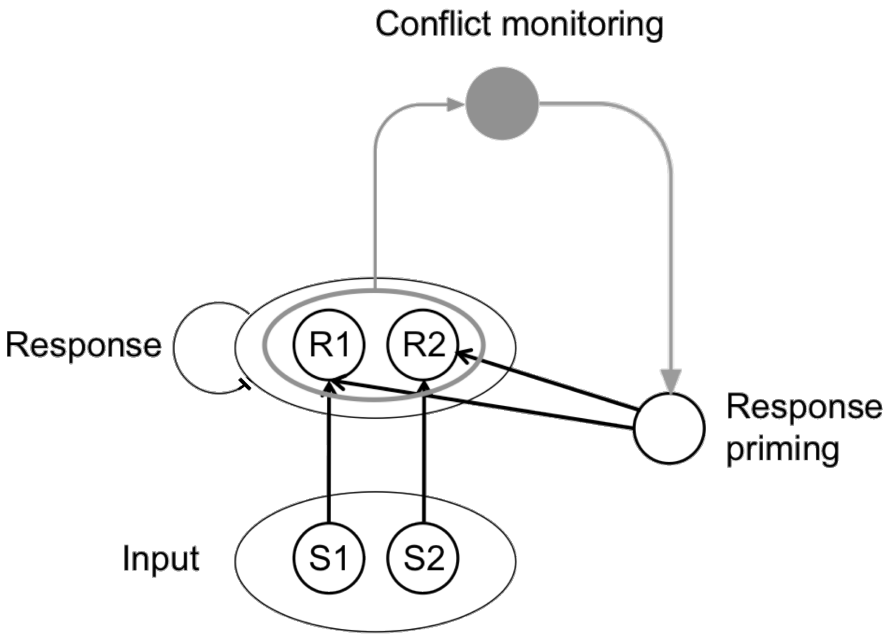

Figure 1: Implementation of the conflict monitoring theory in a simple choice task consisting of two stimuli (S1 and S2) which are associated with two different responses (R1 and R2) based on a model by Usher and McClelland (1995). The conflict monitoring system (depicted in grey) monitors for the occurrence of conflicts between the responses and adjusts the level of control by influencing the amount of response priming.

Conflict is operationally defined in the conflict monitoring model as the simultaneous activation of incompatible response representations. In other words, if a stimulus in an experiment contains information that corresponds to both of two possible responses, crosstalk interference occurs between the representations of these responses, resulting in conflict. Based on Berlyne (1957), Botvinick et al. (2001) proposed that conflict increases with the absolute activation and the number of

competing response representations. Importantly, it is expected to be maximal when all representations are activated with equal strength.

According to the conflict monitoring theory, the monitoring system is located within the anterior cingulate cortex (ACC; see *Figure 2*), an area within the frontal lobes that has been shown to be involved in a variety of cognitive and emotional functions. Traditionally, rostral-ventral areas have been ascribed to affective functions, whereas the dorsal part was believed to play a role in cognitive functioning, including executive functions like the modulation of attention, error detection, working memory, motor control, and monitoring of competition (see Bush, Luu, & Posner, 2000, for a review). Amongst others, the dorsal part of the ACC has connections to the lateral prefrontal cortex, a brain area that is thought to be involved in the implementation of cognitive control (e.g., Kerns et al., 2004; MacDonald, Cohen, Stenger, & Carter, 2000; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; see also Section 1.4.1).

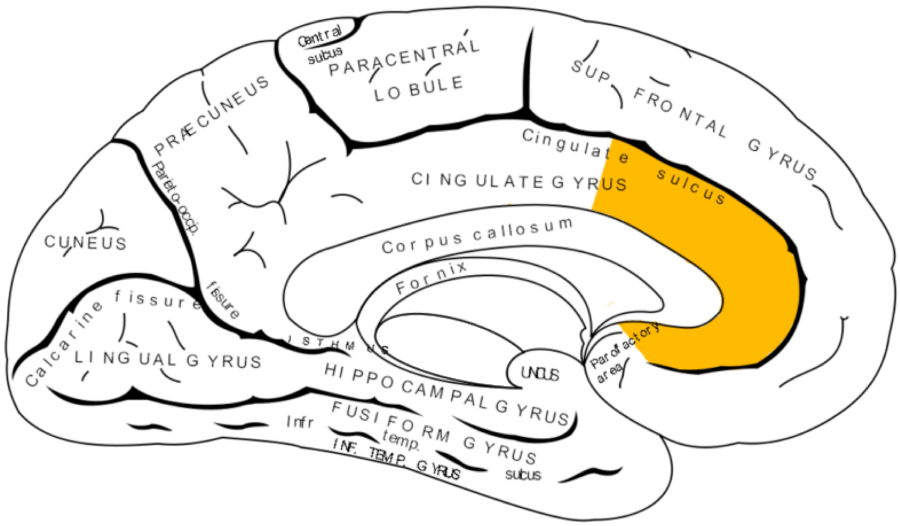

Figure 2: Sagittal view of the brain. The shaded area represents the anterior cingulate cortex (picture source: Brodmann, 2009; adapted by author).

One noteworthy claim of the conflict monitoring theory is that it regards error-related activity in the ACC (see Section 1.4.1 for more details) as a special case of conflict processing and not as being due to the error itself. That is, response conflict associated with error commission is assumed to be responsible for the activation of the ACC. This notion was motivated by the results of a functional

magnetic resonance imaging (fMRI) study by Carter et al. (1998). The authors observed activity in the same area of the ACC not only for errors but also for trials with high response competition. Errors in interference tasks are usually not knowledge errors (i.e. actual mistakes) but slips of action, i.e. errors that the participants are aware of and that occur due to premature responding. During slips in performance the correct response representation receives activation due to continued stimulus processing, while the representation of the actual executed incorrect response is still activated. That is, both incompatible response representations are strongly activated at the same time, leading to high levels of response conflict. This explanation is supported by the fact that these errors are often followed by very quick correction responses (i.e. execution of the correct response immediately after the incorrect response; e.g., Rabbitt, 1966; Ullsperger & von Cramon, 2006), which might be fast due to high correct response activation at the time of incorrect response execution.

Botvinick et al. (2001) have implemented their theory in computational models of a variety of tasks. Predictions of these models have been tested using behavioural, electrophysiological and imaging studies. The results will be presented in the following sections.

1.3 Evaluation and Implementation of Cognitive Control: Behavioural Findings

A large body of research has been concerned with the question of how people maintain task-oriented behaviour in the presence of conflicting information. In the following, I will describe a set of tasks in which participants encounter conflicting information and the consequences of this conflict on their behaviour. Then I will describe two well-known effects, sequential adjustment and post-error slowing, that have been interpreted as adjustments in cognitive control.

1.3.1 Effects of Conflict in Interference Tasks

To investigate assumptions about cognitive control in general and predictions of the conflict monitoring theory in particular, researchers have repeatedly used interference tasks such as the Stroop (Stroop, 1935), the Simon (e.g., Simon, 1990) and the Eriksen flanker task (Eriksen & Eriksen, 1974). These tasks have in common

that they include relevant and irrelevant stimulus aspects, which can potentially activate competing responses, resulting in processing interference or response conflict. In a classic version of the Stroop task, for example, colour words are presented in different ink colours. The participants are required to name the colour of the ink, whilst ignoring the word. Since reading is a highly automated response that is hard to suppress (e.g., LaBerge & Samuels, 1974), the irrelevant stimulus dimension will also be processed to a certain degree. In compatible trials, colour word and ink colour are the same, therefore, activating the same response and making the task very easy. On incompatible trials, on the other hand, colour word and ink colour do not match. In these cases the word interferes with the colour-naming task by activating the incorrect response representation, thereby creating conflict, which leads to higher error rates and slower responses on incompatible than compatible trials (e.g., Alain, McNeely, He, Christensen, & West, 2002; Stroop, 1935; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000; West, 2003; see MacLeod, 1991, for a review).

The Simon task is based on a similar principle; however, in this task conflict does not arise between two different stimulus features but instead between the stimulus and the response location. More specifically, in a typical Simon task trial, a stimulus (e.g., a circle or a square) might be presented on the left or right of a central fixation cross and the participant is required to respond to the shape of the stimulus by pressing a left or right response key, while ignoring the location of the stimulus. An incompatible trial in this task would be, for example, a stimulus on the left of the fixation cross that requires a right hand response. Conversely, stimulus and response location match on compatible trials. Just like in the Stroop task, reaction times and error rates are higher on incompatible than compatible trials (e.g., Burle, Allain, Vidal, & Hasbroucq, 2005; Leuthold & Sommer, 1999; Masaki, Falkenstein, Stürmer, Pinkpank, & Sommer, 2007; Masaki, Murphy, Desjardins, & Segalowitz, 2012; Stürmer, Leuthold, Soetens, Schroter, & Sommer, 2002). This Simon effect can be explained by assuming two separate processing routes (e.g., Kornblum, Hasbroucq, & Osman, 1990; Stürmer et al., 2002). The correct response is deliberately selected via an indirect or conditional route according to task instructions. At the same time, the location of the stimulus automatically activates or primes the spatially corresponding response on a direct or unconditional processing

route. This automatic activation via the direct route facilitates responding on compatible trials but leads to interference on incompatible trials.

In the classical form of the flanker task participants are asked to identify the central letter (target) of a five-letter array by pressing one of two response keys, whilst ignoring the surrounding letters (flankers). On compatible trials target and flankers are identical and, therefore, associated with the same response (e.g., HHHHH and SSSSS). Conversely, on incompatible trials the flankers are associated with the opposite response than the target (e.g., HHSHH and SSHSS). Just like the stimulus location in the Simon task and the colour word in the Stroop task, the flankers are thought to automatically activate the associated response representation, facilitating responding on compatible trials and interfering with responding on incompatible trials, thereby leading to slower reaction times and higher error rates on incompatible compared to compatible trials (e.g., Boksem, Tops, Wester, Meijman, & Lorist, 2006; Davies, Segalowitz, Dywan, & Pailing, 2001; Eriksen & Eriksen, 1974; Scheffers & Coles, 2000). In other words, participants experience response conflict on incompatible trials but not on compatible trials. In other versions of this task arrows or arrowheads in vertical or horizontal alignment were used instead of letters with similar results (e.g., Carbonnell & Falkenstein, 2006; Debener et al., 2005, Endrass, Klawohn, Schuster, & Kathmann, 2008; Ullsperger & von Cramon, 2001, 2006). If anything, compatibility effects can be expected to be enhanced when using arrowheads or arrows instead of letters, because of the inherent spatial information in these stimuli, whereas the association between letter and response side has to be memorized at the beginning of the experiment. When comparing the arrow version and the letter version of the task directly, Wascher, Reinhard, Wauschkuhn, and Verleger (1999) indeed found larger compatibility effects in the arrow version. Furthermore, it has been shown that the compatibility effect can be found for stimulus and response conflict (e.g., van Veen & Carter, 2002; van Veen, Cohen, Botvinick, Stenger, & Carter, 2001; Wendt, Heldmann, Münte, & Kluwe, 2007). More specifically, in a task version with four letters mapped onto two response alternatives, reaction times on compatible trials were faster than reaction times on trials in which the flankers were response-compatible but stimulus-incompatible, i.e. flanker and target letters differed but were associated with the same response (stimulus conflict). Reaction times on response- and stimulus-incompatible trials

were even slower than on just stimulus-incompatible trials (additional effect of response conflict).

Despite conceptual similarities, these interference tasks differ in how the emerging conflict is resolved. In the flanker task, participants can reduce the flanker impact by means of spatial attention, i.e. by narrowing the focus of attention to the target location, whereas this is not possible in the Stroop task, since relevant and irrelevant stimulus aspects are usually presented at the same location (e.g., Magen & Cohen, 2002). Participants might strengthen the colour representation or suppress reading the word instead. In the Simon task, conflict is likely resolved by the suppression of motor activation associated with the direct route (e.g., Stürmer et al., 2002; Stürmer & Leuthold, 2003).

The size of the compatibility effect has been shown to be influenced by several manipulations of the original task versions. Many of these effects can be interpreted in terms of conflict monitoring and cognitive control. For example, a number of studies have demonstrated that the size of the compatibility effect is affected by the frequency of compatible and incompatible trials. When incompatible trials in a flanker task were frequent and participants were assumed to expect high-conflict incompatible trials, interference effects were smaller than when compatible trials were more frequent (e.g., Bartholow et al., 2005; Gratton, Coles, & Donchin, 1992; Purmann, Badde, Luna-Rodriguez, & Wendt, 2011). The same results have been found for the Stroop task (e.g., Carter et al., 2000; Tillman & Wiens, 2011). This effect can be interpreted as a manipulation of the overall level of control. When incompatible trials are frequent, the level of control can be expected to be enhanced, which leads to improved, i.e. faster and more accurate, performance on incompatible trials and, therefore, smaller interference effects. Conversely, when incompatible trials are rare, control might be relaxed, leading to a larger reliance on the facilitatory effect of the irrelevant task dimension on compatible trials and, therefore, to higher interference on incompatible trials. In the Simon task, the compatibility effect has been shown to not only be reduced but even reversed when incompatible trials were frequent (e.g., Hommel, 1994; Stürmer et al., 2002). It is possible that participants use the location of the stimulus strategically, i.e. when incompatible trials are frequent the response side opposite to the stimulus is more likely to be correct and might, therefore, receive additional activation via the indirect route (e.g., Stürmer et

al., 2002). Note that manipulating the overall frequency of compatible and incompatible trials also changes the frequency of trial sequences. For example, when incompatible trials are frequent, an incompatible trial is more likely to be followed by another incompatible than by a compatible trial. Effects of trial sequence, discussed in more detail in Section 1.3.2.1, can therefore confound effects of overall frequency.

In some studies, a neutral condition was included in addition to compatible and incompatible conditions. On neutral trials, the irrelevant stimulus aspect is not associated with any of the response alternatives. Examples for neutral stimuli are a third kind of letter or a symbol in the flanker task, which only serves as flanker but never as target, or colour-neutral words or letter strings in the Stroop task. Neutral trials provide an interesting comparison condition, because they allow the dissociation of the interfering influence of the irrelevant stimulus aspect on incompatible trials and its facilitating influence on compatible trials. In other words, on neutral trials, there is no conflict on the response level, since neutral flankers in the flanker task or non-colour words or letter strings in the Stroop task are not associated with either response. However, the additional response facilitating input from the compatible flankers or the compatible colour word is also not present. The conflict monitoring account, therefore, predicts intermediate reaction times on neutral trials. Empirical findings have mostly been in line with this prediction. Reaction times on neutral trials have usually been found to lie between compatible and incompatible reaction times in the flanker task (e.g., Heil, Osman, Wiegelmann, Rolke, & Hennighausen, 2000; Kopp, Mattler, Goertz, & Rist, 1996; Kopp, Rist, & Mattler, 1996; Mattler, 2003; Van't Ent, 2002; Willemssen, Hoormann, Hohnsbein, & Falkenstein, 2004) and in the Stroop task (e.g., Erickson et al., 2004). However, in some studies reaction times did not differ significantly between compatible and neutral trials (flanker task: e.g., Wild-Wall, Falkenstein, & Hohnsbein, 2008; Stroop task: e.g., West & Alain, 2000). In the following, the difference in behavioural measures between compatible and neutral trials will be referred to as facilitation effect and the difference between neutral and incompatible trials as interference effect.

Another way to manipulate conflict in interferences tasks is the manipulation of stimulus onset asynchrony (SOA), i.e. the interval between the onset of irrelevant

and relevant stimulus aspects.[1] Using a flanker task, Wascher et al. (1999) and Willemssen et al. (2004) found that the compatibility effect was enhanced when the flankers preceded the target by 100 ms compared to a condition with simultaneous flanker and target onset (SOA 0 ms). Increasing the SOA further, did lead to a reduction of the compatibility effect.[2] Flanker effects were still present at an SOA of 400 ms (Willemssen et al., 2004) but not at an SOA of 500 ms (Wascher et al., 1999). This effect can be explained in terms of the conflict monitoring theory. When the flankers precede the target by a short time period, the response that is associated with the flankers is activated before the target-associated response is activated. On trials with long SOAs, there is sufficient time to suppress this preactivation. In the 100 ms SOA, on the other hand, there is not enough time for this kind of suppression; flanker-associated response activity at the time of target-associated response activation is, therefore, larger than in a condition with simultaneous flanker-target onset, leading to enhance conflict and larger compatibility effects. For this reason, the SOA effect can be used to manipulate conflict strength in the flanker task (see also Chapter 4). Effects of SOA are less consistent in the Stroop task. Whereas facilitation effects have been consistently found to be larger when the onset of the colour word preceded the onset of the colour, here background colour, which had to be named (e.g., Appelbaum, Meyerhoff, & Woldorff, 2009; Coderre, Conklin, & van Heuven, 2011; Glaser & Glaser, 1982), interference was sometimes found to be largest for short SOAs (e.g., Appelbaum et al., 2009) and sometimes for simultaneous word-colour onset (e.g., Coderre et al., 2011; Glaser & Glaser, 1982). The conflict monitoring theory cannot account for this asymmetry.

[1] The term SOA has not always been used consistently. Sometimes it refers to the time between the stimulus on one trial and on the next trial; sometimes it refers to the interval between the onset of a cue and the target stimulus display (e.g., Eriksen & Hoffman, 1973); and sometimes it is used to describe the interval between the onsets of different aspects of a stimulus on the same trial (e.g., flankers and target). In this thesis the term SOA always describes the last.

[2] Mattler (2003) also used three different SOAs in a flanker task but did not do a statistical analysis to compare the size of the flanker effects. However, his figures also show the numerically largest effect on reaction times at an SOA 100 ms.

Yet another way to manipulate conflict strength, specifically in the flanker task, is to change the distance between the target and the flankers. If the distance between flankers and target is increased, the flanker influence should be reduced, since a less narrow focus of attention on the target location is needed to prevent the processing of flanker information. Empirical findings are in line with this prediction (e.g., Danielmeier, Wessel, Steinhauser, & Ullsperger, 2009; Kopp, Rist, et al., 1996; Sullivan, 1999). All of these studies showed reduced flanker effects when the distance between target and flankers was increased compared to a condition with narrower spacing.

1.3.2 Control Adjustment Effects

In this section, I will describe how the experience of conflict affects subsequent control adjustments. Braver and colleagues (e.g., Braver, 2012; De Pisapia & Braver, 2006) distinguished between two types of control in their dual mechanisms of control framework: proactive and reactive control. Proactive control is a sustained kind of control, which means that goal-relevant information is kept active to prevent future problems before they occur. Reactive control, on the other hand, describes a transient upregulation of control that is called into action after a problem has occurred and that is supposed to correct the problem and prevent further difficulties. Effects of speed-accuracy instructions can be seen as a manipulation of proactive control. More specifically, when participants are instructed to be as accurate as possible in a task, they will probably have a higher overall level of control than when they are instructed to be fast. Another example of a manipulation of proactive control is the trial type frequency effect described in the previous section. In this section, I will focus on two examples of reactive control: the sequential adjustment effect and post-error slowing. Both of these effects occur after the experience of conflict (in form of incompatible trials and errors, respectively). Although both of these effects have been described in terms of control adjustments, there has been some controversy about whether these effects can be better described in terms of other phenomena (see below).

1.3.2.1 Sequential Adjustment Effect

The term sequential adjustment effect describes the common finding that compatibility effects on reaction times and error rates in interference tasks are reduced following incompatible compared to compatible trials. This effect has been found consistently for the Stroop (e.g., Monti, Weintraub, & Egner, 2010; West & Moore, 2005), the Simon (e.g., Burle et al., 2005; Stürmer & Leuthold, 2003; Stürmer et al., 2002), and the flanker task (e.g., Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Gratton et al., 1992).[3] *Figure 3* shows an illustration of this effect, which has also been labelled Gratton effect after the author who first described it for the flanker task. In the following, I will use the terms sequential adjustment effect and Gratton effect interchangeably.

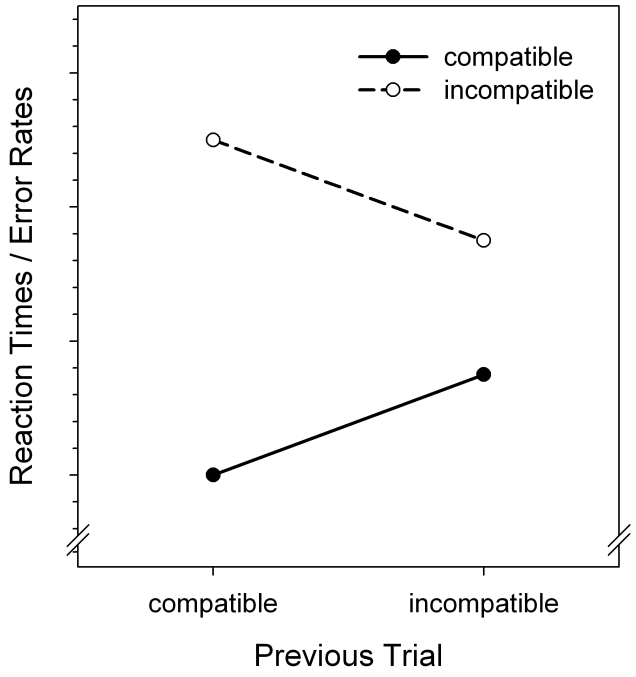

Figure 3: Illustration of the Gratton effect: The difference in reaction time and error rates between compatible and incompatible trials is larger following compatible than following incompatible trials.

[3] There seem to be differences in the extent of the sequential adjustment effect between these tasks. Whereas compatibility effects are usually still present following incompatible trials in the flanker and the Stroop tasks, compatibility effects have sometimes been shown to completely disappear or even reverse after incompatible trials in the Simon task (e.g., Stürmer & Leuthold, 2003; Stürmer et al., 2002).

The size of the Gratton effect can be quantified by subtracting the flanker effect following incompatible trials from the flanker effect following compatible trial: ((c-ic – c-c) – (ic-ic – ic-c)) in which c stands for compatible, ic for incompatible trials, current trial underlined (i.e. c-ic means the current incompatible trial was preceded by a compatible trial). The resulting number can be used as an indicator of the size of the adjustment, for example when comparing different experimental conditions or groups of participants.

In conflict monitoring terminology, the Gratton effect has been explained by a shift in attentional control after the experience of conflict (e.g., Botvinick et al., 2001). More precisely, after the experience of conflict on incompatible trials, participants increase their levels of control by shifting attention towards the relevant aspect of the stimulus and away from irrelevant aspects to avoid further conflict. After the experience of non-conflicting compatible trials, on the other hand, control will be relaxed. *Figure 4* illustrates this point using the example of an incompatible trial in the flanker task (example stimulus: HSH). The left half of the figure shows the situation following a compatible trial. In compliance with the task instructions, spatial attention is focused slightly more on the central position than on the outer positions (indicated by the slightly larger centre circle compared to the left and right circles in the spatial attention layer); however, flankers are processed as well leading to high conflict between the two response alternatives. Following incompatible trials (as illustrated on the right half of the figure), on the other hand, participants focus their attention more on the central letter. Consequently, the target-associated activation in the response layer increases, whereas the flanker-associated activation decreases. As a result, conflict due to incompatible flanker influence is reduced.

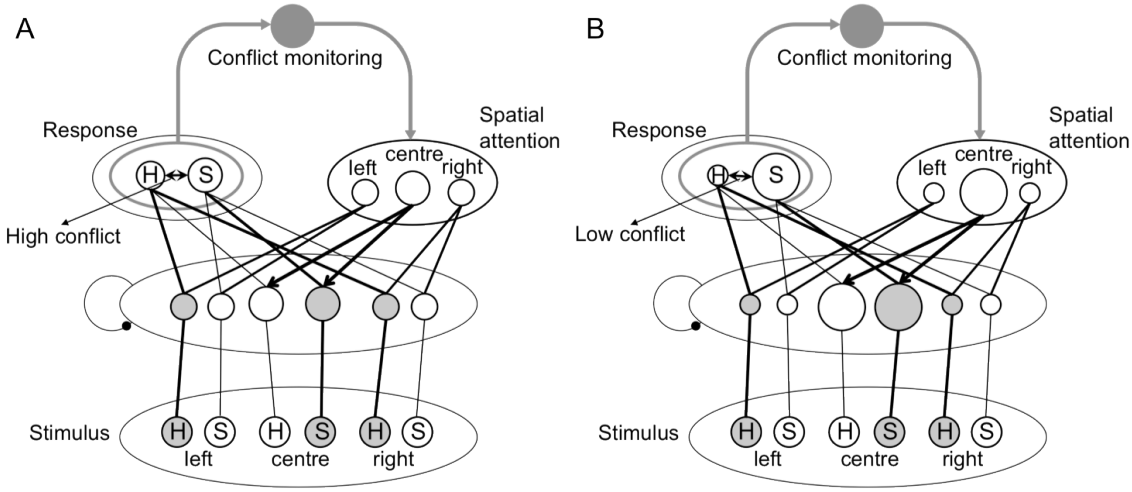

Figure 4: Conflict monitoring theory explanation of the Gratton effect using the example of an incompatible trial (example stimulus: HSH) in the flanker task. The size of the circles represents the level of activation. Conflict is larger following compatible trials (A) than following incompatible trials (B).

This control explanation of the sequential adjustment effect has been challenged. More specifically, Mayr, Awh, and Laurey (2003) argued that a simple memory effect, repetition priming, could explain this pattern of results.[4] Using an arrowhead version of the flanker task, the authors showed that the effect was only present for response repetitions but not for response alternations. For response repetitions, a repetition of the compatibility level (c-c and ic-ic) entails an exact repetition of all stimulus features. Repetition priming can therefore explain the faster and more accurate responses in these conditions compared to the same trial types preceded by the opposite compatibility level (ic-c and c-ic). For response alternations, on the other hand, there are no exact stimulus repetitions. The repetition priming account, therefore, predicts similar compatibility effects following

[4] The repetition priming explanation can be regarded as a special case of feature integration (Hommel, Proctor, & Vu, 2004). According to this view, stimulus and response features are combined into one episodic memory representation. Complete alternations or complete repetitions of all features are therefore processed easily, whereas partial repetitions (for example a repetition of the flankers along with an alternation of the target in the flanker task) are processed more slowly (see Egner, 2007, for a review).

compatible and incompatible trials in this case. However, the control adjustment hypothesis would still predict increased control and a reduced compatibility effect on trials following incompatible compared to compatible trials. The absence of the Gratton effect for response alternations, therefore, argues against the control adjustment explanation.

A number of studies found support for the repetition priming account. For example, Wendt et al. (2007) also found that the Gratton effect in a version of the flanker task disappeared when exact stimulus-response repetitions were excluded from the analysis. Furthermore, Burle et al. (2005) showed that the Gratton effect in the Simon task did not depend on conflict strength, measured as simultaneous electromyographic (EMG) activation of both response hands, on the previous trial. However, other studies showed that the Gratton effect in interference tasks was still present, when exact repetitions were excluded (flanker task: e.g., Clayson & Larson, 2011; Freitas, Banai, & Clark, 2009; Stroop task: e.g., Kerns et al., 2004). In addition, Ullsperger, Bylsma, and Botvinick (2005), using a flanker task, and Stürmer et al. (2002), using a Simon task, showed that sequential adjustment effects were not only present for response repetitions but also for response alternations.[5] The Gratton effect can, therefore, not be fully explained by repetition priming.

Davelaar and Stevens (2009) proposed a compromise between both competing accounts by suggesting that the priming effect is modulated by conflict. More specifically, using a flanker task with additional neutral trials, the authors found that the Gratton effect was not present for response alternations, therefore, supporting the repetition priming account. However, the size of the priming effect did depend on the compatibility of the previous trial. Priming was larger when two incompatible trials followed each other than when two compatible trials followed each other. The authors explained this pattern of results by proposing that the association between stimulus and response is strengthened to a great extent after a correct response on a high-conflict incompatible trial, leading to a large priming

[5] Stürmer et al. (2002) also found effects of repetition priming, i.e. exact repetition trials were processed faster than trials that included any kind of alternation. However, repetition priming could not fully account for sequential adjustment effects.

effect, whereas the association is strengthened to a lesser extent after correct low-conflict compatible trials, leading to a smaller priming effect.

In conclusion, repetition priming seems to play an important role in the emergence of sequential adjustment effects, although it cannot explain the entirety of the research results. Adjustments in control likely also contribute towards the Gratton effect.

1.3.2.2 Error Speed-Up and Post-Error Slowing

Errors in interference and other simple reaction time tasks are usually due to premature responding, i.e. participants do have a representation of what the correct response would have been but responded before stimulus analysis was completed (e.g., Botvinick et al., 2001, Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988; see also Section 1.2). This proposition is in line with results showing that pre-error and error responses are usually faster than correct responses (pre-error/error speed-up effect; e.g., Dudschig & Jentzsch, 2009; Gehring & Fencsik, 2001; Rabbitt, 1966) and sometimes followed by very fast spontaneous correction responses (e.g., Fiehler, Ullsperger, & von Cramon, 2005; Rabbitt & Rodgers, 1977). Correct responses following errors (post-error trials), on the other hand, have repeatedly been found to be slower than correct responses following another correct response (post-correct trials; e.g., Brewer & Smith, 1984; Jentzsch & Dudschig, 2009; Laming, 1968; Rabbitt; 1966). This finding has been labelled the post-error slowing effect. Frequently the slowing in reaction times is accompanied by an increase in accuracy (e.g., Brewer & Smith, 1984; Hester, Barre, Mattingley, Foxe, & Garavan, 2007; Jentzsch & Leuthold, 2006; Laming, 1979), although this has not always been observed (e.g., Hajcak & Simons, 2008; Nunez Castellar, Kuhn, Fias, & Notebaert, 2010). Hajcak, McDonald, and Simons (2003) noticed, however, that the lack of a significant post-error accuracy increase might be due to a ceiling effect, since accuracy in the used tasks is usually at a high level. Despite the lack of a significant accuracy effect, the authors observed a positive correlation between post-error slowing and post-error accuracy.

Some evidence points towards the existence of a link between error awareness and post-error slowing. For example, Nieuwenhuis, Ridderinkhof, Blom,

Band, and Kok (2001) did find slowing for perceived but not for unperceived errors.[6] Hewig, Coles, Trippe, Hecht, and Miltner (2011) showed that post-error slowing was related to the subjective but not to objective response correctness. Furthermore, Endrass et al. (2008) observed slowing only for full but not for partial errors, the latter of which were likely not perceived as errors. It has also been shown that post-error slowing is larger when accuracy is emphasized over speed (e.g., Jentzsch & Leuthold, 2006).

Traditionally, the post-error slowing effect has been interpreted as a strategic adaptation of a response criterion to more conservative levels in order to prevent further errors (speed-accuracy tradeoff; e.g., Brewer & Smith, 1984; Jentzsch & Leuthold, 2006; Laming, 1968; Saunders & Jentzsch, in press). This interpretation is in line with the conflict monitoring theory, which predicts an increase in cognitive control following conflict associated with an error (e.g., Botvinick et al., 2001). However, Notebaert and colleagues recently proposed an alternative hypothesis, the orienting account of post-error slowing (Notebaert et al., 2009). The orienting account is based on the observation that post-error slowing is not always accompanied by an increase in accuracy. The authors propose that, rather than an adjustment in cognitive control, post-error slowing might represent an attentional orienting response to the rare and, therefore, unexpected occurrence of an error and a subsequent reorientation to the task. This interpretation is supported by data showing post-correct slowing in conditions when errors are more frequent than correct responses (Notebaert et al., 2009; Nunez Castellar et al., 2010). However, other data contradict this account. For example, Vocat, Pourtois, and Vuilleumier (2008) observed post-error slowing in a task in which errors and correct responses were equally frequent. Furthermore, the orienting account cannot explain why post-error slowing is sometimes accompanied by an accuracy increase (see above). Further evidence for the criterion adjustment approach was provided by a modelling study by Dutilh et al. (2012), who showed that post-error slowing in their data set could be

[6] Endrass, Franke, and Kathmann (2005) did not find a significant interaction between slowing and error awareness in a similar paradigm. However, the authors compared post-error trials to errors instead of post-correct trials, which might have masked potential effects.

modelled well by assuming increased response caution. Distraction of attention as assumed in the orienting account, on the other hand, did not contribute to the effect.

Jentzsch and Dudschig (2009) showed that the mechanisms contributing to the post-error slowing effect might depend on the time between a response and the subsequent stimulus onset. More specifically, the authors showed that slowing was larger for short response-stimulus intervals (RSIs; 50 ms or 100 ms) and smaller but still significant for longer RSIs (1000 ms). Post-error accuracy, on the other hand, was smaller than post-correct accuracy for short RSIs but numerically larger for long RSIs (see also Dudschig & Jentzsch, 2009). These results indicate that the response criterion adjustment account might only be applicable when participants have enough time between an error and the next stimulus. When time is too short, other mechanisms like error monitoring, attention orienting, or the persistence of the problem that caused the error (e.g., Gehring, Goss, Coles, Meyer, & Donchin, 1993; Gehring & Fencsik, 2001) might be responsible for post-error slowing. However, it seems likely that, at least at long RSIs, post-error slowing is due to a strategic adjustment in control.

1.4 Evaluation and Implementation of Cognitive Control: Neurophysiological Findings

1.4.1 Imaging Studies

Several studies have investigated the neural correlates of the evaluation and implementation of cognitive control using fMRI. One brain region that has been repeatedly found to be involved in the processing of errors is the anterior cingulate cortex (ACC; e.g., Braver, Barch, Gray, Molfese, & Snyder, 2001; Carter et al., 1998; Garavan, Ross, Murphy, Roche, & Stein, 2002; Hester et al., 2007; Kerns et al., 2004; Kiehl, Liddle, & Hopfinger, 2000; Ullsperger & von Cramon, 2001), a finding that is also in line with results from research using event-related potentials (see next section). Carter et al. (1998) found that this region was not only involved in error processing but also in the processing of response competition, which has led to the formulation of the conflict monitoring theory of ACC function (Botvinick et al., 2001; see also Section 1.2). Several studies support this claim. For example, ACC activity has been found to be larger for incompatible than compatible trials in the flanker and Stroop tasks (e.g., Botvinick et al., 1999; MacDonald et al., 2000) and

larger for the less frequent (and therefore higher conflict) of two different response options across several tasks (Braver et al., 2001). Furthermore, van Veen et al. (2001) showed that ACC activity was increased for response- and stimulus-incompatible but not for just stimulus-incompatible trials compared to compatible trials. Therefore, the authors concluded that the ACC is responsive to response conflict. Interestingly, Carter et al. (2000) observed that the compatibility effect in ACC activity was only present when high-conflict incompatible trials were rare and the overall level of control, therefore, expected to be low. When incompatible trials were frequent and the overall level of control, therefore, probably higher, the compatibility effect in ACC activity disappeared. Furthermore, it has been observed that the Gratton effect was mirrored in ACC activity (e.g., Botvinick et al., 1999; Kerns et al., 2004); that is, ACC activation associated with incompatible trials was smaller following another incompatible trial than following a compatible trial. Kerns et al. (2004) also found a correlation indicating larger adjustment in individuals with stronger ACC activation. However, it remains controversial whether conflict and error processing are performed by the same brain areas.[7] Some studies failed to find conflict-related ACC activity and found areas in the supplementary motor area (SMA) or pre-SMA to be related to conflict instead (e.g., Garavan, Ross, Kaufman, & Stein, 2003; Kiehl et al., 2000; Ullsperger & von Cramon 2001). Others found evidence for some overlap between error and conflict processing within the ACC but also additional specifically error-related activity in the rostral ACC (e.g., Mathalon, Whitfield, & Ford, 2003). Furthermore, Erickson et al. (2004) found that ACC activity did not reflect the size of the compatibility effect in behaviour, when comparing the first and the second half of their experiment.

[7] This question has not only been addressed in imaging studies but also in a study using time-frequency analysis of electroencephalographic data (Nigbur, Ivanova, & Stürmer, 2011). The authors found that theta power (i.e. activity in the frequency band from 4 Hz to 7 Hz) differentiated between different kinds of conflicts (e.g., between conflicts in the Simon and in the flanker task). In the source analysis, theta power dipoles were more ventral for stimulus conflict than for response- and error-related conflicts, pointing towards a specialization of areas within the frontal cortex for different kinds of conflict.

If ACC activation indeed reflects the detection of conflict that is associated with error commission and post-error slowing is an adjustment in control triggered by this conflict (see 1.3.2.2), ACC activation should predict subsequent post-error slowing. In line with this hypothesis, Garavan et al. (2002) and Kerns et al. (2004) found that errors that were followed by more response slowing were associated with larger ACC activity. Hester et al. (2007), on the other hand, did find ACC activity following an error but not during an error to be associated with post-error slowing. This research question has been investigated more extensively in studies using event-related potentials (see next section).

Whereas the ACC has been thought to be responsible for evaluative aspects of control, control implementation (i.e. the regulative aspect of control) has been associated with the dorsolateral prefrontal cortex (DLPFC). MacDonald et al. (2000) observed greater DLPFC activity under high-control than under low-control requirements in a task-switching version of the Stroop task. More specifically, participants received instructions on a trial-by-trial basis to either name the colour or read the word of a subsequent Stroop stimulus. Since colour naming is more difficult than the highly automated word reading task, the colour naming instruction should lead to an increase in control in preparation for the task. In line with this prediction, instruction-related DLPFC activity was found to be larger for the colour naming than for the word reading task. Furthermore, participants with larger DLPFC activity showed smaller compatibility effects. Kerns et al. (2004) confirmed this interpretation by showing that larger adjustments in both the Gratton effect and in post-error slowing coincided with greater DLPFC activity. In addition, Erickson et al. (2004) found a positive association between ACC activity and DLPFC activity, at least in the first half of their experiment, which is in line with the interpretation that conflict detected by the ACC leads to control implementation in the DLPFC. This association disappeared in the second half of the experiment; possibly because control regulation within the DLPFC improved with practice, which reduced the need for evaluative control signals from the ACC.

1.4.2 Error- and Conflict-Associated Event-Related Potentials

Event-related potentials (ERPs) have been an important method in the investigation of cognitive control. In Chapter 2, I will explain in more detail what an ERP is and

how it is measured. Here, I will focus on the contribution that ERP research has made in the field of cognitive control.

A large part of the ERP research on cognitive control originates in the investigation of error processing. Especially two error-related ERP components have been investigated thoroughly: the error negativity (Ne; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991), also called error-related negativity (ERN; Gehring et al., 1993), and the error positivity (Pe; e.g., Falkenstein et al., 1991; Leuthold & Sommer, 1999; Nieuwenhuis et al., 2001). The Ne/ERN, depicted in *Figure 5*, is a negative peak in the response-locked ERP with an onset around the time of the commission of an incorrect response and maximum amplitude about 50 ms to 100 ms thereafter. It has a fronto-central distribution, with a maximum at electrode Cz or FCz; and its source has repeatedly been localized within the vicinity of the ACC (e.g., Alain et al., 2002; Dehaene, Posner, & Tucker, 1994; Endrass et al., 2008; Masaki et al., 2012; Mathewson, Dywan, & Segalowitz, 2005; O'Connell, et al., 2007; van Veen & Carter, 2002; Vocat et al., 2008), although a source in the supplementary motor area has also been considered possible (e.g., Dehaene et al., 1994; Herrmann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004).

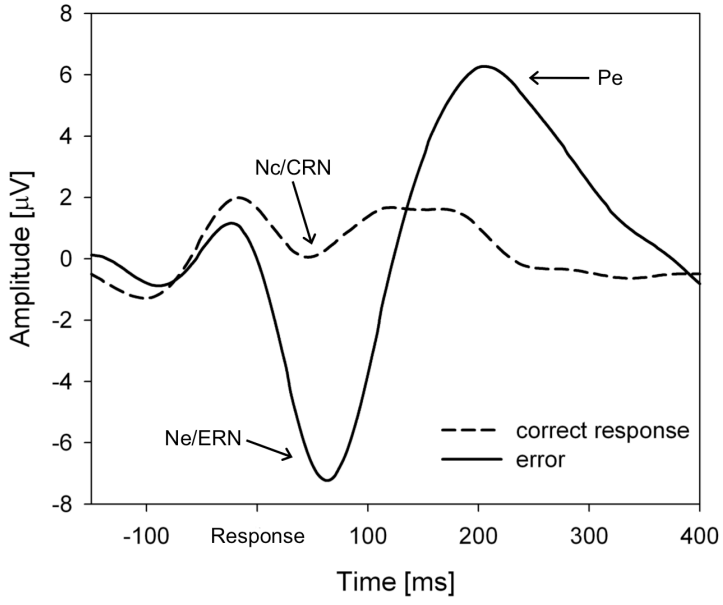

Figure 5: The Ne/ERN is a negative peak in the ERP with maximum amplitude about 50 ms to 100 ms after the onset of an incorrect response. The Nc/CRN is a much smaller peak at the same latency that occurs on correct response trials. The Ne/ERN is followed by a positive peak - the Pe.

Originally, the Ne/ERN was thought to be elicited by a comparison process between the representations of the actually executed response and the required response (e.g., Bernstein, Scheffers, & Coles, 1995; Falkenstein et al., 1991). When this comparison process reveals a mismatch between the two representations, the error is detected and the Ne/ERN signal generated (e.g., Coles, Scheffers, & Holroyd, 2001). The conflict monitoring theory, on the other hand, proposed that the Ne/ERN is not elicited by an explicit response comparison process but rather by the simultaneous activation of incompatible response representations. The resulting conflict between these simultaneous activations is thought to be sufficient to elicit the Ne/ERN; an actual comparison process between response representations is not needed. Error detection is therefore not necessary for the emergence of an Ne/ERN. Yet another theory proposes that the Ne/ERN reflects a reinforcement learning signal that is sent to the ACC via the mesencephalic dopamine system, indicating that an outcome of an action is worse than expected (Holroyd & Coles, 2002). According to this view, the commission of an error leads to a phasic decrease in the activity of the mesencephalic dopamine system, which in turn leads to a disinhibition of neurons within the ACC where the Ne/ERN is elicited.[8]

The amplitude of the Ne/ERN is larger when errors are rare than when errors occur frequently (e.g., Hajcak et al., 2003; Hajcak, McDonald, & Simons, 2004; Herrmann et al., 2004; Holroyd & Coles, 2002). Furthermore, the Ne/ERN is larger when participants are instructed to emphasize accuracy over speed than vice versa (e.g., Arbel & Donchin, 2009; Gehring et al., 1993; Themanson, Hillman, et al., 2008; Themanson, Pontifex, & Hillman, 2008). This effect has been explained by an increased salience of errors when accuracy is important (e.g., Gehring et al., 1993). It is also in agreement with predictions of the reinforcement learning theory, since

[8] Botvinick (2007) extended the conflict monitoring theory to make it compatible with decision making accounts of ACC function, such as the reinforcement learning theory, by proposing that conflict can function as an aversive teaching signal that people learn to avoid. Nevertheless, both theories still differ in where they locate the actual decision making process (within the ACC or elsewhere; cf. Botvinick, 2007) and in how they explain the generation of the Ne/ERN (transmission of reinforcement learning signal vs. manifestation of conflict; cf. Holroyd & Coles, 2002).

errors are less frequent and therefore probably more unexpected in task versions in which accuracy is emphasized than in speeded versions. As Yeung, Botvinick, and Cohen (2004) pointed out, the effect could also be explained by a shift in attentional focus. If accuracy is of subjective importance, participants might focus more on the target and ignore distracting information (e.g., the flankers in a flanker task). Since errors occur mostly on incompatible trials, this focus on the relevant task aspect leads to a faster build-up of correct activity following the error, while the incorrect response representation is still active, and, therefore, to larger conflict.

There is some debate over the question whether the amplitude of the Ne/ERN is influenced by the participants' awareness of error commission. Whereas some studies have shown a reduction of Ne/ERN amplitude for unaware compared to aware errors (e.g., Hewig et al., 2011; Scheffers & Coles, 2000), others did find no difference (e.g., Endrass et al., 2005; Nieuwenhuis et al., 2001; O'Connell et al., 2007). Differences between the tasks that were used in these studies might explain the diverging results. For example, Hewig et al. (2011) as well as Scheffers and Coles (2000) used very difficult tasks in which a large portion of unaware errors might have been due to mistakes rather than slips of action, i.e. errors occurred because participants did not have a representation of the correct response and not due to premature responding. Endrass et al. (2005) and Nieuwenhuis et al. (2001), on the other hand, used tasks in which participants were required to carry out specific eye movements. It is, therefore, possible that unaware errors in these two studies represent instances, in which participants did have a representation of the required correct response but were not aware of the actually executed response. The presence of a representation of the correct response might be a necessary precondition for the occurrence of an Ne/ERN, whereas conscious awareness of the actual executed response is not needed. This hypothesis cannot explain the Ne/ERN for unaware errors in the study by O'Connell et al. (2007) though. In this study, participants were required to respond to compatible Stroop stimuli and to withhold their response for incompatible stimuli or when the same stimulus was repeated. When participants committed an error, they had to indicate this on the next trial instead of responding to the task. Difficulties with this awareness reporting procedure might have led to a number of aware errors counted as unaware, which would explain the Ne/ERN on these trials.

The conflict monitoring interpretation of the Ne/ERN has been challenged by studies measuring conflict directly as simultaneous activation of incompatible responses alternatives via force keys or EMG activity. For example, Carbonnell and Falkenstein (2006) investigated errors with co-activation of both response alternatives in a flanker task. The authors compared full errors (i.e. trials with superthreshold incorrect activation) and partial errors (i.e. trials with subthreshold incorrect activation) and found that full errors were associated with more conflict. The Ne/ERN amplitude, on the other hand, did not differ between full and partial errors. Furthermore, Masaki et al. (2007) compared an easy (high stimulus discriminability) and a hard (low stimulus discriminability) version of the Simon task. As the authors predicted, error-associated conflict, measured as the product of correct and incorrect EMG activation in an error trial, was larger in the easy than in the hard version.[9] Ne/ERN amplitude, however, did not differ between conditions. Finally, Burle, Roger, Allain, Vidal, and Hasbroucq (2008) sorted error trials in a flanker task by the amount of conflict (measured as temporal overlap between correct and incorrect EMG activation) and found that Ne/ERN amplitude decreased rather than increased with increasing conflict.

Danielmeier et al. (2009), on the other hand, did find support for the conflict monitoring view. Using a version of the Eriksen flanker task, the authors manipulated conflict by varying the distance between target and flanker arrows. As mentioned in Section 1.3.1, compatibility effects were larger when the flankers were close than when they were far from the target. Their computational model of the conflict monitoring theory predicted a larger Ne/ERN for the low-conflict condition with the far flankers than for the high-conflict condition with close flankers due to differences in post-error correct activation. More specifically, when the distance between target and flankers is increased, flanker-associated activation immediately after error commission will be smaller than when the flankers are close. Since most errors occur on incompatible trials, reduced flanker processing after the error increases correct activation due to continued target influence while the incorrect

[9] When stimulus alternatives are hard to discriminate, the correct response should receive less activation on an error trial than when stimulus alternatives are easy to discriminate. Incorrect response activation should be similar in the hard and the easy version.

response is still active; conflict should therefore be larger when correct activation is large. This prediction was confirmed in their experimental data.

In some studies a small negativity occurring in the same time window as the Ne/ERN has been observed on correct trials (e.g., Bartholow et al., 2005; Bonnefond, Doignon-Camus, Hoeft, & Dufour, 2011; Coles et al., 2001; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Kray, Eppinger, & Mecklinger, 2005; Vidal et al., 2000). This negativity, depicted in *Figure 5*, has sometimes been called correct negativity (Nc) or correct-related negativity (CRN**)**. This finding has provided a challenge to the error detection account of the Ne/ERN, since this activity seems to be less specific to errors than previously assumed (e.g., Vidal et al., 2000). However, Coles et al. (2001) argued that the Nc/CRN could be explained by error processing on correct trials, for example when a participant considers a late response as an error (e.g., Luu, Flaisch, & Tucker, 2000), when participants are unsure about their response accuracy (e.g., Scheffers & Coles, 2000), or when subthreshold activation of the incorrect response occurs (e.g., Burle et al., 2005). Furthermore, Nc/CRN activity could also be due to contamination by stimulus-associated activity when reaction time variability is low (Coles et al., 2001). Following this argumentation, both the error detection theory and the reinforcement learning theory can explain the occurrence of the Nc/CRN.

Some researchers assumed that the conflict monitoring theory would predict that the Nc/CRN reflects conflict on correct trials and tested this assumption by investigating Nc/CRN amplitude under different levels of conflict (e.g., Bartholow et al., 2005; Falkenstein et al., 2000). Both of these studies failed to find an influence of conflict on this component and interpreted this as evidence against the conflict monitoring theory. However, proponents of the conflict monitoring theory argued that their theory does predict a different time course of conflict on correct compared to incorrect trials. Conflict on correct trials is actually expected to be maximal just before the response and not thereafter (e.g., Botvinick et al., 2001; Yeung et al., 2004). For example, in an incompatible trial in the flanker task, flanker- and target-associated activation start to rise after stimulus presentation. On correct trials, target-associated activation is larger than flanker-associated activation at the time of the response. Conflict between these two response representations should not rise any further at this point, since the response was correct and there is no need for continued

stimulus processing that would cause the response activations to increase. Conflict on correct trials might be reflected in a different component of the ERP, i.e. the N2 (see below; Yeung et al., 2004). Nevertheless, the conflict monitoring theory cannot explain the Nc/CRN any better than the error detection or the reinforcement learning theory.

Another component related to the Ne/ERN is the feedback-related negativity (FRN)[10], first described by Miltner, Braun, and Coles (1997). The FRN is a negative peak with a fronto-central distribution, a possible source in the ACC or SMA (Miltner et al., 1997) and a maximum amplitude about 250 ms after participants received feedback that they have committed an error or that their behaviour has resulted in negative consequences (e.g., Holroyd, Hajcak, & Larsen, 2006; Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003; Miltner et al., 1997). The fact that negative feedback does elicit an Ne/ERN-like component has played an important role in the formulation of the reinforcement learning theory (Holroyd & Coles, 2002). This theory is based on the assumption that the FRN represents the same underlying processes as the Ne/ERN. Since the reinforcement learning signal is assumed to be elicited when the system first notices that an outcome is worst than expected, an Ne/ERN-like signal should emerge when a person first notices an error, i.e. after the response if it is identifiable as an error or after error feedback if the response cannot immediately be identified as an error (first-indicator hypothesis). Stahl (2010) tested this prediction and showed that the FRN only occurred when the feedback was informative, i.e. when participants were not able to internally detect the error. Errors that were internally detectable, on the other hand, did elicit an Ne/ERN but no FRN.

The Ne/ERN is usually followed by a positive peak - the error positivity (Pe; see *Figure 5*). The Pe reaches its peak amplitude 200 ms to 500 ms after the commission of an error. Some researchers reported a fronto-central distribution of the Pe similar to the Ne/ERN (e.g., Boksem et al., 2006; Davies et al., 2001; Hajcak et al., 2004; Herrmann et al., 2004; Vidal et al., 2000); others found a more posterior distribution (e.g., Band & Kok, 2000; Bartholow et al., 2005; Endrass et al., 2005, Falkenstein et al., 1991; Themanson, Hillman, et al., 2008). Similarly, source

[10] Both Nc/CRN and FRN have sometimes been referred to as Ne/ERN without any further distinctions. However, in this thesis the term Ne/ERN always refers to response-locked activity on incorrect trials.

localizations of the Pe have also provided inconsistent results. The Pe has been explained by a single dipole within the ACC at a more posterior location than for the Ne/ERN (e.g., Herrmann et al., 2004; Vocat et al., 2008; West & Travers, 2008) or by two dipoles, one at the same location as for the Ne/ERN and another one posterior to that point (e.g., Ladouceur, Dahl, & Carter, 2007). Just like the Ne/ERN the Pe has been found to be reduced under speed compared to accuracy instructions (e.g., Themanson, Hillman, et al., 2008).

It has been proposed that the Pe might reflect an affective reaction to the occurrence of an error (e.g., Falkenstein et al., 2000; van Veen & Carter, 2002). However, Overbeek, Nieuwenhuis, and Ridderinkhof (2005) conclude in their review that there is only little support for this hypothesis. Furthermore, one study that compared participants with high and low negative affect found smaller, rather than increased, Pe amplitudes for the high affect group (Hajcak et al., 2003). Another hypothesis of the functional significance of the Pe, that it represents the conscious recognition of an error, has received more support. Several studies have demonstrated that aware errors elicit a Pe of larger amplitude than unaware errors (e.g., Endrass et al., 2005; Nieuwenhuis et al., 2001; see Overbeek et al., 2005, for a review).

Some researchers have separated the Pe into two subcomponents according to its timing and/or distribution resulting in an early Pe with a fronto-central distribution, similar to the Ne/ERN, and a late Pe with a more posterior distribution (e.g., Arbel & Donchin, 2009; Hewig et al., 2011; O'Connell et al., 2007; Ullsperger & von Cramon, 2006; van Veen & Carter, 2002). A possible source of the early Pe was found within the caudal ACC, whereas possible sources of the late Pe lay within the rostral ACC and the superior parietal cortex (van Veen & Carter, 2002). Both subcomponents were differentially affected by experimental manipulation. For example, the early Pe has been shown to be larger when participants were required to report errors than when they had to correct them, whereas the amplitude of the late Pe was not affected (Ullsperger & von Cramon, 2006). The posterior but not the frontal Pe was affected by speed vs. accuracy instructions (Arbel & Donchin, 2009). Similarly, only the late Pe but not the early Pe has been shown to be related to error awareness (Hewig et al., 2011; O'Connell et al., 2007). Due to spatial and temporally similarities, it has been suggested that the late Pe might represent a P3-like reaction

to the error (e.g., Arbel & Donchin, 2009; Davies et al., 2001; Leuthold & Sommer, 1999;[11] see also Overbeek et al., 2005). Davies et al. (2001) showed that the Pe was indeed correlated with the P3 on correct trials. The late Pe might therefore represent error recognition and updating of the error context (Leuthold & Sommer, 1999). This interpretation is in line with the findings of a relation between error awareness and Pe amplitude.

Both the Ne/ERN and the Pe have been linked to post-error adjustments. For example, Gehring et al. (1993) reported longer reaction times and higher correction rates following error trials with larger Ne/ERN, compared to error trials with smaller Ne/ERN. Other studies also found a link between Ne/ERN amplitude and post-error slowing (e.g., Debener et al., 2005; Hewig et al., 2011; Hirsh & Inzlicht, 2010; Ladouceur et al., 2007; West & Travers, 2008), whereas others did not confirm this association (e.g., Alain et al., 2002; Dudschig & Jentzsch, 2009; Gehring & Fencsik, 2001; Nieuwenhuis et al., 2001; Stemmer, Segalowitz, Witzke, & Schönle, 2003). It is worth mentioning that some of the studies reporting a significant relation between the Ne/ERN and post-error slowing did correlate the amplitude with post-error reaction times instead of reaction time differences between trials following errors and following correct trials (e.g., Debener et al., 2005; Gehring et al., 1993; Ladouceur et al., 2007). This method can simulate an apparent correlation that would not have been found if reaction time differences had been used. This issue will be discussed in more detail in Section 6.3.

Just like for the Ne/ERN, the data on the link between Pe amplitude and post-error slowing have been inconclusive. Whereas some researches did find a positive relationship between the Pe and slowing (e.g., Boksem et al., 2006; Hajcak et al., 2003; Nieuwenhuis et al., 2001), others did not (e.g., Fiehler et al., 2005; Hewig et al., 2011; Stemmer et al., 2003; see Overbeek et al., 2005, for a review). A possible reason for these diverging results is that error awareness is linked to both post-error slowing and Pe amplitude and might therefore serve as a mediator between the Pe amplitude and post-error slowing.

[11] Of these three studies only one (Arbel & Donchin, 2009) investigated both subcomponents separately. However, the Pe in the study by Leuthold and Sommer (1999) seems to correspond to the late Pe. The Pe in the study by Davies et al. (2001) is not easily identifiable as either early or late Pe.

Another ERP component of interest in the investigation of cognitive control is the N2 or N200. The term N2 describes the second negative peak in the stimulus-locked ERP and has been observed in a variety of experimental settings. Folstein and van Petten (2008) distinguish three different subtypes in their review of the N2. The subtype of interest in this context is what they label the "control" N2. The "control" N2 peaks about 200 ms to 350 ms after stimulus onset and has a fronto-central distribution with maximum amplitudes usually measured at electrodes Fz, FCz, or Cz. Source analyses have repeatedly localized a dipole for this component within the ACC, similar to the source of the Ne/ERN (e.g., Ladouceur et al., 2007; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; van Veen & Carter, 2002; Yeung et al., 2004).

The N2 component has been investigated frequently in different variations of the go/nogo task. In a basic version of this task, participants are presented with two different stimuli (visually or auditory) and they are asked to respond to one and to withhold, or inhibit, their response to the other one (e.g., Dirnberger, Lang, & Lindinger, 2010; Falkenstein, Hoormann, & Hohnsbein, 2002; Kiehl et al., 2000; Mathalon, Whitfield, et al., 2003; Nieuwenhuis et al., 2003). The go/nogo task can be interpreted in terms of response conflict (e.g., Nieuwenhuis et al., 2003). However, other than in interference tasks, conflict in the go/nogo tasks does not emerge between two different response alternatives but between the tendency to respond and the requirement to withhold the response. The N2 has repeatedly been shown to be larger for nogo than go trials in this task (e.g., Band, Ridderinkhof, & van der Molen, 2003; Easdon, Izenberg, Armilio, Yu, & Alain, 2005; Falkenstein, Hoormann, & Hohnsbein, 1999; Falkenstein et al., 2002; Nieuwenhuis et al., 2003). It has also been shown to be larger for incompatible than compatible trials in the Eriksen flanker task (e.g., Bartholow et al., 2005; Boksem et al., 2006; Danielmeier et al., 2009; Freitas et al., 2009; Heil et al., 2000; Ladouceur et al., 2007; Van 't Ent, 2002). The N2 on neutral trials has been found to be of similar size as on compatible trials (e.g., Heil et al., 2000; Kopp, Rist, et al., 1996; Wild-Wall et al., 2008). Furthermore, the N2

might also be larger for incompatible than compatible trials in the Stroop and Simon tasks (e.g., Holmes & Pizzagalli, 2008; Kray et al., 2005; Masaki et al., 2012).[12]

It has been suggested that the N2 might represent the inhibition process associated with withholding a predominant response on nogo trial (e.g., Falkenstein et al., 1999). However, this view has been criticized because a reversal of stimulus frequencies in a go/nogo task, i.e. frequent nogo stimuli and infrequent go stimuli, did reveal enhanced N2 amplitudes to the rare stimuli, although no response had to be inhibited (Nieuwenhuis et al., 2003). Furthermore, the N2 was also enhanced when a response with maximal force had to be executed instead of withholding a response (Donkers & van Boxtel, 2004). These findings have led to the suggestion that the N2 might not represent inhibition but response conflict on correct trials instead (e.g., Donkers & van Boxtel, 2004; Nieuwenhuis et al., 2003; Yeung et al., 2004), an interpretation that is in line with an N2 source in the ACC and the finding of N2 enhancement on incompatible trials in interference tasks. The conflict interpretation of the N2 is supported by the observation of a reduced compatibility effect on N2 amplitude when the spacing between target and flankers was increased, thereby mirroring the effect of this manipulation on reaction times (Danielmeier et al., 2009; Kopp, Rist, et al., 1996). Furthermore, the N2 has been shown to be reduced under conditions of frequent conflict when cognitive control was expected to be high (Purmann et al., 2011). Freitas et al. (2009) found that the Gratton effect was mirrored in the N2 amplitudes, i.e. N2 amplitude on incompatible trials was reduced following incompatible compared to compatible trials. However, just like for the behavioural data (see Section 1.3), there is a possible confound with facilitatory effects due to stimulus repetition. Wendt et al., (2007) indeed showed that the Gratton effect on N2 amplitude disappeared when identical stimulus-response repetitions were excluded. Clayson and Larson (2011), on the other hand, found a Gratton effect on the N2, even when exact repetitions were excluded. Furthermore, it remains controversial whether the N2 reflects stimulus conflict, response conflict or both. In a study by van Veen and Carter (2002), the N2 amplitude was only affected

[12] The evidence for the Simon and the Stroop task is less conclusive than for the flanker task. For example, Masaki et al. (2012) only found a trend towards enhanced N2 amplitudes for incompatible trials in their comparison of both tasks.

by a combination of both kinds of conflict, whereas Wendt et al. (2007) showed that stimulus conflict alone was enough to enhance N2 amplitudes.

One prediction that follows from the conflict monitoring account is that N2 amplitude should be maximal immediately before the response because this is the time of maximal response conflict (Yeung et al., 2004). Nieuwenhuis et al. (2003) and Yeung et al. (2004) did indeed find a small N2-like component immediately before response onset. However, stimulus-locked and response-locked N2s have not been directly compared.

Another prediction following from the conflict monitoring theory is that N2 and Ne/ERN should be related, since they are both assumed to reflect the same underlying process, namely conflict monitoring, on correct and incorrect trials. This notion is supported by the similar topography and source of these two components (see above). Mathalon, Whitfield, et al. (2003) showed that the nogo-N2 and the Ne/ERN were indeed related and associated with overlapping areas of activation in the ACC. However, the Ne/ERN was associated with additional areas within the ACC when N2-related variance aspects where statistically removed from the Ne/ERN. Other studies did not find an association between N2 and Ne/ERN (e.g., Davies et al., 2001). Furthermore, Masaki et al. (2012) observed that the N2 and the Ne/ERN co-exist on slow partial error trials, whereas both components seemed to overlap for faster responses. These findings argue against the conclusion that N2 and Ne/ERN represent the same function.

In summary, there are three main ERP components of interest in the investigation of cognitive control: the Ne/ERN, the Pe and the N2. The Ne/ERN might represent error detection, conflict monitoring or reinforcement learning. The Pe can be divided into an early frontal and a later posterior subcomponent. The late Pe is probably associated with conscious error processing and might reflect a P3-like reaction to the error. The N2 is most likely associated with stimulus or response conflict processing on correct trials.

1.5 Age-Related Changes in Cognitive Control

It is a widely known fact that some intellectual skills decline with increasing age, whereas others, such as verbal abilities, remain largely intact (e.g., Desjardins & Warnke, 2012; Stuart-Hamilton, 1999). For example, there usually is a general

increase in reaction times for older compared to younger adults (e.g., Falkenstein et al., 2000; Mathalon, Bennett et al., 2003). Some researchers reported higher error rates as well (e.g., Mathewson et al., 2005), whereas others found no differences (e.g., Falkenstein et al., 2000; Falkenstein, Hoormann, & Hohnsbein, 2001; Mathalon, Bennett et al., 2003). Older adults have also been shown to have poorer episodic memory than younger adults (e.g., Li, Nilsson, & Wu, 2004) and they might have more difficulties to ignore irrelevant information (inhibition deficit; e.g., Grady, 1998; Kok, 1999).

Several structural and functional changes occur in the ageing brain. For example, Good et al. (2001) found decreases in grey matter volume with increasing age, especially in the frontal and the parietal cortex. Furthermore, older adults showed decreased activity in the default mode network, i.e. the network of brain areas that are active when an individual is resting but awake, compared to young adults (Damoiseaux et al., 2008). However, default mode network activity stayed relatively stable over a period of eight years within a group of older adults (Beason-Held, Kraut, & Resnick, 2009). Age-related changes have also been found in the dopamine system, such as a reduced dopamine transporter and receptor density (see Cropley, Fujita, Innis, & Nathan, 2006, for a review). These changes in the dopaminergic system have been shown to be associated with performance on a variety of cognitive tests measuring episodic memory and executive functions (e.g., Erixon-Lindroth et al., 2005).

Structural and functional differences between young and older adults have been found especially in the frontal lobes (e.g., Grady, 1998; West, 1996), which has led to the formulation of the frontal lobe theory of cognitive ageing (West, 1996). This theory states that, because of differential neural decline in the frontal lobes, cognitive functions that rely on this area (e.g., executive functions) are more vulnerable to ageing than other functions (e.g., lower level computational processes such as feature extraction). Band, Ridderinkhof, and Segalowitz (2002) have criticised this theory, however, since not all skills related to cognitive control are equally impaired when people age. Furthermore, not all areas within the frontal lobes are affected by ageing to the same degree. Altogether, although this view might be too narrow to explain all cognitive changes, there are certainly age-related differences that are associated with frontal lobe functions and the ACC in particular.

For example, Sharp, Scott, Mehta, and Wise (2006) observed an age-related increase in ACC activity in a semantic and a syllable decision task. The authors interpreted this as an increase in control implementation; however, according to the conflict monitoring theory, this finding would represent an increase in conflict monitoring instead. Milham et al. (2002), on the other hand, observed increased ACC activity in older compared to young adults only on compatible trials in a Stroop task. More specifically, the ACC was activated on compatible and incompatible trials for older adults, whereas it was selectively activated on incompatible trials but not on compatible trials for younger adults. The authors concluded that attentional control is compromised in older adults because the mere presence of irrelevant information, i.e. a colour word, was enough to activate the ACC, even when colour word and ink colour were not conflicting and associated with the same response. In contrast, Rosano et al. (2005) did not find differences in ACC activation between young and older adults in a POP (Preparing to Overcome Prepotency) task, in which a colour cue indicated whether a compatible or incompatible response had to be performed in reaction to an arrow target. Both participant groups showed increased activity on incompatible trials as compared to compatible trials.

1.5.1 Effects of Age on the Performance in Interference Tasks

Considering the described changes in ACC functioning with increasing age, one should expect behavioural measures in interference tasks to also be affected, since performance in these tasks relies heavily on executive functions. Several researchers have indeed reported larger compatibility effects for older compared to young adults in the flanker task (e.g., Zeef & Kok, 1993; Zeef, Sonke, Kok, Buiten, & Kenemans, 1996), the Stroop task (e.g., Mager et al., 2007; Milham et al., 2002; West, 2004; West & Moore, 2005), and the Simon task (e.g., Bialystok, Craik, Klein, & Viswanathan, 2004; Kubo-Kawai & Kawai, 2010; van der Lubbe & Verleger, 2002). These findings suggest stronger interference by distracting information with increasing age due to a decrease in the effectiveness of attentional control (e.g., Zeef & Kok, 1993; Milham et al., 2002; Mani, Bedwell, & Miller, 2005). In other words, older adults seem to have difficulties to focus their attention on the relevant aspect of a stimulus, while ignoring irrelevant information. However, there appear to be differences in the reliability of this effect between the three interference tasks.

Several authors did not find significant interactions between age and compatibility in the flanker task (e.g., Mathewson et al., 2005; Nieuwenhuis et al., 2002; Wild-Wall et al., 2008). Furthermore, it has been suggested that the finding of an age-related increase in the compatibility effect might be due to general response slowing in older adults. Since differences between conditions increase with increasing reaction times, general slowing might have feigned differences in the compatibility effect. Indeed, Verhaeghen and de Meersman (1998) concluded in their meta-analysis that the apparent age-related increase in the Stroop effect is an artefact of overall slower reaction times. Similarely, Sullivan (1999) did find no differences in facilitation in a flanker task between young and older participants when correcting for the general reaction time increase. Additionally, there were no differences between young adults and younger-old adults (M = 67.2 years) in interference; older-old adults (M = 86.6 years) showed a reduction in interference in one of the task conditions. The age-related increase in the Simon effect, on the other hand, appears to be more reliable and has also been found when reaction times were corrected for general response slowing (e.g., van der Lubbe & Verleger, 2002; Kubo-Kawai & Kawai, 2010). Furthermore, Kawai, Kubo-Kawai, Kubo, Terazawa, and Masataka (2012) did compare age effects in the Simon task and the flanker task in the same sample of participants and found that the Simon effect was increased in older adults, whereas the flanker effect was of similar size in both groups. Older adults might therefore have more difficulties in resolving conflict by suppression of motor activation associated with the direct route in the Simon task than in resolving flanker-associated conflict by focussing spatial attention on the target.

Age-related changes in executive functions may not only influence the effects of conflict on a same-trial basis; they might also decrease the effectiveness of control adjustments after the experience of conflict or errors, such as the Gratton or the post-error slowing effect. Only few researchers have studied age-related changes in the Gratton effect. West and Moore (2005) found comparable sequential adjustment effects for young and old participants in the Stroop task. Monti et al. (2010), on the other hand, reported a reduced Gratton effect for older adults compared to young adults using a version of the Stroop task, in which participants had to classify pictures of faces as male or female. However, the same participants showed preserved adjustment when an emotional Stroop task was used. According to the

authors, these results can either be explained by a motivational shift towards emotional information processing in the elderly or by differences in the extent of neural degradation of the circuits involved in control adjustment in emotional and non-emotional tasks. To my knowledge, age-related changes of the Gratton effect in the other interference tasks have so far not been investigated, although this would be an interesting research question considering the differences in conflict resolution (see Section 1.3.1) and the differential effects of ageing on the compatibility effect in these tasks.

Age-related changes in post-error slowing have been studied more extensively. Some studies reported an increase in slowing for older adults (e.g., Band & Kok, 2000; Falkenstein et al., 2000), whereas others did not find any significant difference between the age groups (e.g., Beste, Willemssen, Saft, & Falkenstein, 2009; Nessler, Friedman, Johnson, & Bersick, 2007; Nieuwenhuis et al., 2002; Themanson, Hillman, & Curtin, 2006). Interestingly, Gehring and Knight (2000) and West and Moore (2005) found an age-related increase in post-error slowing in their original analyses; however, that difference disappeared when they controlled for general slowing by using proportional slowing scores or log transformed data, respectively. These findings of an age-related increase of slowing or no age-related difference are surprising, considering the postulated difficulties in error processing in older adults derived from Ne/ERN research (see Section 1.5.2). If an error is not adequately detected or processed, post-error slowing would be expected to be reduced. Findings of larger slowing in older adults might instead be related to greater subjective importance of accuracy with increasing age. This interpretation would be in line with findings of increased post-error slowing when participants are instructed to be as accurate as possible compared to a speed instruction (e.g., Jentzsch & Leuthold, 2006).

1.5.2 Effects of Age on Error- and Conflict-Associated ERPs

Age-related neurophysiological changes have often been investigated in studies using ERPs. It is a well-established finding that older adults show an amplitude reduction of the Ne/ERN compared to young adults across a variety of different tasks, including the flanker and the Stroop task (e.g., Band & Kok, 2000; Beste et al., 2009; Falkenstein et al., 2001; Gehring & Knight, 2000; Hoffmann & Falkenstein,

2011; Mathalon, Bennett et al., 2003; Mathewson et al., 2005; Nieuwenhuis et al., 2002; Themanson et al., 2006; West, 2004). Eppinger, Kray, Mock, and Mecklinger (2008) proposed that age difference in the Ne/ERN amplitude might be an artefact of performance differences between the groups. More specifically, older adults might commit more errors, as they perhaps do not have an intact representation of the correct response. According to the different theories of the Ne/ERN (see Section 1.4.2), this representation would be a necessary prerequisite for the emergence of the Ne/ERN. Furthermore, it has been shown that the amplitude of the Ne/ERN decreases, when errors are more frequent (e.g., Hajcak et al., 2003, 2004; Herrmann et al., 2004; Holroyd & Coles, 2002; see also Section 1.4.2). However, of the aforementioned studies only Band and Kok (2000) and Mathewson et al. (2005) found an increase in error rate for their older participant groups. In most studies, both age groups performed at the same accuracy level (Beste et al., 2009; Falkenstein et al., 2001; Gehring & Knight, 2000; Hoffmann & Falkenstein, 2011; Mathalon, Bennett et al., 2003) or the older age groups even showed reduced error rates (Nieuwenhuis et al., 2002). It is therefore unlikely that age-related reductions in Ne/ERN amplitude were due to accuracy differences, and it seems justified to conclude that older adults show deficits in error detection, conflict monitoring and/or reinforcement learning (cf. Section 1.4.2).

Some evidence suggests that a repetitive work environment might accelerate the ageing process reflected in Ne/ERN amplitude. Gajewski et al. (2010) showed amplitude reductions in middle-aged participants (48 to 58 years of age) who were working on a car assembly line. Participants of the same age group who worked in the same company but in positions with more flexible work demands (e.g., service and maintenance) did not show Ne/ERN reductions compared to young adults.

One important methodological consideration when investigating group differences in Ne/ERN amplitude is that analyses are often conducted using difference waves. Therefore, age-related amplitude reductions might be due to a reduction in error-related activity or to an increase in correct-related activity, i.e. the Nc/CRN. Such an increase in Nc/CRN amplitude might reflect a less discriminatory error evaluation system or increased response uncertainty in older adults, leading to error processing on correct response trials. However, results about age-related changes in the Nc/CRN are inconclusive. Whereas Beste et al. (2009) did find no

difference between their age groups, Eppinger et al. (2008) found larger Nc/CRN amplitudes for older than younger adults, and Mathalon, Bennett et al. (2003) found the opposite, i.e. smaller amplitudes for older than younger adults. Differences in the Nc/CRN between age groups were not dependent on accuracy differences in any of these studies. Therefore, the amplitude of the Nc/CRN might depend more on specific task demands (e.g., difficulty) than on age.

Age-related effects on the amplitude of the FRN have mostly been investigated within the framework of the reinforcement learning theory. According to this theory, the Ne/ERN and the FRN reflect the same underlying process, namely a reinforcement learning signal that is sent to the ACC via the mesencephalic dopamine system, indicating that an outcome is worse than expected (Holroyd & Coles, 2002; see also Section 1.4.2). Therefore, the FRN is expected to decline in amplitude with increasing age, just like the Ne/ERN. In line with this prediction, the FRN has consistently been found to be reduced in older compared to younger participants in a variety of learning tasks, in which the correct stimulus-response mapping had to be inferred by trial and error (e.g., Eppinger et al., 2008; Nieuwenhuis et al., 2002; Pietschmann, Simon, Endrass, & Kathmann, 2008). This finding can be explained in the reinforcement learning theory framework. The age-related reduction in dopamine transporter and receptor density (Cropley et al. 2006) might lead to overall weaker mesencephalic dopamine signals. In this case, the phasic decrease in the activity of the mesencephalic dopamine system in response to errors might be weaker in older than young adults, which leads to a smaller disinhibition of ACC neurons and therefore to smaller FRN (and possibly Ne/ERN) amplitudes. In support of this interpretation, Nieuwenhuis et al. (2002) showed that the age effects in their study (i.e. reduced accuracy, FRN amplitude, and Ne/ERN amplitude in older compared to young adults) could indeed be modelled by reducing the value of the parameter in the Holroyd and Coles (2002) model that reflects the phasic activity of the dopamine system.

Effects of ageing on the Pe have not been studied extensively, although age effects on this component might give insight into differences in error awareness between young and old adults. The few studies that have investigated the Pe have found inconsistent results. For example, Band and Kok, (2000) and Mathewson et al. (2005) found reduced Pe amplitudes in older compared to young adults, whereas

Mathalon, Bennett, et al. (2003) found no age-related differences. None of these studies distinguished between an early and a late subcomponent, although the Pe in the study by Band and Kok (2000) seems to represent the late one. The components in the other two studies cannot be easily identified as either early or late Pe, since the authors investigated activity over several electrode sites. Furthermore, Mathalon, Bennett, et al. (2003) averaged amplitudes over a long time window for their analysis, covering both early and late aspects of the Pe. Using such a long time interval might have obscured effects that might have been found if peak measures and shorter time intervals had been used. These differences in the analysis of the Pe between studies might potentially explain the discrepant results.

Another component of interest in the investigation of ageing is the N2. Since this component is assumed to reflect conflict monitoring processes, age-related differences in the size of the compatibility effect in interference tasks should be reflected in the amplitude of this component. However, the results of age-related effects on the N2 in interference paradigms are inconsistent. Using the Eriksen flanker task, Nieuwenhuis et al. (2002) found no differences in N2 amplitude or latency between their participant groups; Wild-Wall et al. (2008), on the other hand, reported a reduction in N2 amplitude for older adults in the same task. Kray et al. (2005) did not find any age effects on N2 amplitude in a Stroop task; however, N2 latency was significantly longer for older than four young adults. None of theses studies found age-related differences in compatibility effects in behavioural measures. Similar result have been obtained using go/nogo task. Whereas Czigler, Csibra, and Ambro (1996) found an age-related reduction of the nogo-N2, Falkenstein et al. (2002) found no significant differences. Both studies reported a significant delay of the N2 component for older adults.

In summary, whereas age-related reductions in Ne/ERN and FRN amplitude are well-established findings, results of age effects on the Pe and the N2 are inconclusive. Further research is needed to tease apart age effects on the early and late subcomponents of the Pe.

1.6 The Current Project

In this thesis I will investigate two main research questions. First, Chapters 3 and 4 are concerned with the question how conflict strength influences the mechanisms

underlying cognitive control. Second, Chapters 5 and 6 explore how cognitive control processes are influenced by ageing.

In order to investigate the first of these research questions, I manipulated conflict strength within the Eriksen flanker task using two different methods. The first approach was to independently manipulate the contrast of target and flanker letters to reduce or increase flanker influence. Chapter 3 describes an ERP study and a behavioural follow-up study using this contrast manipulation. The second approach was to use different stimulus onset asynchronies to manipulate flanker-associated response activation and to include neutral flankers to separate facilitation and interference effects. A behavioural pilot study and an ERP study using this method are described in Chapter 4. The manipulations and the resulting hypotheses will be described in more detail in the respective chapters. Furthermore, I examined effects of conflict strength on measures of control adjustment, i.e. the Gratton effect and post-error slowing, since the conflict monitoring theory predicts larger adjustment following stronger conflict. Additionally, I studied effects of conflict strength on ERP components that have been associated with conflict processing and cognitive control.

In order to evaluate age-related effects on cognitive control processes, I included a group of middle-aged participants in the previously mentioned contrast manipulation ERP study. Age-related changes in cognitive control have rarely been examined in a middle age range; most studies focussed on adults over the age of 60 years. Chapter 5 describes effects of ageing in the middle age range on behavioural measures of conflict processing and control adjustments and on conflict- and error-related ERP components. Furthermore, I investigated early and late aspects of the Pe separately. Chapter 6 is concerned with the single-trial analysis of the Ne/ERN in the same data set, to study the underlying reasons for the well-established finding of age-related amplitude reductions in this component. This amplitude reduction might be either due to generally weaker error signals and, therefore, reduced Ne/ERN amplitudes on all trials or, alternatively, due to lapses in error detection, which would lead to the absence of an Ne/ERN on some trials and normal sized Ne/ERN amplitudes on others trials. The averaging approach that is commonly used in the analysis of ERPs (see Section 2.2) would conceal this kind of variation between

trials. Using single-trial analysis can provide useful information about these underlying processes.

Before describing empirical data, I am going to give some background information about the EEG technique used in this project in the following chapter.

2 General Methods - Electroencephalography

Some of the experiments in this thesis use electroencephalography (EEG) in addition to behavioural data, such as reaction times and error rates. EEG refers to the technique of measuring electric brain activity, first described by Hans Berger (1929). If measured at the surface of the head, as in this case, EEG provides a non-invasive method to measure brain activity with a high temporal resolution (within the range of milliseconds). This chapter gives an overview of how EEG signals emerge and how they are measured and analysed.

2.1 The EEG Signal

There are two kinds of electric potentials in the brain, action potentials and postsynaptic potentials. Action potentials are short electric spikes that travel from the cell body along the axons of neurons towards a synapse. These signals cannot be measured using EEG. Postsynaptic potentials, on the other hand, are changes in the membrane potential of the postsynaptic cell. One postsynaptic potential alone is too small to be measurable at the surface of the head. However, under certain conditions the activity of several neurons can add up. According to Seifert (2005) these conditions are (1) a large number of neurons (at least 10 000), (2) a parallel geometric arrangement of these neurons, (3) synchrony, i.e. all electric fields must emerge at the same time to sum up, and (4) proximity to the surface of the head. If these conditions are fulfilled, a so-called local field potential is created. This local field potential can be described as a dipole, called equivalent current dipole. If the equivalent current dipole is perpendicular to the surface of the head, it can be measured using EEG[13] (see Luck, 2005, and Seifert, 2005, for overviews). Because of these restrictions, only activity of certain brain areas is accessible with EEG. The pyramidal cells of the cortex fulfil the requirements; therefore, most of the activity measured on the surface of the head originates from there.

Despite the summation of the signal of several thousands of cells, the electric potential on the surface of the head is still very small, i.e. in the microvolt range. The voltage on the scalp is measured using silver chloride (Ag/AgCl) electrodes,

[13] Local field potentials that are parallel to the surface of the head can be measured with magnetoencephalography, a technique that uses the magnetic field that emerges around the dipoles to measure brain activity.

arranged on the surface of the head according to different standardized systems. The most common of these systems is the International 10-20 layout, which was used in this thesis and is presented in *Figure 6*. Electrodes are mounted in an elastic cap, which is placed on the participants' head. Electrolyte gel is used to assure contact between the surface of the head and the electrodes, and to reduce the impedance. Additional electrodes are placed around the eyes to measure eye movements and blinks. The signal that is measured by the electrodes is transmitted to the amplifier, which amplifies the difference between two electrically active points. For this reason a reference electrode has to be defined. In the current setup electrode CMS was used as recording reference; during analysis the data were re-referenced to average reference. Other common recording reference points are the mastoid bones behind each ear or the ear lobes. The continuous EEG signal is digitized during recording. In this thesis the used data-sampling rate was 256 Hz.

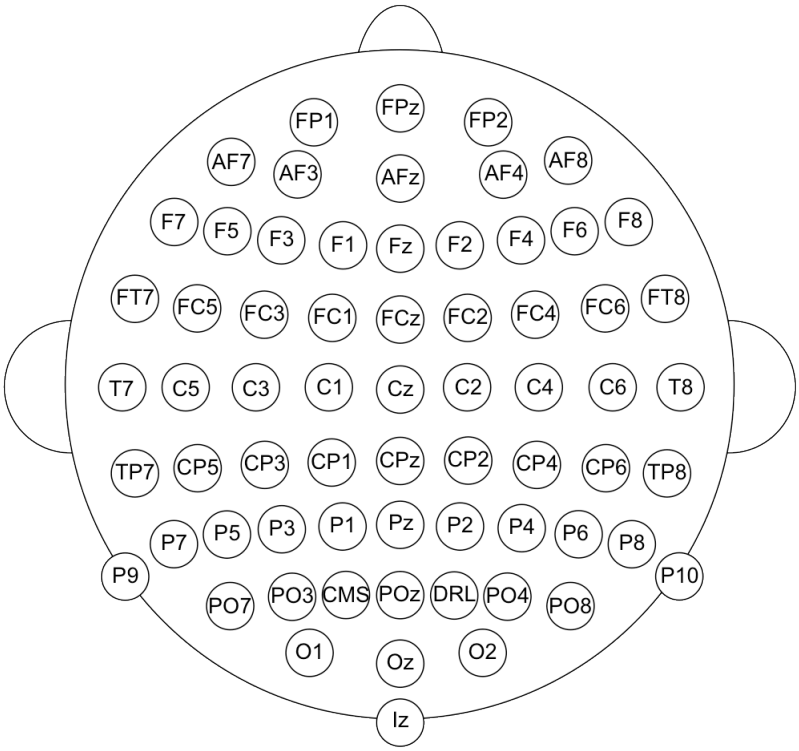

Figure 6: Top view of the International 10-20 electrode layout that was used in this thesis. The top of the graph corresponds to the front of the head. Capital letters describe the position on the head. Electrodes along the midline are denoted by the letter z. Electrodes on the left are donated by odd number, electrodes on the right by even numbers. Electrodes CMS (Common Mode Sense) and DRL (Driven Right Leg) serve as ground electrodes.

The measurement of the EEG signal is complicated by artefacts, i.e. signals in the recording that are not due to brain activity. Common artefacts are eye movements and blinks, other muscle movements, the heartbeat, changes in skin conductance due to sweating and alpha activity. Some of those, such as eye movements and blinks, are easy to correct using mathematical algorithms (see Section 2.2). Others are harder to remove; and usually the segment of data with this kind of artefact has to be excluded from the analysis.

2.2 From the continuous EEG to the ERP

Several processing steps are necessary to convert the digitized recorded EEG into an event-related potential. Eye movement and blink artefacts can be removed using artefact correction methods. There are a variety of different methods that can be used for this purpose (see Croft & Barry, 2000, for a review). The method used in this thesis is implemented in BESA (Version 5.0.6) and uses a source model approach to distinguish artefact topographies from brain activity topographies (Ille, Berg, & Scherg, 2002). Using principal component analysis, the data are then separated into artefact and signal components; and the estimated artefact signals for horizontal and vertical eye movements and blinks are subtracted from the originally recorded signal.

In a next step, the artefact-corrected signal is cut into short epochs, time-locked to an event of interest, such as the onset of a stimulus or a certain kind of response. Segments that still contain artefacts (e.g., large sudden changes in amplitude) need to be excluded from further analysis. This can be done by visual inspection of the data or by using automated processes. The remaining segments associated with the same event are averaged. The rationale behind the averaging approach is that activity, which is associated with the event, should be equivalent on every trial, whereas noise in the data is randomly distributed across the epoch and should, therefore, disappear during the averaging process. The resulting waveform is what is called an ERP.

In a next step, the ERP needs to be baseline corrected. This means that a period in which no event-related brain activity is expected is defined as a baseline interval. The average of this interval is computed and subtracted from each time point of the remainder of the signal for the purpose of making different conditions comparable. A common baseline length is 100 ms to 200 ms although shorter or

longer intervals can be used. The baseline interval is usually set shortly before the onset of the event of interest. In order to further reduce possible artefacts, filters can be applied to the data. High-pass or low cutoff filters remove slow drifts in the recording that might occur due to changes in skin conductance. Low-pass or high cutoff filters, on the other hand, eliminate high frequency artefacts such as muscle activity.

The positive and negative peaks in the ERPs are referred to as components of the ERP. These ERP components can give useful information about processes occurring while participants process stimuli and responses. Reaction times can be seen as an end product of these underlying processes. The amplitude of a component is an indicator of the extent of neural activation associated with a certain cognitive process; its latency reflects the time needed for this process (e.g., Gehring, Gratton, Coles, & Donchin, 1992; Rugg & Coles, 1995). Whereas latency is always measured as the time between the event of interest and the peak of a component, there are different ways of measuring component amplitude. A common way is to measure the peak amplitude compared to baseline or compared to another peak (e.g., the preceding peak). For these methods clearly defined peaks are needed. However, for some components a clear peak cannot be identified. In these cases an area measure, meaning the average amplitude of a predefined time interval, can be used instead.

For display purposes the individual ERPs are usually averaged over several participants. The result is called a grand average. Furthermore, difference waves can be calculated by subtracting the ERP of one condition (e.g., correct response trials) from the ERP of another condition (e.g., error trails) in order to visualize differences between conditions.

2.3 Other relevant ERP components

In the following empirical chapters (see Chapters 3 and 5), I will investigate two further ERP components, the P1 and the N1, which have not been mentioned in the introduction.

The P1 and N1 components describe the first positive (P1) and the first negative peak (N1) in an ERP. In visual tasks, these components are maximal over posterior electrode sites and reflect early visual processing of the stimuli. The P1 typically peaks about 80 ms to 130 ms after stimulus onset; the N1 peaks later, about

150 ms to 200 ms after stimulus onset. Both P1 and N1 have been shown to originate within the lateral extrastriate cortex (e.g., Finnigan, O'Connell, Cummins, Broughton, & Robertson, 2011; Di Russo, Martinez, & Hillyard, 2003), a brain area within the occipital lobes that lies adjacent to the primary visual cortex. Luck, Heinze, Mangun, and Hillyard (1990) suggested that the P1 might reflect early sensory processing at a location that is already attended, whereas the N1 might reflect the orienting of attention towards a relevant stimulus.

The amplitude of the P1 and N1 is sensitive to processes of spatial attention. Both components have been shown to be larger when a stimulus occurs in an attended compared to an unattended location (e.g., Di Russo et al., 2003; Luck et al., 1990; Wijers, Lange, Mulder, & Mulder, 1997). Furthermore, the components are sensitive to stimulus contrast. In a study by Jentzsch, Leuthold, and Ulrich (2007) both P1 and N1 showed increased latencies for low contrast compared to high contrast stimuli. Johannes, Münte, Heinze, and Mangun (1995) found a latency shift only for the N1. The P1, on the other hand, showed a reduction in amplitude for low compared to high contrast stimuli.

3 Effects of Stimulus Contrast in the Eriksen Flanker Task

The first data chapter describes two experiments in which the contrast of the stimuli in a version of the Eriksen flanker task has been manipulated in order to manipulate conflict strength. The first part describes an EEG experiment in which the contrast conditions were presented in a blocked fashion. The second part shows a behavioural follow-up study in which contrast stimuli presentation was randomized. Both experiments will be discussed together in the third part.

3.1 Blocked Presentation of Contrast Conditions: An ERP Study

The aim of the first experiment was to manipulate conflict in the flanker task to investigate the effects of conflict strength on control adjustments like the Gratton effect and post-error slowing and on conflict-related ERP components. In order to achieve this, I manipulated the contrast of target and flanker letters independently. In accordance with Berlyne's (1957; see Section 1.2) conflict definitions, the following predictions regarding conflict on incompatible trials were made: 1. Conflict was expected to be larger when all letters were of high contrast than when they were of low contrast, since the absolute activation of response representations should be higher. 2. Conflict was expected to be increased compared to the standard version of the task when the target had low contrast and the flankers had high contrast, because the correct target-associated response representation should be relatively less activated than the incorrect flanker-associated response representation. 3. Conflict was expected to be lowest when the target was of high and the flankers of low contrast due to the relatively high activation of the target-associated response representation. Conditions with higher conflict were expected to show larger compatibility effects and also larger N2 amplitudes. In addition to the usual stimulus-locked analysis of the N2, the component was also analysed in the response-locked ERPs to test predictions of the conflict monitoring theory that link the N2 more closely to the response than to the stimulus (e.g., Nieuwenhuis et al., 2003; Yeung et al., 2004). Furthermore, I analysed the Gratton effect separately for response alternations and response repetitions in order to investigate whether the conflict monitoring account (Botvinick et al., 2001) or the priming account (Mayr et al., 2003) provide a better explanation for this effect. If the Gratton effect were indeed triggered by conflict adaptation effects, it should be present in both response

repetition and response alternation trials; and it would be expected to be larger for high conflict conditions. A similar logic applies to post-error slowing and the Ne/ERN. If the Ne/ERN indeed represented conflict as suggested by Botvinick et al. (2001) and post-error slowing were related to the Ne/ERN (see also Section 1.4.2), both might be expected to be influenced by conflict strength. Furthermore, early and late aspects of the Pe were investigated. Additionally, I analysed effects of the contrast manipulation on early visual ERP components, i.e. the P1 and the N1, since both have been shown to be modulated by stimulus contrast (e.g., Jentzsch et al., 2007; Johannes et al., 1995; see also Section 2.3).

3.1.1 Methods

3.1.1.1 Participants

Twenty-four young adults (M = 21.5 years, range 18 to 31 years, 14 women) were tested in a single session of approximately two hours' duration. All participants had normal or corrected-to-normal vision, gave written informed consent, and received payment of £10. Four participants of the original sample had to be replaced because of artefacts in the electrophysiological recording or because they did not commit enough errors for the analysis (inclusion criterion: at least five ERP trials in every condition of interest). The study was approved by the University Teaching and Research Ethics Committee (UTREC) of the University of St Andrews (approval code: PS5099).

3.1.1.2 Stimuli and Apparatus

The stimuli were presented on an Envy 17-inch monitor at a viewing distance of about 80 cm using Experimental Run Time System software (ERTS; version 3.22). Responses were recorded with two response keys, mounted 15 cm apart in horizontal direction. Participants used the index finger of each hand to indicate their response. The stimuli consisted of five-letter-strings composed of the letters S and H; each of which was associated with one response key. The assignment of letter to response alternatives was counter-balanced across participants. Half of the stimuli were composed of identical letters (compatible, SSSSS or HHHHH); in the other half target and flanker letters belonged to opposite response sets (incompatible, SSHSS or HHSHH). The approximate stimulus size was 7 x 30 mm. Stimuli were presented in

black (0.46 cd/m^2) or grey (44.32 cd/m^2) on a white background (66.31 cd/m^2). The contrast of target and flankers was manipulated independently, resulting in four contrast conditions: dark target – dark flankers, dark target – light flankers, light target – dark flankers and light target – light flankers.

EEG activity was recorded using a BIOSEMI Active-Two amplifier with 72 Ag/AgCl electrodes. Four of these electrodes, one at the outer canthus of and one under each eye, were used to record electrooccculographic (EOG) activity. The usual ground electrode was replaced by a Common Mode Sense (CMS) active electrode and Driven Right Leg (DRL) passive electrode feedback loop. The CMS also served as the recording reference. Data were recorded at a sampling rate of 256 Hz.

3.1.1.3 Procedure and Design

Participants were asked to identify the central letter of the presented letter string by pressing the corresponding response key. Each stimulus was presented until a response was executed. If no response was made within 1500 ms, the next trial was started. The response-stimulus interval (RSI) was set at 1000 ms.

Stimuli were presented in blocks of 64 trials, separately for the four contrast conditions, starting with four additional non-recorded warm-up trials. The order of trials within a block was randomized. The four contrast conditions were presented in sets of five blocks each, with every set preceded by a short practice block of 16 trials, resulting in 20 experimental blocks and 1280 trials overall. The order of sets was counter-balanced across participants using a Latin square procedure. Every block ended with the presentation of a feedback screen, providing information about the participants' average reaction time (in ms) and their error rate (in %). Participants were instructed to respond as quickly as possible while keeping up an error rate of about 10%. Between blocks they were asked to speed up when error rates were lower, or slow down when they were higher, than 10%. During the presentation of this feedback screen, participants had the opportunity to rest. Pressing either response key started the next block.

3.1.1.4 Data Analysis

Only trials with reaction times between 150 ms and 1500 ms were included in the analyses. Post-error accuracy was determined by dividing the number of errors

following an error by the total number of trials following an error and multiplied by one hundred ((E-E / (E-C + E-E)) * 100 with E = error and C = correct response, current trial underlined). This was compared to the number of errors following correct responses divided by the total number of trials following correct responses and multiplied by one hundred ((C-E / (C-C + C-E)) * 100).

The EEG was re-referenced off-line to the average reference. Eye movement artefacts were corrected using a source model approach implemented in BESA (Version 5.0.6). The continuous EEG was segmented into epochs of 1200 ms length, starting 200 ms before the onset of the stimulus (stimulus-locked ERPs), 200 ms before the response (response-locked ERPs for the analysis of error-related components), or 800 ms before the response (response-locked analysis of the N2). Trials containing amplitudes larger than 100 μV or a gradient larger than 75 μV were rejected; as were trials with a signal lower than 0.032 μV. A 15 Hz high cutoff filter was applied. The baseline was set to -200 ms to 0 ms for stimulus-locked ERPs, to -150 ms to -50 ms for response-locked ERPs for the error-related component analysis, and to -800 ms to -700 ms for the response-locked analysis of the N2, respectively. Epochs were averaged separately for compatible and incompatible trials, correct and incorrect responses, and the four contrast conditions. Subsequent analysis steps were executed using Konstanz Format software. Statistical analyses of the P1 and N1 components were conducted in the stimulus-locked ERPs at electrodes PO7 and PO8. The peak search window for the P1 ranged from 40 ms to 150 ms after stimulus onset; the time window for the N1 ranged from 110 ms to 250 ms after stimulus onset. All other components of the ERP were analysed in the difference waves; incompatible minus compatible for the N2 and incorrect minus correct for the error-related ERP components. Peak amplitude and latency of the N2 were determined at electrode FCz where the difference was maximal in the time window from 250 ms to 370 ms after stimulus onset. The time window for the response-locked analysis of the N2 ranged from 210 ms to 60 ms before the response. Peak amplitude and latency of the Ne/ERN were determined at electrode FCz in the time window of 20 ms to 110 ms post-response. Peak amplitude and latency of the early Pe were analysed at electrode Cz in the window of 150 ms to 330 ms. The average amplitude of the late Pe was determined at electrode Pz in the window of 250 ms to 450 ms following the response. I used the average amplitude of this component

instead of the peak amplitude, because the peak is less clearly defined than for the other components. For this reason, there will be no latency measures reported for the late Pe.

All data were analysed using repeated-measures ANOVAs. Conservative Huynh-Feldt tests were used throughout. Adjusted *p*-values are reported, along with the uncorrected degrees of freedom. Bonferroni corrected *p*-values are reported for all post hoc analyses.

3.1.2 Results

3.1.2.1 Behavioural Data

3.1.2.1.1 Compatibility Effects

Compatibility effects on reaction times and error rates were analysed with repeated-measures ANOVAs with the within-subject factors Compatibility (compatible, incompatible), Target Contrast (dark, light), and Flanker Contrast (dark, light). Only trials with correct responses on the current and the previous trial were included in the reaction time analysis. Reaction times and error rates for compatible and incompatible trials in the four contrast conditions are shown in *Figure 7*.

Reaction times: Participants responded faster on compatible (358 ms) than incompatible trials (401 ms), $F(1, 23) = 169.73$, $p < .001$, $\eta_p^2 = .88$. They also responded faster to dark (374 ms) than to light targets (385 ms), $F(1, 23) = 10.77$, $p = .003$, $\eta_p^2 = .32$. There was no significant main effect of flanker contrast ($p > .10$) but a significant interaction between target and flanker contrast, $F(1, 23) = 16.89$, $p < .001$, $\eta_p^2 = .42$. Post hoc tests showed that participants were only faster for dark targets compared to light targets when the flankers were light (368 ms and 389 ms, respectively), $F(1, 23) = 29.26$, $p < .001$, $\eta_p^2 = .56$, but not when they were dark (381 ms and 382 ms, respectively; $p > .10$). Neither target nor flanker contrast interacted with the compatibility effect (both *p*s > .10). However, there was a trend towards a three-way interaction $F(1, 23) = 3.84$, $p = .062$, $\eta_p^2 = .14$. The compatibility effect (incompatible minus compatible) was numerically smaller for light flankers (37 ms) than dark flankers when targets were dark (45 ms) but not when targets were light (46 ms and 42 ms, respectively). However, post hoc test did not reach significance (both *p*s > .10).

Error rates: Participants made more errors on incompatible trials (16.7%) than on compatible trials (4.7%), $F(1, 23) = 187.04$, $p < .001$, $\eta_p^2 = .89$. Error rates were not significantly different for dark and light targets or dark and light flankers. The interaction of target and flanker contrast was not significant either. The size of the compatibility effect was not influenced by either target contrast or flanker contrast or their combination (all *p*s > .10).

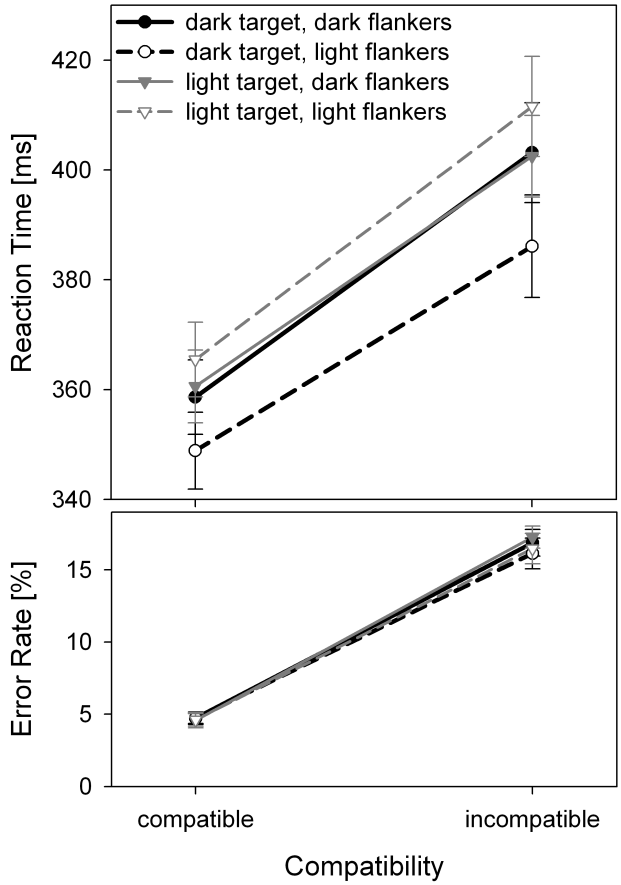

Figure 7: Compatibility effect on reaction times (top) and error rates (bottom) in the four contrast conditions.[14]

3.1.2.1.2 Sequential Adjustment Effects

Figure 8 shows the Gratton effect for the different contrast conditions for response alternations and repetitions. This effect was analysed by including the factors Trial N-1 Compatibility (compatible, incompatible) and Response Sequence (alternation,

[14] Error bars in this figure and all following figures throughout the thesis represent the standard error of the mean.

repetition) in the ANOVA design. Only interactions including the Trial N-1 Compatibility factor will be reported in this section.

Reaction times: Participants responded faster for response repetitions (373 ms) than for response alternations (388 ms), $F(1, 23) = 5.64$, $p = .026$, $\eta_p^2 = .20$. Generally, reaction times were shorter when the previous trial was compatible (378 ms) than when it was incompatible (383 ms), $F(1, 23) = 21.33$, $p < .001$, $\eta_p^2 = .48$. However, this difference was only present for response alternations (9 ms) but not for repetitions (0 ms), as indicated by the interaction of these two factors, $F(1, 23) = 10.58$, $p = .004$, $\eta_p^2 = .32$. There was a significant interaction between Target Contrast, Flanker Contrast and Trial N-1 Compatibility, $F(1, 23) = 9.99$, $p = .004$, $\eta_p^2 = .30$. Importantly, there was a significant interaction between Trial N-1 Compatibility and Current Trial Compatibility, $F(1, 23) = 35.93$, $p < .001$, $\eta_p^2 = .61$, reflecting the presence of the Gratton effect. The compatibility effect (incompatible minus compatible) was reduced following incompatible as compared to compatible trials (35 ms and 51 ms, respectively). A significant three-way interaction with the factor Response Sequence, $F(1, 23) = 86.66$, $p < .001$, $\eta_p^2 = .79$, was due to the fact that the Gratton effect ((c-ic – c-c) – (ic-ic – ic-c)) was only present for response repetitions (34 ms), $F(1, 23) = 79.63$, $p < .001$, $\eta_p^2 = .78$, but not for response alternations (-4 ms; $p > .10$). A significant interaction between Target Contrast, Flanker Contrast, Trial N-1 Compatibility and Current Trial Compatibility, $F(1, 23) = 17.97$, $p < .001$, $\eta_p^2 = .44$, showed that the size of the Gratton effect was influenced by the contrast manipulation. This effect was further modulated by the Response Sequence as indicated by a significant five-way interaction, $F(1, 23) = 34.89$, $p < .001$, $\eta_p^2 = .60$. Post hoc tests showed that the interaction of Target Contrast, Flanker Contrast, Trial N-1 Compatibility and Current Trial Compatibility was only significant for response repetitions, $F(1, 23) = 52.26$, $p < .001$, $\eta_p^2 = .69$, but not for response alternations ($p > .10$). As can be seen in *Table 1* and *Figure 8*, the Gratton effect was smaller in the mixed contrast conditions than in the conditions where target and flankers were of the same contrast for response repetitions. All other interactions including the factor Trial N-1 Compatibility did not reach significance (all ps > .10).

Table 1: Gratton effect size ((c-ic – c-c) – (ic-ic – ic-c)) for response alternations and repetitions in the four contrast conditions for reaction times (ms).

	Dark targets		Light targets	
Response Sequence	Dark flankers	Light flankers	Dark flankers	Light flankers
Alternation	-6 ms	-6 ms	3 ms	-8 ms
Repetition	50 ms	21 ms	15 ms	53 ms

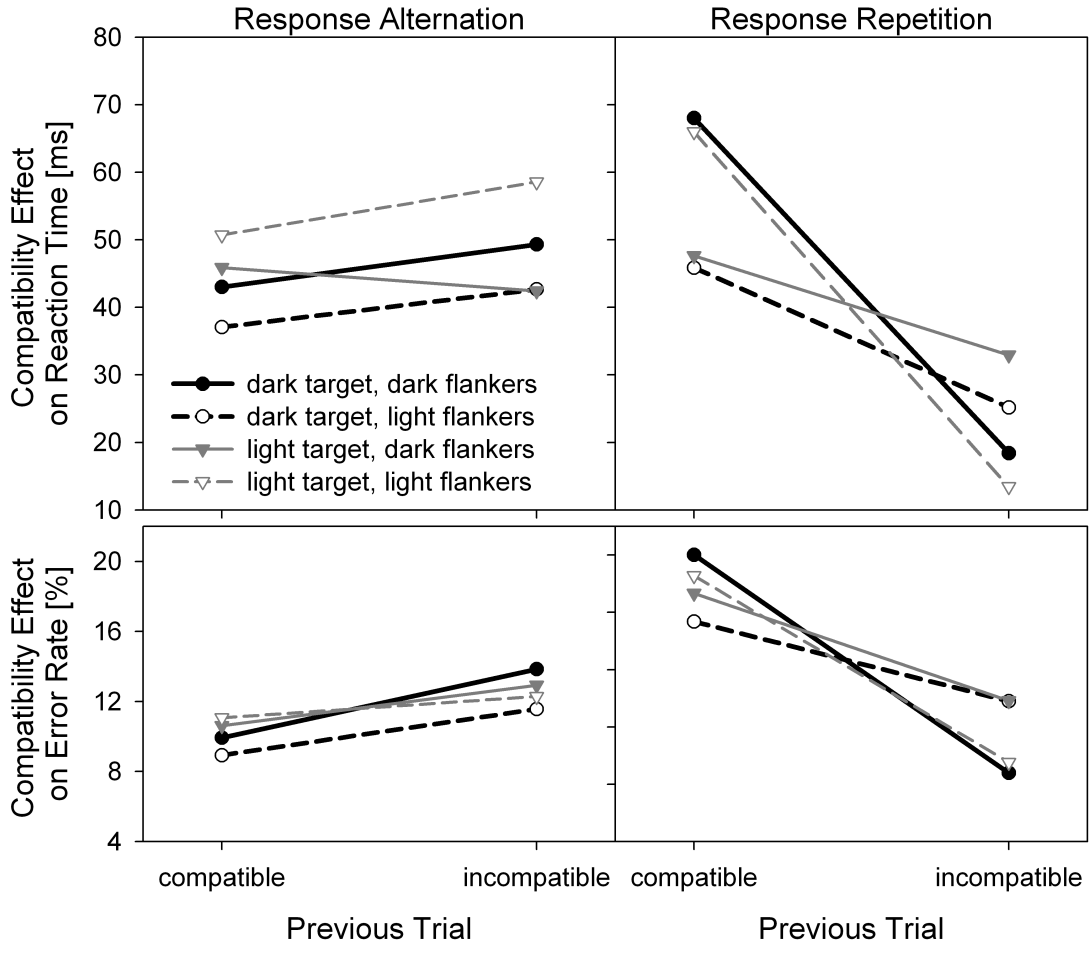

Figure 8: Gratton effect for reaction times (top) and error rates (bottom): Compatibility effect size (incompatible minus compatible) following compatible and incompatible trials for response alternations (left) and repetitions (right) in the four contrast conditions. A steeper slope indicates a larger Gratton effect.

Error rates: There was no difference in error rate between response alternations and repetitions ($p > .10$). Participants made more errors when the previous trial was compatible (11.1%) than when it was incompatible (10.2%), $F(1, 23) = 4.96$, $p = .036$, $\eta_p^2 = .18$. However, this effect interacted with the factor Response Sequence, $F(1, 23) = 35.87$, $p < .001$, $\eta_p^2 = .61$. Post hoc tests showed that only for response repetitions participants did make more errors when the previous trial had been compatible (12.6%) than when it had been incompatible (9.2%), $F(23) = 47.99$, $p < .001$, $\eta_p^2 = .68$. There was no significant difference for response alternations (9.7% and 11.2%, respectively), $F(1, 23) = 5.20$, $p = .064$, $\eta_p^2 = .19$. There was a smaller compatibility effect on error rates (incompatible minus compatible) following incompatible (10.1%) than compatible trials (14.0%), resulting in a significant Gratton effect, $F(1, 23) = 19.90$, $p < .001$, $\eta_p^2 = .46$. A three-way interaction occurred between this Gratton effect and the factor Response Sequence, $F(1, 23) = 58.06$, $p < .001$, $\eta_p^2 = .72$. Post hoc tests showed that the Gratton effect ((c-ic – c-c) – (ic-ic – ic-c)) was only present for response repetitions (10.3%), $F(1, 23) = 63.40$, $p < .001$, $\eta_p^2 = .73$, but not for response alternations (-2.5%), $F(1, 23) = 5.02$, $p = .070$, $\eta_p^2 = .18$. The Gratton effect was influenced by the contrast manipulation as shown by a significant interaction of the factors Target Contrast, Flanker Contrast, Trial N-1 Compatibility and Current Trial Compatibility, $F(1, 23) = 5.21$, $p = .032$, $\eta_p^2 = .19$. A significant five-way interaction, $F(1, 23) = 7.96$, $p = .010$, $\eta_p^2 = .26$, showed that the influence of the contrast manipulation on the Gratton effect was different for response alternations and repetitions. Post hoc tests showed that the interaction of Target Contrast, Flanker Contrast, Trial N-1 Compatibility and Current Trial Compatibility was only significant for response repetitions, $F(1, 23) = 11.23$, $p = .006$, $\eta_p^2 = .33$, but not for response alternations ($p > .10$). The respective adjustments in error rate are shown in *Table 2*. As can be seen in *Figure 8*, the adjustment for response repetitions was smaller in the mixed contrast conditions than in the same contrast conditions.

Table 2: Gratton effect size ((c-ic – c-c) – (ic-ic – ic-c)) for response alternations and repetitions in the four contrast conditions for error rates (%).

	Dark targets		Light targets	
Response Sequence	Dark flankers	Light flankers	Dark flankers	Light flankers
Alternation	-3.9%	-2.6%	-2.3%	-1.2%
Repetition	15.2%	5.5%	7.5%	13.0%

3.1.2.1.3 Error and Post-Error Effects

Figure 9 shows the reaction times for correct responses following another correct response (post-correct), for errors following a correct response (error) and for correct responses following an erroneous response (post-error). Only trials following incompatible trials were included in this analysis because participants did not commit enough errors on compatible trials. Error speed-up and post-error slowing were analysed in a repeated-measures ANOVA with the within-subjects factors Trial Type (post-correct, error, post-error), Target Contrast (dark, light) and Flanker Contrast (dark, light). Post-error accuracy was analysed using a repeated-measures ANOVA with the within-subjects factors Trial N-1 Accuracy (correct, incorrect), Target Contrast (dark, light), and Flanker Contrast (dark, light).

Reaction times: The main effect of Trial Type was significant, $F(2, 46) = 105.10$, $p < .001$, $\eta_p^2 = .82$. Post hoc tests confirmed the expected post-error slowing effect, $F(1, 23) = 29.05$, $p < .001$, $\eta_p^2 = .56$, and error speed-up effect, $F(1, 23) = 115.53$, $p < .001$, $\eta_p^2 = .83$; post-error trials (407 ms) were significantly slower and error trials (327 ms) were significantly faster than post-correct trials (380 ms). There were no significant interactions of Trials Type and any of the contrast manipulations (all ps > .10).

Error rates: Participants committed less errors following an error (6.9%) than following a correct response (8.8%), $F(1, 23) = 6.60$, $p = .017$, $\eta_p^2 = .22$, indicating increased post-error accuracy. No other effects were significant (all $p > .10$).

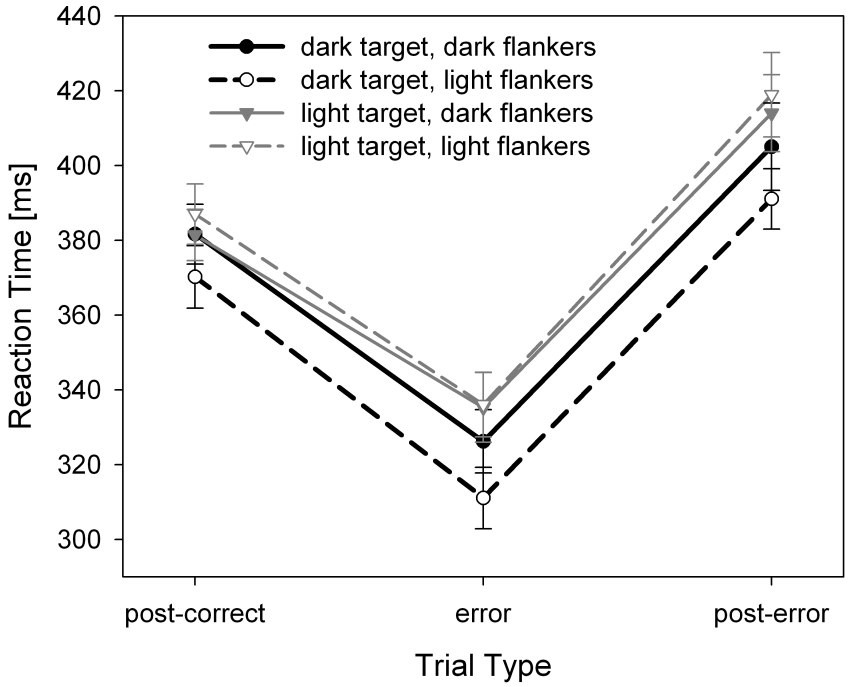

Figure 9: Error speed-up and post-error slowing in the four contrast conditions.

3.1.2.2 Electrophysiological Data

3.1.2.2.1 P1 and N1

Figure 10 shows the stimulus-locked P1-N1 complex at electrodes PO7 and PO8. Amplitude and latency of P1 and N1 were analysed in the stimulus-locked ERPs using repeated-measures ANOVAs with the within-subjects factors Hemisphere (left, right), Target Contrast (dark, light), Flanker Contrast (dark, light) and Compatibility (compatible, incompatible).

P1: The P1 peaked about 103 ms after stimulus onset. It was larger for dark flankers (+4.86 μV) than for light flankers (+4.51 μV), $F(1, 23) = 7.67$, $p = .011$, $\eta_p^2 = .25$. None of the other amplitude effects reached significance (all *p*s > .05). A significant interaction between Hemisphere and Flanker Contrast for P1 latency, $F(1, 23) = 5.66$, $p = .026$, $\eta_p^2 = .20$, was due to a later P1 peak for light than dark flankers (102 ms and 96 ms, respectively) over the right, $F(1, 23) = 20.07$, $p < .001$, $\eta_p^2 = .47$, but not over the left hemisphere (107 ms and 108 ms, respectively; $p > .10$). There was a significant interaction between Target Contrast and Compatibility, $F(1, 23) = 4.43$, $p = .047$, $\eta_p^2 = .16$, which in turn interacted with the factor Hemisphere, $F(1, 23) = 4.36$, $p = .048$, $\eta_p^2 = .16$. Post hoc tests showed that the Target Contrast X Compatibility interaction was only significant over the left,

$F(1, 23) = 6.77$, $p = .032$, $\eta_p^2 = .23$, but not over the right hemisphere ($p > .10$). All other latency effects did not reach significance (all ps > .05).

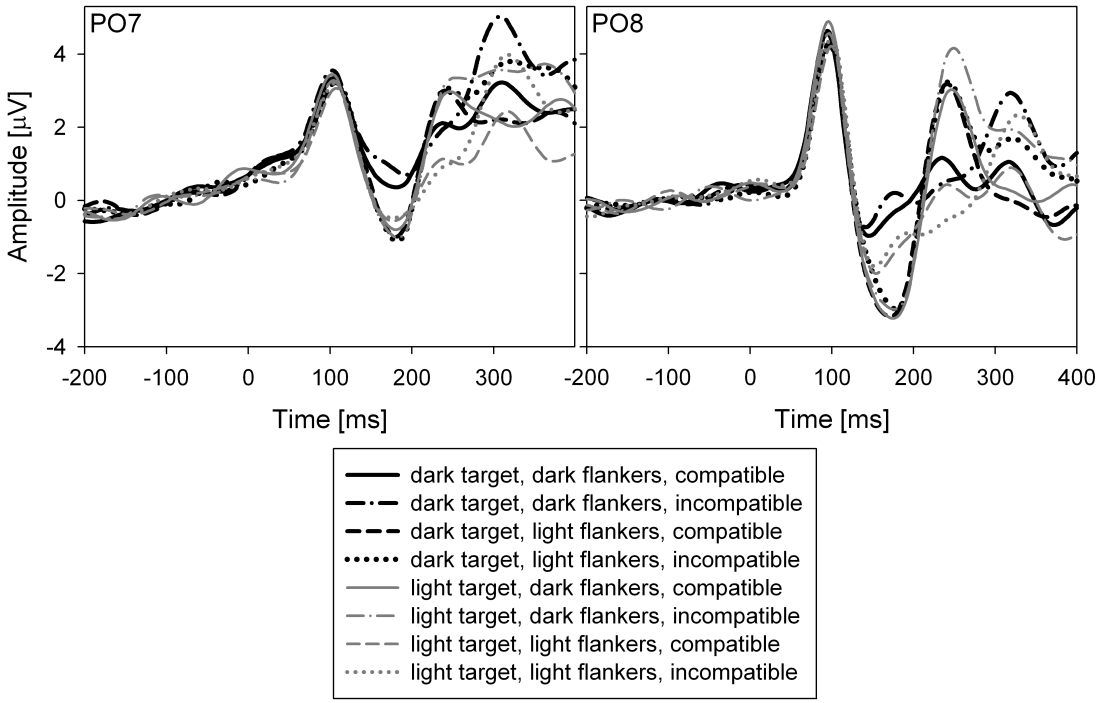

Figure 10: P1 and N1 for compatible and incompatible trials at electrodes PO7 and PO8 in the four contrast conditions.

N1: The N1 peaked about 177 ms after stimulus onset. Its amplitude was larger over the right (-4.19 µV) than over the left hemisphere (-2.03 µV), $F(1, 23) = 6.67$, $p = .017$, $\eta_p^2 = .23$. The N1 was significantly larger for light (-3.36 µV) than for dark flankers (-2.86 µV), $F(1, 23) = 5.17$, $p = .033$, $\eta_p^2 = .18$. A significant interaction between Target Contrast and Flanker Contrast, $F(1, 23) = 5.29$, $p = .031$, $\eta_p^2 = .19$, was due to significantly less negative amplitudes for dark than light targets when the flankers were dark (-2.35 µV and -3.36 µV, respectively), $F(1, 23) = 8.92$, $p = .013$, $\eta_p^2 = .28$, but not when they were light (-3.47 µV and -3.25 µV, respectively; $p > .10$). None of the other amplitude effects reached significance (all ps > .05). The N1 peaked significantly later for light (180 ms) than for dark targets (174 ms), $F(1, 23) = 12.73$, $p = .002$, $\eta_p^2 = .36$, and for light (180 ms) than for dark flankers (174 ms), $F(1, 23) = 16.00$, $p = .001$, $\eta_p^2 = .41$. No other effects reached significance (all ps > .05).

3.1.2.2.2 N2

Peak latency and amplitude of the N2 were analysed in the stimulus- and response-locked difference waves (see *Figure 11*) using repeated-measures ANOVA with the within-subjects factors Target contrast (dark, light) and Flanker Contrast (dark, light). Furthermore, the amplitude of the N2 was compared across stimulus- and response-locked analyses by including the factor Analysis Type (stimulus-locked, response-locked) in the ANOVA design.

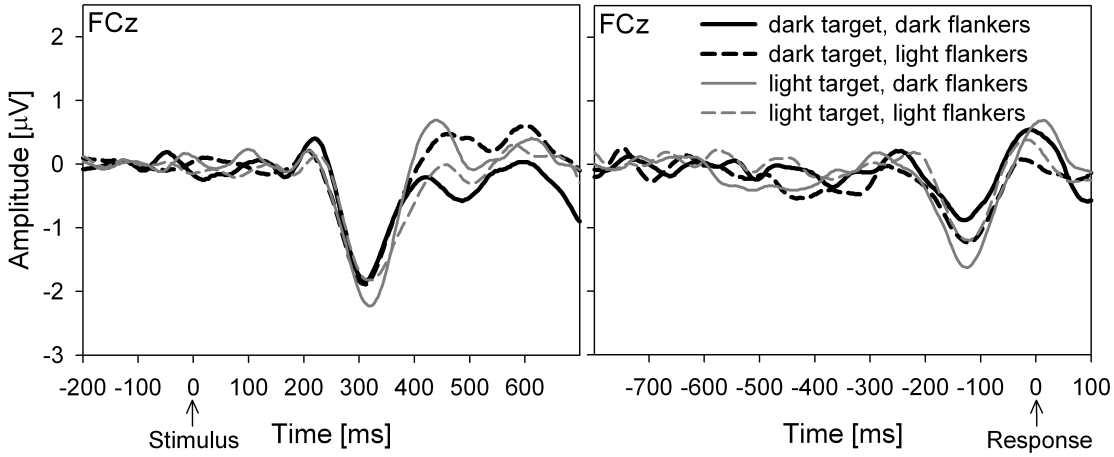

Figure 11: Stimulus-locked (left) and response-locked (right) difference wave (incompatible minus compatible) at electrode FCz showing the N2 in the four contrast conditions.

Stimulus-locked: The stimulus-locked N2 reached its maximum amplitude 316 ms after stimulus onset. Although the amplitude was numerically larger in the condition with light targets and dark flankers none of the amplitude effects reached significance (all *p*s > .10). However, there was a significant main effect of Target Contrast on latency, $F(1, 23) = 5.49$, $p = .028$, $\eta_p^2 = .19$. The N2 peaked earlier for dark targets (312 ms) than for light targets (320 ms). Neither the main effect of Flanker Contrast nor the interaction of both factors reached significance (both *p*s > .10).

Response-locked: The response-locked N2 peaked 135 ms before the response. There were no significant amplitude differences between the contrast

conditions (all *p*s > .10). There also were no significant latency effects in the response-locked analysis (all *p*s > .10).

Stimulus- vs. response-locked: Overall, the N2 was more negative in the stimulus-locked than in the response-locked analysis, $F(1, 23) = 10.19$, $p = .004$, $\eta_p^2 = .31$. None of the other effects reached significance (all *p*s > .10).

3.1.2.2.3 Error-Related ERP Components

Figure 12 shows topographic maps of the Ne/ERN, early Pe, and late Pe in the contrast condition with dark target and flankers as an example. *Figure 13* shows the response-locked difference waves (incorrect minus correct) for incompatible trials at electrodes Fz, FCz, Cz, and Pz. Peak latency and amplitude of the Ne/ERN and the early Pe as well as the average amplitude of the late Pe were analysed using repeated-measures ANOVAs with the within-subjects factors Target Contrast (dark, light) and Flanker Contrast (dark, light).

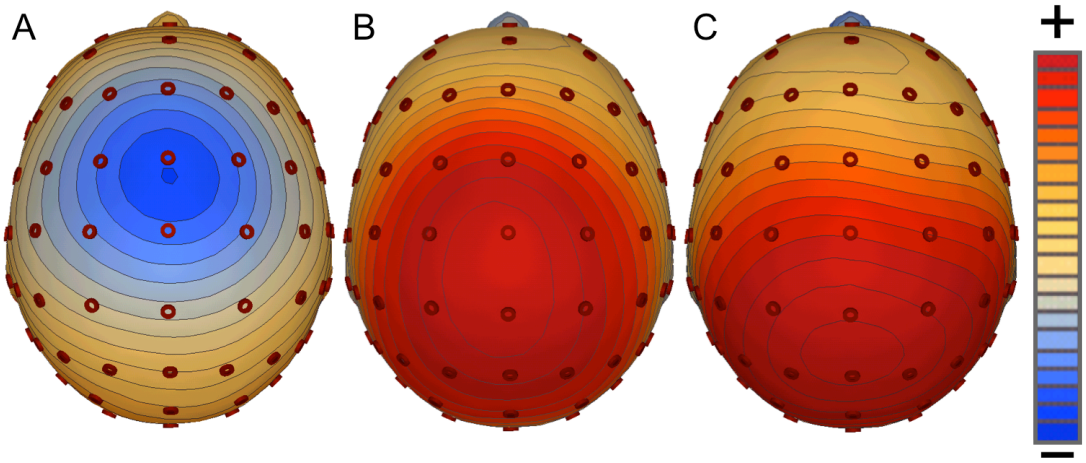

Figure 12: Exemplary topographic maps of the Ne/ERN at 65 ms (A), the early Pe at 245 ms (B) and the late Pe at 350 ms (C) post-response in the condition with dark target and flankers. Shown are the difference waves (incorrect minus correct) with 0.80 µV/step.

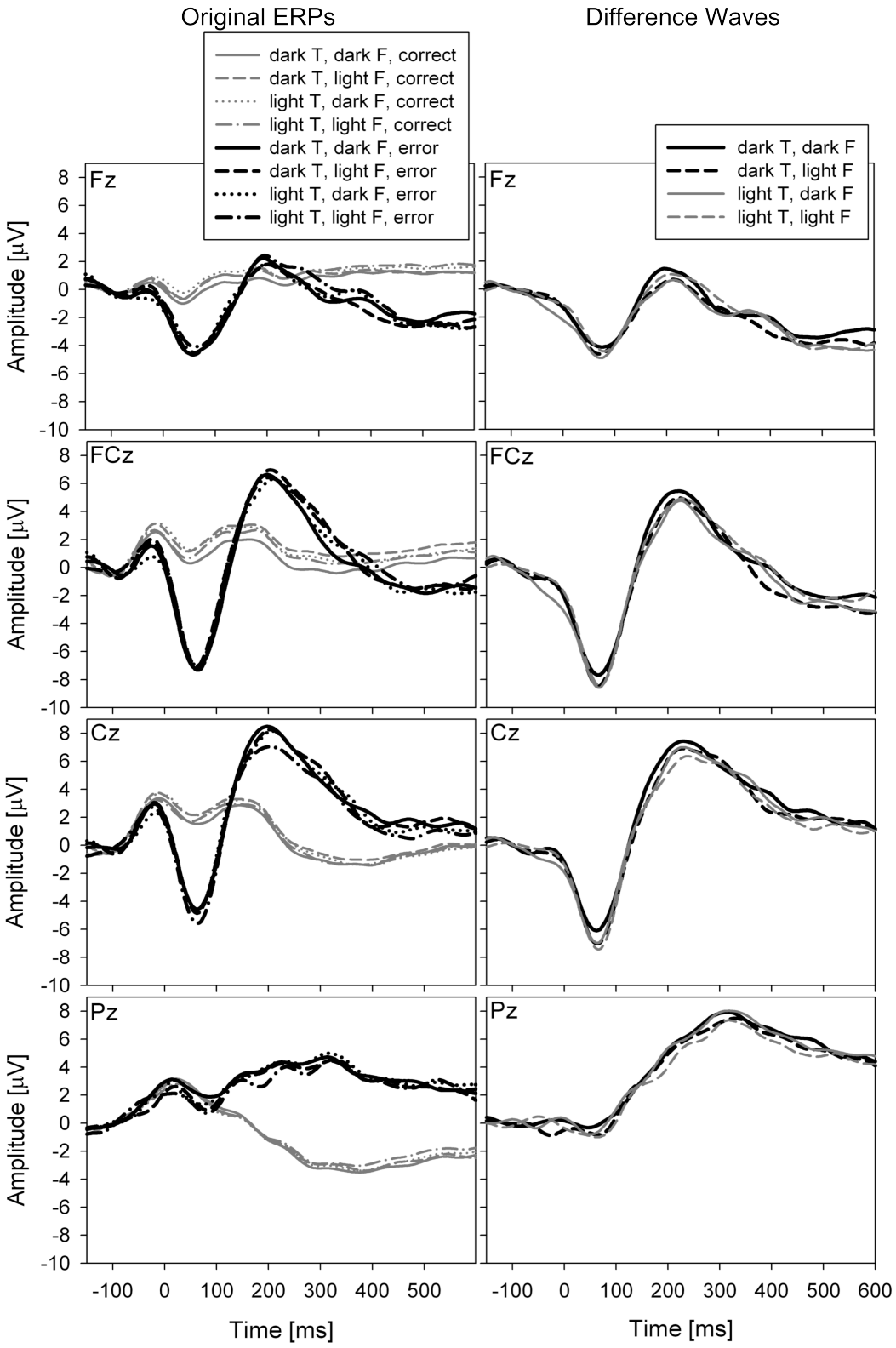

Figure 13: Response-locked ERPs for incompatible trials in the four contrast conditions at electrodes Fz, FCz, Cz, and Pz (T = target; F = flankers). Left: Correct (grey lines) and incorrect (black lines) response trials. Right: Difference waves (incorrect minus correct).

Ne/ERN: The Ne/ERN reached its maximum amplitude at about 66 ms after the response. Neither target nor flanker contrast nor their combination affected the amplitude of the ERN (all $p > .10$). There were also no significant latency differences between any of the conditions (all $p > .10$).

Early Pe: The early Pe peaked at 246 ms after the response. Neither amplitude nor latency was affected by the contrast manipulation (all ps $> .10$).

Late Pe: The amplitude of the late Pe was not affected by the contrast manipulation (all ps $> .10$).

3.1.3 Summary of the Main Results

The present study did show the standard compatibility, error speed-up, and post-error slowing effects. Post-error slowing was accompanied by an increase in post-error accuracy. The Gratton effect was only present for response repetitions but not for response alternations, thereby replicating an earlier finding of Mayr et al. (2003). The functional meaning of this result will be discussed in more detail in Section 3.3. The contrast manipulation did influence some of the behavioural effects. The compatibility effect was numerically but not significantly smaller in the condition with dark targets and light flankers compared to the other conditions. Note that this is the condition in which the least conflict was expected. The Gratton effect for response repetitions was reduced in conditions with mixed target-flanker contrast compared to conditions with the same contrast. This finding is not in line with the predictions of the conflict monitoring theory and will be discussed in more detail in Section 3.3. Neither error speed-up nor post-error slowing was affected by the contrast manipulation.

The P1 component peaked later for light than dark flankers over the right hemisphere and was also reduced in amplitude for light compared to dark flankers. The N1 peaked later for light than dark flankers as well as targets and was smaller for dark than light targets when flankers were dark. The stimulus-locked N2 peaked later for light than dark targets. The latency of this component might be linked to the reaction time, since participants also responded slower to light than dark targets. Furthermore, the latency effect was not present in the response-locked analysis of the N2. None of the error-related components were affected by the contrast manipulation.

3.2 Randomized Presentation of Contrast Conditions

The contrast conditions in the experiment described in Section 3.1 were presented in separate blocks. This was done to facilitate the analysis of sequential adjustment and post-error slowing effects. More specifically, the previous trial contrast did not have to be taken into consideration, since it was always the same as the current trial contrast. However, it is possible that the reported results were affected by the fact that participants might have used different response strategies (e.g., response thresholds) for different contrast conditions. This might have obscured certain patterns in the data that would be visible in a non-blocked, randomized design. Therefore, I replicated the behavioural part of the previous experiment with the four contrast conditions presented in a fully randomized order. The effects of contrast on the compatibility effect, sequential adjustment effects, and post-error slowing were investigated.

3.2.1 Methods

3.2.1.1 Participants

Twenty-four psychology students, aged 18 to 26 years (M = 19.4 years, 16 women), which had not participated in the first study, took part in this experiment. All participants had normal or corrected-to-normal vision, and gave written consent. The majority received course credits for their participation, the remaining participants volunteered as a favour to the experimenter. The study was approved by the University Teaching and Research Ethics Committee (UTREC) of the University of St Andrews (approval code: PS5099).

3.2.1.2 Stimuli and Apparatus

Stimuli and apparatus were the same as in the first study. However, in this experiment EEG activity was not recorded.

3.2.1.3 Procedure and Design

Procedure and design were identical to the first study with the exception of the following: In this experiment, the four contrast conditions were presented randomly within each block. The session started with a practice block of 32 trials, followed by twelve experimental blocks of 112 trials, each of which started with additional four

non-recorded warm-up trials. The overall number of recorded trials was 1344. Participants filled in a short questionnaire about their demographic information before they proceeded to the experiment.

3.2.1.4 Data Analysis

As in the first study, trials with reaction times under 150 ms and over 1500 ms were excluded from the analyses. All data were analysed using repeated-measures ANOVAs. Conservative Huynh-Feldt tests were used throughout. Adjusted *p*-values are reported, along with the uncorrected degrees of freedom. Bonferroni corrected *p*-values are reported for all post hoc analyses. Note that only the contrast of the previous trial was included in the analysis of sequential adjustment effects and post-error effects. The analyses are therefore not identical to the ones described in Sections 3.1.2.1.2 and 3.1.2.1.3, since in that study current and previous trial contrast were always identical.

3.2.2 Results

3.2.2.1 Compatibility Effects

Error rates and reaction times are presented in *Figure 14* and were analysed using repeated-measures ANOVAs with the within-subject factors Compatibility (compatible, incompatible), Target Contrast (dark, light) and Flanker Contrast (dark, light). Only trials with correct responses on the previous and the current trial were included in the reaction times analysis.

Reaction times: Participants responded significantly faster to compatible (368 ms) than to incompatible trials (411 ms), $F(1, 23) = 204.95$, $p < .001$, $\eta_p^2 = .90$. Reaction times for dark targets (385 ms) were faster than for light targets (394 ms), $F(1, 23) = 53.98$, $p < .001$, $\eta_p^2 = .70$, and reaction times for light flankers (388 ms) were faster than for dark flankers (391 ms), $F(1, 23) = 11.97$, $p = .002$, $\eta_p^2 = .34$. The significant Target Contrast X Flanker Contrast interaction, $F(1, 23) = 9.19$, $p = .006$, $\eta_p^2 = .29$, reflects that participants only responded significantly faster to dark than to light targets when the flankers were light (380 ms and 395 ms, respectively), $F(1, 23) = 51.90$, $p < .001$, $\eta_p^2 = .69$, but not when the flankers were dark (390 ms and 393 ms, respectively; $p > .10$). The compatibility effect (incompatible minus compatible) was larger for light targets (47 ms) than for dark

targets (41 ms), $F(1, 23) = 6.46$, $p = .018$, $\eta_p^2 = .22$, and for dark flankers (49 ms) than for light flankers (39 ms), $F(1, 23) = 12.57$, $p = .002$, $\eta_p^2 = .35$. A significant three-way interaction, $F(1, 23) = 11.37$, $p = .003$, $\eta_p^2 = .33$, reflected a smaller compatibility effect for light than dark flankers when targets were dark (51 ms and 31 ms, respectively), $F(1, 23) = 22.55$, $p < .001$, $\eta_p^2 = .50$, but not when targets were light (47 ms and 46 ms, respectively; $p > .10$).

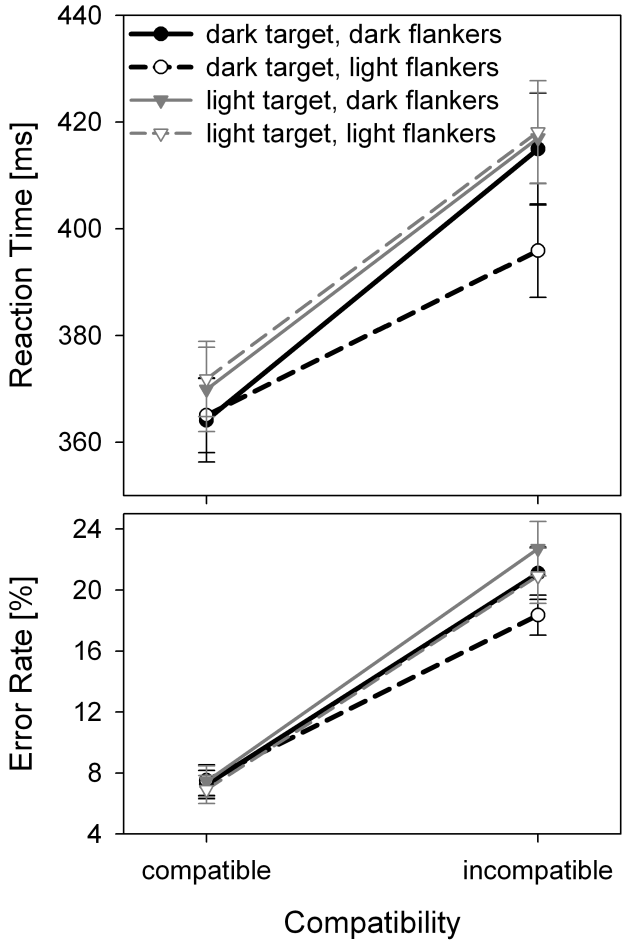

Figure 14: Compatibility effect on reaction times and error rates in the four contrast conditions.

Error rates: Overall participants committed an average of 14.0% errors, 7.3% on compatible trials and 20.8% on incompatible trials. This compatibility effect was significant, $F(1, 23) = 147.32$, $p < .001$, $\eta_p^2 = .87$. Participants also committed more errors for light targets (14.5%) than dark targets (13.6%), $F(1, 23) = 6.05$, $p = .022$, $\eta_p^2 = .21$, and for dark flankers (14.6%) than light flankers (13.4%), $F(1, 23) = 7.96$,

p = .010, η_p^2 = .26. The compatibility effect (incompatible minus compatible) was larger for light targets (14.6%) than for dark targets (12.3%), $F(1, 23) = 5.60$, p = .027, η_p^2 = .20, and also larger for dark flankers (14.6%) than for light flankers (12.4%), $F(1, 23) = 9.25$, p = .006, η_p^2 = .29. There was no significant interaction of the two contrast types and no significant three-way interaction (both ps > .10).

3.2.2.2 Sequential Adjustment Effects

Sequential adjustment effects (see *Figure 15*) were analysed using repeated-measures ANOVAs with the within-subject factors Trial N-1 Compatibility (compatible, incompatible), Current Trial Compatibility (compatible, incompatible), Trial N-1 Target Contrast (dark, light), Trial N-1 Flanker Contrast (dark, light) and Response Sequence (alternation, repetition). Only interactions involving the factor Trial N-1 Compatibility are reported in this section. The contrast of the current trial was not taken into account, since realizing all possible combinations of trial N-1 and trial N contrast would result in conditions with an insufficient number of trials. Furthermore, the contrast of the previous trial is of greater importance than current trial contrast in the analysis of sequential effects because the size of the Gratton effect would be expected to be influenced by previous trial contrast if contrast indeed manipulates conflict and the Gratton effect were due to control adjustments following the experience of conflict.

Reaction times: Participants reacted overall faster when the previous trial had been compatible (387 ms) than when it had been incompatible (394 ms), $F(1, 23) = 42.05$, $p < .001$, η_p^2 = .65. This effect was modulated by the contrast manipulation as indicated by a significant three-way interaction between Trial N-1 Compatibility, Trial N-1 Target Contrast and Trial N-1 Flanker Contrast, $F(1, 23) = 4.68$, p = .041, η_p^2 = .17. Importantly, there was a significant interaction between Trial N-1 and Current Trial Compatibility, $F(1, 23) = 29.64$, $p < .001$, η_p^2 = .56, representing the Gratton effect, which in turn interacted with the factor Response Sequence, $F(1, 23) = 47.72$, $p < .001$, η_p^2 = .68. Post hoc tests showed that there was only a significant Gratton effect ((c-ic – c-c) – (ic-ic – ic-c)) for response repetitions (35 ms), $F(1, 23) = 61.25$, $p < .001$, η_p^2 = .73, but not for response alternations (-6 ms; $p > .10$). None of the other effects reached significance (all ps > .05).

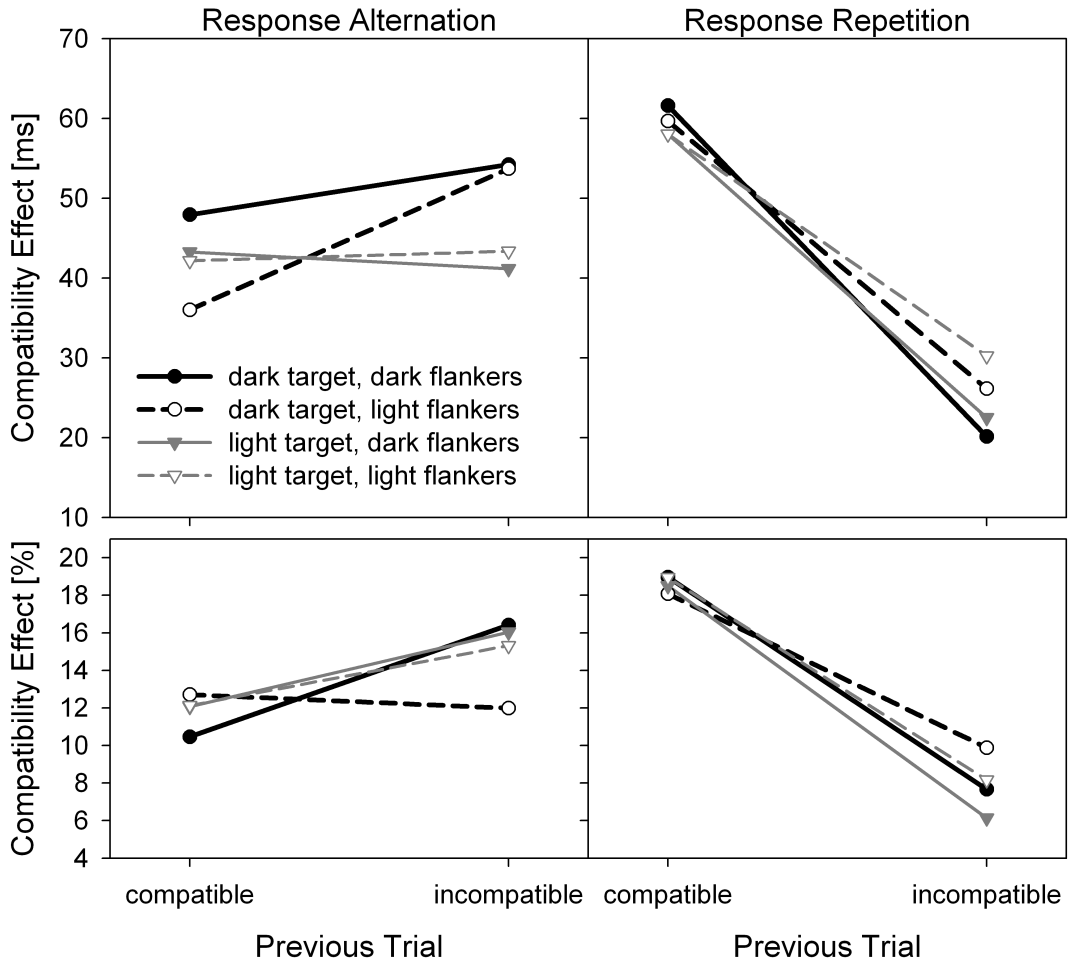

Figure 15: Gratton effect for reaction times (top) and error rates (bottom): Compatibility effect size (incompatible minus compatible) following compatible and incompatible trials for response alternations (left) and repetitions (right) in the four contrast conditions. A steeper slope indicates a larger Gratton effect. Note that the contrast condition refers to trial N-1.

Error rates: There was a significant interaction between Trial N-1 Compatibility and Response Sequence, $F(1, 23) = 19.38$, $p < .001$, $\eta_p^2 = .46$; participants made more errors following incompatible (16.4%) than compatible trials (13.6%) for response alternations, $F(1, 23) = 15.84$, $p = .002$, $\eta_p^2 = .41$, but less errors following incompatible (11.8%) than compatible trials (14.4%) for response repetitions, $F(1, 23) = 8.41$, $p = .016$, $\eta_p^2 = .27$. There also was a significant Gratton effect (interaction of Trial N-1 Compatibility and Current Trial Compatibility), $F(1, 23) = 9.56$, $p = .005$, $\eta_p^2 = .29$, that in turn interacted with the factor Response Sequence, $F(1, 23) = 46.87$, $p < .001$, $\eta_p^2 = .67$. Post hoc tests showed a significant

Gratton effect ((c-ic – c-c) – (ic-ic – ic-c)) for response repetitions (10.7%), $F(1, 23) = 32.19$, $p < .001$, $\eta_p^2 = .58$, and a reverse Gratton effect for response alternations (-3.1%), $F(1, 23) = 6.45$, $p = .037$, $\eta_p^2 = .22$. No other effects reached significant levels (all *p*s > .05).

3.2.2.3 Error and Post-Error Effects

Reaction times for correct responses following another correct response (post-correct), errors following a correct response (error) and correct responses following an erroneous response (post-error) are shown in *Figure 16*. Error speed-up and post-error slowing were analysed in a repeated-measures ANOVA with the within-subjects factors Trial Type (post-correct, error, post-error), Trial N-1 Target Contrast (dark, light), and Trial N-1 Flanker Contrast (dark, light). Post-error accuracy was analysed using a repeated-measures ANOVA with the within-subjects factors Trial N-1 Accuracy (correct, incorrect), Trial N-1 Target Contrast (dark, light), and Trial N-1 Flanker Contrast (dark, light). Only trials following incompatible trials were included in these analyses.

Reaction times: The main effect of Trial Type was significant, $F(2, 46) = 107.74$, $p < .001$, $\eta_p^2 = .82$. Post hoc tests showed that post-error trials (420 ms) were significantly slower, $F(1, 23) = 25.11$, $p < .001$, $\eta_p^2 = .52$, and error trials (337 ms) were significantly faster than post-correct trials (392 ms), $F(1, 23) = 216.38$, $p < .001$, $\eta_p^2 = .90$, thereby confirming the expected post-error slowing and error speed-up effects. Participants reacted faster following light flankers (380 ms) than following dark flankers (386 ms), $F(1, 23) = 6.30$, $p = .020$, $\eta_p^2 = .22$. None of the other effects reached significance (all *p*s > .10).

Error rates: Participants committed less errors following incorrect (9.3%) than following correct responses (11.9%), $F(1, 23) = 10.77$, $p = .003$, $\eta_p^2 = .32$, indicating increased post-error accuracy. None of the other effects reached significance (all *p*s > .10).

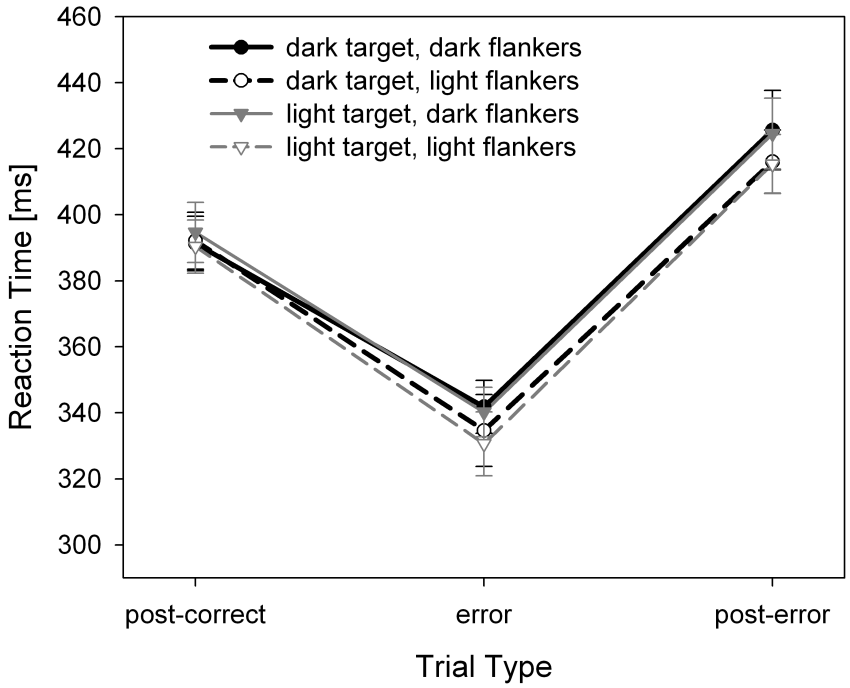

Figure 16: Error speed-up and post-error slowing in the four contrast conditions. Note that the contrast condition refers to trial N-1.

3.3 Discussion

The aim of this chapter was to investigate the effects of conflict size on behavioural control adjustment effects and conflict- and error-related ERP components. Overall, the contrast manipulation was partly successful in influencing conflict. In the ERP study, the compatibility effect was slightly, but not significantly, smaller for light than dark flankers when targets were dark. This effect became significant when the randomized design of the behavioural study was used. The compatibility effect was significantly reduced for light compared to dark flankers when the targets were dark. As expected, reducing the contrast of the flanker letters compared to the target reduced the amount of conflict in information processing. This is in line with the conflict monitoring theory, which predicts reduced conflict when the incompatible response representation associated with the flanker letters receives less activation than the target-associated response representation (e.g. Berlyne, 1957; Botvinick et al., 2001). On the other hand, there was no significant difference for dark and light flankers when the target was light. Therefore, the contrast manipulation failed to increase conflict by reducing the contrast of the target letter relative to the flankers. A possible explanation for this is that a pop-out effect occurred in trials with light

targets and dark flankers that facilitated the task and counteracted the effects of conflict. That is, a light target letter between dark flanker letters might stand out and, therefore, it might have been easier for participants to focus on the target, reducing the distracting influence of the flankers. Nevertheless, the randomized presentation of contrast conditions was clearly more successful in manipulating conflict than the blocked design. A possible reason for this is that participants got used to the contrast in the blocked design and the visual system adjusted itself accordingly. Indeed it has been shown that training with low contrast stimuli over the course of several weeks can improve visual performance (Polat et al., 2012). It is possible that similar processes on a much shorter time scale have been at work in this experiment thereby reducing the effect of contrast.

The contrast manipulation had effects on both the P1 and the N1 component. The latency of the N1 was longer for light than dark flankers and for light than dark targets. The P1 also peaked later for light compared to dark flankers, at least over the right hemisphere. These findings indicate prolonged early visual processing for stimuli with reduced contrast. The amplitude effects of P1 and N1 are less clear. Whereas the P1 amplitude was larger for dark than light flankers, the N1 amplitude was smaller for dark than light flankers and especially small in the condition where all letters were dark.

The results of the N2 analysis revealed a clear presence of this component in all conflict trials. Since it had been suggested previously that the N2 should in fact be time-locked to the response (e.g., Nieuwenhuis et al., 2003; Yeung et al., 2004), the N2 was also analysed response-locked. Overall, the N2 had smaller amplitudes in the response-locked analysis, probably due to increased latency jitter, contradicting the hypothesis that the N2 represents response conflict on correct trials as suggested by Yeung et al. (2004), since conflict should be maximal immediately before the response. The amplitude of the N2 did not differ between the contrast conditions. However, this result should be treated with caution, since the conflict manipulation was not successful in the ERP study.

The analysis of sequential adjustment effects revealed some surprising results. In both studies, Gratton effects were only present for response repetitions but not for response alternations, thereby confirming the results of Mayr et al. (2003). The Gratton effect in the two studies described in this chapter seems to be due to

stimulus-specific repetition priming, as suggested by Mayr and colleagues, and not due to conflict adaptation as assumed in other studies (e.g., Botvinick et al., 1999; Botvinick et al., 2001). That is, compatible trials might be faster following compatible than incompatible trials and incompatible trials might be faster following incompatible than compatible trials because the exact same stimulus is repeated in these cases, leading to facilitated processing of the current trial due to episodic memory effects. Furthermore, the Gratton effect did not vary as a function of previous trial conflict size, further supporting the suggestion that the Gratton effect reported here is not linked to adaptive control adjustments after high conflict. More specifically, although stimulus contrast influenced conflict size in the behavioural study, as indicated by its effect on the compatibility effect size, there were no significant interactions between stimulus contrast on the previous trial and previous and current trial compatibility. Decreased conflict on incompatible trials with a dark target and light flankers should have resulted in a smaller increase in attentional control than in other contrast conditions and, therefore, a smaller decrease in the compatibility effect on the following trial, which was not found. Interestingly, the contrast manipulation did affect the Gratton effect for response repetitions in the ERP study with the blocked stimulus contrast design. Sequential adjustment was reduced in the blocks with mixed target-flanker contrast (dark target – light flankers, light target – dark flankers) compared to the blocks where target and flankers were of the same contrast (dark target – dark flankers, light target – light flankers). A possible explanation for this effect could be that memory traces were stronger due to perceptual grouping when all letters had the same contrast (e.g., Ben-Av & Sagi, 1995; Palmer, Brooks, & Nelson, 2003) than when contrast was mixed. Stronger memory traces would lead to stronger priming effects in cases where the exact same stimulus is repeated, i.e. for response repetitions when a compatible trial follows another compatible trial and when an incompatible trial follows another incompatible trial. Faster reaction times in these two cases for conditions with the same contrast compared to conditions with mixed contrast would lead to a larger Gratton effect. Interestingly, this could also explain why this effect of contrast on priming was only found in the blocked but not in the randomized design. More specifically, since contrast conditions were randomized in the behavioural study, exact stimulus repetitions were rare. Even in conditions were the same letter stimulus was repeated

the contrast of both stimuli differed in most cases, thereby eliminating the effect of stronger memory traces in same-contrast conditions.

The analysis of error-related behavioural data in both studies showed the expected speed-up on error trials and the post-error slowing effect. Post-error slowing was accompanied by an increase in post-error accuracy compared to trials following a correct response. This pattern of results is compatible with the hypothesis that post-error slowing is due to an adjustment of a response criterion to a more conservative level to avoid further errors (e.g., Brewer & Smith, 1984; Jentzsch & Leuthold, 2006; Laming, 1968). Error speed-up and post-error slowing were not affected by the contrast manipulation in either of the two studies. According to the conflict monitoring theory, post-error slowing represents an adjustment of control due to the conflict associated with an error. Errors in the condition with reduced conflict (i.e., dark target – light flankers) should have been associated with more conflict in a time window immediately following the error than errors in the condition with dark targets and flankers, since the correct response representation can be expected to receive more activation due to continued stimulus processing after the response (cf. Danielmeier et al., 2009). Accordingly, post-error control adjustments would have been predicted to be enhanced, especially in the behavioural study where contrast significantly influenced conflict size; however, this was not the case. It seems that the internal error signal triggered post-error adjustments to the same degree in all contrast conditions.

The results of the error-related ERP analysis showed three distinct components with different topographies: the Ne/ERN with a fronto-central distribution, an early Pe with a more central distribution, and a late Pe with a posterior distribution. None of these components were affected by the contrast manipulation. Whereas the conflict monitoring theory does not make any predictions about the error positivities, the Ne/ERN was predicted to be increased in amplitude in the low conflict condition (cf. Danielmeier et al., 2009, and Section 1.4.2). However, the lack of significant effects of contrast on the Ne/ERN should be interpreted with caution, since its effects on the compatibility effect did not reach significance either.

In conclusion, the two studies show that stimulus contrast can be used to manipulate conflict in the flanker task, however, not as easily and effectively as

expected. Randomizing the contrast conditions within blocks, instead of using a blocked contrast design, increased the influence of contrast on the compatibility effect. Nevertheless, the contrast manipulation was only effective in decreasing conflict relative to the standard version of the task when the influence of the flankers was weakened but not in increasing conflict when the influence of the target relative to the flankers was weakened. Reducing the absolute activation of response representations by reducing flanker and target contrast also did not seem to influence conflict strength. Therefore, I will take a different approach to manipulate conflict in the following chapter. Furthermore, the current experiments showed that the Gratton effect is probably not due to control adjustments but to response priming and that sequential adjustment effects should therefore be analysed separately for response alternations and repetitions.

4 Effects of Stimulus Onset Asynchrony in the Eriksen Flanker Task

The aim of the two studies described in this chapter was to find a different way to manipulate conflict strength in the flanker task within subjects. Previous research has shown that the interval between flanker and target onset (stimulus onset asynchrony, SOA; see *Figure 17*A for an illustration of that concept) influences the size of the compatibility effect; the effect has been shown to be largest when flanker onset precedes target onset by about 100 ms (e.g., Mattler, 2003; Wascher et al., 1999; Willemssen et al., 2004). Although these authors did not interpret their results in terms of the conflict monitoring theory, it could be argued that conflict might be enhanced when flankers are presented shortly before the target, since the response representation associated with the flankers receives earlier and, therefore, stronger activation, which is expected to interfere with the target-associated response representation when flankers and target are incompatible. When the SOA interval gets too long, flanker-related activity might already be partly inhibited by the time the target is presented. Accordingly, compatibility effects should get smaller again with larger SOAs. Results of previous studies are in line with these predictions (e.g., Mattler, 2003; Wascher et al., 1999; Willemssen et al., 2004; see also Section 1.3.1). In the two studies described in this chapter, I used three different SOAs: 0 ms (i.e., simultaneous flanker and target onset), 100 ms and 200 ms. The predicted amount of conflict in these three SOA conditions is depicted in *Figure 17*B. Furthermore, I included neutral flankers in addition to compatible and incompatible flankers, i.e. flanker letters that are not associated with either response hand. According to the conflict monitoring theory, reaction times and error rates should be intermediate in this neutral condition because the flankers should neither facilitate nor interfere with target processing. The current chapter describes a behavioural pilot study, investigating effects of SOA on the compatibility effect, the Gratton effect, and post-error effects, as well as an ERP study additionally investigating effects of conflict on the N2 component and error-related ERP components.

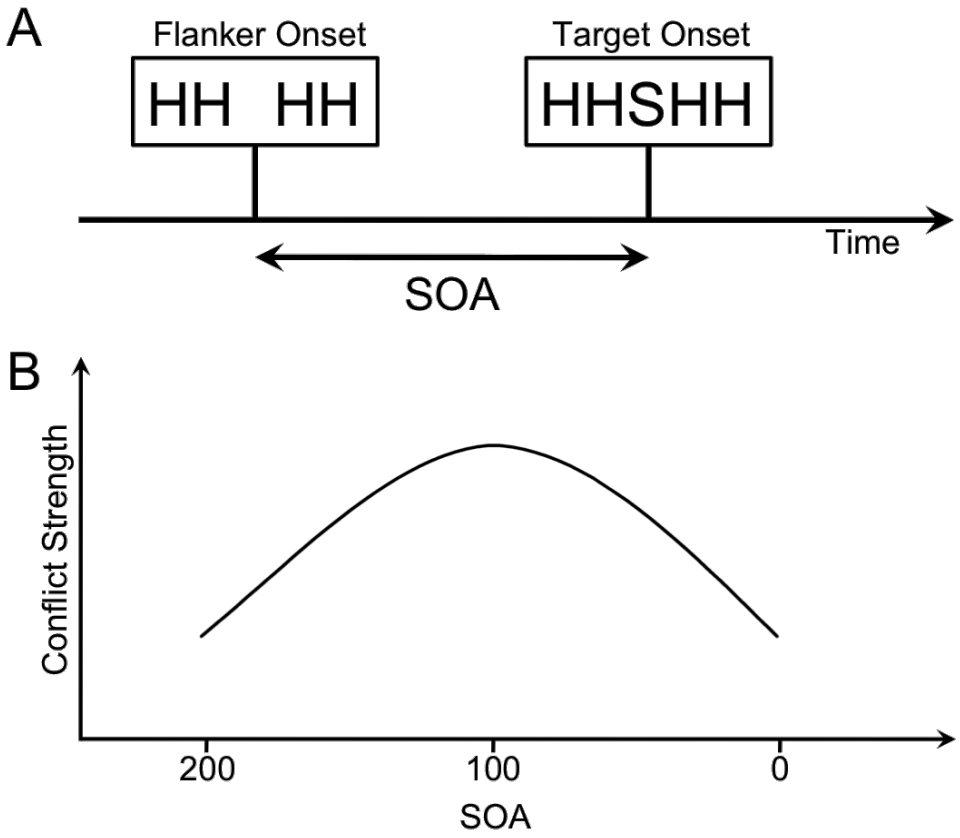

Figure 17: Schematic illustration of stimulus onset asynchrony (A) and predicted conflict strength for the different SOA conditions used in this chapter (B).

4.1 Behavioural Pilot Study

This pilot study was carried out in order to establish that the expected behavioural effects were present in the paradigm before conducting a more extensive ERP study. Methods and results of this study will be presented in this section. The results will be discussed in Section 4.3 together with the results of the ERP study.

4.1.1 Methods

4.1.1.1 Participants

Eighteen psychology undergraduate students, aged 18 to 21 years (M = 19.1 years, 16 women), were tested in this experiment in a single session of about 50 minutes duration. They all had normal or corrected-to-normal vision, gave written consent, and received course credits for their participation. The study was approved by the University Teaching and Research Ethics Committee (UTREC) of the University of St Andrews (approval code: PS5099).

4.1.1.2 Stimuli and Apparatus

The same apparatus as in the first experiment was used for stimulus presentation and response indication. The stimuli consisted of five-letter-strings using the letters S, H, and O. One of these letters was assigned to left hand responses, another one to right hand responses, and the third one was neutral, i.e. not associated with either response alternative. The assignment of letters to response hands was counter-balanced across participants. The central letter (target) always corresponded to a response hand; the flanker letters were identical (compatible), neutral, or belonged to the opposite response set (incompatible). All three kinds of stimuli appeared with the same frequency and were presented in black (0.46 cd/m^2) on white background (66.31 cd/m^2). The approximate stimulus size was 45 x 10 mm.

4.1.1.3 Procedure and Design

Participants completed 18 blocks of 72 trials in randomized order, resulting in 1296 trials overall. Every block started with six non-recorded warm-up trials. In six blocks target and flankers appeared at the same time (SOA 0), in another six blocks flankers appeared 100 ms before the target (SOA 100), and in the remaining six blocks flanker onset preceded target onset by 200 ms (SOA 200). Blocks were presented alternating between SOAs. The order of blocks was counter-balanced across participants using a Latin square procedure. Before starting with the experimental blocks, participants completed three short practice blocks consisting of 18 trials, one block for each SOA.

As in the previous experiments, participants were asked to identify the central letter by pressing the corresponding response key. Stimuli were presented until the participant responded or, if no response was made, until 1500 ms after target onset. The RSI was 1000 ms, i.e. flanker onset of the next trial occurred 1000 ms after response execution. Instructions and feedback were the same as in the first experiment.

4.1.1.4 Data Analysis

Only trials with reaction times between 150 ms and 1500 ms were included in the analyses. As in the first two studies, post-error accuracy was determined by dividing the number of errors following an error by the total number of trials following an

error and multiplied by one hundred. This was then compared to the number of errors following correct responses divided by the total number of trials following correct responses and multiplied by one hundred.

All data were analysed using repeated-measures ANOVAs. Conservative Huynh-Feldt tests were used throughout. Adjusted *p*-values are reported, along with the uncorrected degrees of freedom. Bonferroni corrected *p*-values are reported for all post hoc analyses.

4.1.2 Results

4.1.2.1 Compatibility Effects

Compatibility effects on error rates and reaction times are shown in *Figure 18* and were analysed using repeated-measures ANOVAs with the factors Compatibility (compatible, neutral, incompatible) and SOA (0 ms, 100 ms, 200 ms). Only trials with correct responses on the current and the previous trial were included in the reaction time analysis.

Reaction times: As can be seen in *Figure 18* (top panel), there was a main effect of Compatibility on reaction time, $F(2, 34) = 228.65$, $p < .001$, $\eta_p^2 = .56$. Reaction times increased reliably from compatible (316 ms) to neutral (348 ms), $F(1, 17) = 117.87$, $p < .001$, $\eta_p^2 = .87$, and from neutral to incompatible trials (376 ms), $F(1, 17) = 146.12$, $p < .001$, $\eta_p^2 = .89$. There also was a main effect of the factor SOA, $F(2, 34) = 46.63$, $p < .001$, $\eta_p^2 = .73$, due to a reliable decrease in reaction time from SOA 0 (370 ms) over SOA 100 (343 ms) to SOA 200 (327 ms), Fs$(1, 17) \geq 21.04$, ps $< .001$, η_p^2s $\geq .55$. Importantly, there was a significant interaction between the factors Compatibility and SOA, $F(4, 68) = 21.38$, $p < .001$, $\eta_p^2 = .56$. Post hoc analyses showed that facilitation (neutral minus compatible) was smaller in the SOA 0 condition (12 ms) than in the SOA 100 (44 ms), $F(1, 17) = 64.82$, $p < .001$, $\eta_p^2 = .79$, and SOA 200 (40 ms) conditions, $F(1, 17) = 29.36$, $p < .001$, $\eta_p^2 = .63$. The facilitation effect did not differ between SOA 100 and SOA 200 ($p > .10$). Although interference (incompatible minus neutral) was numerically larger in the SOA 100 condition (36 ms) than in the SOA 0 and SOA 200 conditions (25 ms and 23 ms, respectively), the differences did not reach significance (all $ps > .10$).

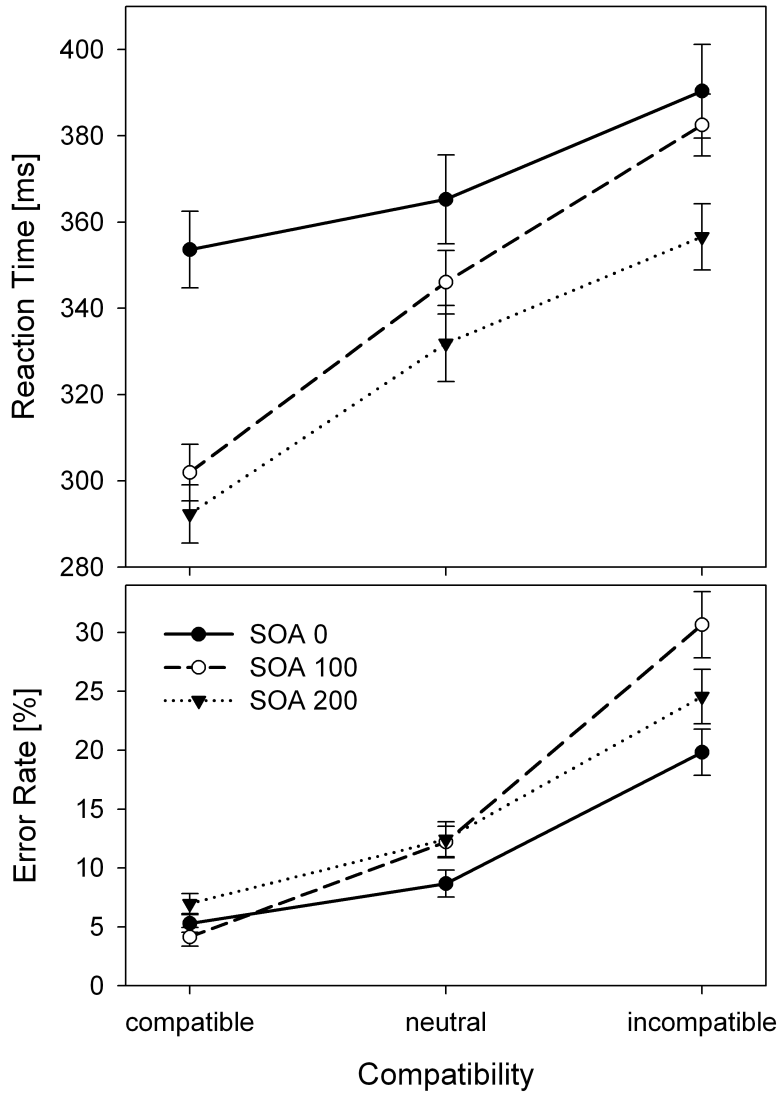

Figure 18: Compatibility effects on reaction times (top) and error rates (bottom) in the three SOA conditions.

Error rates: Participants committed 5.5% errors on compatible trials, 11.1% errors on neutral trials and 25.0% errors on incompatible trials, $F(2, 34) = 97.60$, $p < .001$, $\eta_p^2 = .85$. Post-hoc tests showed that the error rate reliably increased from compatible to neutral trials, $F(1, 17) = 48.62$, $p < .001$, $\eta_p^2 = .74$, and from neutral to incompatible trials, $F(1, 17) = 114.80$, $p < .001$, $\eta_p^2 = .87$. Differences in error rates were also found for the three SOAs, $F(2, 34) = 11.82$, $p = .001$, $\eta_p^2 = .41$. The error rate of 11.3% for SOA 0 was significantly lower than the error rate of 15.7% for SOA 100, $F(1, 17) = 23.18$, $p < .001$, $\eta_p^2 = .58$, and the error rate of 14.6% for SOA 200, $F(1, 17) = 10.25$, $p = .016$, $\eta_p^2 = .38$. The error rates of SOA 100 and SOA 200

did not differ significantly ($p > .10$). Importantly, there was a significant interaction between SOA and Compatibility, $F(4, 68) = 11.36$, $p < .001$, $\eta_p^2 = .40$. Post hoc test showed that facilitation (neutral minus compatible) was larger in the SOA 100 than in the SOA 0 condition, $F(1, 17) = 13.35$, $p = .012$, $\eta_p^2 = .44$. There were no significant differences in facilitation between SOA 0 and SOA 200 or between SOA 100 and SOA 200 (both ps $> .10$). Interference (incompatible minus neutral) was larger for SOA 100 than for SOA 0, $F(1, 17) = 10.91$, $p = .025$, $\eta_p^2 = .39$, and SOA 200, $F(1, 17) = 12.44$, $p = .016$, $\eta_p^2 = .42$, but did not differ significantly between SOA 0 and SOA 200 ($p > .10$).

4.1.2.2 Sequential Adjustment Effects

Sequential adjustment effects on reaction times (*Figure 19*) and error rates (*Figure 20*) were analysed using repeated-measures ANOVAs with the within-subjects factors Trial N-1 Compatibility (compatible, neutral, incompatible), Current Trial Compatibility (compatible, neutral, incompatible), SOA (0 ms, 100 ms, 200 ms), and Response Sequence (alternation, repetition). Only trials with correct responses on the current and the previous trial were included in the reaction time analysis. Only interactions including the factor Trial N-1 Compatibility will be reported in this section.

Reaction times: Participants responded faster for response repetitions (341 ms) than alternations (354 ms), $F(1, 17) = 6.96$, $p = .017$, $\eta_p^2 = .29$. There was a significant main effect of Trial N-1 Compatibility, $F(2, 34) = 6.72$, $p = .003$, $\eta_p^2 = .28$, and a significant interaction between Trial N-1 Compatibility, SOA and Response Sequence, $F(4, 68) = 3.24$, $p = .027$, $\eta_p^2 = .16$. Importantly, the interaction between Trial N-1 Compatibility and Current Trial Compatibility was significant, $F(4, 68) = 12.98$, $p < .001$, $\eta_p^2 = .43$, and in turn interacted with the factor Response Sequence, $F(4, 68) = 13.83$, $p < .001$, $\eta_p^2 = .45$. Post hoc tests showed significant Trial N-1 Compatibility X Current Trial Compatibility interactions for response repetitions, $F(4, 68) = 23.26$, $p < .001$, $\eta_p^2 = .58$, as well as response alternations, $F(4, 68) = 4.30$, $p = .007$, $\eta_p^2 = .20$. *Figure 19* and further post hoc tests suggested that the Trial N-1 Compatibility X Current Trial Compatibility interaction for response alternations was not due to the typical Gratton effect (i.e., a reduced compatibility effect following incompatible compared to compatible trials) but due to

occurrences on neutral trials.[15] None of the other effects involving factor Trial N-1 Compatibility reached significance (all *p*s > .10).

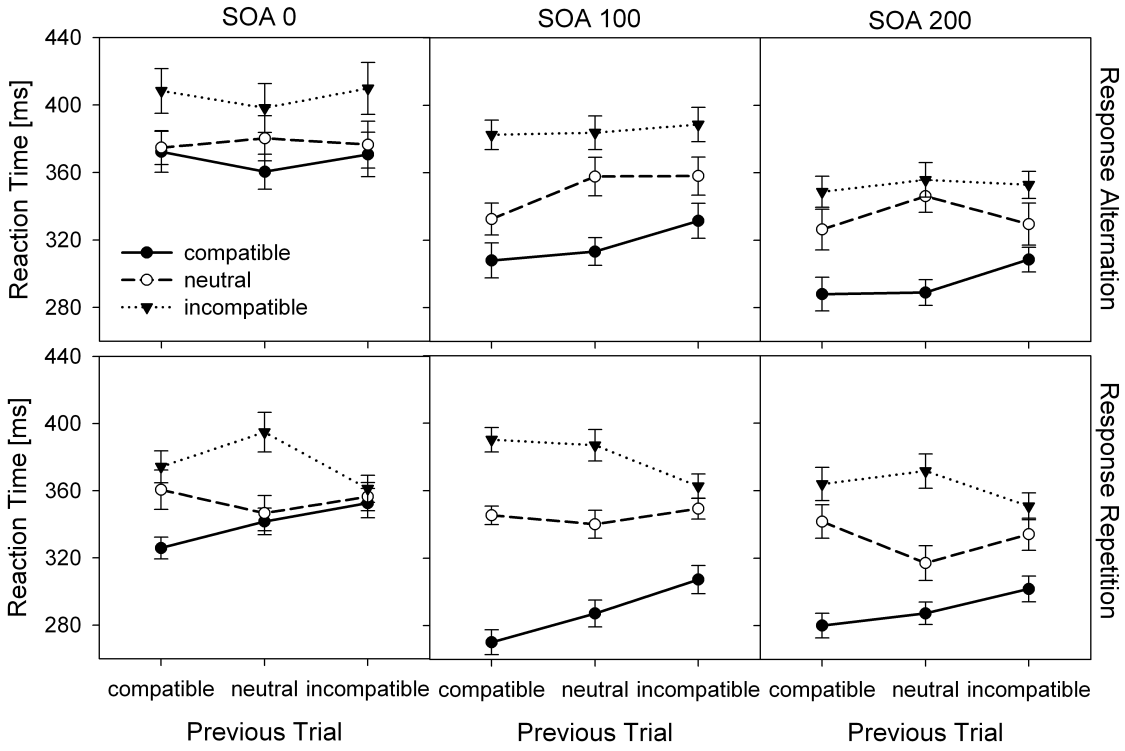

Figure 19: Gratton effect: Reaction times as a function previous and current trial compatibility in the three SOA conditions for response alternations (top) and response repetitions (bottom).

Error rates: There was a significant effect of Trial N-1 Compatibility, $F(2, 34) = 5.06$, $p = .012$, $\eta_p^2 = .23$, and a significant interaction between this factor and the factor Response Sequence, $F(2, 34) = 5.25$, $p = .010$, $\eta_p^2 = .24$. Importantly, the interaction between Trial N-1 Compatibility and Current Trial Compatibility was significant, $F(4, 68) = 14.02$, $p < .001$, $\eta_p^2 = .45$, and interacted in turn with the factor Response Sequence, $F(4, 68) = 9.90$, $p < .001$, $\eta_p^2 = .37$. Although the Gratton effect ((c-ic – c-c) – (ic-ic – ic-c)) was numerically smaller for response alternations

[15] When current neutral trials were excluded from the analysis the interaction between Trial N-1 Compatibility and Current Trial Compatibility was only significant for response repetitions, $F(2, 34) = 28.22$, $p < .001$, $\eta_p^2 = .62$, but not for response alternations ($p > .10$).

(3.2%) than repetitions (14.8%), post hoc tests showed that the Trial N-1 Compatibility X Current Trial Compatibility interaction was still significant for response alternations, $F(4, 68) = 3.76$, $p = .039$, $\eta_p^2 = .18$, not only for repetitions, $F(4, 68) = 22.16$, $p < .001$, $\eta_p^2 = .57$. As can be seen in *Figure 20*, the interaction for response alternations was not due to the usual Gratton effect (i.e., a smaller compatibility effect following incompatible than compatible trials) but due to occurrences on neutral trials. This was confirmed by further post hoc tests.[16] None of the other effects involving the factor Trial N-1 Compatibility reached significance (all *p*s > .10).

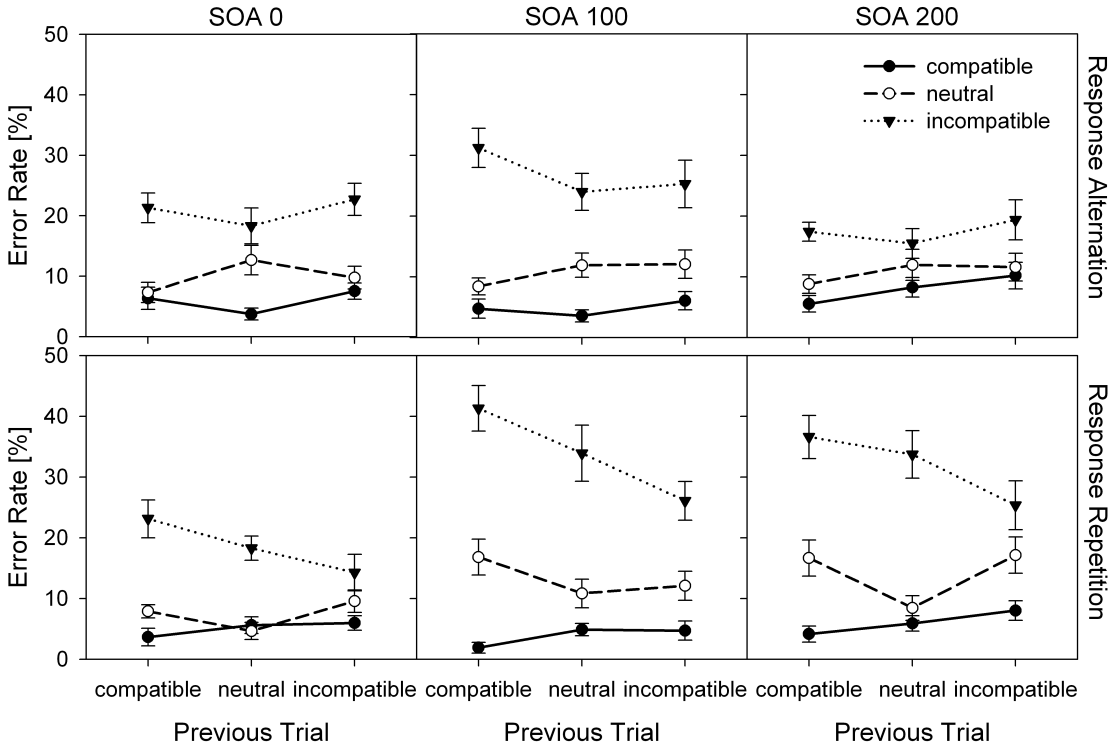

Figure 20: Gratton effect: Error rates as a function of previous and current trial compatibility in the three SOA conditions for response alternations (top) and response repetitions (bottom).

[16] When current neutral trials were excluded from the analysis, the Trial N-1 Compatibility X Current Trial Compatibility interaction did only reach significance for response repetitions, $F(2, 34) = 36.62$, $p < .001$, $\eta_p^2 = .68$, but not for response alternations ($p > .10$).

4.1.2.3 Error and Post-Error Effects

Figure 21 shows the reaction times for correct responses following correct responses (post-correct), errors following correct responses (error), and correct responses following an error (post-error). Error speed-up and post-error slowing were analysed using a repeated-measures ANOVA with the within-subjects factors Trial Type (post-correct, error, post-error) and SOA (0 ms, 100 ms, 200 ms). Post-error accuracy was analysed using a repeated-measures ANOVA with the within-subjects factors Trial N-1 Accuracy (correct, incorrect) and SOA (0 ms, 100 ms, 200 ms). Only trials following incompatible trials were included in these analyses because extremely few errors were committed on compatible trials.

Reaction times: A significant main effect of Trial Type, $F(2, 34) = 120.05$, $p < .001$, $\eta_p^2 = .88$, indicated significant error-speed-up and post-error slowing effects. Error trials (287 ms) were significantly faster, $F(1, 17) = 163.67$, $p < .001$, $\eta_p^2 = .91$, and post-error trials (367 ms) were significantly slower, $F(1, 17) = 15.37$, $p = .002$, $\eta_p^2 = .48$, than post-correct trials (348 ms). The main effect of SOA was also significant, $F(2, 34) = 36.50$, $p < .001$, $\eta_p^2 = .68$, as well as the interaction of both factors, $F(4, 68) = 3.63$, $p = .018$, $\eta_p^2 = .18$. Post hoc test showed that the error speed-up effect (post-correct minus error) tended to be larger for SOA 100 (77 ms) than SOA 0 (45 ms), $F(1, 17) = 7.63$, $p = .080$, $\eta_p^2 = .31$. Error speed-up in SOA 200 (62 ms) did not differ significantly from the other two SOAs (both *p*s > .10). Post-error slowing (post-error minus post-correct) was larger for SOA 0 (35 ms) than SOA 100 (11 ms), $F(1, 17) = 12.50$, $p = .015$, $\eta_p^2 = .42$, and marginally larger for SOA 0 than SOA 200 (10 ms), $F(1, 17) = 7.86$, $p = .073$, $\eta_p^2 = .32$. Post-error slowing did not differ significantly between SOA 100 and SOA 200 ($p > .10$).

Error rates: Participant committed 10.4% errors following correct trials and 9.8% errors following incorrect trials. None of the effects reached significance (all *p*s > .05).

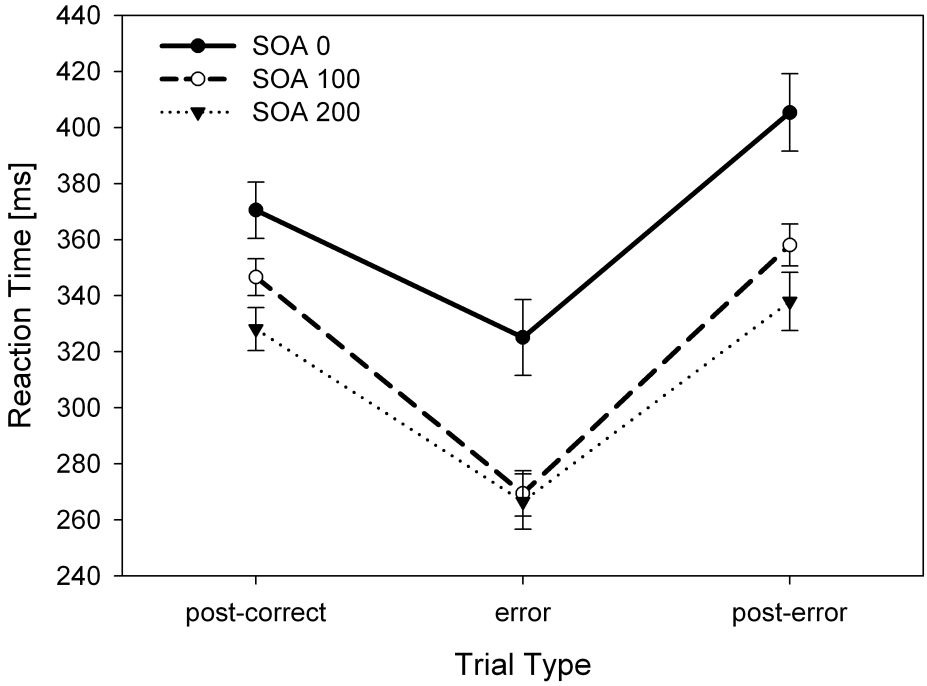

Figure 21: Error speed-up and post-error slowing in the three SOA conditions.

4.1.3 Summary of the Main Results

The pilot study showed that the conflict manipulation using different SOAs was successful. As expected compatibility effects were largest in the SOA 100 condition. Whereas the Gratton effect was not influenced by the SOA manipulation, error speed-up and post-error slowing showed differential effects for the different SOAs. Error speed-up tended to be enhanced in the SOA 100 condition. Post-error slowing was largest for SOA 0 and smaller for longer SOAs. These effects will be discussed in more detail in Section 4.3, in combination with the results of the ERP study.

4.2 ERP Study

The same design as in the pilot was used for the EEG study with the following alterations. A fixation cross, time-locked to target onset, was presented before the stimulus. This was included because the onset of the flankers may serve as a warning stimulus that triggers motor preparation on trials with an SOA larger than 0 ms. This preactivation would not be present on trials where target and flankers appear at the same time and is therefore a possible confound. Furthermore, the interval between response execution and target onset (not flanker onset as in the pilot study) was kept constant across SOA conditions.

4.2.1 Methods

4.2.1.1 Participants

Eighteen young adults (M = 21.2 years, range 19 to 25 years, 11 women) were tested in a single session of about 90 minutes duration. They all reported having normal or corrected-to-normal vision, gave written consent, and were paid £8 for their participation. None of them had participated in the pilot study. One person of the original sample had to be replaced because of excessive EEG artefacts. The study was approved by the University Teaching and Research Ethics Committee (UTREC) of the University of St Andrews (approval code: PS5099).

4.2.1.2 Stimuli and Apparatus

Stimuli and the apparatus for stimulus presentation and response indication were the same as in the pilot study (see Section 4.1.1.2). EEG activity was recorded with a BIOSEMI Active-Two amplifier using the same settings as in the first experiment (see Section 3.1.1.2).

4.2.1.3 Procedure and Design

The same procedure and design as in the pilot study were used, with the following exceptions. A fixation point, consisting of a small plus sign (2 x 2 mm), was presented 700 ms before target onset. It stayed on the screen until stimulus onset (SOA 0) or flanker onset (SOA 100 and SOA 200), respectively. The interval between response execution and fixation cross onset, was set to 600 ms. As a result, the interval between response execution and target onset was 1300 ms. The number of trials within a block was increased to 96, resulting in 1728 trials overall.

4.2.1.4 Data Analysis

Only trials with reaction times between 150 ms and 1500 ms were included in the analyses. Trials with two responses on the same trial (double reactions) were excluded. Post-error accuracy was determined by dividing the number of errors following an error by the total number of trials following an error and multiplied by one hundred. This was compared to the number of errors following correct responses divided by the total number of trials following correct responses and multiplied by one hundred.

The same settings as in the first ERP study were used for the EEG analysis, with the following exceptions. Epochs were averaged separately for compatible, incompatible, and neutral trials, correct and incorrect responses and the three SOA conditions. Peak amplitude and latency of the N2 were analysed in the original ERPs in a time window from 200 ms to 350 ms after target onset and also in the difference waves (incompatible minus compatible and neutral minus compatible) at electrode FCz in a time window from 150 ms to 350 ms after target onset. The difference waves were additionally analysed in the response-locked ERPs in a time window reaching from 300 ms to 50 ms before the response. Error-related ERPs were analysed in response-locked difference waves (incorrect minus correct). Peak amplitude and latency of the Ne/ERN were determined at electrode FCz in the time window of 20 ms to 120 ms following response onset. The early Pe peak amplitude and latency were determined at electrode Cz in the window of 140 ms to 260 ms following the response. The average amplitude of the late Pe was measured at electrode Pz, using a time window from 250 ms to 450 ms post-response.

Conservative Huynh-Feldt tests were used throughout. Adjusted *p*-values are reported, along with the uncorrected degrees of freedom. Bonferroni corrected *p*-values are reported for all post hoc analyses.

4.2.2 Results

4.2.2.1 Behavioural Data

4.2.2.1.1 Compatibility Effects

Compatibility effects on error rates and reaction times, shown in *Figure 22*, were analysed using repeated-measures ANOVAs with the within-subjects factors Compatibility (compatible, neutral, incompatible) and SOA (0 ms, 100 ms, 200 ms). Only trials with correct responses on the previous and the current trial were included in the analysis of reaction times.

Reaction times: A significant main effect of Compatibility, $F(2, 34) = 209.86$, $p < .001$, $\eta_p^2 = .93$, was due to a reliable increase in reaction time from compatible (300 ms) to neutral (322 ms), $F(1, 17) = 102.85$, $p < .001$, $\eta_p^2 = .86$, and from neutral to incompatible (344 ms) trials, $F(1, 17) = 202.13$, $p < .001$, $\eta_p^2 = .92$. There also was a significant main effect of SOA, $F(2, 34) = 56.19$, $p < .001$, $\eta_p^2 = .77$. Participants responded faster in the SOA 200 (306 ms) condition than in the SOA

100 (318 ms), $F(1, 17) = 24.50$, $p < .001$, $\eta_p^2 = .59$, and SOA 0 (342 ms) conditions, $F(1, 17) = 77.17$, $p < .001$, $\eta_p^2 = .82$. They also responded faster for SOA 100 than SOA 0, $F(1, 17) = 43.12$, $p < .001$, $\eta_p^2 = .72$. Importantly, the interaction of both factors reached significance, $F(4, 68) = 25.06$, $p < .001$, $\eta_p^2 = .60$. Post hoc analyses showed that facilitation (neutral minus compatible) was larger for SOA 100 (38 ms) than SOA 0 (7 ms), $F(1, 17) = 79.45$, $p < .001$, $\eta_p^2 = .82$, and SOA 200 (21 ms), $F(1, 17) = 20.79$, $p = .002$, $\eta_p^2 = .55$. It was also larger for SOA 200 than SOA 0, $F(1, 17) = 8.99$, $p = .048$, $\eta_p^2 = .35$. Interference (incompatible minus neutral) was larger at SOA 100 (29 ms) than SOA 200 (17 ms), $F(1, 17) = 22.87$, $p = .001$, $\eta_p^2 = .57$. Interference at SOA 0 (20 ms) did not differ significantly form the other two SOAs (both ps > .10).

Error rates: A significant main effect of Compatibility, $F(2, 34) = 138.60$, $p < .001$, $\eta_p^2 = .89$, was due to a reliable increase in error rate from compatible (5.4%) to neutral (10.0%) to incompatible (22.1%) trials, Fs$(1, 17) \geq 53.75$, ps < .001, η_p^2s $\geq .76$. There also was a significant main effect of SOA, $F(2, 34) = 17.80$, $p < .001$, $\eta_p^2 = .51$, showing smaller error rates for SOA 0 (9.5%) than SOA 100 (14.5%), $F(1, 17) = 31.75$, $p < .001$, $\eta_p^2 = .65$, and SOA 200 (13.4%), $F(1, 17) = 33.03$, $p < .001$, $\eta_p^2 = .66$. The error rates for SOA 100 and SOA 200 did not differ significantly ($p > .10$). Importantly, there was a significant interaction between both factors, $F(4, 68) = 11.97$, $p < .001$, $\eta_p^2 = .41$. Post hoc tests showed that facilitation (neutral minus compatible) was larger for SOA 100 (8.2%) than for SOA 0 (2.5%), $F(1, 17) = 25.87$, $p < .001$, $\eta_p^2 = .60$, and SOA 200 (3.3%), $F(1, 17) = 16.80$, $p = .004$, $\eta_p^2 = .50$, but did not differ between SOA 0 and SOA 200 ($p > .10$). Interference (incompatible minus neutral) tended to be larger for SOA 100 (16.0%) than SOA 0 (9.1%), $F(1, 17) = 7.67$, $p = .079$, $\eta_p^2 = .31$. Interference for SOA 200 (11.0%) did not differ significantly from the other two SOAs (both ps > .10).

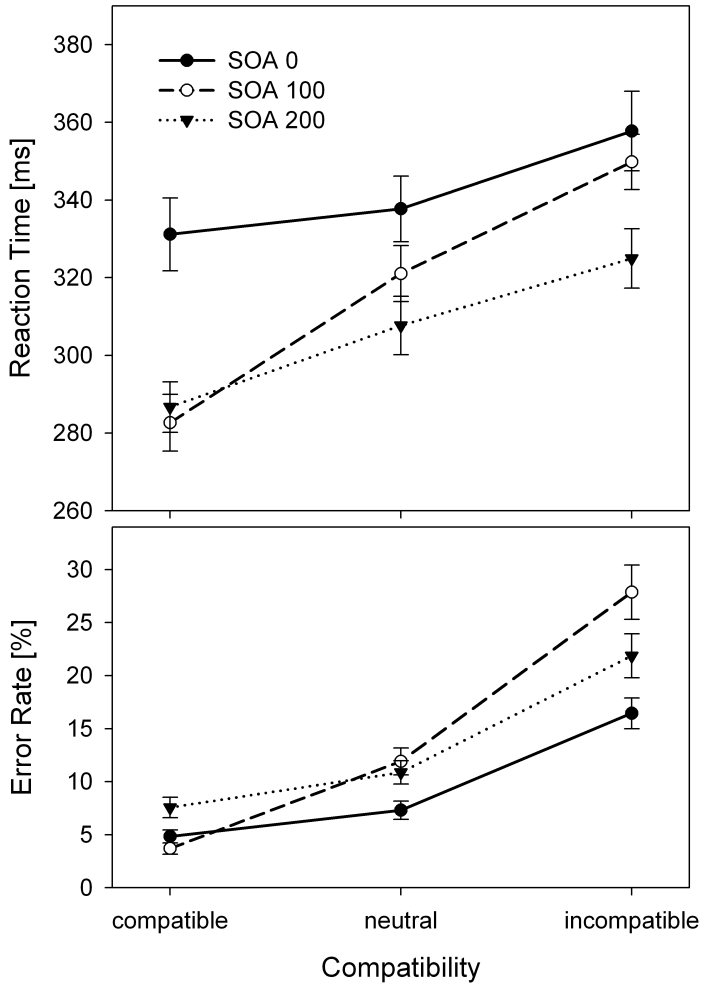

Figure 22: Compatibility effects on reaction times (top) and error rates (bottom) in the three SOA conditions.

4.2.2.1.2 Sequential Adjustment Effects

Sequential adjustment effects on reaction times (*Figure 23*) and error rates (*Figure 24*) were analysed using repeated-measures ANOVAs with the within-subjects factors Trial N-1 Compatibility (compatible, neutral, incompatible), Current Trial Compatibility (compatible, neutral, incompatible), SOA (0 ms, 100 ms, 200 ms) and Response Sequence (alternation, repetition). Only trials with correct responses on the previous and the current trial were included in the analysis of reaction times. Only interaction involving the factor Trial N-1 Compatibility will be reported in this section.

Reaction times: Overall, response repetitions (314 ms) were faster than alternations (331 ms), $F(1, 17) = 8.26$, $p = .011$, $\eta_p^2 = .33$. There also was a

significant main effect of Trial N-1 Compatibility, $F(2, 34) = 16.96$, $p < .001$, $\eta_p^2 = .50$, and a significant interaction between Trial N-1 Compatibility and Response Sequence, $F(2, 34) = 9.31$, $p = .001$, $\eta_p^2 = .35$. Importantly, there was a significant interaction between Trial N-1 Compatibility and Current Trial Compatibility, $F(4, 68) = 16.84$, $p < .001$, $\eta_p^2 = .50$, which in turn interacted with the factor Response Sequence, $F(4, 68) = 9.47$, $p < .001$, $\eta_p^2 = .36$. Post hoc tests showed a significant Trial N-1 Compatibility X Current Trial Compatibility interaction for response repetition, $F(4, 68) = 25.17$, $p < .001$, $\eta_p^2 = .60$, whereas the interaction was only marginally significant for response alternations, $F(4, 68) = 2.97$, $p = .051$, $\eta_p^2 = .15$.[17] The respective size of the Gratton effect ((c-ic – c-c) – (ic-ic – ic-c)) was 32 ms for response repetitions and 11 ms for response alternations. Furthermore, there were significant interactions between Trial N-1 Compatibility, SOA and Response Sequence, $F(4, 68) = 5.60$, $p = .001$, $\eta_p^2 = .25$, and between Trial N-1 Compatibility, Current Trial Compatibility and SOA, $F(8, 136) = 4.17$, $p < .001$, $\eta_p^2 = .20$. The latter was due to the fact that there were significant Trial N-1 Compatibility X Current Trial Compatibility interactions for SOA 100, $F(4, 68) = 7.72$, $p < .001$, $\eta_p^2 = .31$, and SOA 200, $F(4, 68) = 13.10$, $p < .001$, $\eta_p^2 = .44$, but not for SOA 0 ($p > .10$). The respective size of the Gratton effect ((c-ic – c-c) – (ic-ic – ic-c)) was 2 ms for SOA 0, 26 ms for SOA 100 and 36 ms for SOA 200. None of the other effects involving the factor Trial N-1 Compatibility reached significance (all ps > .10).

[17] When current neutral trials were excluded from the analysis, the trend towards an interaction between Trial N-1 Compatibility and Current Trial Compatibility for response alternations still remained, $F(2, 34) = 3.78$, $p = .066$, $\eta_p^2 = .18$.

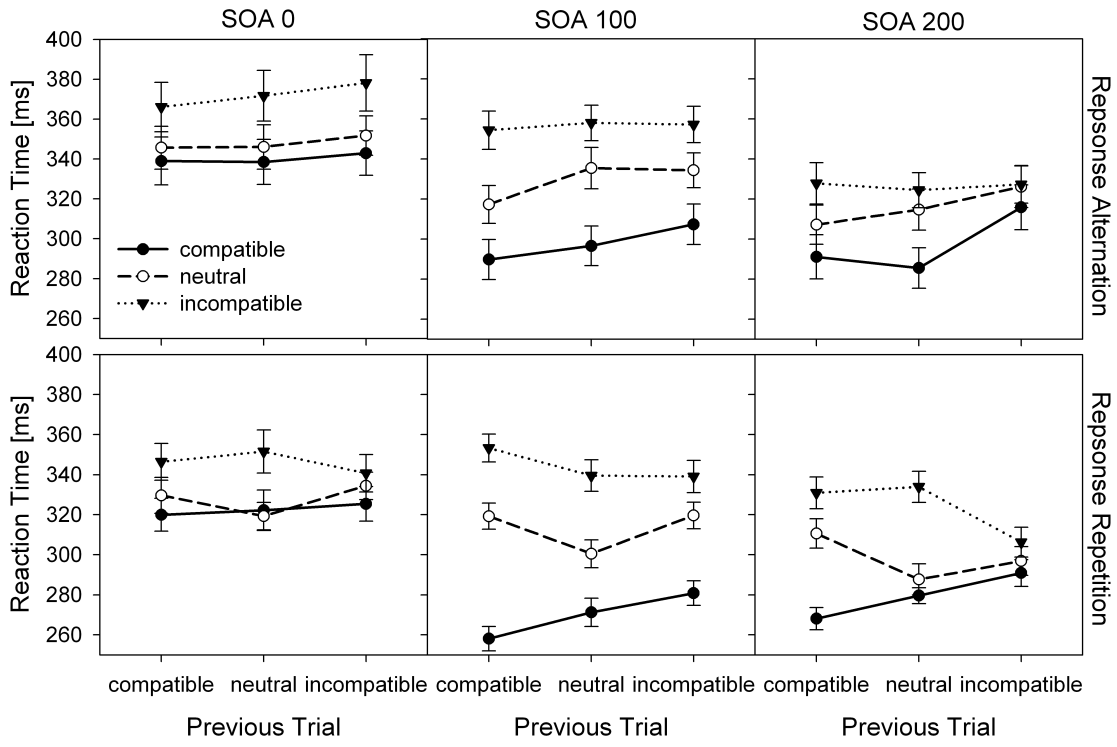

Figure 23: Gratton effect: Reaction times as a function of previous and current trial compatibility in the three SOA conditions for response alternations (top) and response repetitions (bottom).

Error rates: There were no significant main effects of Trial N-1 Compatibility or Response Sequence (both *p*s > .10), however, their interaction reached significance, $F(2, 34) = 16.86$, $p < .001$, $\eta_p^2 = .50$. Importantly, there was a significant interaction between Trial N-1 Compatibility and Current Trial Compatibility, $F(4, 68) = 4.52$, $p = .003$, $\eta_p^2 = .21$, which in turn interacted with the factor Response Sequence, $F(4, 68) = 3.22$, $p = .019$, $\eta_p^2 = .16$. Post hoc tests showed that the Trial N-1 Compatibility X Current Trial Compatibility interaction was only significant for response repetitions, $F(4, 68) = 7.25$, $p < .001$, $\eta_p^2 = .30$, but not for response alternations ($p > .10$). There were trends towards interactions between Trial N-1 Compatibility, SOA and Response Sequence, $F(4, 68) = 2.46$, $p = .053$, $\eta_p^2 = .13$, and Trial N-1 Compatibility, Current Trial Compatibility and SOA, $F(8, 136) = 2.12$, $p = .055$, $\eta_p^2 = .11$. The latter was due to the fact that there were significant Trial N-1 Compatibility X Current Trial Compatibility interactions for SOA 100, $F(4, 68) = 4.18$, $p = .018$, $\eta_p^2 = .20$, and SOA 200, $F(4, 68) = 3.71$,

p = .038, η_p^2 = .18, but not for SOA 0 (p > .10). None of the other effects involving the factor Trial N-1 Compatibility reached significance (all ps > .10).

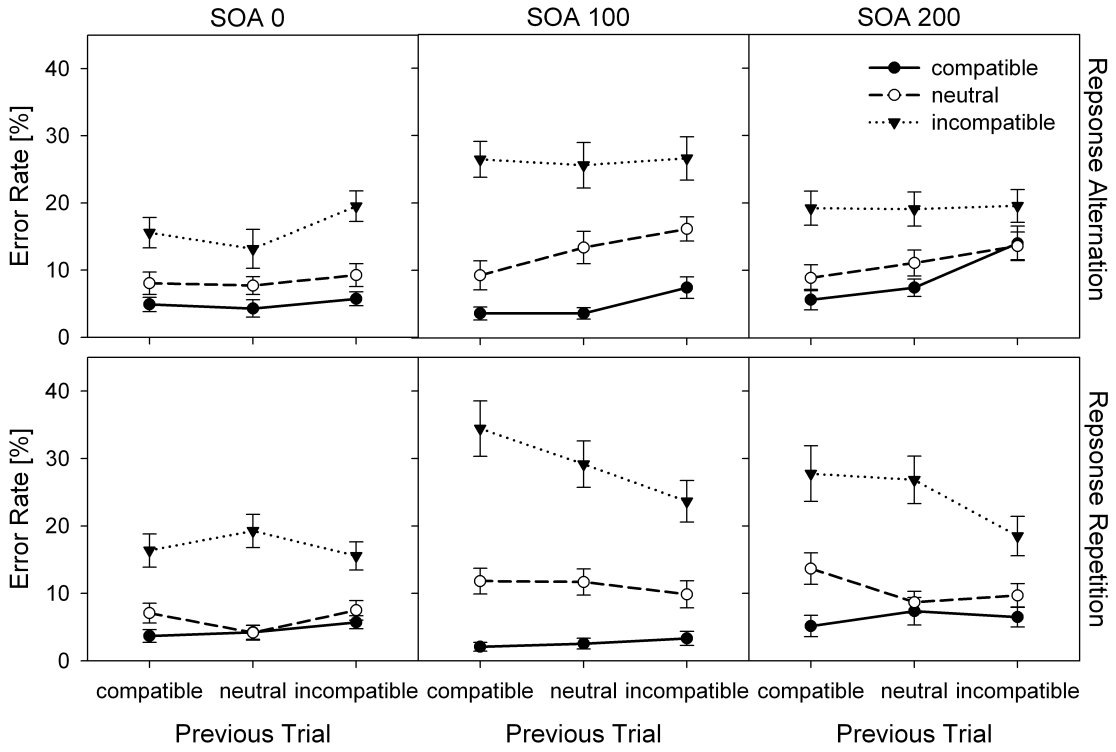

Figure 24: Gratton effect: Error rates as a function of previous and current trial compatibility in the three SOA conditions for response alternations (top) and response repetitions (bottom).

4.2.2.1.3 Error and Post-Error Effects

Reaction times for correct responses following correct responses (post-correct), errors following correct responses (error), and correct responses following an error (post-error) can be seen in *Figure 25*. Error speed-up and post-error slowing were analysed using a repeated-measures ANOVA with the within-subjects factors Trial Type (post-correct, error, post-error) and SOA (0 ms, 100 ms, 200 ms). Post-error accuracy was analysed using a repeated-measures ANOVA with the within-subjects factors Trial N-1 Accuracy (correct, incorrect) and SOA (0 ms, 100 ms, 200 ms). Only trials following incompatible trials were included in these analyses.

Reaction times: There was a significant effect of Trial Type, $F(2, 34) = 83.62$, $p < .001$, $\eta_p^2 = .83$. Post hoc tests showed that the error speed-up effect (post-correct minus error) was significant, $F(1, 17) = 102.82$, $p < .001$,

η_p^2 = .86, however, post-error slowing (post-error minus post-correct) was not (p > .10). There also was a significant main effect of SOA, $F(2, 34) = 39.69$, $p < .001$, $\eta_p^2 = .70$, as well as a significant interaction between Trial Type and SOA, $F(4, 68) = 4.24$, $p = .008$, $\eta_p^2 = .20$. Post hoc tests showed that error speed-up was larger for SOA 100 (70 ms) than for SOA 0 (48 ms), $F(1, 17) = 15.62$, $p = .006$, $\eta_p^2 = .48$, and SOA 200 (51 ms), $F(1, 17) = 14.96$, $p = .007$, $\eta_p^2 = .47$. Error speed-up for SOA 0 and SOA 200 did not differ significantly (p > .10). There were no significant interactions between post-error slowing and SOA (all ps > .10).

Error rates: The error rates for trials following correct (9.7%) and following incorrect (9.8%) responses did not differ significantly (p > .10). The main effect of SOA and the interaction of both factors were not significant either (both ps > .10).

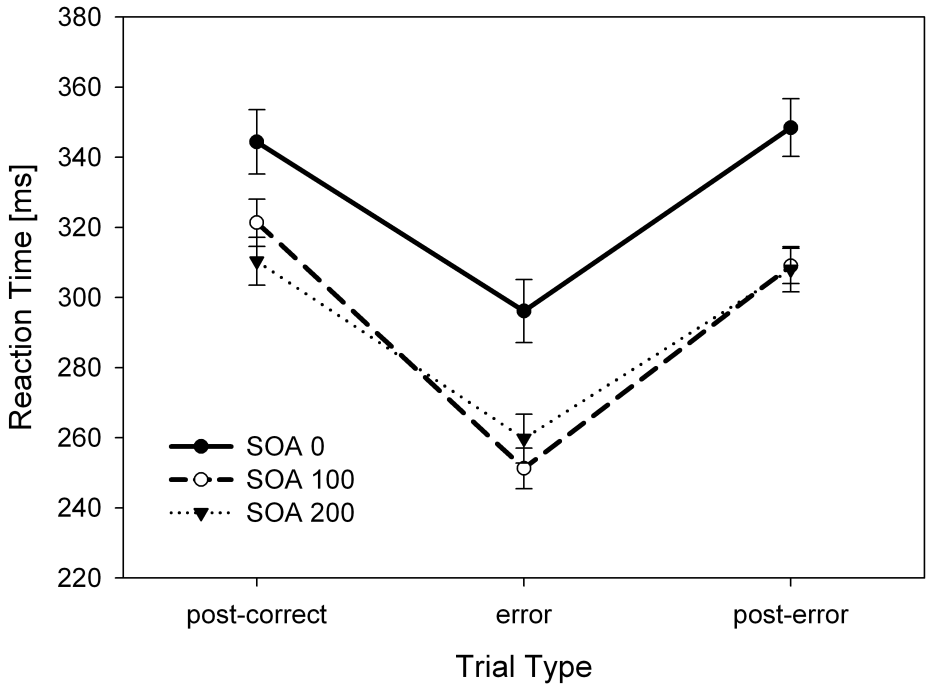

Figure 25: Error speed-up and post-error slowing in the three SOA conditions.

4.2.2.2 Electrophysiological data

4.2.2.2.1 N2

Figure 26 shows the target-locked ERPs for compatible, neutral, and incompatible trials on the left and the difference waves (neutral minus compatible and incompatible minus compatible) on the right. Latency and amplitude of the N2 were analysed in the original ERPs using repeated-measures ANOVAs with the within-

subjects factors Compatibility (compatible, neutral, incompatible) and SOA (0 ms, 100 ms, 200 ms), as well as in the difference waves using repeated-measures ANOVAs with the within-subjects factors Contrast Type (neutral minus compatible, incompatible minus compatible) and SOA (0 ms, 100 ms, 200 ms). Furthermore, the amplitude of the N2 was compared between target-locked and response-locked difference waves (see *Figure 27*) using a repeated-measures ANOVA with the within-subjects factors Analysis Type (target-locked, response-locked), Contrast Type (neutral minus compatible, incompatible minus compatible), and SOA (0 ms, 100 ms, 200 ms).

Original ERPs: The N2 peaked about 275 ms after target onset. The main effect of Compatibility on amplitude was significant, $F(2, 34) = 13.08$, $p < .001$, $\eta_p^2 = .44$. Post hoc test showed that the N2 was smaller for compatible (-1.9 μV) than for neutral (-2.8 μV), $F(1, 17) = 11.40$, $p = .011$, $\eta_p^2 = .40$, and incompatible trials (-3.3 μV), $F(1, 17) = 19.54$, $p = .001$, $\eta_p^2 = .54$. The amplitude difference between neutral and incompatible trials did not reach significance ($p > .10$). The main effect of SOA on amplitude was also significant, $F(2, 34) = 4.80$, $p = .015$, $\eta_p^2 = .22$. The N2 was larger for SOA 0 (-3.4 μV) than SOA 200 (-2.1 μV), $F(1, 17) = 9.94$, $p = .017$, $\eta_p^2 = .37$. SOA 100 (-2.5 μV) did not differ significantly from the other two SOAs (both *p*s > .10). The interaction of both factors did not reach significance ($p > .10$). There also was a significant main effect of SOA on N2 latency, $F(2, 34) = 5.78$, $p = .012$, $\eta_p^2 = .25$. The N2 peaked earlier for SOA 200 (263 ms) than for SOA 0 (280 ms), $F(1, 17) = 18.61$, $p = .001$, $\eta_p^2 = .52$, and SOA 100 (282 ms), $F(1, 17) = 7.37$, $p = .044$, $\eta_p^2 = .30$. The latency of SOA 0 and SOA 100 did not differ significantly ($p > .10$). Neither the main effect of Compatibility nor the interaction of both factors reached significance (both *p*s > .10).

Target-locked difference waves: As the right panel of *Figure 26* shows, the N2 was larger in the incompatible-compatible (-2.4 μV) than the neutral-compatible contrast (-1.7 μV), $F(1, 17) = 9.71$, $p = .006$, $\eta_p^2 = .36$. The main effect of SOA on amplitude approached significance, $F(2, 34) = 3.45$, $p = .051$, $\eta_p^2 = .17$, however, none of the post hoc comparisons were significant (all *p*s > .10). There was a trend towards a significant interaction of both factors, $F(2, 34) = 2.85$, $p = .072$, $\eta_p^2 = .14$. Post hoc test did show no significant effects (all *p*s > .05). The N2 peaked earlier in the incompatible-compatible (247 ms) than the neutral-compatible contrast (262 ms),

$F(1, 17) = 5.22$, $p = .036$, $\eta_p^2 = .24$. There also was a significant main effect of SOA, $F(2, 34) = 4.27$, $p = .022$, $\eta_p^2 = .20$. Post hoc tests showed that the N2 peaked earlier for SOA 200 (233 ms) than SOA 0 (272 ms), $F(1, 17) = 7.48$, $p = .042$, $\eta_p^2 = .31$. The N2 latency of SOA 100 (258 ms) did not differ significantly from the other two SOAs (both *p*s > .10). The interaction did not reach significance ($p > .10$).

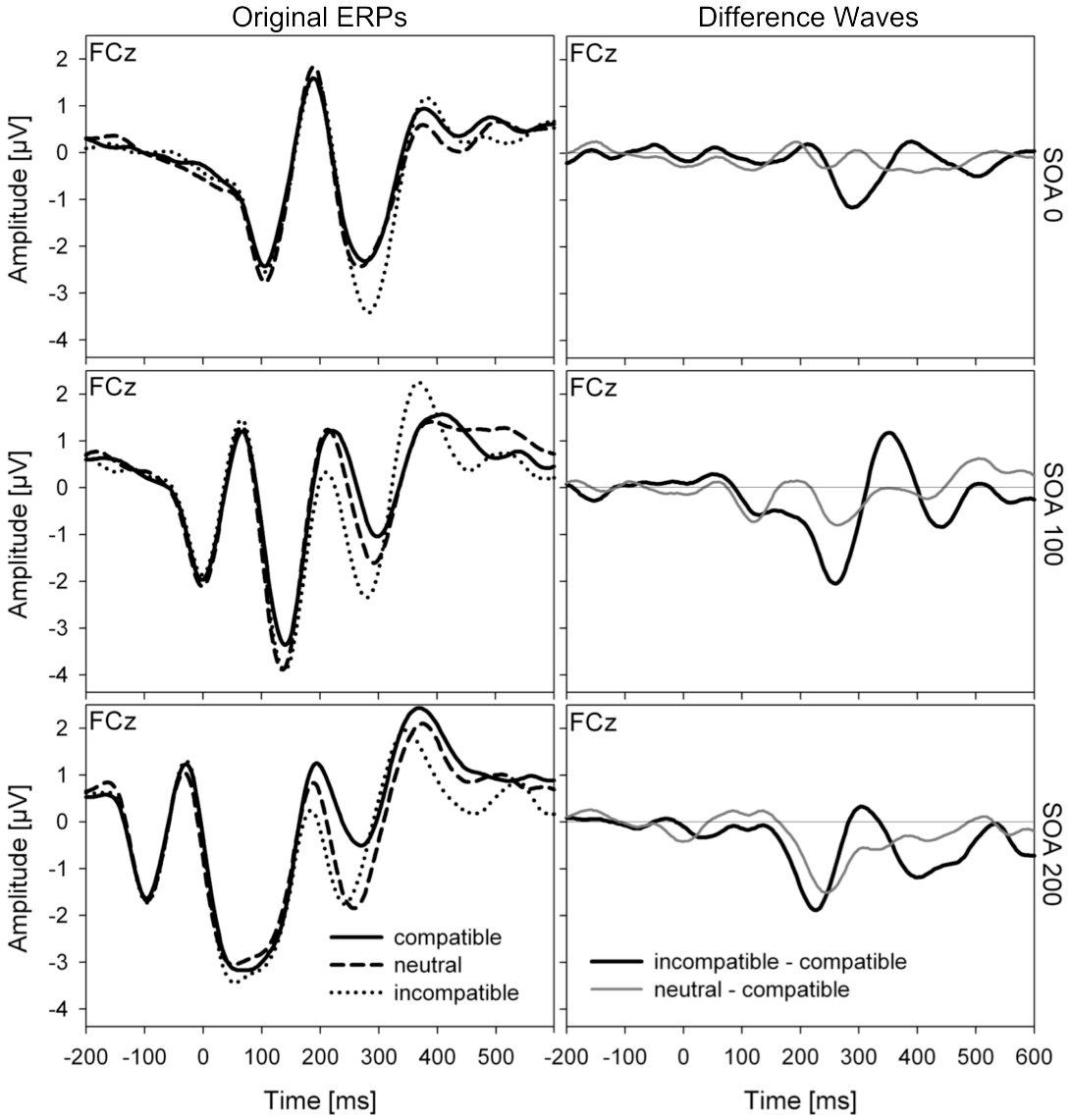

Figure 26: Target-locked ERPs for SOA 0 (top), SOA 100 (middle) and SOA 200 (bottom) at electrode FCz. Left: Compatible, neutral and incompatible trials. Right: Difference waves (incompatible minus compatible and neutral minus compatible).

Target- vs. response-locked difference waves: As can be seen in *Figure 27*, the N2 was larger in the target-locked (-2.0 µV) than in the response-locked

(-1.4 μV) analysis, $F(1, 17) = 8.40$, $p = .010$, $\eta_p^2 = .33$. The N2 had a larger amplitude in the incompatible-compatible (-2.0 μV) than the neutral-compatible contrast (-1.5 μV), $F(1, 17) = 7.27$, $p = .015$, $\eta_p^2 = .30$. The main effect of SOA was significant, $F(2, 34) = 3.77$, $p = .035$, $\eta_p^2 = .18$, however, none of the post hoc comparisons reached significance (all $ps \geq .10$). There was a significant interaction between the factors Analysis Type and Contrast Type, $F(1, 17) = 6.24$, $p = .023$, $\eta_p^2 = .27$, indicating that the difference between the incompatible-compatible and the neutral-compatible contrast was only significant in the target-locked, $F(1, 17) = 9.71$, $p = .013$, $\eta_p^2 = .36$, but not in the response-locked analysis ($p > .10$). None of the other interaction reached significance (all $ps > .10$).

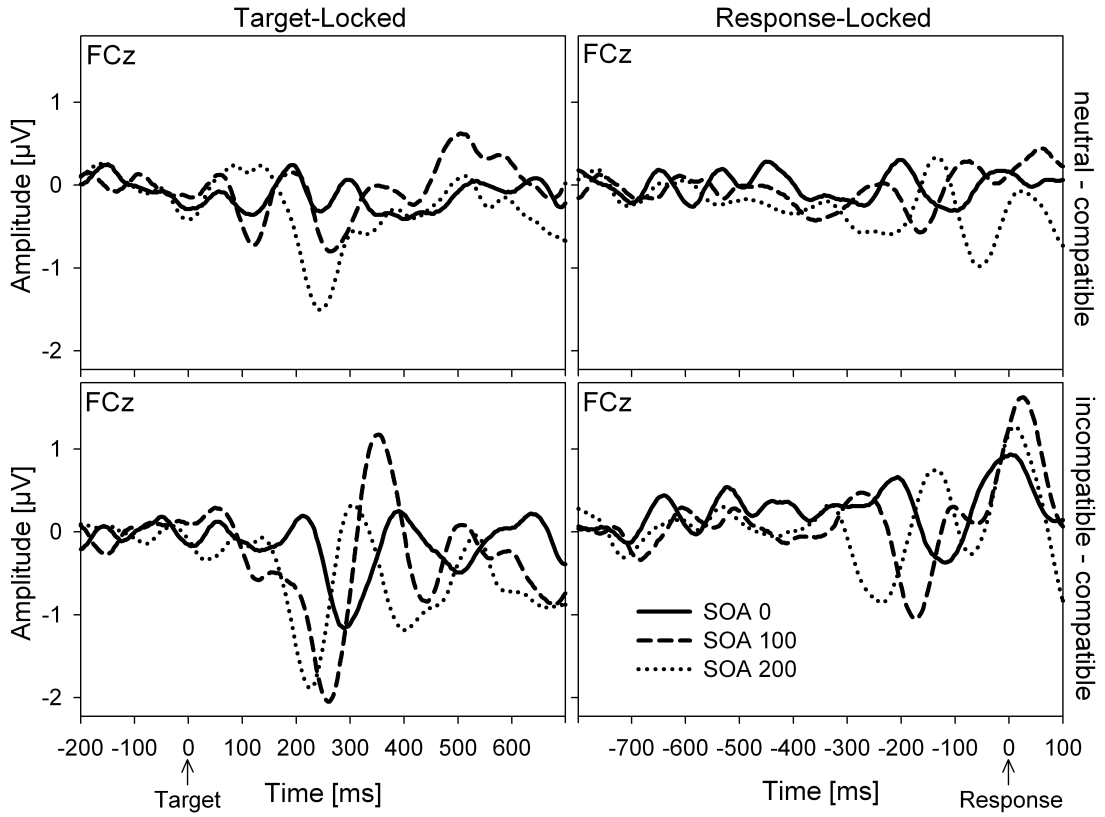

Figure 27: Target-locked (left) and response-locked (right) difference waves (top: neutral minus compatible; bottom; incompatible minus compatible) at electrode FCz in the three SOA conditions.

4.2.2.2.2 Error-Related ERP Components

Figure 28 shows topographic maps at the time point of maximum amplitude of the Ne/ERN, the early Pe, and the late Pe in the SOA 0 condition. *Figure 29* shows the

response-locked ERPs for correct and incorrect responses, as well as the difference waves (incorrect minus correct) for incompatible trials in the three SOA conditions at electrodes Fz, FCz, Cz, and Pz. Peak latency and amplitude of the Ne/ERN and the early Pe, as well as the average amplitude of the late Pe were analysed in the difference waves using repeated-measures ANOVAs with the within-subjects factor SOA (0 ms, 100 ms, 200 ms).

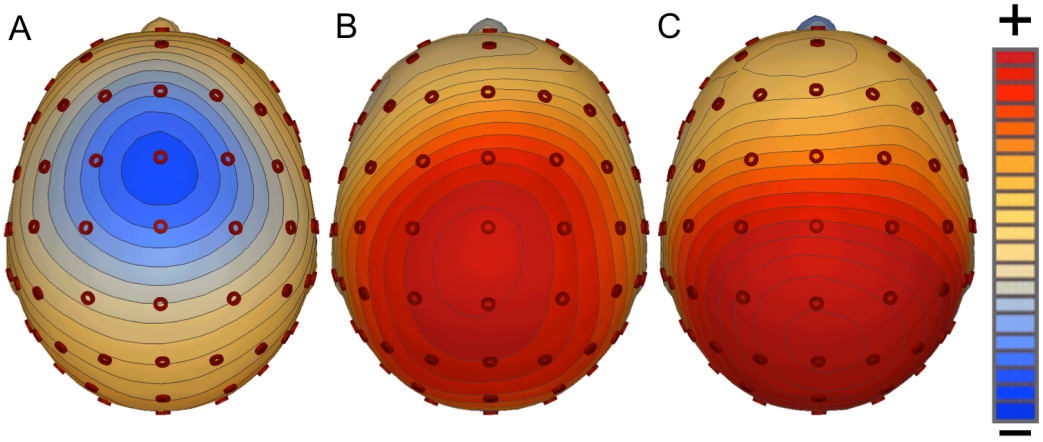

Figure 28: Exemplary topographic maps of the Ne/ERN at 65 ms (A), the early Pe at 205 ms (B) and the late Pe at 350 ms (C) post-response for the SOA 0 condition. Shown are the difference waves (incorrect minus correct) with 0.80 µV/step.

Ne/ERN: The Ne/ERN peaked 65 ms after response onset. There were no significant effects of SOA on Ne/ERN amplitude or latency (both *p*s > .10).

Early Pe: The early Pe reached its maximum amplitude 204 ms after the response. There were no significant effects of the SOA manipulation on either amplitude or latency (both *p*s > .10).[18]

Late Pe: The amplitude of the late Pe was not affected by the SOA manipulation ($p > .10$).

[18] When analysed at electrode FCz, the main effect of SOA on amplitude approached significance, $F(2, 34) = 3.31$, $p = .051$, $\eta_p^2 = .16$. However, none of the post hoc comparisons were significant (all *p*s > .10).

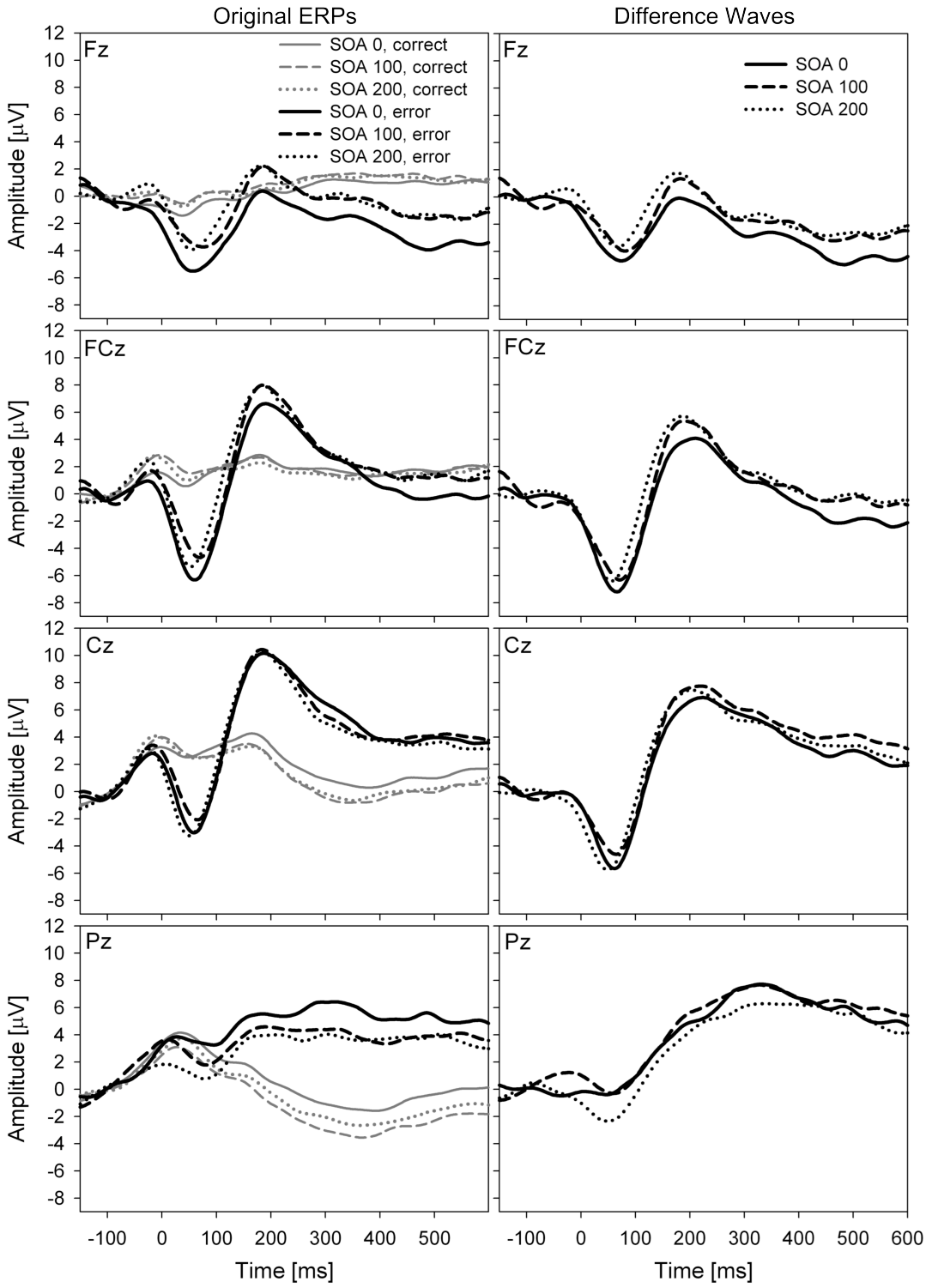

Figure 29: Response-locked ERPs for incompatible trials in the three SOA conditions at electrodes Fz, FCz, Cz, and Pz. Left: Correct (grey lines) and incorrect (black lines) response trials. Right: Difference waves (incorrect minus correct).

4.3 Discussion

The aim of the two experiments described in this chapter was to manipulate conflict strength in the flanker task, in order to investigate effects of conflict on control adjustment effects and ERP components. Overall, the SOA and compatibility manipulations showed the expected effects on error rates and reaction times in both studies. Error rates and reaction times increased reliably from compatible over neutral to incompatible trials. The compatibility effect was reflected in the N2 amplitudes of the ERP study; compatible trials showed the smallest amplitudes and incompatible the largest. The N2 amplitude of neutral trials lay between compatible and incompatible trials, although the difference between incompatible and neutral did not reach significance. Overall, these findings confirm that the amplitude of the N2 is related to conflict. Furthermore, reaction times decreased with increasing SOA, and error rates were larger for the longer SOAs compared to an SOA of 0 ms. This might reflect a speed-accuracy tradeoff, indicating that participants emphasized speed over accuracy to a larger degree at longer SOAs. Alternatively, when flankers are presented before the target, flanker onset might serve as a warning stimulus that allows unspecific motor preparation leading to faster response times. Increased flanker processing in the longer SOA conditions might explain the increase in error rate, since differences in error rate between SOAs were mostly due to errors on incompatible trials, as suggested by *Figure 18* and *Figure 22*. The effects of SOA were reflected in N2 latency of the difference waves, i.e. the latency was shortest in the SOA 200 condition and longest for SOA 0. Importantly, the compatibility effect was affected by the SOA manipulation. As can be seen in *Figure 18* and *Figure 22*, effects of compatibility were generally larger in the SOA 100 condition compared to the other two SOAs, thereby replicating previous findings (e.g., Mattler, 2003; Wascher et al., 1999; Willemssen et al., 2004). Thus, the SOA manipulation successfully influenced conflict strength.

Importantly, conflict strength influenced the size of the Gratton effect in the ERP study as indicated by a significant three-way interaction between previous and current trial compatibility and SOA. The Gratton effect was virtually nonexistent in

the SOA 0 condition but present for both SOA 100 and SOA 200.[19] The interaction was not influenced by the response sequence, indicating that this effect was not due to repetition priming effects. However, just like in the two studies described in the previous chapter, repetition priming did play a role in the generation of the Gratton effect. In both studies, the Gratton effect on error rates and reaction times was reduced for response alternations compared to response repetitions. In the pilot study, the previous and current trial compatibility interaction for response alternations disappeared when current neutral trials were not taken into consideration, indicating that the interaction was not due to the traditional Gratton effect (i.e., reduced compatibility effect following incompatible compared to following compatible trials) but due to occurrences on neutral trials (see below for a discussion of this finding). In the ERP study, on the other hand, although the Gratton effect was reduced for response alternations compared to response repetitions, it was still marginally significant even when current neutral trials were excluded from the analysis. Therefore, even though repetition priming contributed to the Gratton effect, it cannot fully account for the observed pattern of sequential adjustment effects. Conflict adaptation apparently also contributed to this effect.

Both studies showed the expected speed-up for error trials compared to correct trials. The error speed-up was numerically largest for SOA 100 in the pilot study. This effect became significant in the ERP study. Considering that most errors were due to misleading flanker information on incompatible trials and flanker influence was largest in the SOA 100 condition, the fastest errors can be expected in the SOA 100 condition. Surprisingly, post-error slowing was only present in the pilot study. It is possible that the slightly longer RSI in the ERP study was too long for slowing to occur, since it has been shown previously that post-error slowing gets smaller with increasing RSI (e.g., Dudschig & Jentzsch, 2009). In the pilot study, post-error slowing was larger for SOA 0 than for the other two SOAs. This might also be due to differences in RSI. As mentioned in Section 4.1.1.3, the RSI was set to flanker and not target onset resulting in the shortest response-target interval for SOA 0 and longest in SOA 200. The fact that error speed-up was affected by the SOA

[19] Although the same interaction did not reach significance in the pilot study, the Gratton effect ((c-ic – c-c) – (ic-ic – ic-c)) was numerically largest in the SOA 100 condition.

manipulation in the ERP study, whereas post-error slowing was not, suggests that these two effects were due to independent mechanisms. This interpretation is in line with the finding that the post-error slowing effect is affected by the RSI, whereas error speed-up is not (Dudschig & Jentzsch, 2009). The authors concluded that post-error slowing represents a strategic adjustment when sufficient time is available, while error speed-up might be due to automatic lowering of response thresholds following correct responses. In the current experiment, lowered response thresholds may have led to increased processing of flanker information and therefore faster errors, especially in the SOA 100 condition. However, the current results have to be interpreted with caution since the post-error slowing effect was confounded by the RSI in the pilot study and not significant in the ERP study.

Just like in the ERP study of the previous chapter, the analysis of error-related ERPs showed three distinct components: a fronto-central Ne/ERN, a central early Pe, and a more posterior late Pe. Neither amplitude nor latency of these components was affected by the SOA manipulation. Interestingly, there was a clear Ne/ERN present despite the absence of post-error slowing, arguing against a possible association between these processes.

Although the amplitude of the N2 showed the predicted compatibility effects, the analysis also yielded some unexpected results. In the original ERPs, the N2 was largest in the SOA 0 condition, even though conflict was largest at SOA 100. However, this result should be treated with caution, since components associated with flanker and target processing overlap differently in the three SOA conditions. Therefore, in these circumstances it is a more reliable method to investigate N2 effects in difference waves. The amplitude of the incompatible-compatible difference wave followed the expected pattern, with amplitudes being numerically largest in the SOA 100 condition where conflict was largest. However, the effect did not reach significance. This might be due to a lack of statistical power. It is possible that the amplitude differences would be significant in an experiment with a larger sample size.

Another unexpected result was that the N2 on neutral trials was larger than on compatible trials but did not differ significantly from the N2 on incompatible trials. Previous studies had found the opposite, i.e. similar N2 amplitudes on compatible and neutral trials and increased N2 amplitudes on incompatible trials (e.g., Heil et al.,

2000; Kopp, Rist, et al., 1996; Wild-Wall et al., 2008). Differences in SOA between these studies (SOA 0: Heil et al., 2000; Wild-Wall et al., 2008; SOA 100: Kopp, Rist, et al., 1996) and the current experiment might explain this discrepancy. As *Figure 26* shows, the neutral-compatible difference wave was larger for the longer SOAs and virtually absent for SOA 0, reflecting an increase in neutral trial N2 amplitude with increasing SOA. Although this effect was not significant, it nonetheless shows that the unexpectedly large N2 on neutral trials might be due to the SOA 200 condition. At SOA 0 the compatible and the neutral N2 do not seem to differ in amplitude.

As expected, the comparison of the stimulus- and response-locked N2 showed reduced amplitudes in the response-locked ERPs, just like in the ERP study described in the previous chapter. This result strongly suggests that the N2 represents processes associated with the stimuli and not with the response. These findings, therefore, contradict the suggestion by Yeung et al. (2004) that the N2 reflects response conflict on correct trials, since their model predicts maximum conflict and N2 amplitude immediately before response onset (see also Section 1.4.2).

As mentioned earlier, neutral trials seemed to have been responsible for the significant previous and current trial compatibility interaction for response alternations in the pilot study, since the interaction disappeared when neutral trials were excluded. The pattern of results for sequential adjustment effects of neutral stimuli in both studies can be explained by the feature integration account. As can be seen in Figures 19, 20, 23, and 24, for response repetitions neutral trial responses were especially fast and accurate when they were preceded by another neutral trial. This is not surprising, since these cases constitute exact stimulus-response repetitions and repetition priming should, therefore, be high. For response alternations, on the other hand, a repetition of a neutral trial did lead to increased reaction times and error rates. On these trial sequences, the flankers were repeated but the response changed; they are therefore an example of partial feature repetition. According to the feature integration account such partial feature repetitions lead to processing difficulties due to the fact that stimulus and response are combined within one episodic memory representation. For example, the stimulus OOSOO (with O being the neutral flanker letter) might be associated with a left hand response. If the following stimulus is OOHOO, the repetition of the flankers automatically coactivates the left hand

response. A complete alternation of all features (e.g., when a neutral trial (OOHOO) is preceded by a compatible (SSSSS) or incompatible (HHSHH) trial) should also be processed easily, since there is no previous feature binding that needs to be overcome.

In conclusion, varying SOA was successful in manipulating conflict strength in the flanker task. Sequential adjustment was affected by conflict strength, although repetition priming and feature integration also contribute towards this effect. The N2 seems to be sensitive to conflict, even though effects were small and not always significant. The error-related components, on the other hand, were clearly not affected by the manipulation of conflict and seem to represent error-specific activity.

5 Age-Related Changes in Conflict and Error Processing[20]

One of the aims of my PhD project was to investigate age-related changes in conflict and error processing and in cognitive control adjustments. Previous research in this area has mostly focussed on participants above 60 years of age and compared their performance to that of young adults (see Section 1.5). Relatively little is known about adults in a middle age range, i.e., aged 40 to 60 years. Considering that adults of that age are usually still active in the workspace, changes in cognitive control in this age group are of great practical relevance. For that reason, I included a middle-aged participant group in the experiment described in Section 3.1. Since preliminary analyses revealed that the contrast manipulation (see Chapter 3 for details) did not have any differential effects in the two age groups, the contrast conditions were combined for the purpose of this chapter.

One problem when investigating age-related changes in this subject area is that many older people take antidepressant medication and that this drug has been shown to decrease the amplitude of the Ne/ERN (e.g., Endrass et al., 2008). Therefore, the finding of an age-related reduction of the Ne/ERN might be in fact due to differences in medication usage between the groups. For this reason, I assessed medication usage in the middle-aged sample and excluded participants who were taking psychoactive drugs.

5.1 Methods

5.1.1 Participants

Twenty-eight young (M = 21.4 years, range 18 to 31 years, 15 women) and 29 middle-aged adults (M = 49.1 years, range 41 to 59 years, 18 women) were tested in a single session of approximately two hours duration. The young group included the 24 participants of the ERP study described in Section 3.1 and the four additional participants that were not included in that study due to an insufficient number of errors. Since the four contrast conditions were combined in this study, their number of errors was sufficient for the analysis. Two additional middle-aged participants had to be excluded from the analysis because they were taking antidepressant medication. All remaining middle-aged participants reported to be free of psychoactive

[20] Parts of this chapter have been published in Brain Research (Strozyk & Jentzsch, 2012).

medication. Their average forward digit span (Wechsler, 1945) was 7.0 (range 5 to 8) and the average backward digit span (ibid.) was 5.6 (range 3 to 7). All participants had normal or corrected-to-normal vision, gave written informed consent, and received payment of £10. The study was approved by the University Teaching and Research Ethics Committee (UTREC) of the University of St Andrews (approval code: PS5099).

5.1.2 Stimuli and Apparatus

Stimuli and apparatus were the same as described in Section 3.1.1.2.

5.1.3 Procedure and Design

Procedure and design were the same as described in Section 3.1.1.3, with the exception that middle-aged participants completed a digit span task before proceeding to the main experiment, in order to ensure intact working memory functions, and a short questionnaire assessing their medication usage.

5.1.4 Data Analysis

The data analysis was as described in Section 3.1.1.4 with the exception that the data was pooled over the four contrast conditions. This was done because a preliminary analysis of the behavioural data showed no significant interactions between the contrast conditions and age group (all *p*s > .05).

Statistical analyses of the P1 and N1 components were conducted in the stimulus-locked ERPs at electrodes PO7 and PO8. The peak search window for the P1 ranged from 40 ms to 150 ms after stimulus onset; the time window for the N1 ranged from 110 ms to 220 ms after stimulus onset. Latency and amplitude of the N2 were analysed in the stimulus- and response-locked difference waves (incompatible minus compatible) at electrode FCz. The peak search windows reached from 260 ms to 390 ms after stimulus onset and from 210 ms to 40 ms before the response, respectively. Error-related ERP components were analysed in the difference waves (incorrect minus correct). Peak amplitude and latency of the Ne/ERN were determined at electrode FCz in a time window reaching from 20 ms to 120 ms after the response. Peak amplitude and latency of the early Pe were measured at electrode Cz in a time window reaching from 150 ms to 390 ms post-response. The mean

amplitude of the late Pe was measured at electrode Pz in a time window from 250 ms to 450 ms. The early and late Pe components were additionally analysed in the original ERPs, using the same time windows.

5.2 Results

5.2.1 Behavioural Data

5.2.1.1 Compatibility Effects

Compatibility effects on error rates and reaction times, depicted in *Figure 30*, were analysed using mixed ANOVAs with the within-subjects factor Compatibility (compatible, incompatible) and the between-subjects factor Age Group (young, middle-aged). Only trials with correct responses on the previous and the current trial were included in the analysis of reaction times.

Reaction times: Responses were generally faster on compatible (377 ms) than incompatible trials (424 ms), $F(1,55) = 529.33$, $p < .001$, $\eta_p^2 = .91$. Young participants responded faster than middle-aged participants (384 ms and 416 ms, respectively), $F(1,55) = 6.43$, $p = .014$, $\eta_p^2 = .11$. The interaction of both factors was significant, $F(1,55) = 4.12$, $p = .047$, $\eta_p^2 = .07$, indicating a slightly larger compatibility effect for middle-aged (51 ms) than young participants (42 ms).[21]

Error rates: Participants committed more errors on incompatible (15.1%) than on compatible trials (4.3%), $F(1,55) = 351.40$, $p < .001$, $\eta_p^2 = .87$. Young participants committed 10.3% errors and middle-aged participants committed 9.1% errors on average. However, neither the main effect of Age Group nor the interaction of both factors reached significance (both *p*s >.10).

[21] The interaction was not significant when the overall reaction time difference between groups was controlled, using log-normalized data ($p > .10$).

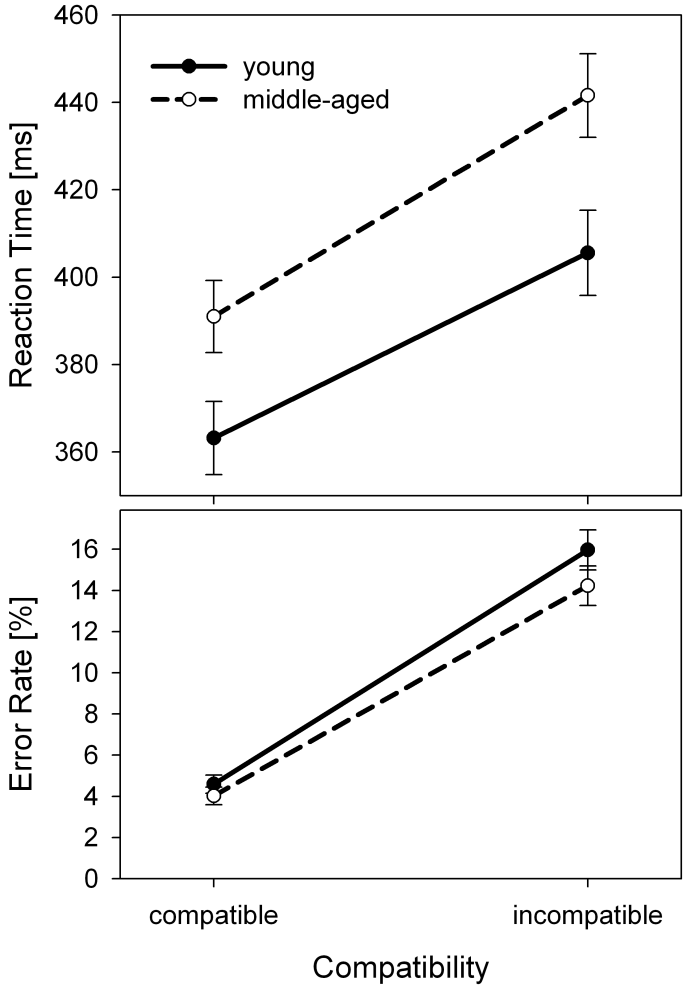

Figure 30: Compatibility effects on reaction times (top) and error rates (bottom) for young and middle-aged participants.

5.2.1.2 Sequential Adjustment Effects

Sequential adjustment effects on error rates and reaction times (see *Figure 31*) were analysed using mixed ANOVAs with the within-subjects factors Response Sequence (alternation, repetition), Trial N-1 Compatibility (compatible, incompatible), and Current Trial Compatibility (compatible, incompatible) and the between-subjects factor Age Group (young, middle-aged). Only trials with correct responses on the previous and the current trial were included in these analyses. Only interaction including the factor Trial N-1 Compatibility will be reported in this section.

Reaction times: Response repetitions (394 ms) were faster than alternations (407 ms), $F(1, 55) = 11.58$, $p = .001$, $\eta_p^2 = .17$. Responses were also faster following compatible (399 ms) than following incompatible trials (403 ms), $F(1, 55) = 24.48$, $p < .001$, $\eta_p^2 = .31$. However, this was only true for response alternations (403 ms

and 411 ms, respectively) and not for response repetitions (both 394 ms), as shown by a significant interaction between these two factors, $F(1, 55) = 10.43$, $p = .002$, $\eta_p^2 = .16$. Importantly, the interaction between Trial N-1 Compatibility and Current Trial Compatibility was significant, $F(1, 55) = 109.06$, $p < .001$, $\eta_p^2 = .67$, and interacted in turn with the factor Response Sequence, $F(1, 55) = 203.48$, $p < .001$, $\eta_p^2 = .79$. Post hoc test showed significant Trial N-1 Compatibility X Current Trial Compatibility interactions for both response alternations, $F(1, 55) = 16.60$, $p < .001$, $\eta_p^2 = .23$, and response repetitions, $F(1, 55) = 231.97$, $p < .001$, $\eta_p^2 = .81$. However, as can bee seen in the top panel of *Figure 31*, the typical Gratton effect ((c-ic – c-c) – (ic-ic – ic-c)) of a reduced compatibility effect following incompatible trials compared to following compatible trials was only present for response repetitions (39 ms). Response alternations showed a reversed Gratton effect instead (-7.4 ms).

Interestingly, the four-way interaction of Trial N-1 Compatibility, Current Trial Compatibility, Age Group and Response Sequence was also significant, $F(1, 55) = 9.87$, $p = .003$, $\eta_p^2 = .15$.[22] Post hoc tests showed a significant interaction between Trial N-1 Compatibility, Current Trial Compatibility, and Age Group for response repetitions, $F(1, 55) = 6.03$, $p = .035$, $\eta_p^2 = .10$, which was due to larger Gratton effect for middle-aged (45 ms) than for young participants (33 ms). The same interaction was only marginally significant for response alternations, $F(1, 55) = 4.68$, $p = .070$, $\eta_p^2 = .08$. None of the other effects including the factor Trial N-1 Compatibility reached significance (all *p*s > .10).

Error rates: Overall, participants committed more errors on trials following compatible (10.2%) than following incompatible trials (9.1%), $F(1, 55) = 18.75$, $p < .001$, $\eta_p^2 = .25$. A significant interaction between Trial N-1 Compatibility and Response Sequence, $F(1, 55) = 72.95$, $p < .001$, $\eta_p^2 = .57$, showed that this was only true for response repetitions (11.3% and 7.6%, respectively), $F(1, 55) = 107.86$, $p < .001$, $\eta_p^2 = .66$, whereas response alternations showed the opposite effect, i.e. smaller error rates following compatible (9.2%) than incompatible trials (10.7%), $F(1, 55) = 12.20$, $p = .002$, $\eta_p^2 = .18$. Importantly, there was a significant interaction between Trial N-1 Compatibility and Current Trial Compatibility representing the

[22] The four-way interaction remained significant when controlling for the general reaction time difference between groups, using log-normalized data, $F(1,55) = 8.74$, $p = .005$, $\eta_p^2 = .14$.

Gratton effect, $F(1, 55) = 58.62$, $p < .001$, $\eta_p^2 = .52$, which in turn interacted with the factor Response Sequence, $F(1, 55) = 120.00$, $p < .001$, $\eta_p^2 = .69$. Post hoc tests showed significant Trial N-1 Compatibility X Current Trial Compatibility interactions for both response alternations, $F(1, 55) = 13.20$, $p = .001$, $\eta_p^2 = .19$, and repetitions, $F(1, 55) = 141.76$, $p < .001$, $\eta_p^2 = .72$. However, as can be seen in the bottom panel of *Figure 31*, the typical Gratton effect ((c-ic – c-c) – (ic-ic – ic-c)) of a reduced compatibility effect following incompatible compared to compatible trials was only present for response repetitions (10.0%), whereas response alternations showed a reversed Gratton effect (-2.4%), i.e., an increased compatibility effect following incompatible trials. None of the other effects including the factor Trial N-1 Compatibility reached significance (all *p*s > .10).

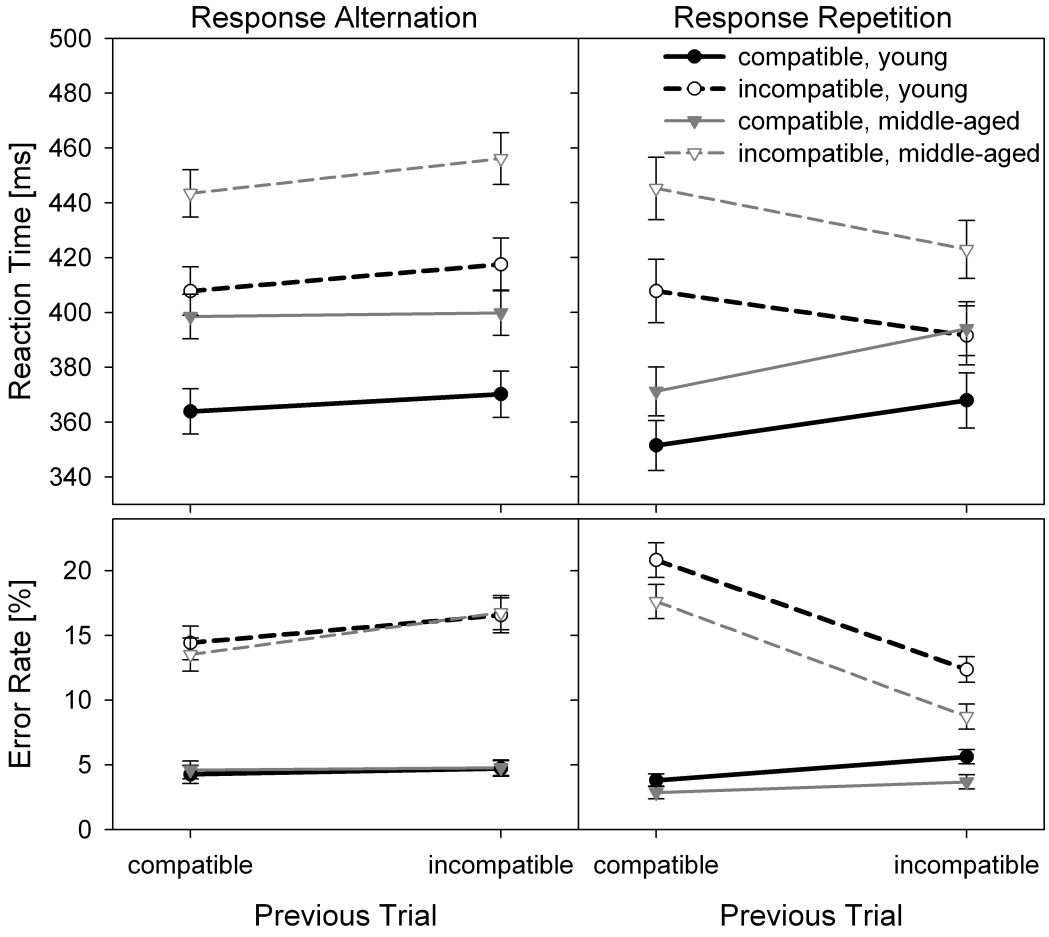

Figure 31: Gratton effect: Reaction times (top) and error rates (bottom) as a function of previous and current trial compatibility for young (black lines) and middle-aged (grey lines) participants for response alternations (left) and repetitions (right).

5.2.1.3 Error- and Post-Error Effects

Figure 32 shows the reaction times for correct trials following another correct response (post-correct), error trials following a correct response (error), and correct trials following an error (post-error) for both participant groups. Error speed-up and post-error slowing were analysed using a mixed ANOVA with the within-subjects factor Trial Type (post-correct, error, post-error) and the between-subjects factor Age Group (young, middle-aged). Post-error accuracy was analysed using a mixed ANOVA with the within-subjects factor Trial N-1 Accuracy (correct, incorrect) and the between-subjects factor Age Group (young, middle-aged). Since many more errors were made on incompatible than compatible trials, only trials following incompatible trials were included in these analyses.

Reaction times: There was a significant main effect of Trial Type, $F(2, 110) = 189.35$, $p < .001$, $\eta_p^2 = .78$, due to significant error speed-up and post-error slowing effects. Error responses (353 ms) were significantly faster, $F(1, 55) = 163.28$, $p < .001$, $\eta_p^2 = .75$, and post-error responses (431 ms) were significantly slower than post-correct responses (401 ms), $F(1, 55) = 69.95$, $p < .001$, $\eta_p^2 = .56$. Young participants were faster overall (374 ms) than middle-aged participants (415 ms), $F(1, 55) = 10.59$, $p = .002$, $\eta_p^2 = .16$. The interaction between both factors did not reach significance ($p > .10$).

Error rates: Participants committed less errors on post-error trials (6.4%) than post-correct trials (8.1%), $F(1, 55) = 11.05$, $p = .002$, $\eta_p^2 = .17$, suggesting a general increase in accuracy following errors. The main effect of Age Group and the interaction between both factors did not reach significance (both ps $> .10$).

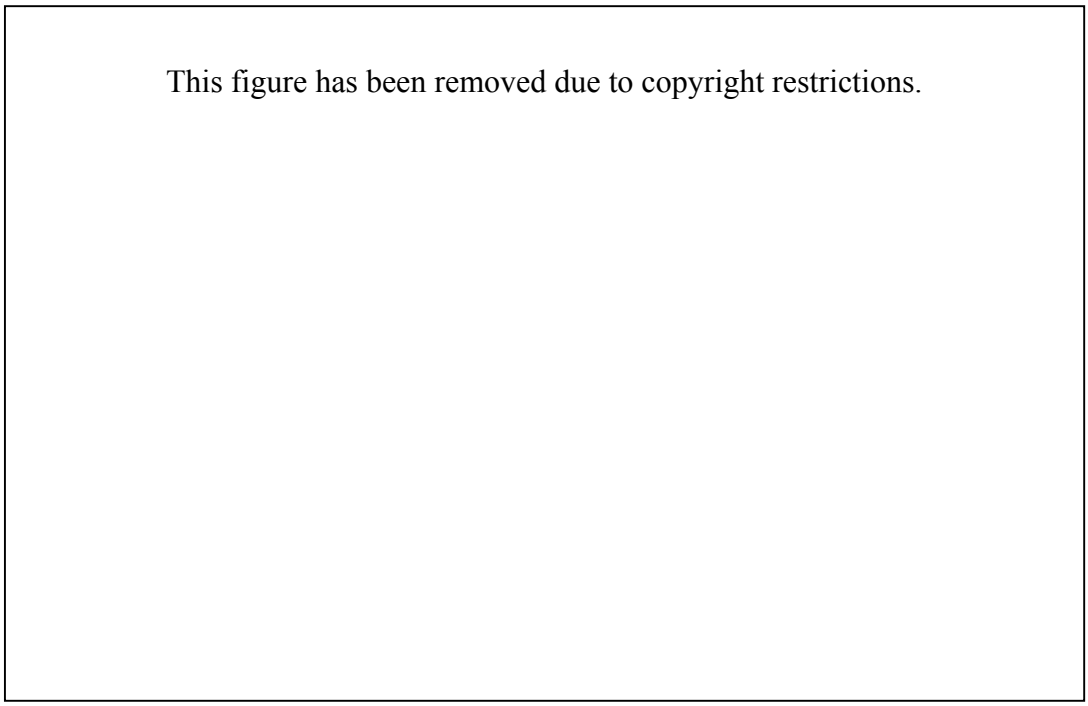

Figure 32: Error speed-up and post-error slowing for young and middle-aged participants.

5.2.2 Electrophysiological Data

5.2.2.1 P1 and N1

The early visual components P1 and N1 for young and middle-aged participants are shown in *Figure 33*. Peak amplitude and latency of these components were analysed using mixed ANOVAs with the within-subjects factors Hemisphere (left, right) and Compatibility (compatible, incompatible) and the between-subjects factor Age Group (young, middle-aged).

P1: The P1 peaked 103 ms after stimulus onset. It was larger for young (+4.2 μV) than for middle-aged participants (+2.8 μV), $F(1, 55) = 5.01$, $p = .029$, $\eta_p^2 = .08$. None of the other amplitude effects reached significance (all *p*s > .10). The P1 peaked earlier over the right (100 ms) than the left hemisphere (105 ms), $F(1, 55) = 4.60$, $p = .036$, $\eta_p^2 = .08$. None of the other effects on latency were significant (all *p*s > .05).

N1: The N1 peaked 166 ms after stimulus onset. It was larger over the right (-4.4 μV) than over the left hemisphere (-3.4 μV), $F(1, 55) = 5.75$, $p = .020$, $\eta_p^2 = .10$. There was a significant interaction between Hemisphere and Compatibility, $F(1, 55) = 6.20$, $p = .016$, $\eta_p^2 = .10$. However, post hoc test showed no significant

compatibility effect over either hemisphere (both *p*s > .10). Overall, the N1 was larger for middle-aged (-5.2 μV) than for young participants (-2.7 μV), $F(1, 55) = 8.76$, $p = .005$, $\eta_p^2 = .14$. A significant Hemisphere X Age Group interaction, $F(1, 55) = 5.71$, $p = .020$, $\eta_p^2 = .09$, indicated that the age effect was only significant over the left, $F(1, 55) = 17.66$, $p < .001$, $\eta_p^2 = .24$, but not over the right hemisphere ($p > .10$). None of the other amplitude effects reached significance (all *p*s > .10). For N1 latency, there were a significant Compatibility X Age Group interaction, $F(1, 55) = 6.76$, $p = .012$, $\eta_p^2 = .11$, and a marginally significant Hemisphere X Age Group interaction, $F(1, 55) = 3.77$, $p = .057$, $\eta_p^2 = .06$. However, post hoc test revealed no significant age effects in either of the conditions (all *p*s > .10). None of the other latency effects reached significance (all *p*s > .10).

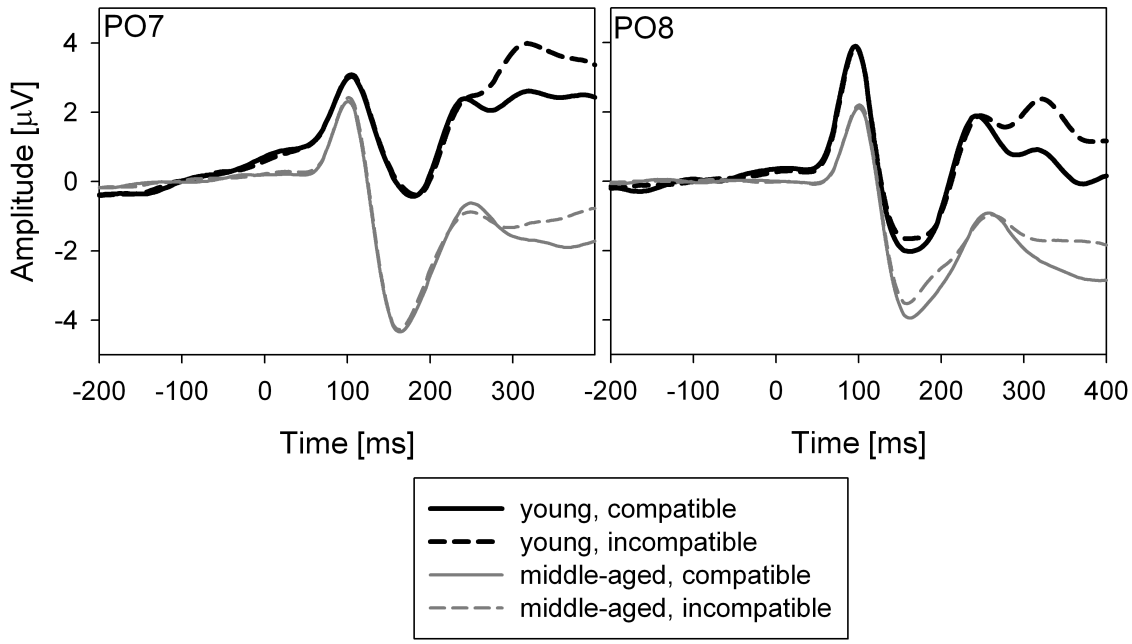

Figure 33: P1 and N1 for compatible and incompatible trials at electrodes PO7 and PO8 for young and middle-aged participants.

5.2.2.2 N2

Figure 34 shows the stimulus- and response-locked difference waves (incompatible minus compatible) for young and middle-aged participants at electrode FCz. Amplitude and latency of the N2 were analysed in the stimulus- and response-locked difference waves using two-tailed independent-samples *t*-tests. The amplitude of the N2 was also compared across stimulus- and response-locked waves using a mixed

ANOVA with the within-subjects factor Analysis Type (stimulus-locked, response-locked) and the between-subjects factor Age Group (young, middle-aged).

Stimulus-locked: The stimulus-locked N2 peaked 325 ms after stimulus onset. There was no significant difference in amplitude between the groups ($p > .10$). The N2 peaked significantly later for middle-aged (339 ms) than for young participants (311 ms), $t(55) = -3.59$, $p = .001$.

Response-locked: The response-locked N2 peaked 129 ms before response onset. It was larger for young (-1.5 µV) than for middle-aged participants (-0.6 µV), $t(55) = -3.18$, $p = .002$. There was no significant difference in latency between the groups ($p > .10$).

Stimulus- vs. response-locked: The N2 was significantly larger in the stimulus-locked (-1.9 µV) than in the response-locked analysis (-1.1 µV), $F(1, 55) = 40.82$, $p < .001$, $\eta_p^2 = .43$. The main effect of Age Group was marginally significant, $F(1, 55) = 3.71$, $p = .059$, $\eta_p^2 = .06$. The interaction of both factors was significant, $F(1, 55) = 7.04$, $p = .010$, $\eta_p^2 = .11$, due to the fact that there was only a significant age difference in the response-locked analysis (see above).

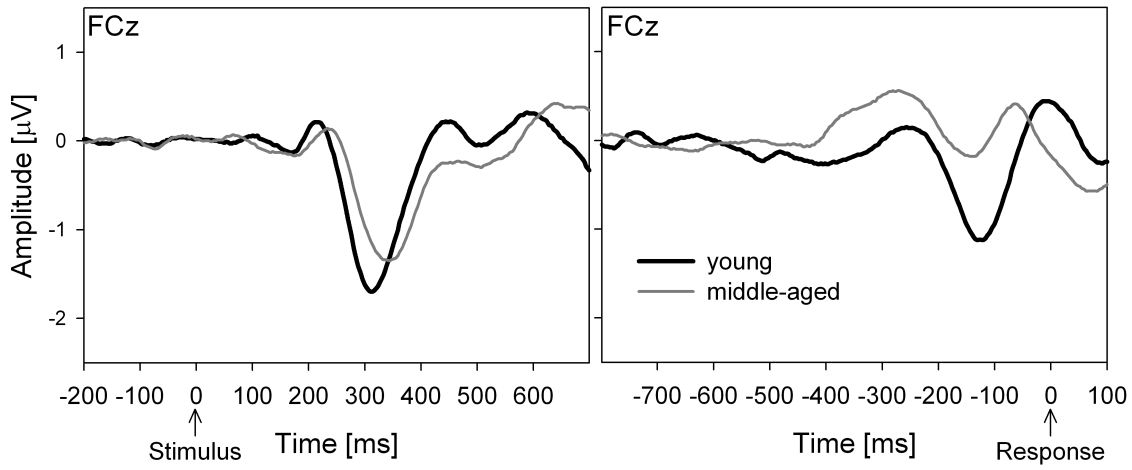

Figure 34: Stimulus-locked (left) and response-locked (right) difference waves (incompatible minus compatible) at electrode FCz for young and middle-aged participants.

5.2.2.3 Error-Related ERP Components

Figure 35 shows topographic maps at the time points of the respective peak amplitudes of Ne/ERN, early Pe, and late Pe for young and middle-aged participants. *Figure 36* shows the response-locked ERPs for correct and incorrect responses as well as the difference waves (incorrect minus correct) on incompatible trials for young and middle-aged participants at electrodes Fz, FCz, Cz, and Pz. Peak latency and amplitude of the Ne/ERN and the early Pe, as well as the average amplitude of the late Pe, were analysed in the difference waves using two-tailed independent-samples *t*-tests. The amplitude of the early and late Pe were additionally analysed in the original ERPs using mixed ANOVAs with the within-subjects factor Response Accuracy (correct, incorrect) and the between-subjects factor Age Group (young, middle-aged). The latency of the early Pe was not analysed in the original ERPs because there was no clearly identifiable peak for correct responses.

Figure 35: Topographic maps of the Ne/ERN at 70 ms (A), the early Pe at 245 ms for young and 280 ms for middle-aged participants (B) and the late Pe at 350 ms (C) post-response for young (top) and middle-aged (bottom) participants. Shown are the difference waves (incorrect minus correct) with 0.80 µV/step.

Ne/ERN: The Ne/ERN peaked 70 ms after response onset. It had a larger (i.e., more negative) amplitude for young (-8.4 μV) than for middle-aged participants (-5.1 μV), $t(55) = -3.37$, $p = .001$. There was no significant difference in latency between the groups ($p > .10$).

Early Pe: The early Pe in the difference waves was significantly larger for young (+8.4 μV) than for middle-aged participants (+5.7 μV), $t(55) = 2.47$, $p = .017$. It also peaked earlier for young (243 ms) than for middle-aged participants (282 ms), $t(55) = -2.82$, $p = .007$. In the analysis of the original ERPs, the main effect of Response Accuracy on amplitude was significant, $F(1, 55) = 74.77$, $p < .001$, $\eta_p^2 = .58$, whereas the main effect of Age Group was not ($p > .10$). Importantly, the interaction of both factors was significant, $F(1, 55) = 6.67$, $p = .012$, $\eta_p^2 = .11$. Post hoc tests showed a significant group differences for incorrect responses, $t(55) = 2.76$, $p = .016$, but not for correct responses ($p > .10$).

Late Pe: In the difference waves, the late Pe was larger for young (+6.6 μV) than for middle-aged participants (+3.8 μV), $t(55) = 3.53$, $p = .001$. In the analysis of the original ERPs, there were significant main effects of Response Accuracy, $F(1, 55) = 183.49$, $p < .001$, $\eta_p^2 = .78$, and Age Group, $F(1, 55) = 4.75$, $p = .034$, $\eta_p^2 = .08$, as well as significant interaction, $F(1, 55) = 12.59$, $p = .001$, $\eta_p^2 = .19$. Post hoc tests showed a significant group difference for correct response trials, $t(55) = -3.81$, $p < .001$, but not for incorrect response trials ($p > .10$).

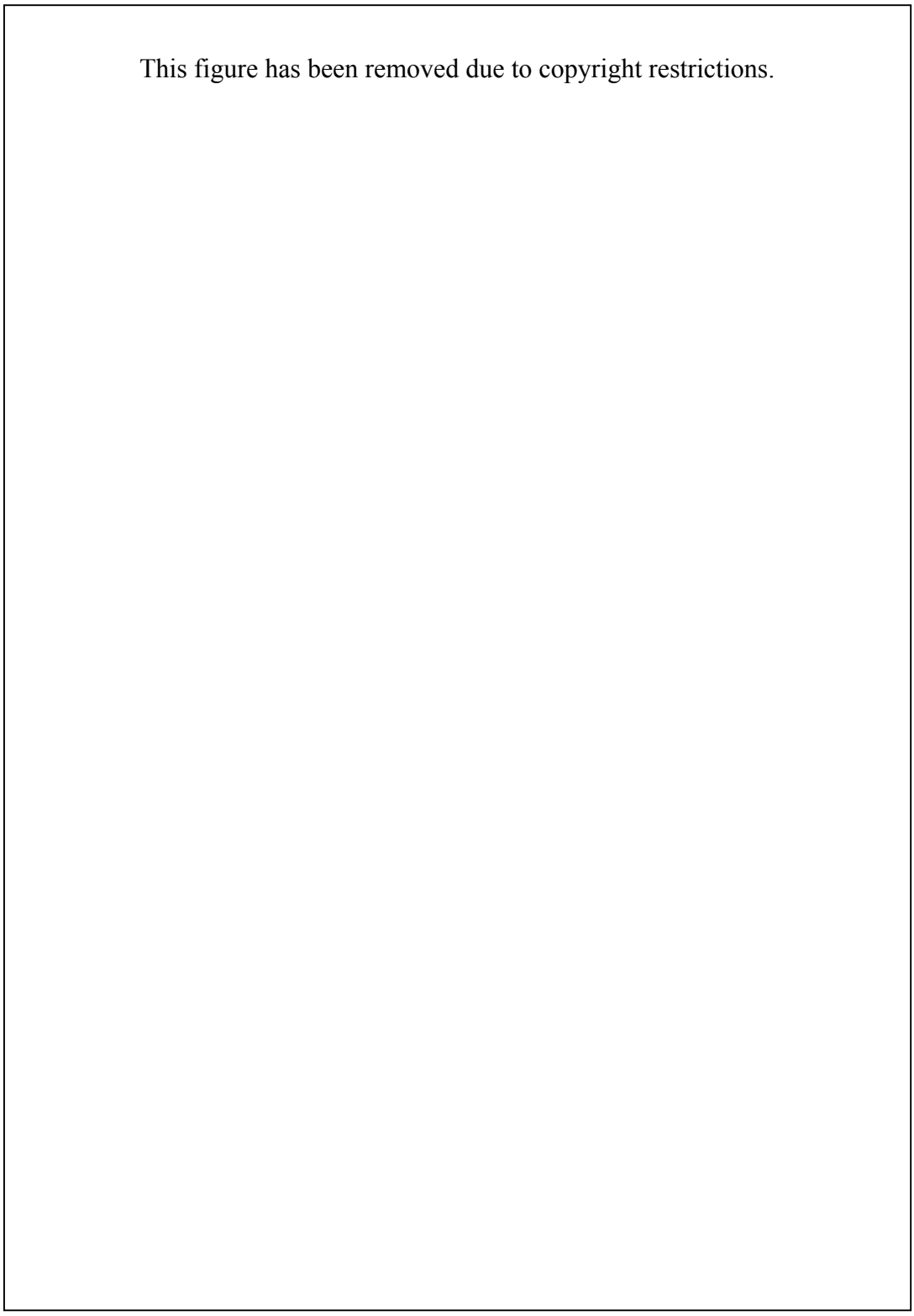

Figure 36: Response-locked ERPs for incompatible trials for young (black lines) and middle-aged (grey lines) participants at electrodes Fz, FCz, Cz, and Pz. Left: Correct (dashed lines) and incorrect (solid lines) response trials. Right: Difference waves (incorrect minus correct).

5.3 Discussion

This chapter aimed to investigate whether age-related changes in cognitive control, as described in the literature (see Section 1.5), are already present in middle-aged adults. There was a slight increase in the compatibility effect on reaction times for middle-aged compared to young participants. However, this increase was likely due to the general increase in reaction time for middle-aged participants, since the difference in the compatibility effect disappeared when the data were log-normalized. The N2 component, which is assumed to reflect conflict related to the compatibility effect, was clearly present in the stimulus-locked analysis for both age groups and did not differ in amplitude. However, there was a significant reduction in N2 amplitude for the middle-aged compared to the young group in the response-locked analysis. This result should be treated with caution though, since *Figure 34* (right panel) shows that the difference waves began to diverge between groups prior to the time window of the N2 analysis. Furthermore, as in the previous chapters, the comparison between stimulus- and response-locked analyses showed that the N2 was larger in the stimulus-locked difference waves and, therefore, most likely represents processes associated with stimulus rather than response processing. One possible explanation for the observed pattern of results is that the peak latency of the N2 showed a greater variability for middle-aged than young participants. This variability would be even further enhanced in the response-locked analysis, thus leading to the significant group difference in amplitude. This interpretation is indeed supported by the observed standard deviation of the stimulus-locked N2 peak latencies in both groups, which was larger for middle-aged (35 ms) than for young (24 ms) participants. This difference was even larger in the response-locked data (57 ms and 32 ms, respectively).

The stimulus-locked N2 was delayed in middle-aged compared to young adults. This is especially interesting, since there were no latency differences between young and middle-aged participants in the early visual components P1 and N1. In previous studies, both P1 and N1 have been shown to have longer latencies for older compared to young adults (both P1 and N1: Curran, Hills, Patterson, & Strauss, 2001; just the P1: Finnigan et al., 2011), which has been interpreted as delayed sensory processing in older age groups. It seems that in the middle-aged group of the current study visual processing speed was not reduced compared to the young group.

Therefore, it appears likely that the age difference in processing speed was specific to a later stage, i.e. the processing of stimulus conflict reflected by the N2 component.

The pattern of results found for the amplitude of the early visual components, i.e. an age-related decrease in the P1 amplitude along with an increase in N1 amplitude, has been reported previously in a study investigating older adults over the age of 60 years, using a memory task (Finnigan et al., 2011). The authors concluded that attentional suppression, reflected in the P1 amplitude, was less efficient in older adults, whereas orienting of attention towards the stimuli, reflected in N1 amplitude, was enhanced to maintain the level of performance. Similar processes might have been at work in the present study. Middle-aged participants might have been less efficient at suppressing flanker information, which they compensated by putting more effort into focussing on the target letter (cf. Wild-Wall et al., 2008). Along with the prolonged conflict processing, this resulted in overall slower reaction times but otherwise equal performance compared to young participants. One possible limitation of the data is that potential group differences in the contingent negative variation (CNV; Walter, Cooper, Aldridge, McCallum, & Winter, 1964) in the baseline interval might have impacted the P1 amplitude. This might have enhanced the difference between the groups and simulated an apparent age effect, especially over the left hemisphere where the difference is rather small (see *Figure 33*). However, the group differences in N1 amplitude appear to be too large to be caused by CNV carryover effects, suggesting that this interpretation of the data is unlikely. The hemisphere differences of the early visual components, i.e. earlier P1 latency and larger N1 amplitude over the right than over the left hemisphere, could be caused by the nature of the stimuli that were used. Since the stimuli were letter strings and the usual reading direction for most participants was from left to right, it is possible that participants paid overall more and earlier attention to the left side of the stimuli, resulting in earlier and larger activity in the right hemisphere of the brain.

Just like in the studies described in Chapters 3 and 4, the typical Gratton effect on error rates and reaction times was only present for response repetitions, suggesting that repetition priming plays an important role in the emergence of this effect. In the current study the Gratton effect was even reversed for response alternations, especially for middle-aged participants; compatibilitfy effects were

larger following incompatible than following compatible trials due to increased reaction times and error rates when two incompatible trials followed each other (see *Figure 31*). Neither the conflict monitoring, the repetition priming nor the feature integration account can explain this reversal of the Gratton effect. However, negative priming occurring on response alternation trial sequences might be able to account for this finding (see Section 7.1.2). For response repetitions, the Gratton effect was larger for middle-aged than for young participants indicating larger repetition priming in the older group. Larger repetition priming for older adults has been shown before (e.g., Witthöft, Sander, Süß, & Wittmann, 2009) and has been interpreted as a deficit in the inhibition of residual activation from the previous trial. More specifically, it seems likely that middle-aged participants profited more from exact stimulus-response repetitions than young adults did, because they took longer to deactivate the response representation of the previous trial.

Both young and middle-aged participants showed significant error speed-up and post-error slowing. Post-error slowing was accompanied by an increase in accuracy suggesting that participants adjusted their response criterion to more conservative levels following errors. There were no significant differences between the groups in either error speed-up or post-error slowing. That is, not only did middle-aged participants show similar automatic response threshold lowering leading to an error as young participants, they also were able to strategically adjust their response thresholds following errors as well as young adults (for a more detailed discussion of automatic and strategic response threshold adjustments see Section 4.3).

In contrast, there were age-related differences in error processing as indicated by the error-related ERP components. Although all error-related ERP components were present and showed their typical topographic distributions in both age groups, middle-aged participants had significantly smaller Ne/ERN and early Pe amplitudes, as well as a longer early Pe latency, compared to young participants. Reduced Ne/ERN amplitudes are a common finding in older adults (see Section 1.5.2); however, reduced amplitudes in a middle-aged sample have only been shown in one previous study (Gajewski et al., 2010) to my knowledge. In that study Ne/ERN amplitude reductions were restricted to middle-aged participants with a highly repetitive work environment. Considering that most middle-aged participants of the

current study were either PhD students or employees of the university, a repetitive work environment seems to be an unlikely explanation for the current findings. It is noteworthy that the Ne/ERN group differences were due to differences in error-related activity and not due to processes on correct trials. The left panel of *Figure 36* clearly shows that the ERPs for correct responses did not differ between groups in the time window of the Ne/ERN. Therefore, it is unlikely that group differences were due to increased error uncertainty in middle-aged adults as had been suggested previously (e.g., Band & Kok, 2000). If older adults were less sure about whether they committed an error or not than young adults, an increase in Nc/CRN amplitude would have been expected.

To my knowledge, this study is the first to investigate age effects on early and late aspects of the Pe separately. The results confirm the importance of distinguishing between these two subcomponents. The early Pe had a longer latency for middle-aged compared to young adults, possibly indicating prolonged error processing in the older age group. Furthermore, the early Pe was reduced in amplitude for middle-aged participants. The analysis of the original ERPs confirmed that the difference was indeed due to error- and not correct-related activity. The late Pe, on the other hand, was reduced in amplitude for middle-aged participants in the analysis of the difference waves; however, the analysis of the original ERPs revealed that the difference was actually due to occurrences on correct trials, whereas the late Pe on incorrect trials was of similar size in both groups. It is possible that the correct-related activity represents a CNV, indicating greater preparatory activity for the next trial in younger than older adults. However, the posterior distribution of this activity is rather atypical for the CNV (cf. Walter et al., 1964). Furthermore, the same difference in preparatory activity between groups should have also been expected on incorrect trials.

In conclusion, although middle-aged participants in this study responded slower overall and showed an increase in repetition priming compared to young adults, their performance was not more susceptible to conflicting information. The physiological data, on the other hand, showed age-related changes already at this early stage of ageing. Although early sensory processing was not yet delayed, the amplitude differences between the groups suggested changes in visual processing. Conflict processing took longer in middle-aged than in young participants.

Physiological indicators of error processing were already compromised in middle-aged participants. However, post-error adjustments were not affected. These findings argue against a previously suggested (e.g., Gehring et al., 1993; Nieuwenhuis et al., 2001) direct functional link between these ERP components and post-error slowing.

6 Single-Trial Analysis of Age Effects on the Ne/ERN[23]

The ERP data in the previous chapters were analysed using the conventional averaging approach described in Chapter 2. While this approach is a good way to reduce noise in the data, it also has some disadvantages. For example, it has to be assumed that the activity associated with an event is the same every time that event occurs. However, it has been shown that this is not always the case and that some ERP components, as well as task performance, can change over the course of a long experiment with increasing fatigue (e.g., Lorist et al., 2000), at least when motivation is low (Bonnefond et al., 2011). More importantly in the present context[24], amplitude reductions in one condition or group compared to another may be either due to smaller amplitudes on all trials or due to fully sized components on some trials and no signal on other trials. The averaging approach would cover up this kind of variation between trials. As mentioned in Section 1.6, the finding of a reduced Ne/ERN amplitude in older and middle-aged (see previous chapter) compared to young adults, is one example where this kind of logic applies. This reduction in amplitude might be due to a smaller error detection signal on all trials (error detection signal reduction) or due to failure to detect errors on some trials leading to the absence of the Ne/ERN selectively on these trials (lapses in error detection), while showing a fully sized signal on other trials.

For this reason, I performed a single-trial analysis on the Ne/ERN data described in the previous chapter to investigate the amplitude distribution of this component. If the age-related amplitude reduction were due to lapses in error detection, the distribution would be expected to be wider for middle-aged than for young participants due to the fact that it would be consisting of two distributions, i.e. a signal and a no-signal distribution, whereas the young participants' distribution would be expected to only consist of the signal distribution. *Figure 37* illustrates this

[23] Parts of this chapter have been published in Brain Research (Strozyk & Jentzsch, 2012).

[24] Studies finding effects of time-on-task usually used long experiments with no or very short breaks. In the current studies, on the other hand, participants were allowed to take longer breaks between blocks, and they were aware that the experimenter was monitoring their performance, which should have kept their motivation at a high level.

point. The black dashed line represents cases in which no Ne/ERN signal occurred; it therefore has a mean of zero. The black solid line represents trials in which an Ne/ERN occurred. The sum of the two Gaussians curves, represented in grey, has a larger variation than each of the individual curves. Therefore, a larger variance in the middle-aged than the young sample would be expected if the amplitude reduction were indeed due to more frequent lapses in error detection. On the other hand, if the amplitude reduction were due to reduced error detection signals, the opposite outcome would be predicted. That is, the young participants' distribution would be expected to have a larger variance than the middle-aged participants' distribution, because it has been shown that neural signals with higher amplitudes usually are more variable (e.g., Tolhurst, Movshon, & Thompson, 1981; Wiener, Oram, Liu, & Richmond, 2001). In addition to the distributions, I also analysed correlations between the single-trial Ne/ERN and a variety of error-related reaction times (pre-error, error, and post-error reaction time, as well as the post-error/pre-error and post-error/error differences), in order to investigate the proposed relationship between the Ne/ERN amplitude and post-error slowing (e.g., Gehring et al., 1993).

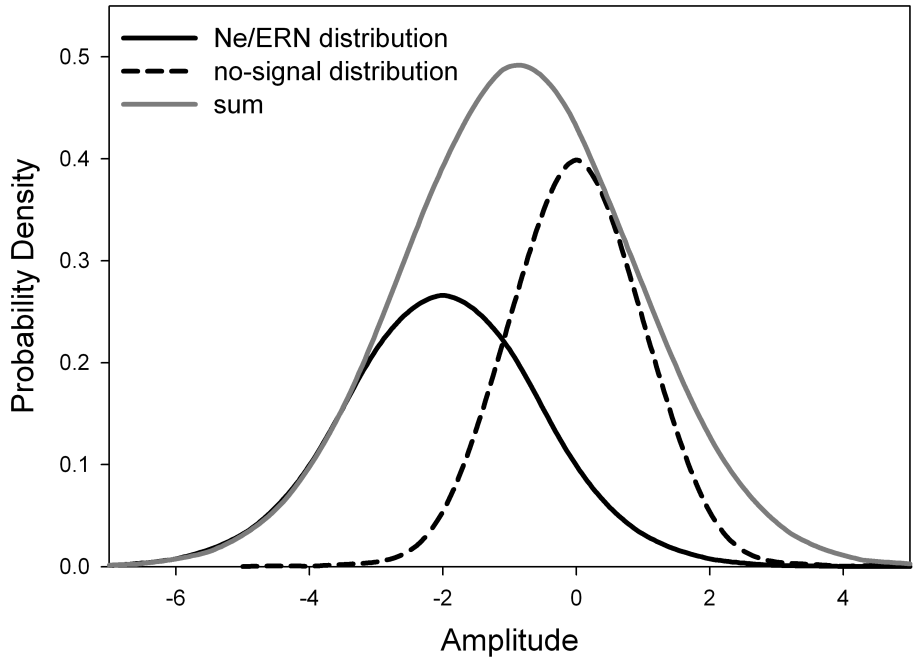

Figure 37: Illustration of the hypothesis that middle-aged participants show reduced Ne/ERN amplitudes due to more frequent lapses in error detection. The summation of the Ne/ERN and the no-signal distribution leads to a larger variation compared to the signal distribution alone.

6.1 Methods

6.1.1 Participants

The single-trial analysis was performed on a subset of the participants described in Chapter 5, who had a sufficient number of artefact free error trials (N > 40). The remaining participants were 23 young (M = 21.7 years, range 18 to 31 years, 13 women) and 19 middle-aged adults (M = 48.2 years, range 41 to 57 years, 10 women).

6.1.2 Stimuli, Apparatus, Procedure and Design

The data from the experiment described in Chapters 3 and 5 were used for single-trial analysis. Details of the experimental settings and procedure are described in these chapters.

6.1.3 Data Analysis

Eye movement artefacts in the EEG data were corrected using the same method as in the analysis of the averages. The continuous EEG was segmented into epochs of 1800 ms length, starting 1000 ms before the onset of an incorrect response. Epochs containing artefacts were rejected after manual inspection. Each participant's peak latency of the Ne/ERN was determined in the individual average. The single-trial amplitude was then measured as the average of a time window of about 28 ms duration around the individual's peak latency at electrode FCz, using a baseline correction interval from 150 ms to 50 ms before response onset. The mean amplitude of the time window from 1000 ms to 900 ms before the response was used as a no-signal comparison measure. Considering that the average reaction time was around 400 ms and that the RSI was 1000 ms long, the no-signal interval was expected to lie about halfway between the response to the previous trial and the stimulus onset of the current trial. The next step was to fit a Gaussian curve into the single-trial Ne/ERN amplitude distribution and the no-signal distribution for each participant using MATLAB (Mathworks). R^2 was calculated as a measure of goodness of fit. The mean μ and the standard deviation σ of the fitted curves were analysed using mixed ANOVAs. Conservative Huynh-Feldt tests were used throughout. Adjusted *p*-values are reported, along with the uncorrected degrees of freedom. Bonferroni corrected *p*-values are reported for all post hoc analyses.

6.2 Results

6.2.1 Analysis of the parameters of the fitted curves

Figure 38 shows the single-trial data distribution and corresponding Gaussian fits for the Ne/ERN amplitude and the amplitude of the no-signal comparison interval. *Figure 39* depicts the four Gaussian fits in one graph for comparison purposes. The average R^2s for the Ne/ERN fits were .79 for young and .65 for middle-aged participants, respectively. The average R^2s for the no-signal fits were .86 for young and .81 for middle-aged participants, respectively. Mean μ and standard deviation σ of the fitted curves were analysed with mixed ANOVAs including the within-subjects factor Distribution Type (signal, no-signal) and the between-subjects factor Age Group (young, middle-aged).

Mean μ: The mean of the fitted curves was more negative in the signal than in the no-signal distribution (-5.21 μV and -0.69 μV, respectively), $F(1, 40) = 73.00$, $p < .001$, $\eta_p^2 = .65$. Overall, the mean of young and middle-aged participants did not differ significantly ($p > .10$); however, the interaction of both factors was significant, $F(1, 40) = 23.86$, $p < .001$, $\eta_p^2 = .37$. Post hoc test showed that the mean of the signal distribution was more negative for young (-6.06 μV) than for middle-aged participants (-4.18 μV), $t(40) = -2.36$, $p = .047$, whereas the mean of the no-signal distribution was more negative for middle-aged (-2.34 μV) than for young participants (+0.68 μV), $t(40) = 3.30$, $p = .004$.

Standard deviation σ: The standard deviation of the fitted curves was larger for the signal than for the no-signal distributions (+7.38 μV and +5.59 μV, respectively), $F(1, 40) = 72.42$, $p < .001$, $\eta_p^2 = .64$, and larger for young than for middle-aged participants (+7.38 μV and +5.41 μV, respectively), $F(1, 40) = 18.70$, $p < .001$, $\eta_p^2 = .32$. The factors did not interact significantly ($p > .10$).

Correlations between these measures: The signal distribution's mean was significantly correlated with its standards deviation, $r = -.384$, $p = .012$, showing that larger, i.e. more negative, Ne/ERN amplitudes were associated with more variation. The standard deviation of signal and no-signal distributions were positively correlated, $r = +.795$ $p < .001$. None of the other correlations reached significant levels (all ps > .10).

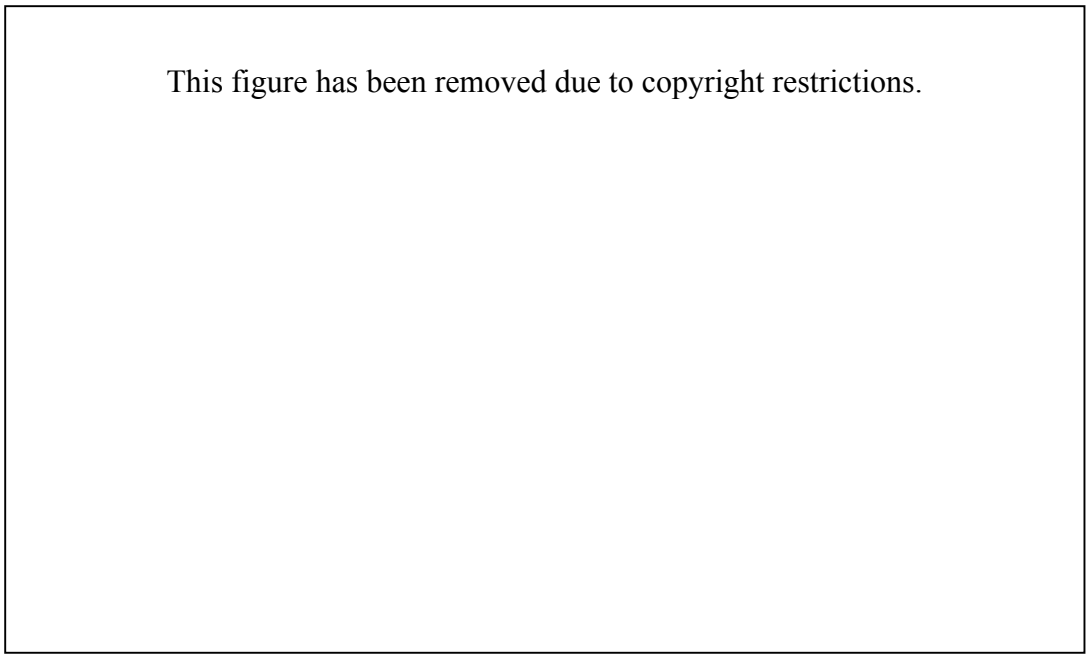

Figure 38: Data distribution and Gaussian fit for young and middle-aged participants for the Ne/ERN signal interval (left) and the no-signal comparison interval (right).

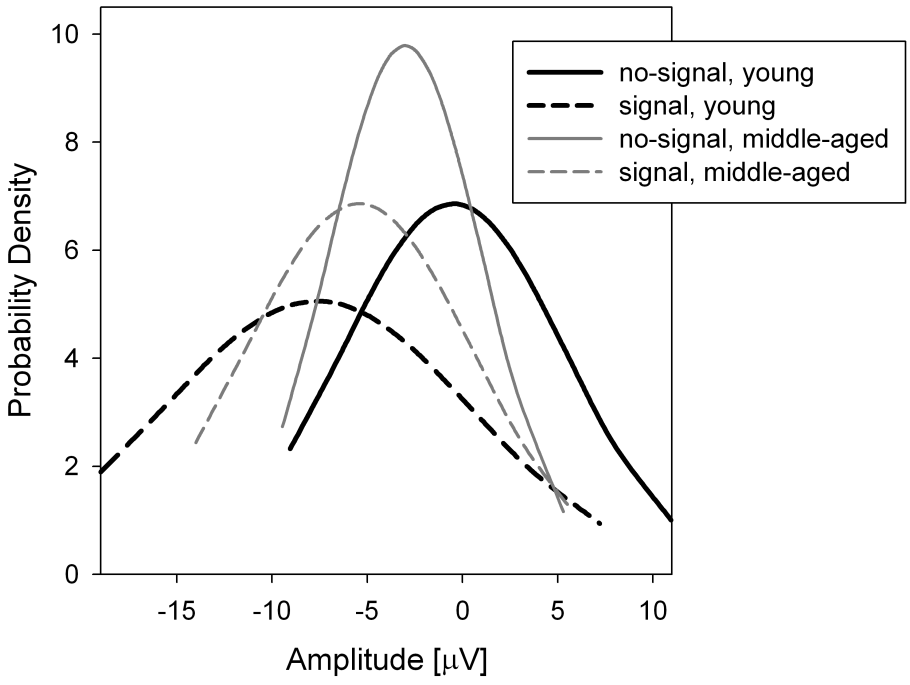

Figure 39: Gaussian fits of the signal (dashed lines) and no-signal (solid lines) distributions for young and middle-aged participants.

6.2.2 Correlations between single-trial Ne/ERN amplitude and reaction times

The amplitude of the single-trial Ne/ERN was correlated with the reaction times on trials preceding the error, the error trials themselves, and the trials following errors, as well as with the differences between post- and pre-error trials and post-error and error trials. Correlations were calculated for every individual and then averaged using Fisher's Z transformation. The back-transformed correlation coefficients are shown in *Table 3*. None of the correlations reached significance (all *p*s > .10).

Table 3: Correlations of the single-trial Ne/ERN amplitude with pre-error, error and post-error reaction times as well as the post-error minus pre-error and post-error minus error reaction time differences for young and middle-aged participants and for both age groups together.

	Pre-error	Error	Post-error	Post-error – pre-error	Post-error – error
Young	-.017	.007	.006	.020	.001
Middle-aged	.031	.007	-.048	-.024	-.044
Overall	.004	.007	-.017	-.011	-.019

6.3 Discussion

The main reason for analysing the amplitude of the Ne/ERN on a single-trial basis was to compare the distribution between groups, in order to investigate whether the age-related amplitude reduction described in the previous chapter was due to smaller error signals in middle-aged compared to young participants or due to more frequent lapses in error detection. The no-signal comparison interval was introduced to validate the method and to obtain a measure of the general noise in the data. Overall, the data show that both signal and no-signal distribution could be reasonably well fitted using Gaussian curves. As expected, the mean of the signal distribution was more negative than the mean of the no-signal distribution. Furthermore, the mean of the signal distribution was more negative for the young than for the middle-aged participants, reflecting the reduction in Ne/ERN amplitude. More importantly, the

standard deviation of the signal distribution was smaller for middle-aged than young participants. This result is in line with the interpretation that middle-aged participants had an overall smaller error signal than young participants rather than more frequent lapses in error detection, since a summation of a no-signal and an Ne/ERN signal distribution would have resulted in an overall wider distribution (see *Figure 37*). The standard deviation of the signal distribution was larger than that of the no-signal distribution, and there was a significant correlation between signal mean and signal standard deviation, both indicating larger variability for trials with larger amplitudes. These findings are in line with previous research showing that larger signals are associated with larger variability (e.g., Tollhurst et al., 1981; Wiener et al., 2001). In conclusion, the smaller standard deviation of the Ne/ERN amplitude distribution, along with the less negative mean for middle-aged compared to young adults, can be explained by an overall smaller error detection signal in the older participant group.

The analysis of the no-signal distribution yielded a surprising result. As no task-related activity was expected for this time interval, the mean of this distribution was anticipated to centre around zero. This was the case for the young participants; however, the mean of the distribution for the middle-aged participants was slightly negative. It is possible that this amplitude difference reflects an increased CNV in middle-aged compared to young participants, indicating increased task preparation in the older group. The response-locked ERPs for correct responses, shown in *Figure 36* in the previous chapter, indeed show negative activity for middle-aged participants starting around 400 ms after response onset, which does not seem to be present in young participants. This interpretation is in line with results of a study by Wild-Wall, Hohnsbein, and Falkenstein (2007), who found a similar increase in CNV at frontal electrodes in their middle-aged participant group, using a visual search task. It seems to be at odds with the observation of a possible age-related increase in CNV amplitude at electrode Pz described in Chapter 5 though. However, age-related changes in preparatory activity reflected in the CNV are not very consistent and seem to vary according to task demands (e.g., Gajewski et al., 2010; Wild-Wall et al., 2007). For example, Ferrandez and Pouthas (2001) found increased CNV amplitudes for middle-aged compared to young adults at central electrodes and the opposite effect at frontal electrodes in a time estimation task. Therefore, it appears possible that the CNV age-effect might be reversed when comparing anterior

and posterior electrodes. The smaller standard deviation of the no-signal distribution for middle-aged compared to young participants might indicate that middle-aged participants as a group were very invested in the task and therefore all showed great preparatory activity, whereas the younger group showed more variability in motivation and therefore in preparatory activity. However, this is highly speculative and cannot be tested in the current data set.

The amplitude of the single-trial Ne/ERN did not correlate significantly with either the reaction time on the trial preceding the error, the error reaction time itself, or the post-error reaction time. The correlations between the reaction time differences and Ne/ERN amplitude also did not reach significance. This finding clearly contradicts accounts like the conflict monitoring theory (Botvinick et al., 2001) that propose a close relationship between the Ne/ERN and post-error slowing. As mentioned in Section 1.4.2, previous studies that reported significant correlations between the average Ne/ERN amplitude and post-error slowing usually used post-error reaction times for their analyses (e.g., Debener et al., 2005; Gehring et al., 1993, Ladouceur et al., 2007). In these cases, it is possible that general fluctuations in reaction times and Ne/ERN amplitude simulate an apparent correlation that would not be present if difference scores (post-error minus post-correct) had been used. Additional correlational analyses of the averaged Ne/ERN data presented in Chapter 5 confirmed this interpretation.[25] There were no significant correlations between the Ne/ERN amplitude and post-error slowing (expressed as a reaction time difference) in either the difference waves or the original ERPs. However, the Ne/ERN amplitude in the difference waves correlated with both post-error reaction time and post-correct reaction time, indicating that it is the overall reaction time and not specifically post-error slowing that is associated with this component. Interestingly, these correlations

[25] There were no significant correlations between post-error slowing (post-error minus post-correct) and the Ne/ERN amplitude in the averaged data, neither in the difference waves nor in the original ERPs (both *p*s >. 10). The correlations between Ne/ERN amplitude and post-error as well as post-correct reaction times reached significance in the difference waves (r = .403, p = .002 and r = .417, p = .001, respectively) but not in the original ERPs (both *p*s > .10). The correlations between the Nc/CRN amplitude and post-error reaction time as well as post-correct reaction time reached significance (r = -.378, p = .004 and r = -.314, p = .017, respectively).

were not driven by activity on incorrect but on correct trials, as shown by the fact that the Nc/CRN amplitude in the original ERPs, but not the Ne/ERN amplitude, correlated with these reaction time measures. Longer reaction times were associated with larger (more negative) Nc/CRN amplitudes, possibly indicating that participants with slower reaction times showed more error monitoring on correct trials.

In conclusion, the single-trial analysis of the Ne/ERN showed that the age-related amplitude reduction of this component was most likely driven by generally weaker error signals and not by lapses in error detection. Furthermore, the amplitude of the Ne/ERN does not seem to be predictive of the amount of subsequent post-error slowing. Instead the Ne/ERN might trigger post-error adjustments in an all-or-none fashion. The reduced error signal for middle-aged participant might still be strong enough to trigger normal post-error slowing, which could explain the apparent dissociation of reduced Ne/ERN amplitude along with unchanged post-error slowing described in the previous chapter. However, this account cannot explain reports of post-error slowing in the absence of an Ne/ERN (e.g., Band & Kok, 2000).

7 General Discussion

This project was concerned with the investigation of effects of conflict strength and ageing on cognitive control. Chapters 3 and 4 described two different approaches to manipulate conflict strength in the Eriksen flanker task, i.e. variation of target and flanker contrast and of stimulus onset asynchrony. Effects of ageing on behavioural and electrophysiological measures of cognitive control were reported in Chapters 5 and 6. In the following, I will discuss these two issues separately, before turning towards the subject of a possible functional link between the Ne/ERN and subsequent behavioural adjustments.

7.1 Conflict Strength and Cognitive Control

7.1.1 The Manipulation of Conflict Strength

The studies described in Chapters 3 and 4 showed that, under the right circumstances, both the independent manipulation of flanker and target contrast, and the manipulation of SOA, can be used to affect conflict strength in the Eriksen flanker task. In the ERP study described in Chapter 3 the contrast of flankers and target was manipulated in a block-wise manner. Based on the conflict definitions of Berlyne (1957), conflict was expected to be enhanced when the target was light and flankers were dark and reduced when the target was dark and flankers were light, compared to the standard condition of all dark letters. In the follow-up study the same contrast conditions were used but instead of varying them block-wise, they were presented in a randomized fashion within each block. Reducing the contrast of the flanker letters compared to the target letter diminished the compatibility effect numerically in the ERP study and significantly in the follow-up study. The smaller effect in the blocked design might have been due to the fact that participants adapted to the contrast condition at hand by adjusting contrast sensitivity at a basic visual level. Alternatively, different contrast conditions might have been associated with different levels of proactive control. For example, the condition with dark target and light flankers might have been perceived as easy, leading to an overall lower level of proactive control than in other conditions. Differences in proactive control between contrast conditions are only possible in a blocked (ERP study) but not in a randomized design (follow-up study), potentially explaining the differences between the two experiments. Contrary to the prediction of increased conflict strength in the

condition with light targets and dark flankers, compatibility effects were not increased in either of the two studies. It is possible that a pop-out effect on these trials facilitated focussing on the target letter and counteracted the effects of conflict (see Section 3.3 for details).

In the two studies described in Chapter 4, conflict was manipulated by using three different SOAs; i.e. flanker onset was simultaneous with target onset (SOA 0) or preceded it by 100 ms or 200 ms (SOA 100 and SOA 200, respectively). In accordance with previous findings (Wascher et al., 1999; Willemssen et al., 2004), compatibility effects were largest at SOA 100. Earlier flanker onset in that condition most likely did lead to increased flanker-associated response activation compared to SOA 0, and therefore more interference on incompatible trials and more facilitation on compatible trials. At the longest SOA (200 ms), flanker-associated activation was probably already partly inhibited at the time of target onset, leading to smaller compatibility effects than at SOA 100. This interpretation is supported by findings of Wascher et al. (1999) and Willemssen et al. (2004) who reported largest flanker-associated incorrect motor activation, measured using lateralized readiness potentials, for incompatible trials for SOA 100 compared to simultaneous flanker-target onset and longer SOAs (500 ms and 400 ms, respectively).

Although the findings of Wascher et al. (1999) and Willemssen et al. (2004) can be interpreted in terms of conflict strength, the authors did not set out to test predictions of the conflict monitoring theory. Therefore, they did not investigate effects of conflict strength on control adjustment effects. To my knowledge, effects of flanker and target contrast on the compatibility effect have not been investigated previously. However, studies using different spacing between target and flankers (e.g., Danielmeier et al., 2009; Kopp, Rist, et al., 1996; Sullivan, 1999) followed a similar approach to the manipulation of conflict. In these studies, flanker influence was reduced by increasing the space between target and flankers, while in the current studies, flanker influence was reduced by decreasing flanker contrast. None of the spacing studies investigated influences of conflict strength on control adjustment effects either.

7.1.2 Effects of Conflict Strength on Control Adjustment Effects

The conflict monitoring theory explains the sequential adjustment or Gratton effect as a shift in attentional control after the experience of conflict (e.g., Botvinick et al., 2001). Based on this assumption, control adjustments should be influenced by conflict strength. More specifically, strong conflict should lead to larger adjustments than weak conflict. The compatibility effect should therefore be reduced to a larger degree following incompatible trials in a high conflict than in a low conflict condition.

Sequential adjustment effects were present in all four experiments described in Chapters 3 and 4. The compatibility effect (incompatible minus compatible) was smaller following incompatible than following compatible trials. However, this effect was influenced by the response sequence. In all four experiments there was a large Gratton effect for response repetitions, which was greatly reduced for response alternations. This finding indicates that repetition priming played an important role in the emergence of the Gratton effect. More specifically, in accordance with previous findings (e.g., Mayr et al., 2003), responses were especially fast and accurate when the exact same stimulus was repeated (i.e. c-c and ic-ic for response repetitions).

Nevertheless, control adjustments due to the experience of conflict seem to also contribute towards sequential adjustment effects. Several researchers have found a Gratton effect even when repetition priming was not possible (flanker task: e.g., Freitas et al., 2009; Clayson & Larson, 2011; Ullsperger et al., 2005; Stroop task: e.g., Kerns et al., 2004; Simon task: Stürmer et al., 2002). In the current experiments the Gratton effect remained marginally significant for response alternations in the ERP study described in Chapter 4. More importantly, the sequential adjustment effect was influenced by the SOA manipulation in that study. The Gratton effect ((c-ic – c-c) – (ic-ic – ic-c)) was smaller in the SOA 0 condition than in the conditions with longer SOAs, and this effect occurred independent of the response sequence.[26] The same effect did not reach significance in the pilot study; however,

[26] The effect of SOA on the Gratton effect cannot be explained by differences in associative strength between the stimulus and the response as proposed by Davelaar and Stevens (2009). If the amount of priming were dependent on the amount of conflict on the previous trial, increased priming would be predicted for SOA 100 compared to the other SOA conditions. However, this effect should have been

the Gratton effect was numerically largest at SOA 100. This pattern of results can be best explained by the conflict monitoring theory. Conflict on incompatible trials was largest at SOA 100, as shown by the increased compatibility effects for this SOA. Control adjustments should, therefore, be largest in this condition too.

The contrast manipulation in the blocked design study described in Chapter 3 also influenced the size of the Gratton effect. However, in this case the effect was specific to response repetitions and not present for response alternations. Therefore, it appears unlikely that this result was driven by conflict adaptation, since conflict effects should have been present for both response repetitions and alternations. More specifically, the Gratton effect for response repetitions was larger for stimulus arrays in which target and flanker contrast were the same than for arrays with mixed contrast in the blocked design study. When contrast conditions were presented in a within-block randomized fashion, on the other hand, there were no differences in adjustment for the four different target-flanker contrasts. This difference between experiments can be explained by repetition priming assuming that perceptual grouping did lead to stronger memory traces in conditions of same contrast. Since repetitions of the same contrast condition were less frequent in the follow-up study due to the randomized design, stronger memory traces did not lead to larger priming effects (see Section 3.3 for details).

While the separation of response repetitions and alternations provides a convenient method to disentangle effects of repetition priming and control adjustment, it also has the disadvantage that negative priming effects may mask control adjustment effects on alternation trial sequences (Egner, 2007; Ullsperger et al, 2005). More specifically, when a response had to be inhibited on the previous trial and has to be activated on the current trial, responses are slower compared to a control condition without repetition of any kind of stimulus aspect (e.g., Stadler & Hogan, 1996). In the current experiments, this would be the case for ic-ic and ic-c trial sequences for response alternations (e.g., HHSHH-SSHSS and HHSHH-HHHHH). The negative priming effect is even larger when, additionally, the previously activated response has to be inhibited on the current trial (ic-ic response alternations in the current experiments; Stadler & Hogan, 1996). Negative priming

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specific to response repetitions, since repetition priming does not occur for response alternations.

can therefore explain the reverse Gratton effect for response alternations on error rates in the contrast experiments.

In conclusion, sequential adjustment effects in the flanker task cannot be explained by either the conflict monitoring theory or repetition priming alone. Both control adjustments and priming effects contributed towards the Gratton effect in the current experiments. Future studies should therefore account for both by either separating between response repetitions and alternations or by using larger stimulus sets that include fewer repetitions of stimulus aspects, and thereby reduce effects of priming.

The other control adjustment effect investigated in this project was post-error slowing. Traditionally, post-error slowing has been explained as a strategic shift of a response criterion to more conservative levels to avoid further errors (e.g., Brewer & Smith, 1984; Jentzsch & Leuthold, 2006; Laming, 1968; Saunders & Jentzsch, in press). Post-error responses are therefore expected to not only be slower but also more accurate than post-correct responses. In terms of the conflict monitoring theory, this effect reflects an increase in cognitive control after the experience of conflict associated with an error, leading to a reduction in response priming (e.g., based on flanker information in the flanker task or colour word information in the Stroop task) and, therefore, longer reaction times. Alternatively, Notebaert et al. (2009) proposed that post-error slowing does not reflect changes in control but rather an orienting response towards the error as a surprising event and subsequent reorientation to the task. Importantly, according to this theory accuracy should not be increased after an error.

Post-error slowing was present in all experiments, except for the ERP study with SOA manipulation described in Chapter 4. The lack of slowing in that experiment was likely due to the increased RSI associated with the addition of the fixation point (see Sections 4.2.1.3 and 4.3 for details). In the studies described in Chapter 3, slowing was accompanied by an increase in accuracy, arguing against the orienting account of post-error slowing and in favour of the strategic adjustment hypothesis. Contrary to the two contrast manipulation studies, post-error accuracy did not differ from post-correct accuracy in the SOA pilot study. It is possible that this was due to an RSI confound. The interval between response and target onset did depend on the SOA condition in this study. In accordance with previous research

showing that post-error slowing decreases with increasing RSI (e.g., Dudschig & Jentzsch, 2009), slowing was largest for SOA 0 and largely reduced for longer SOAs In fact, post-error slowing was only significant for SOA 0 but not for the longer SOAs.[27] Since the strategic adjustment account of post-error slowing predicts an increase in post-error accuracy due to shift towards a more conservative response criterion following an error, an increase in accuracy should only be expected for SOA 0, which may have led to the overall insignificant effect of accuracy.[28]

According to the conflict monitoring theory, the amount of post-error slowing as a control adjustment effect should depend on the degree of conflict associated with an error. However, as current results show, post-error slowing does not seem to be modulated by conflict strength. Although flanker effects were reduced in the condition with dark targets and light flankers in the follow-up study described in Chapter 3, post-error slowing was of similar size in all contrast conditions.[29] As most errors occur on incompatible trials, continued stimulus processing after the response should have led to more correct response activation in a time window immediately following the error, leading to larger post-error conflict in this low conflict condition than in the other conditions (cf. Danielmeier et al., 2009). Since the adjustment of control is expected to depend on the amount of conflict, post-error slowing should have been increased. This finding argues against the conflict explanation of post-error slowing but is compatible with the idea of a strategic adjustment in response criterion. Error speed-up, on the other hand, was affected by conflict strength in the

[27] Post hoc tests for post-error slowing in the three SOA conditions showed the following results: SOA 0: $F(1, 17) = 20.77$, $p < .001$, $\eta_p^2 = .55$; SOA 100: $F(1, 17) = 6.03$, $p = .075$, $\eta_p^2 = .26$; SOA 200: $p > .10$.

[28] Although not significant, inspection of the data did indeed show a reduction in error rate for SOA 0 from 10.1% post-correct to 7.2% post-error. For SOAs 100 and 200 the error rates were more similar (SOA 100: 9.8% and 10.8%; SOA 200: 11.2% and 11.4% respectively).

[29] The size of the post-error slowing effect did not vary with conflict strength in the other experiments either. However, these results have to be treated with caution because the conflict strength manipulation was not successful in the ERP study of chapter 3, post-error slowing was confounded by differences in RSI in the pilot study in chapter 4, and slowing was not significant in the ERP study in chapter 4.

SOA experiments. Conditions with high conflict on incompatible trials seem to lead to especially fast errors due to increased flanker interference.

7.1.3 Effects of Conflict Strength on Error- and Conflict-Associated ERPs

The conflict monitoring theory proposed that the Ne/ERN reflects conflict associated with an error. That is, the simultaneous activation of incompatible response representations, which occurs during errors that are due to premature responding, is thought to elicit this component. Conflict associated with correct responses, on the other hand, is thought to be reflected in the amplitude of the N2 (e.g., Yeung et al., 2004; see also Section 1.4.2). In agreement with these hypotheses, conflict strength in the flanker tasks investigated in the current experiments should have influenced the amplitude of these components.

The amplitude of the N2 was expected to be enhanced for high relative to low conflict conditions. In line with this prediction and with previous findings (e.g., Bartholow et al., 2005; Boksem et al., 2005; Danielmeier et al., 2009; Freitas et al., 2009; Heil et al., 2000; Ladouceur et al., 2007; Van ’t Ent, 2002), the N2 was enhanced for incompatible compared to compatible trials in both ERP studies. The amplitude was not further modulated by the contrast manipulation in the first study; however, neither were behavioural conflict effects. In the second ERP study, the amplitude of the incompatible-compatible difference wave followed the expected pattern with numerically largest N2 for SOA 100; however, this effect was not significant. Furthermore, the N2 was larger in the stimulus-locked than in the response-locked analysis in both studies. Taken together, these results indicated that the N2 might not reflect response conflict, since that should have been largest immediately before the response (Botvinick et al., 2001; Yeung et al., 2004). Instead, the N2 seems to reflect processes associated more closely with the stimulus, possibly semantic conflict associated with stimulus processing. This interpretation is in line with the finding of an enhanced N2 for a condition with stimulus conflict but without response conflict (Wendt et al., 2007; see also Section 1.4.2).

The predictions for the amplitude of the Ne/ERN are less straightforward than for the N2. As Danielmeier et al. (2009) pointed out, Ne/ERN amplitude was expected to be smaller in a high conflict conditions with flankers close to the target than in a low conflict condition with more distant flankers. Continued stimulus

processing following an error was expected to lead to larger post-error conflict due to increased correct response activation in the condition with smaller flanker influence (see also Section 1.4.2). Since the contrast manipulation in the studies of Chapter 3 was also varying conflict strength by changing flanker influence, similar effects of conflict on Ne/ERN amplitude as in the Danielmeier study should have been expected. The SOA manipulation, on the other hand, did vary conflict size by giving flanker processing a temporal advantage over target processing. At the time of the response, stimuli of different SOAs did not differ. Post-error correct activation should therefore not differ between these conditions. However, it could be argued that errors on incompatible trials in the SOA 100 condition might be associated with increased incorrect activation compared to the other SOAs. Conflict associated with an error should therefore be larger, leading to increased Ne/ERN amplitudes in this condition.

The Ne/ERN was not influenced by the conflict manipulation in either of the studies. This might not be very surprising in the first study, considering that the contrast manipulation did not affect behavioural measures of conflict either. However, in the second study behavioural conflict effects were largest at SOA 100, while the Ne/ERN did not distinguish between conditions. This finding is in line with results from a study by Masaki et al. (2007), in which the size of the compatibility effect in a Simon task was also not reflected in Ne/ERN amplitude. The reduced Ne/ERN amplitude for stimuli with close compared to far flanker-target distance in the study by Danielmeier et al. (2009) might alternatively be explained by a difference in error rate between the conditions. Participants committed more errors for the close than for the far spacing, which may have led to a decrease in Ne/ERN amplitudes (cf. Hajcak et al., 2003, 2004; Herrmann et al., 2004; Holroyd & Coles, 2002).

The current findings contradict the hypothesis that the Ne/ERN reflects the degree of conflict between incompatible response representations. Instead, the Ne/ERN may be triggered in an all-or-none fashion. That is, as soon as the conflict reaches a certain threshold, a fully sized Ne/ERN is elicited. However, the current findings are also compatible with the hypotheses that the Ne/ERN represents the detection of an error (e.g., Bernstein et al., 1995; Falkenstein et al., 1991) or the transmission of a reinforcement learning signal (Holroyd & Coles, 2002).

7.2 Ageing and Cognitive Control

Chapters 5 and 6 were concerned with the question how cognitive control processes are affected by ageing. Whereas previous research has focussed mostly on older adults above the age of 60 years, I investigated age-related differences in a younger sample of middle-aged adults (40 to 60 years). Using this relatively younger group of participants might give some insight into the early development of changes in cognitive control in an age group that is still active in the workspace.

Middle-aged participants responded overall slower than young participants, while maintaining a comparable error rate. The increase in reaction time was, therefore, not due to differences in speed-accuracy tradeoff, since a stronger emphasis on accuracy in middle-aged compared to young adults should have resulted in reduced error rates. The compatibility effect did not differ between age groups when controlling for the general increase in reaction times, suggesting that there was no generic difference in the amount of interference between young and middle-aged participants. In line with this finding, there were no significant amplitude differences in the stimulus-locked N2 between the groups either. The N2 had a longer latency for middle-aged than for young adults, probably indicating prolonged stimulus or semantic conflict processing. This interpretation is supported by the fact that early visual components (i.e. P1 and N1) were not delayed for middle-aged compared to young participants, indicating normal sensory processing speed. The age-related latency increase for the N2 must, therefore, have been specific to a later processing stage (see Section 5.3 for details). In sum, middle-aged participants needed longer to resolve conflict as suggested by the longer N2 latency, but they were able to resolve it as well as younger participants, as there were no differences in the compatibility effect.

Age-related changes in sequential adjustment effects have not been studied extensively. The few studies that did investigate effects of age on the Gratton effect using the Stroop task (West & Moore, 2005; Monti et al., 2010) have come to inconclusive results (see Section 1.5.1). None of these studies controlled for effects of priming by comparing response repetitions and alternations. In the study described in Chapter 5, the overall Gratton effect did not interact significantly with the factor age group. However, when investigating response repetitions and alternations separately, some differential effects emerged. The Gratton effect was larger for

middle-aged than young participants for response repetitions, indicating increased repetition priming for the older age group. The same interaction was only marginally significant for response alternations. As *Figure 31* shows, the reverse Gratton effect was slightly larger for middle-aged compared to young participants, possibly indicating a small increase in negative priming. This is a surprising finding considering that negative priming has usually been found to be reduced with increased age (e.g., May, Kane, & Hasher, 1995; Witthöft et al., 2009). In conclusion, control adjustment was still intact in middle-aged adults. Any age-related differences in sequential adjustment effects were due to priming effects.

This conclusion of intact control adjustment effects is also supported by the post-error slowing results. Both age groups showed similar amounts of error speed-up and post-error slowing. Post-error slowing was accompanied by an increase in accuracy in young and middle-aged participants, providing further support for the strategic adjustment hypothesis of post-error slowing. Interestingly, physiological indicators of error detection were affected by ageing. The Ne/ERN was reduced in amplitude for middle-aged compared to young participants, suggesting that physiological changes in error processing can be found at an earlier age than previously known. The single-trial analysis described in Chapter 6 showed that this amplitude reduction was not due to lapses in error detection but rather reflected a general reduction in the strength of the error signal, possibly due to decreased dopaminergic activity. The early Pe was also reduced in amplitude for middle-aged participants. As suggested previously (e.g., van Veen & Carter, 2002), the early Pe might be related to the same underlying processes as the Ne/ERN. However, the slightly more posterior distribution of the early Pe compared to the Ne/ERN argues against this suggestion. Furthermore, similarly to the N2 results, the peak amplitude of the early Pe was delayed in middle-aged relative to young participants, indicating prolonged error monitoring processes in this age group. The late Pe, on the other hand, was of comparable size in both groups. It therefore appears that middle-aged participants were as aware of their errors as younger participants. Taken together, the current results show the importance of distinguishing between an early and a late subcomponent of the Pe, since both were differentially affected by ageing.

In conclusion, although middle-aged participants showed some changes in electrophysiological indicators of error detection and conflict processing, they

seemed to be able to compensate for them, since these changes did not manifest themselves in behaviour. The only significant behavioural changes present in this age group were overall slower response times and an increase in repetition priming.

7.3 The Relationship Between the Ne/ERN and Post-Error Slowing

Previous research has suggested that the size of the Ne/ERN might be related to post-error slowing (e.g., Debener et al., 2005; Gehring et al., 1993; Hewig et al., 2011; Hirsh & Inzlicht, 2010; Ladouceur et al., 2007; West & Travers, 2008). Although the investigation of this link was not one of the main aims of this research project, the current data can shed some light on this question. First, post-error slowing did not differ between the age groups in the study described in Chapter 5, while the Ne/ERN amplitude did. This finding argues against a close link between the amplitude of this component and post-error slowing. Second, I investigated correlations between the single-trial Ne/ERN amplitude and reaction times on the error trial, on the trial following the error and the trial preceding the error, as well as the post-error/pre-error and post-error/error differences in reaction times. None of these correlations reached significance, in either the individual age groups nor overall. Furthermore, additional correlational analyses between the Ne/ERN and the Nc/CRN in the averaged ERPs and reaction times revealed that previous findings of such an association might have been due to a relationship between the Nc/CRN and post-error as well as post-correct reaction times. Unless correlations are analysed between the Ne/ERN in the original ERPs, instead of the difference waves, and the reaction time difference between post-error and post-correct trials, the association between Nc/CRN and reaction times might have simulated an apparent relationship between the Ne/ERN and post-error slowing. However, it remains possible that the Ne/ERN triggers post-error slowing in an all-or-none fashion. That is, if the error signal reaches a certain threshold, slowing might occur. Any further increases in the signal might not lead to further differences in response slowing. However, this account cannot explain results showing post-error slowing under conditions in which no significant Ne/ERN occurred (e.g., Alain et al., 2002; Band & Kok, 2000). It is therefore likely that the Ne/ERN and post-error slowing are not directly related.

7.4 General Conclusions

In this thesis, predictions of the conflict monitoring theory were tested by manipulating conflict strength in the Eriksen flanker task. Overall, the predictions could be partly confirmed. Compatibility effects were reduced for light flankers when the target was dark, at least when contrast conditions were presented in a randomized fashion. As expected, compatibility effects were largest at an SOA of 100 ms. These conflict effects appear to be represented in the amplitude of the N2. However, conflict reflected in this component seems to be more closely associated with stimulus processing than with response execution. Sequential adjustment effects were affected by conflict strength to a certain degree, although this effect is difficult to investigate due to overlapping effects of repetition priming and negative priming. Nevertheless, the overall pattern of results followed the predictions of the conflict monitoring theory. The theory's hypotheses concerning error processing, on the other hand, could not be confirmed. Both the Ne/ERN and the post-error slowing effect have been explained in terms of conflict associated with the occurrence of an error (Botvinick et al., 2001). As such, Ne/ERN amplitude and the amount of post-error slowing should have been expected to be affected by the degree of conflict. However, neither of them was influenced by the conflict strength manipulation. Errors seem to constitute a special case of information processing, which is in line with findings of studies showing that activation of certain brain areas within the rostral ACC is specific to errors (e.g., Mathalon, Whitfield, et al., 2003).

Furthermore, this research project showed that effects of ageing on physiological indicators of conflict and error processing could be found at an earlier stage than previously known. Interestingly, these changes did not manifest themselves in behaviour. Although middle-aged adults responded overall slower than young adults, conflict did not affect their performance in a different way. Therefore, it appears that at this early stage of ageing, people can successfully compensate for changes in brain activity.

References

Alain, C., McNeely, H. E., He, Y., Christensen, B. K., & West, R. (2002). Neurophysiological evidence of error-monitoring deficits in patients with schizophrenia. *Cerebral Cortex, 12*(8), 840-846. doi: 10.1093/cercor/12.8.840

Appelbaum, L. G., Meyerhoff, K. L., & Woldorff, M. G. (2009). Priming and Backward Influences in the Human Brain: Processing Interactions during the Stroop Interference Effect. *Cerebral Cortex, 19*(11), 2508-2521. doi: 10.1093/cercor/bhp036

Arbel, Y., & Donchin, E. (2009). Parsing the componential structure of post-error ERPs: A principal component analysis of ERPs following errors. *Psychophysiology, 46*(6), 1179-1189. doi: 10.1111/j.1469-8986.2009.00857.x

Band, G. P. H., & Kok, A. (2000). Age effects on response monitoring in a mental-rotation task. *Biological Psychology, 51*(2-3), 201-221. doi: 10.1016/S0301-0511(99)00038-1

Band, G. P. H., Ridderinkhof, K. R., & Segalowitz, S. (2002). Explaining neurocognitive aging: Is one factor enough? *Brain and Cognition, 49*(3), 259-267. doi: 10.1006/breg.2001.1499

Band, G. P. H., Ridderinkhof, K. R., & van der Molen, M. W. (2003). Speed-accuracy modulation in case of conflict: the roles of activation and inhibition. *Psychological Research-Psychologische Forschung, 67*(4), 266-279. doi: 10.1007/s00426-002-0127-0

Bartholow, B. D., Pearson, M. A., Dickter, C. L., Sher, K. J., Fabiani, M., & Gratton, G. (2005). Strategic control and medial frontal negativity: Beyond errors and response conflict. *Psychophysiology, 42*(1), 33-42. doi: 10.1111/j.1469-8986.2005.00258.x

Beason-Held, L. L., Kraut, M. A., & Resnick, S. M. (2009). Stability of Default-Mode Network Activity in the Aging Brain. *Brain Imaging and Behavior, 3*(2), 123-131. doi: 10.1007/s11682-008-9054-z

Ben-Av, M. B., & Sagi, D. (1995). Perceptual grouping by similarity and proximity - Experimental results can be predicted by intensity autocorrelations. *Vision Research, 35*(6), 853-866. doi: 10.1016/0042-6989(94)00173-j

Berger, H. (1929). Über das Elektrenkephalogramm des Menschen. *Archiv für Psychiatrie und Nervenkrankheiten, 87*, 527-570. doi: 10.1007/BF01797193

Berlyne, D. E. (1957). Uncertainty and conflict: A point of contact between information-theory and behavior-theory concepts. *Psychological Review, 64*(6, Part 1), 329-339. doi: 10.1037/h0041135

Bernstein, P. S., Scheffers, M. K., & Coles, M. G. H. (1995). Where did I go wrong - a psychophysiological analysis of error-detection. *Journal of Experimental Psychology-Human Perception and Performance, 21*(6), 1312-1322. doi: 10.1037//0096-1523.21.6.1312

Beste, C., Willemssen, R., Saft, C., & Falkenstein, M. (2009). Error processing in normal aging and in basal ganglia disorders. *Neuroscience, 159*(1), 143-149. doi: 10.1016/j.neuroscience.2008.12.030

Bialystok, E., Craik, F. I. M., Klein, R., & Viswanathan, M. (2004). Bilingualism, aging, and cognitive control: Evidence from the Simon task. *Psychology and Aging, 19*(2), 290-303. doi: 10.1037/0882-7974.19.2.290

Boksem, M. A. S., Tops, M., Wester, A. E., Meijman, T. F., & Lorist, M. M. (2006). Error-related ERP components and individual differences in punishment and reward sensitivity. *Brain Research, 1101*, 92-101. doi: 10.1016/j.brainres.2006.05.004

Bonnefond, A., Doignon-Camus, N., Hoeft, A., & Dufour, A. (2011). Impact of motivation on cognitive control in the context of vigilance lowering: An ERP study. *Brain and Cognition, 77*(3), 464-471. doi: 10.1016/j.bandc.2011.08.010

Botvinick, M. M. (2007). Conflict monitoring and decision making: Reconciling two perspectives on anterior cingulate function. *Cognitive Affective & Behavioral Neuroscience, 7*(4), 356-366. doi: 10.3758/CABN.7.4.356

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*(3), 624-652. doi: 10.1037//0033-295x.108.3.624

Botvinick, M. M., Cohen, J. D., & Carter, C. S. (2004). Conflict monitoring and anterior cingulate cortex: an update. *Trends in Cognitive Sciences, 8*(12), 539-546. doi: 10.1016/j.tics.2004.10.003

Botvinick, M. M., Nystrom, L. E., Fissell, K., Carter, C. S., & Cohen, J. D. (1999). Conflict monitoring versus selection-for-action in anterior cingulate cortex. *Nature, 402*(6758), 179-181. doi: 10.1038/46035

Braver, T. S. (2012). The variable nature of cognitive control: a dual mechanisms framework. *Trends in Cognitive Sciences, 16*(2), 106-113. doi: 10.1016/j.tics.2011.12.010

Braver, T. S., Barch, D. M., Gray, J. R., Molfese, D. L., & Snyder, A. (2001). Anterior cingulate cortex and response conflict: Effects of frequency, inhibition and errors. *Cerebral Cortex, 11*(9), 825-836. doi: 10.1093/cercor/11.9.825

Brewer, N., & Smith, G. A. (1984). How normal and retarded individuals monitor and regulate speed and accuracy of responding in serial choice tasks. *Journal of Experimental Psychology-General, 113*(1), 71-93. doi: 10.1037//0096-3445.113.1.71

Brodmann, M. (2009). Anterior cingulate cortex. Coloured by was_a_bee. Retrieved 19 September, 2012, from http://commons.wikimedia.org/wiki/File:Gray727_anterior_cingulate_cortex.png

Burle, B., Allain, S., Vidal, F., & Hasbroucq, T. (2005). Sequential compatibility effects and cognitive control: Does conflict really matter? *Journal of Experimental Psychology-Human Perception and Performance, 31*(4), 831-837. doi: 10.1037/0096-1523.31.4.831

Burle, B., Roger, C., Allain, S., Vidal, F., & Hasbroucq, T. (2008). Error negativity does not reflect conflict: A reappraisal of conflict monitoring and anterior cingulate cortex activity. *Journal of Cognitive Neuroscience, 20*(9), 1637-1655. doi: 10.1162/jocn.2008.20110

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4*(6), 215-222. doi: 10.1016/S1364-6613(00)01483-2

Carbonnell, L., & Falkenstein, M. (2006). Does the error negativity reflect the degree of response conflict? *Brain Research, 1095*, 124-130. doi: 10.1016/j.brainres.2006.04.004

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science, 280*(5364), 747-749. doi: 10.1126/science.280.5364.747

Carter, C. S., Macdonald, A. M., Botvinick, M., Ross, L. L., Stenger, V. A., Noll, D., & Cohen, J. D. (2000). Parsing executive processes: Strategic vs. evaluative functions of the anterior cingulate cortex. *Proceedings of the National Academy of Sciences of the United States of America, 97*(4), 1944-1948. doi: 10.1073/pnas.97.4.1944

Clayson, P. E., & Larson, M. J. (2011). Conflict adaptation and sequential trial effects: Support for the conflict monitoring theory. *Neuropsychologia, 49*(7), 1953-1961. doi: 10.1016/j.neuropsychologia.2011.03.023

Coderre, E., Conklin, K., & van Heuven, W. J. B. (2011). Electrophysiological measures of conflict detection and resolution in the Stroop task. *Brain Research, 1413*, 51-59. doi: 10.1016/j.brainres.2011.07.017

Coles, M. G. H., Scheffers, M. K., & Holroyd, C. B. (2001). Why is there an ERN/Ne on correct trials? Response representations, stimulus-related components, and the theory of error-processing. *Biological Psychology, 56*(3), 173-189. doi: 10.1016/S0301-0511(01)00076-X

Croft, R. J., & Barry, R. J. (2000). Removal of ocular artifact from the EEG: a review. *Neurophysiologie Clinique-Clinical Neurophysiology, 30*(1), 5-19. doi: 10.1016/s0987-7053(00)00055-1

Cropley, V. L., Fujita, M., Innis, R. B., & Nathan, P. J. (2006). Molecular imaging of the dopaminergic system and its association with human cognitive function. *Biological Psychiatry, 59*(10), 898-907. doi: 10.1016/j.biopsych.2006.03.004

Curran, T., Hills, A., Patterson, M. B., & Strauss, M. E. (2001). Effects of aging on visuospatial attention: an ERP study. *Neuropsychologia, 39*(3), 288-301. doi: 10.1016/s0028-3932(00)00112-3

Czigler, I., Csibra, G., & Ambro, A. (1996). Aging, stimulus identification and the effect of probability: An event-related potential study. *Biological Psychology, 43*(1), 27-40. doi: 10.1016/0301-0511(95)05173-2

Damoiseaux, J. S., Beckmann, C. F., Arigita, E. J. S., Barkhof, F., Scheltens, P., Stam, C. J., . . . Rombouts, S. (2008). Reduced resting-state brain activity in the "default network" in normal aging. *Cerebral Cortex, 18*(8), 1856-1864. doi: 10.1093/cercor/bhm207

Danielmeier, C., Wessel, J. R., Steinhauser, M., & Ullsperger, M. (2009). Modulation of the error-related negativity by response conflict. *Psychophysiology, 46*(6), 1288-1298. doi: 10.1111/j.1469-8986.2009.00860.x

Davelaar, E. J., & Stevens, J. (2009). Sequential dependencies in the Eriksen flanker task: A direct comparison of two competing accounts. *Psychonomic Bulletin & Review, 16*(1), 121-126. doi: 10.3758/pbr.16.1.121

Davies, P. L., Segalowitz, S. J., Dywan, J., & Pailing, P. E. (2001). Error-negativity and positivity as they relate to other ERP indices of attentional control and stimulus processing. *Biological Psychology, 56*(3), 191-206. doi: 10.1016/s0301-0511(01)00080-1

De Pisapia, N., & Braver, T. S. (2006). A model of dual control mechanisms through anterior cingulate and prefrontal cortex interactions. *Neurocomputing, 69*(10-12), 1322-1326. doi: 10.1016/j.neucom.2005.12.100

Debener, S., Ullsperger, M., Siegel, M., Fiehler, K., von Cramon, D. Y., & Engel, A. K. (2005). Trial-by-trial coupling of concurrent electroencephalogram and functional magnetic resonance imaging identifies the dynamics of performance monitoring. *Journal of Neuroscience, 25*(50), 11730-11737. doi: 10.1523/jneurosci.3286-05.2005

Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error-detection and compensation. *Psychological Science, 5*(5), 303-305. doi: 10.1111/j.1467-9280.1994.tb00630.x

Desjardins, R., & Warnke, A. (2012). Ageing and skills: A review and analysis of skill gain and skill loss over the lifespan and over time. *OECD Education Working Papers*, No. 72, OECD Publishing. doi: http://dx.doi.org/10.1787/5k9csvw87ckh-en

Di Russo, F., Martinez, A., & Hillyard, S. A. (2003). Source analysis of event-related cortical activity during visuo-spatial attention. *Cerebral Cortex, 13*(5), 486-499. doi: 10.1093/cercor/13.5.486

Dirnberger, G., Lang, W., & Lindinger, G. (2010). Differential effects of age and executive functions on the resolution of the contingent negative variation: a reexamination of the frontal aging theory. *Age, 32*(3), 323-335. doi: 10.1007/s11357-010-9134-z

Donkers, F. C. L., & van Boxtel, G. J. M. (2004). The N2 in go/no-go tasks reflects conflict monitoring not response inhibition. *Brain and Cognition, 56*(2), 165-176. doi: 10.1016/j.bandc.2004.04.005

Dudschig, C., & Jentzsch, I. (2009). Speeding before and slowing after errors: Is it all just strategy? *Brain Research, 1296*, 56-62. doi: 10.1016/j.brainres.2009.08.009

Dutilh, G., Vandekerckhove, J., Forstmann, B., Keuleers, E., Brysbaert, M., & Wagenmakers, E.-J. (2012). Testing theories of post-error slowing. *Attention, Perception, & Psychophysics, 74*(2), 454-465. doi: 10.3758/s13414-011-0243-2

Easdon, C., Izenberg, A., Armilio, M. L., Yu, H., & Alain, C. (2005). Alcohol consumption impairs stimulus- and error-related processing during a Go/No-Go Task. *Cognitive Brain Research, 25*(3), 873-883. doi: 10.1016/j.cogbrainres.2005.09.009

Egner, T. (2007). Congruency sequence effects and cognitive control. *Cognitive Affective & Behavioral Neuroscience, 7*(4), 380-390. doi: 10.3758/cabn.7.4.380

Endrass, T., Franke, C., & Kathmann, N. (2005). Error awareness in a saccade countermanding task. *Journal of Psychophysiology, 19*(4), 275-280. doi: 10.1027/0269-8803.19.4.275

Endrass, T., Klawohn, J., Schuster, F., & Kathmann, N. (2008). Overactive performance monitoring in obsessive-compulsive disorder: ERP evidence from correct and erroneous reactions. *Neuropsychologia, 46*(7), 1877-1887. doi: 10.1016/j.neuropsychologia.2007.12.001

Eppinger, B., Kray, J., Mock, B., & Mecklinger, A. (2008). Better or worse than expected? Aging, learning, and the ERN. *Neuropsychologia, 46*(2), 521-539. doi: 10.1016/j.neuropsychologia.2007.09.001

Erickson, K. I., Milham, M. P., Colcombe, S. J., Kramer, A. F., Banich, M. T., Webb, A., & Cohen, N. J. (2004). Behavioral conflict, anterior cingulate cortex, and experiment duration: Implications of diverging data. *Human Brain Mapping, 21*(2), 98-107. doi: 10.1002/hbm.10158

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon identification of a target letter in a nonsearch task. *Perception & Psychophysics, 16*(1), 143-149. doi: 10.3758/BF03203267

Eriksen, C. W., & Hoffman, J. E. (1973). The extent of processing of noise elements during selective encoding from visual displays. *Perception & Psychophysics, 14*(1), 155-160. doi: 10.3758/BF03198630

Erixon-Lindroth, N., Farde, L., Wahlin, T. B. R., Sovago, J., Halldin, C., & Backman, L. (2005). The role of the striatal dopamine transporter in cognitive aging. *Psychiatry Research-Neuroimaging, 138*(1), 1-12. doi: 10.1016/j.pscychresns.2004.09.005

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. 2. Error processing in choice reaction tasks. *Electroencephalography and Clinical Neurophysiology, 78*(6), 447-455. doi: 10.1016/0013-4694(91)90062-9

Falkenstein, M., Hoormann, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: a tutorial. *Biological Psychology, 51*(2-3), 87-107. doi: 10.1016/S0301-0511(99)00031-9

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (1999). ERP components in Go Nogo tasks and their relation to inhibition. *Acta Psychologica, 101*(2-3), 267-291. doi: 10.1016/s0001-6918(99)00008-6

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (2001). Changes of error-related ERPs with age. *Experimental Brain Research, 138*(2), 258-262. doi: 10.1007/s002210100712

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (2002). Inhibition-related ERP components: Variation with modality, age, and time-on-task. *Journal of Psychophysiology, 16*(3), 167-175. doi: 10.1027//0269-8803.16.3.167

Ferrandez, A. M., & Pouthas, V. (2001). Does cerebral activity change in middle-aged adults in a visual discrimination task? *Neurobiology of Aging, 22*(4), 645-657. doi: 10.1016/s0197-4580(00)00254-2

Fiehler, K., Ullsperger, M., & von Cramon, D. Y. (2005). Electrophysiological correlates of error correction. *Psychophysiology, 42*(1), 72-82. doi: 10.1111/j.1469-8986.2005.00265.x

Finnigan, S., O'Connell, R. G., Cummins, T. D. R., Broughton, M., & Robertson, I. H. (2011). ERP measures indicate both attention and working memory encoding decrements in aging. *Psychophysiology, 48*(5), 601-611. doi: 10.1111/j.1469-8986.2010.01128.x

Folstein, J. R., & Van Petten, C. (2008). Influence of cognitive control and mismatch on the N2 component of the ERP: A review. *Psychophysiology, 45*(1), 152-170. doi: 10.1111/j.1469-8986.2007.00602.x

Freitas, A. L., Banai, R., & Clark, S. L. (2009). When cognitive control is calibrated: Event-related potential correlates of adapting to information-processing conflict despite erroneous response preparation. *Psychophysiology, 46*(6), 1226-1233. doi: 10.1111/j.1469-8986.2009.00864.x

Gajewski, P. D., Wild-Wall, N., Schapkin, S. A., Erdmann, U., Freude, G., & Falkenstein, M. (2010). Effects of aging and job demands on cognitive flexibility assessed by task switching. *Biological Psychology, 85*(2), 187-199. doi: 10.1016/j.biopsycho.2010.06.009

Garavan, H., Ross, T. J., Kaufman, J., & Stein, E. A. (2003). A midline dissociation between error-processing and response-conflict monitoring. *Neuroimage, 20*(2), 1132-1139. doi: 10.1016/s1053-8119(03)00334-3

Garavan, H., Ross, T. J., Murphy, K., Roche, R. A. P., & Stein, E. A. (2002). Dissociable executive functions in the dynamic control of behavior: Inhibition, error detection, and correction. *Neuroimage, 17*(4), 1820-1829. doi: 10.1006/nimg.2002.1326

Gehring, W. J., & Fencsik, D. E. (2001). Functions of the medial frontal cortex in the processing of conflict and errors. *Journal of Neuroscience, 21*(23), 9430-9437. doi:

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A neural system for error-detection and compensation. *Psychological Science, 4*(6), 385-390. doi: 10.1111/j.1467-9280.1993.tb00586.x

Gehring, W. J., Gratton, G., Coles, M. G., & Donchin, E. (1992). Probability effects on stimulus evaluation and response processes. *Journal of Experimental*

Psychology: Human Perception and Performance, 18(1), 198-216. doi: 10.1037/0096-1523.18.1.198

Gehring, W. J., & Knight, R. T. (2000). Prefrontal-cingulate interactions in action monitoring. *Nature Neuroscience, 3*(5), 516-520. doi: 10.1038/74899

Glaser, M. O., & Glaser, W. R. (1982). Time course analysis of the Stroop phenomenon. *Journal of Experimental Psychology-Human Perception and Performance, 8*(6), 875-894. doi: 10.1037//0096-1523.8.6.875

Good, C. D., Johnsrude, I. S., Ashburner, J., Henson, R. N. A., Friston, K. J., & Frackowiak, R. S. J. (2001). A voxel-based morphometric study of ageing in 465 normal adult human brains. *Neuroimage, 14*(1), 21-36. doi: 10.1006/nimg.2001.0786

Grady, C. L. (1998). Brain imaging and age-related changes in cognition. *Experimental Gerontology, 33*(7-8), 661-673. doi: 10.1016/S0531-5565(98)00022-9

Gratton, G., Coles, M. G. H., & Donchin, E. (1992). Optimizing the use of information - strategic control of activation of responses. *Journal of Experimental Psychology-General, 121*(4), 480-506. doi: 10.1037/0096-3445.121.4.480

Gratton, G., Coles, M. G. H., Sirevaag, E. J., Eriksen, C. W., & Donchin, E. (1988). Prestimulus and poststimulus activation of response channels - a psychophysiological analysis. *Journal of Experimental Psychology-Human Perception and Performance, 14*(3), 331-344. doi: 10.1037/0096-1523.14.3.331

Hajcak, G., McDonald, N., & Simons, R. F. (2003). To err is autonomic: Error-related brain potentials, ANS activity, and post-error compensatory behavior. *Psychophysiology, 40*(6), 895-903. doi: 10.1111/1469-8986.00107

Hajcak, G., McDonald, N., & Simons, R. F. (2004). Error-related psychophysiology and negative affect. *Brain and Cognition, 56*(2), 189-197. doi: 10.1016/j.bandc.2003.11.001

Hajcak, G., & Simons, R. F. (2008). Oops!.. I did it again: An ERP and behavioral study of double-errors. *Brain and Cognition, 68*(1), 15-21. doi: 10.1016/j.bandc.2008.02.118

Heil, M., Osman, A., Wiegelmann, J., Rolke, B., & Hennighausen, E. (2000). N200 in the Eriksen-task: Inhibitory executive processes? *Journal of Psychophysiology, 14*(4), 218-225. doi: 10.1027//0269-8803.14.4.218

Herrmann, M. J., Römmler, J., Ehlis, A. C., Heidrich, A., & Fallgatter, A. J. (2004). Source localization (LORETA) of the error-related-negativity (ERN/Ne) and positivity (Pe). *Cognitive Brain Research, 20*(2), 294-299. doi: 10.1016/j.cogbrainres.2004.02.013

Hester, R., Barre, N., Mattingley, J. B., Foxe, J. J., & Garavan, H. (2007). Avoiding another mistake: Error and posterror neural activity associated with adaptive posterror behavior change. *Cognitive Affective & Behavioral Neuroscience, 7*(4), 317-326. doi: 10.3758/CABN.7.4.317

Hewig, J., Coles, M. G. H., Trippe, R. H., Hecht, H., & Miltner, W. H. R. (2011). Dissociation of Pe and ERN/Ne in the conscious recognition of an error. *Psychophysiology, 48*(10), 1390-1396. doi: 10.1111/j.1469-8986.2011.01209.x

Hirsh, J. B., & Inzlicht, M. (2010). Error-related negativity predicts academic performance. *Psychophysiology, 47*(1), 192-196. doi: 10.1111/j.1469-8986.2009.00877.x

Hoffmann, S., & Falkenstein, M. (2011). Aging and error processing: Age related increase in the variability of the error-negativity is not accompanied by increase in response variability. *Plos One, 6*(2). doi: 10.1371/journal.pone.0017482

Holmes, A. J., & Pizzagalli, D. A. (2008). Response conflict and frontocingulate dysfunction in unmedicated participants with major depression. *Neuropsychologia, 46*(12), 2904-2913. doi: 10.1016/j.neuropsychologia.2008.05.028

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis. of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review, 109*(4), 679-709. doi: 10.1037//0033-295x.109.4.679

Holroyd, C. B., Hajcak, G., & Larsen, J. T. (2006). The good, the bad and the neutral: Electrophysiological responses to feedback stimuli. *Brain Research, 1105*, 93-101. doi: 10.1016/j.brainres.2005.12.015

Holroyd, C. B., Nieuwenhuis, S., Yeung, N., & Cohen, J. D. (2003). Errors in reward prediction are reflected in the event-related brain potential. *Neuroreport, 14*(18), 2481-2484. doi: 10.1097/01.wnr.0000099601/41403.a5

Hommel, B. (1994). Spontaneous decay of response-code activation. *Psychological Research-Psychologische Forschung, 56*(4), 261-268. doi: 10.1007/bf00419656

Hommel, B., Proctor, R. W., & Vu, K. P. L. (2004). A feature-integration account of sequential effects in the Simon task. *Psychological Research-Psychologische Forschung, 68*(1), 1-17. doi: 10.1007/s00426-003-0132-y

Ille, N., Berg, P., & Scherg, M. (2002). Artifact correction of the ongoing EEG using spatial filters based on artifact and brain signal topographies. *Journal of Clinical Neurophysiology, 19*(2), 113-124. doi: 10.1097/00004691-200203000-00002

Jentzsch, I., & Dudschig, C. (2009). Why do we slow down after an error? Mechanisms underlying the effects of posterror slowing. *Quarterly Journal of Experimental Psychology, 62*(2), 209 - 218. doi: 10.1080/17470210802240655

Jentzsch, I., & Leuthold, H. (2006). Control over speeded actions: A common processing locus for micro- and macro-trade-offs? *Quarterly Journal of Experimental Psychology, 59*(8), 1329-1337. doi: 10.1080/17470210600674394

Jentzsch, I., Leuthold, H., & Ulrich, R. (2007). Decomposing sources of response slowing in the PRP paradigm. *Journal of Experimental Psychology-Human Perception and Performance, 33*(3), 610-626. doi: 10.1037/0096-1523.33.3.610

Johannes, S., Münte, T. F., Heinze, H. J., & Mangun, G. R. (1995). Luminance and spatial attention effects on early visual processing. *Cognitive Brain Research, 2*(3), 189-205. doi: 10.1016/0926-6410(95)90008-X

Kawai, N., Kubo-Kawai, N., Kubo, K., Terazawa, T., & Masataka, N. (2012). Distinct aging effects for two types of inhibition in older adults: a near-infrared spectroscopy study on the Simon task and the flanker task. *Neuroreport, 23*(14), 819-824. doi: 10.1097/WNR.0b013e3283578032

Kerns, J. G., Cohen, J. D., MacDonald, A. W., III, Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior Cingulate conflict monitoring and adjustments in control. *Science, 303*(5660), 1023-1026. doi: 10.1126/science.1089910

Kiehl, K. A., Liddle, P. F., & Hopfinger, J. B. (2000). Error processing and the rostral anterior cingulate: An event-related fMRI study. *Psychophysiology, 37*(2), 216-223. doi: 10.1111/1469-8986.3720216

Kok, A. (1999). Varieties of inhibition: manifestations in cognition, event-related potentials and aging. *Acta Psychologica, 101*(2-3), 129-158. doi: 10.1016/S0001-6918(99)00003-7

Kopp, B., Mattler, U., Goertz, R., & Rist, F. (1996). N2,P3 and the lateralized readiness potential in a nogo task involving selective response priming. *Electroencephalography and Clinical Neurophysiology, 99*(1), 19-27. doi: 10.1016/0921-884X(96)95617-9

Kopp, B., Rist, F., & Mattler, U. (1996). N200 in the flanker task as a neurobehavioral tool for investigating executive control. *Psychophysiology, 33*, 282-294. doi: 10.1111/j.1469-8986.1996.tb00425.x

Kornblum, S., Hasbroucq, T., & Osman, A. (1990). Dimensional overlap: cognitive basis for stimulus-response compatibility - a model and taxonomy. *Psychological Review, 97*(2), 253-270. doi: 10.1037/0033-295x.97.2.253

Kray, J., Eppinger, B., & Mecklinger, A. (2005). Age differences in attentional control: An event-related potential approach. *Psychophysiology, 42*(4), 407-416. doi: 10.1111/j.1469-8986.2005.00298.x

Kubo-Kawai, N., & Kawai, N. (2010). Elimination of the enhanced Simon effect for older adults in a three-choice situation: Ageing and the Simon effect in a go/no-go Simon task. *Quarterly Journal of Experimental Psychology, 63*(3), 452-464. doi: 10.1080/17470210902990829

LaBerge, D., & Samuels, S. J. (1974). Toward a theory of automatic information processing in reading. *Cognitive Psychology, 6*(2), 293-323. doi: 10.1016/0010-0285(74)90015-2

Ladouceur, C. D., Dahl, R. E., & Carter, C. S. (2007). Development of action monitoring through adolescence into adulthood: ERP and source localization. *Developmental Science, 10*(6), 874-891. doi: 10.1111/j.1467-7687.2007.00639.x

Laming, D. (1968). *Information theory of choice-reaction times*. London: Academic Press.

Laming, D. (1979). Choice reaction performance following an error. *Acta Psychologica, 43*(3), 199-224. doi: 10.1016/0001-6918(79)90026-X

Leuthold, H., & Sommer, W. (1999). ERP correlates of error processing in spatial S-R compatibility tasks. *Clinical Neurophysiology, 110*(2), 342-357. doi: 10.1016/S1388-2457(98)00058-3

Li, J. A., Nilsson, L. G., & Wu, Z. Y. (2004). Effects of age and anxiety on episodic memory: Selectivity and variability. *Scandinavian Journal of Psychology, 45*(2), 123-129. doi: 10.1111/j.1467-9450.2004.00387.x

Lorist, M. M., Klein, M., Nieuwenhuis, S., De Jong, R., Mulder, G., & Meijman, T. F. (2000). Mental fatigue and task control: Planning and preparation. *Psychophysiology, 37*(5), 614-625. doi: 10.1017/s004857720099005x

Luck, S. J. (2005). *An introduction to the event-related potential technique*. Cambridge, Massachusetts: The MIT Press.

Luck, S. J., Heinze, H. J., Mangun, G. R., & Hillyard, S. A. (1990). Visual event-related potentials index focused attention within bilateral stimulus arrays. 2. Functional dissociation of P1 and N1 components. *Electroencephalography and Clinical Neurophysiology, 75*(6), 528-542. doi: 10.1016/0013-4694(90)90139-B

Luu, P., Flaisch, T., & Tucker, D. M. (2000). Medial frontal cortex in action monitoring. *Journal of Neuroscience, 20*(1), 464-469.

MacDonald, A. W., III, Cohen, J. D., Stenger, V. A., & Carter, C. S. (2000). Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science, 288*(5472), 1835-1838. doi: 10.1126/science.288.5472.1835

MacLeod, C. M. (1991). Half a century of research on the Stroop effect - an integrative review. *Psychological Bulletin, 109*(2), 163-203. doi: 10.1037//0033-2909.109.2.163

Magen, H., & Cohen, A. (2002). Action-based and vision-based selection of input: Two sources of control. *Psychological Research-Psychologische Forschung, 66*(4), 247-259. doi: 10.1007/s00426-002-0099-0

Mager, R., Bullinger, A. H., Brand, S., Schmidlin, M., Schärli, H., Müller-Spahn, F., . . . Falkenstein, M. (2007). Age-related changes in cognitive conflict processing: An event-related potential study. *Neurobiology of Aging, 28*(12), 1925-1935. doi: 10.1016/j.neurobiolaging.2006.08.001

Mani, T. M., Bedwell, J. S., & Miller, L. S. (2005). Age-related decrements in performance on a brief continuous performance test. *Archives of Clinical Neuropsychology, 20*(5), 575-586. doi: 10.1016/j.acn.2004.12.008

Masaki, H., Falkenstein, M., Stürmer, B., Pinkpank, T., & Sommer, W. (2007). Does the error negativity reflect response conflict strength? Evidence from a Simon task. *Psychophysiology, 44*(4), 579-585. doi: 10.1111/j.1469-8986.2007.00522.x

Masaki, H., Murphy, T. I., Desjardins, J. A., & Segalowitz, S. J. (2012). The error-related negativity associated with different strength of stimulus-response interference. *Clinical Neurophysiology, 123*(4), 689-699. doi: 10.1016/j.clinph.2011.07.043

Mathalon, D. H., Bennett, A., Askari, N., Gray, E. M., Rosenbloom, M. J., & Ford, J. M. (2003). Response-monitoring dysfunction in aging and Alzheimer's disease: an event-related potential study. *Neurobiology of Aging, 24*(5), 675-685. doi: 10.1016/S0197-4580(02)00154-9

Mathalon, D. H., Whitfield, S. L., & Ford, J. M. (2003). Anatomy of an error: ERP and fMRI. *Biological Psychology, 64*(1-2), 119-141. doi: 10.1016/s0301-0511(03)00105-4

Mathewson, K. J., Dywan, J., & Segalowitz, S. J. (2005). Brain bases of error-related ERPs as influenced by age and task. *Biological Psychology, 70*(2), 88-104. doi: 10.1016/j.biopsycho.2004.12.005

Mattler, U. (2003). Delayed flanker effects on lateralized readiness potentials. *Experimental Brain Research, 151*(2), 272-288. doi: 10.1007/s00221-003-1486-5

May, C. P., Kane, M. J., & Hasher, L. (1995). Determinants of negative priming. *Psychological Bulletin, 118*(1), 35-54. doi: 10.1037//0033-2909.118.1.35

Mayr, U., Awh, E., & Laurey, P. (2003). Conflict adaptation effects in the absence of executive control. *Nature Neuroscience, 6*(5), 450-452. doi: 10.1038/nn1051

Milham, M. P., Erickson, K. I., Banich, M. T., Kramer, A. F., Webb, A., Wszalek, T., & Cohen, N. J. (2002). Attentional control in the aging brain: Insights from an fMRI study of the Stroop task. *Brain and Cognition, 49*(3), 277-296. doi: 10.1006/brcg.2001.1501

Miltner, W. H. R., Braun, C. H., & Coles, M. G. H. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: Evidence for a "generic" neural system for error detection. *Journal of Cognitive Neuroscience, 9*(6), 788-798. doi: 10.1162/jocn.1997.9.6.788

Monti, J. M., Weintraub, S., & Egner, T. (2010). Differential age-related decline in conflict-driven task-set shielding from emotional versus non-emotional distracters. *Neuropsychologia, 48*(6), 1697-1706. doi: 10.1016/j.neuropsychologia.2010.02.017

Nessler, D., Friedman, D., Johnson, R., & Bersick, M. (2007). ERPs suggest that age affects cognitive control but not response conflict detection. *Neurobiology of Aging, 28*(11), 1769-1782. doi: 10.1016/j.neurobiolaging.2006.07.011

Nieuwenhuis, S., Ridderinkhof, K. R., Blom, J., Band, G. P. H., & Kok, A. (2001). Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. *Psychophysiology, 38*(5), 752-760. doi: 10.1111/1469-8986.3850752

Nieuwenhuis, S., Ridderinkhof, K. R., Talsma, D., Coles, M. G. H., Holroyd, C. B., Kok, A., & van der Molen, M. W. (2002). A computational account of altered error processing in older age: Dopamine and the error-related negativity. *Cognitive Affective & Behavioral Neuroscience, 2*, 19-36. doi: 10.3758/CABN.2.1.19

Nieuwenhuis, S., Yeung, N., van den Wildenberg, W., & Ridderinkhof, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: Effects of response conflict and trial type frequency. *Cognitive Affective & Behavioral Neuroscience, 3*, 17-26. doi: 10.3758/CABN.3.1.17

Nigbur, R., Ivanova, G., & Stürmer, B. (2011). Theta power as a marker for cognitive interference. *Clinical Neurophysiology, 122*(11), 2185-2194. doi: 10.1016/j.clinph.2011.03.030

Notebaert, W., Houtman, F., Van Opstal, F., Gevers, W., Fias, W., & Verguts, T. (2009). Post-error slowing: An orienting account. *Cognition, 111*(2), 275-279. doi: 10.1016/j.cognition.2009.02.002

Nunez Castellar, E., Kuhn, S., Fias, W., & Notebaert, W. (2010). Outcome expectancy and not accuracy determines posterror slowing: ERP support. *Cognitive Affective & Behavioral Neuroscience, 10*(2), 270-278. doi: 10.3758/cabn.10.2.270

O'Connell, R. G., Dockree, P. M., Bellgrove, M. A., Kelly, S. P., Hester, R., Garavan, H., . . . Foxe, J. J. (2007). The role of cingulate cortex in the detection of errors with and without awareness: a high-density electrical mapping study. *European Journal of Neuroscience, 25*(8), 2571-2579. doi: 10.1111/j.1460-9568.2007.05477.x

Overbeek, T. J. M., Nieuwenhuis, S., & Ridderinkhof, K. R. (2005). Dissociable components of error processing - On the functional significance of the Pe Vis-a-vis the ERN/Ne. *Journal of Psychophysiology, 19*(4), 319-329. doi: 10.1027/0269-8803.19.4.319

Palmer, S. E., Brooks, J. L., & Nelson, R. (2003). When does grouping happen? *Acta Psychologica, 114*(3), 311-330. doi: 10.1016/j.actpsy.2003.06.003

Pietschmann, M., Simon, K., Endrass, T., & Kathmann, N. (2008). Changes of performance monitoring with learning in older and younger adults. *Psychophysiology, 45*(4), 559-568. doi: 10.1111/j.1469-8986.2008.00651.x

Polat, U., Schor, C., Tong, J. L., Zomet, A., Lev, M., Yehezkel, O., . . . Levi, D. M. (2012). Training the brain to overcome the effect of aging on the human eye. *Scientific Reports, 2*. doi: 27810.1038/srep00278

Purmann, S., Badde, S., Luna-Rodriguez, A., & Wendt, M. (2011). Adaptation to Frequent Conflict in the Eriksen Flanker Task An ERP Study. *Journal of Psychophysiology, 25*(2), 50-59. doi: 10.1027/0269-8803/a000041

Rabbitt, P. M. A. (1966). Errors and error correction in choice-response tasks. *Journal of Experimental Psychology, 71*(2), 264-272. doi: 10.1037/h0022853

Rabbitt, P. M. A., & Rodgers, B. (1977). What does a man do after he makes an error - analysis of response programming. *Quarterly Journal of Experimental Psychology, 29*(NOV), 727-743. doi: 10.1080/14640747708400645

Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S. (2004). The role of the medial frontal cortex in cognitive control. *Science, 306*(5695), 443-447. doi: 10.1126/science.1100301

Rosano, C., Aizenstein, H., Cochran, J., Saxton, J., De Kosky, S., Newman, A. B., . . . Carter, C. S. (2005). Functional neuroimaging indicators of successful executive control in the oldest old. *Neuroimage, 28*(4), 881-889. doi: 10.1016/j.neuroimage.2005.05.059

Rugg, M. D., & Coles, M. G. H. (Eds.). (1995). *Electrophysiology of mind: Event-related brain potentials and cognition*. New York: Oxford University Press.

Saunders, B., & Jentzsch, I. (in press). False external feedback modulates posterror slowing and the f-P300: Implications for theories of posterror adjustment. *Psychonomic Bulletin & Review*.

Scheffers, M. K., & Coles, M. G. H. (2000). Performance monitoring in a confusing world: Error-related brain activity, judgments of response accuracy, and types of errors. *Journal of Experimental Psychology-Human Perception and Performance, 26*(1), 141-151. doi: 10.1037/0096-1523.26.1.141

Seifert, J. (2005). *Ereigniskorrelierte EEG-Aktivität*. Langerich: Pabst Science Publishers.

Sharp, D. J., Scott, S. K., Mehta, M. A., & Wise, R. J. S. (2006). The neural correlates of declining performance with age: Evidence for age-related changes in cognitive control. *Cerebral Cortex, 16*(12), 1739-1749. doi: 10.1093/cercor/bhj109

Simon, J. R. (1990). The effects of an irrelevant directional cue on human information processing. In R. Proctor, W. & T. G. Reeve (Eds.), *Stimulus-response compatibility: An integrated perspective* (pp. 31-86). Amsterdam: Elsevier.

Stadler, M. A., & Hogan, M. E. (1996). Varieties of positive and negative priming. *Psychonomic Bulletin & Review, 3*(1), 87-90. doi: 10.3758/bf03210745

Stahl, J. (2010). Error detection and the use of internal and external error indicators: An investigation of the first-indicator hypothesis. *International Journal of Psychophysiology, 77*(1), 43-52. doi: 10.1016/j.ijpsycho.2010.04.005

Stemmer, B., Segalowitz, S. J., Witzke, W., & Schönle, P. W. (2003). Error detection in patients with lesions to the medial prefrontal cortex: an ERP study. *Neuropsychologia, 42*(1), 118-130. doi: 10.1016/s0028-3932(03)00121-0

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*(6), 643-662. doi: 10.1037/h0054651

Strozyk, J. V., & Jentzsch, I. (2012). Weaker error signals do not reduce the effectiveness of post-error adjustments: Comparing error processing in young and middle-aged adults. *Brain Research, 1460*, 41-49. doi: 10.1016/j.brainres.2012.04.028

Stuart-Hamilton, I. (1999). Intellectual changes in late life. In R. T. Woods (Ed.), *Psychological problems of ageing - assessment, treatment and care* (pp. 27-47). Chichester: Wiley.

Stürmer, B., & Leuthold, H. (2003). Control over response priming in visuomotor processing: a lateralized event-related potential study. *Experimental Brain Research, 153*(1), 35-44. doi: 10.1007/s00221-003-1579-1

Stürmer, B., Leuthold, H., Soetens, E., Schroter, H., & Sommer, W. (2002). Control over location-based response activation in the Simon task: Behavioral and electrophysiological evidence. *Journal of Experimental Psychology-Human Perception and Performance, 28*(6), 1345-1363. doi: 10.1037//0096-1523.28.6.1345

Sullivan, M. P. (1999). The functional interaction of visual-perceptual and response mechanisms during selective attention in young adults, young-old adults, and old-old adults. *Perception & Psychophysics, 61*(5), 810-825. doi: 10.3758/BF03206899

Themanson, J. R., Hillman, C. H., & Curtin, J. J. (2006). Age and physical activity influences on action monitoring during task switching. *Neurobiology of Aging, 27*(9), 1335-1345. doi: 10.1016/j.neurbiolaging.2005.07.002

Themanson, J. R., Hillman, C. H., McAuley, E., Buck, S. M., Doerksen, S. E., Morris, K. S., & Pontifex, M. B. (2008). Self-efficacy effects on neuroelectric and behavioral indices of action monitoring in older adults. *Neurobiology of Aging, 29*(7), 1111-1122. doi: 10.1016/j.neurobiolaging.2007.01.004

Themanson, J. R., Pontifex, M. B., & Hillman, C. H. (2008). Fitness and action monitoring: evidence for improved cognitive flexibility in young adults. *Neuroscience, 157*(2), 319-328. doi: 10.1016/j.neuroscience.2008.09.014

Tillman, C. M., & Wiens, S. (2011). Behavioral and ERP indices of response conflict in Stroop and flanker tasks. *Psychophysiology*, no-no. doi: 10.1111/j.1469-8986.2011.01203.x

Tolhurst, D. J., Movshon, J. A., & Thompson, I. D. (1981). The dependence of response amplitude and variance of cat visual cortical neurones on stimulus contrast. *Experimental Brain Research, 41*(3-4), 414-419. doi:

Ullsperger, M., Bylsma, L. M., & Botvinick, M. M. (2005). The conflict adaptation effect: It's not just priming. *Cognitive Affective & Behavioral Neuroscience, 5*(4), 467-472. doi: 10.3758/CABN.5.4.467

Ullsperger, M., & von Cramon, D. Y. (2001). Subprocesses of performance monitoring: A dissociation of error processing and response competition revealed by event-related fMRI and ERPs. *Neuroimage, 14*(6), 1387-1401. doi: 10.1006/nimg.2001.0935

Ullsperger, M., & von Cramon, D. Y. (2006). How does error correction differ from error signaling? An event-related potential study. *Brain Research, 1105*, 102-109. doi: 10.1016/j.brainres.2006.01.007

Usher, M., & McClelland, J. L. (1995). *On the time course of perceptual choice: A model based on principles of neural computation* (Tech. Rep. PDP.CNS.95.5). Carnegie Mellon University, Department of Psychology.

Van't Ent, D. (2002). Perceptual and motor contributions to performance and ERP components after incorrect motor activation in a flanker reaction task. *Clinical Neurophysiology, 113*(2), 270-283. doi: 10.1016/S1388-2457(01)00730-1

Van der Lubbe, R. H. J., & Verleger, R. (2002). Aging and the Simon task. *Psychophysiology, 39*(1), 100-110. doi: 10.1017/s0048577201020042

van Veen, V., & Carter, C. S. (2002). The timing of action-monitoring processes in the anterior cingulate cortex. *Journal of Cognitive Neuroscience, 14*(4), 593-602. doi: 10.1162/08989290260045837

van Veen, V., Cohen, J. D., Botvinick, M. M., Stenger, V. A., & Carter, C. S. (2001). Anterior cingulate cortex, conflict monitoring, and levels of processing. *Neuroimage, 14*(6), 1302-1308. doi: 10.1006/nimg.2001.0923

Verhaeghen, P., & De Meersman, L. (1998). Aging and the stroop effect: A meta-analysis. *Psychology and Aging, 13*(1), 120-126. doi: 10.1037/0882-7974.13.1.120

Vidal, F., Hasbroucq, T., Grapperon, J., & Bonnet, M. (2000). Is the 'error negativity' specific to errors? *Biological Psychology, 51*(2-3), 109-128. doi: 10.1016/S0301-0511(99)00032-0

Vocat, R., Pourtois, G., & Vuilleumier, P. (2008). Unavoidable errors: A spatio-temporal analysis of time-course and neural sources of evoked potentials associated with error processing in a speeded task. *Neuropsychologia, 46*(10), 2545-2555. doi: 10.1016/j.neuropsychologia.2008.04.006

Walter, W. G., Cooper, R., Aldridge, V. J., McCallum, W. C., & Winter, A. L. (1964). Contingent negative variation : An electric sign of sensori-motor association and expectancy in the human brain. *Nature, 203*(4943), 380-384. doi: 10.1038/203380a0

Wascher, E., Reinhard, M., Wauschkuhn, B., & Verleger, R. (1999). Spatial S-R compatibility with centrally presented stimuli: An event-related asymmetry study on dimensional overlap. *Journal of Cognitive Neuroscience, 11*(2), 214-229. doi: 10.1162/089892999563346

Wendt, M., Heldmann, M., Münte, T. F., & Kluwe, R. H. (2007). Disentangling sequential effects of stimulus- and response-related conflict and stimulus-response repetition using brain potentials. *Journal of Cognitive Neuroscience, 19*(7), 1104-1112. doi: 10.1162/jocn.2007.19.7.1104

West, R. (1996). An application of prefrontal cortex function theory to cognitive aging. *Psychological Bulletin, 120*(2), 272-292. doi: 10.1037/0033-2909.120.2.272

West, R. (2003). Neural correlates of cognitive control and conflict detection in the Stroop and digit-location tasks. *Neuropsychologia, 41*(8), 1122-1135. doi: 10.1016/S0028-3932(02)00297-X

West, R. (2004). The effects of aging on controlled attention and conflict processing in the Stroop task. *Journal of Cognitive Neuroscience, 16*(1), 103-113. doi: 10.1162/089892904322755593

West, R., & Alain, C. (2000). Effects of task context and fluctuations of attention on neural activity supporting performance of the Stroop task. *Brain Research, 873*(1), 102-111. doi: 10.1016/S0006-8993(00)02530-0

West, R., & Moore, K. (2005). Adjustments of cognitive control in younger and older adults. *Cortex, 41*(4), 570-581. doi: 10.1016/S0010-9452(08)70197-7

West, R., & Travers, S. (2008). Tracking the temporal dynamics of updating cognitive control: An examination of error processing. *Cerebral Cortex, 18*(5), 1112-1124. doi: 10.1093/cercor/bhm142

Wiener, M. C., Oram, M. W., Liu, Z., & Richmond, B. J. (2001). Consistency of encoding in monkey visual cortex. *Journal of Neuroscience, 21*(20), 8210-8221.

Wijers, A. A., Lange, J. J., Mulder, G., & Mulder, L. J. M. (1997). An ERP study of visual spatial attention and letter target detection for isoluminant and nonisoluminant stimuli. *Psychophysiology, 34*(5), 553-565. doi: 10.1111/j.1469-8986.1997.tb01742.x

Wild-Wall, N., Falkenstein, M., & Hohnsbein, J. (2008). Flanker interference in young and older participants as reflected in event-related potentials. *Brain Research, 1211*, 72-84. doi: 10.1016/j.brainres.2008.03.025

Wild-Wall, N., Hohnsbein, J., & Falkenstein, M. (2007). Effects of ageing on cognitive task preparation as reflected by event-related potentials. *Clinical Neurophysiology, 118*(3), 558-569. doi: 10.1016/j.clinph.2006.09.005

Willemssen, R., Hoormann, J., Hohnsbein, J., & Falkenstein, M. (2004). Central and parietal event-related lateralizations in a flanker task. *Psychophysiology, 41*(5), 762-771. doi: 10.1111/j.1469-8986.2004.00201.x

Witthöft, M., Sander, N., Süβ, H. M., & Wittmann, W. W. (2009). Adult Age Differences in Inhibitory Processes and their Predictive Validity for Fluid Intelligence. *Aging Neuropsychology and Cognition, 16*(2), 133-163. doi: 10.1080/13825580802348554

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review, 111*(4), 931-959. doi: 10.1037/0033-295x.111.4.931

Zeef, E. J., & Kok, A. (1993). Age-related differences in the timing of stimulus and response processes during visual selective attention - performance and psychophysiological analyses. *Psychophysiology, 30*(2), 138-151. doi: 10.1111/j.1469-8986.1993.tb01727.x

Zeef, E. J., Sonke, C. J., Kok, A., Buiten, M. M., & Kenemans, J. L. (1996). Perceptual factors affecting age-related differences in focused attention: Performance and psychophysiological analyses. *Psychophysiology, 33*(5), 555-565. doi: 10.1111/j.1469-8986.1996.tb02432.x

Appendices

Appendix I:

Ethical approval forms

Appendix II:

Copy of Strozyk, J. V. & Jentzsch, I. (2012). Weaker error signals do not reduce the effectiveness of post-error adjustments: Comparing error processing in young and middle-aged adults. *Brain Research, 1460*, 41-49.

24 November 2008

Ethics Reference No: *Please quote this ref on all correspondence*	**PS5099**
Project Title:	**Conflict processing in different age groups**
Researchers Name(s):	Jessica Strozyk
Supervisor(s):	Dr I Jentzsch

Thank you for submitting your application which was considered at the Psychology School Ethics Committee meeting on the 21 November 2008. The following documents were reviewed:

1. Ethical Application Form 19 November 2008
2. Participant Information Sheet 19 November 2008
3. Consent Form 19 November 2008
4. Debriefing Form 20 November 2008
5. Questionnaires 19 November 2008
6. Advertisement 20 November 2008

The University Teaching and Research Ethics Committee (UTREC) approves this study from an ethical point of view. Please note that where approval is given by a School Ethics Committee that committee is part of UTREC and is delegated to act for UTREC.

Approval is given for completion within the stated time period. Projects, which have not commenced within the time given must be re-submitted to your School Ethics Committee.

You must inform your School Ethics Committee when the research has been completed. If you are unable to complete your research within the validation period, you will be required to write to your School Ethics Committee and to UTREC (where approval was given by UTREC) to request an extension or you will need to re-apply.

Any serious adverse events or significant change which occurs in connection with this study and/or which may alter its ethical consideration, must be reported immediately to the School Ethics Committee, and an Ethical Amendment Form submitted where appropriate.

Approval is given on the understanding that the 'Guidelines for Ethical Research Practice' (http://www.st-andrews.ac.uk/media/UTRECguidelines%20Feb%2008.pdf) are adhered to.

Yours sincerely

On behalf of the Convenor of the School Ethics Committee OR Convener of UTREC

University of St Andrews

University Teaching and Research Ethics Committee
School of Psychology

25 August 2009

Ethics Reference No: *Please quote this ref on all correspondence*	**PS5099**
Project Title:	**Conflict processing in different age groups**
Researchers Name(s):	**Jessica Strozyk**
Supervisor(s):	**Dr Ines Jentzsch**

Thank you for submitting your application which was considered by the School Ethics Committee. The following documents were reviewed:

1. Ethical Amendment Form 25 August 2009
2. Participant Advertisement 25 August 2009
3. Debriefing Form 25 August 2009
4. Questionnaires 25 August 2009

The University Teaching and Research Ethics Committee (UTREC) approves this study from an ethical point of view. Please note that where approval is given by a School Ethics Committee that committee is part of UTREC and is delegated to act for UTREC.

Approval is given for three years. Projects, which have not commenced within two years of original approval, must be re-submitted to your School Ethics Committee.

You must inform your School Ethics Committee when the research has been completed. If you are unable to complete your research within the 3 three year validation period, you will be required to write to your School Ethics Committee and to UTREC (where approval was given by UTREC) to request an extension or you will need to re-apply.

Any serious adverse events or significant change which occurs in connection with this study and/or which may alter its ethical consideration, must be reported immediately to the School Ethics Committee, and an Ethical Amendment Form submitted where appropriate.

Approval is given on the understanding that the 'Guidelines for Ethical Research Practice' (http://www.st-andrews.ac.uk/media/UTRECguidelines%20Feb%2008.pdf) are adhered to.

Yours sincerely

Convenor of the School Ethics Committee OR Convener of UTREC

Ccs Dr Ines Jentzsch

Supervisor
School Ethics Committee

University of St Andrews

University Teaching and Research Ethics Committee
School of Psychology

02 December 2009

Ethics Reference No: *Please quote this ref on all correspondence*	PS5099
Project Title:	Conflict processing in different age groups
Researchers Name(s):	Jessica Strozyk
Supervisor(s):	Dr Ines Jentzsch

Thank you for submitting your application which was considered at the School Ethics Committee meeting on the 2nd December 2009. The following documents were reviewed:

1. Ethical Amendment Form 02/12/2009
2. Debriefing Forms 02/12/2009

The University Teaching and Research Ethics Committee (UTREC) approves this study from an ethical point of view. Please note that where approval is given by a School Ethics Committee that committee is part of UTREC and is delegated to act for UTREC.

Approval is given for three years. Projects, which have not commenced within two years of original approval, must be re-submitted to your School Ethics Committee.

You must inform your School Ethics Committee when the research has been completed. If you are unable to complete your research within the 3 three year validation period, you will be required to write to your School Ethics Committee and to UTREC (where approval was given by UTREC) to request an extension or you will need to re-apply.

Any serious adverse events or significant change which occurs in connection with this study and/or which may alter its ethical consideration, must be reported immediately to the School Ethics Committee, and an Ethical Amendment Form submitted where appropriate.

Approval is given on the understanding that the 'Guidelines for Ethical Research Practice' (http://www.st-andrews.ac.uk/media/UTRECguidelines%20Feb%2008.pdf) are adhered to.

Yours sincerely

Convenor of the School Ethics Committee OR Convener of UTREC

Ccs Dr Ines Jentzsch (Supervisor)
School Ethics Committee

University of St Andrews

University Teaching and Research Ethics Committee
School of Psychology

7 April 2010

Ethics Reference No: *Please quote this ref on all correspondence*	PS5099
Project Title:	Conflict processing in different age groups
Researchers Name(s):	Jessica Strozyk
Supervisor(s):	Dr Ines Jentzsch

Thank you for submitting your application which was considered at the Psychology School Ethics Committee meeting on the 7th April 2010. The following documents were reviewed:

1. Ethical Amendment Form 07/04/2010
2. Debriefing Form 07/04/2010
3. Questionnaire 07/04/2010

The University Teaching and Research Ethics Committee (UTREC) approves this study from an ethical point of view. Please note that where approval is given by a School Ethics Committee that committee is part of UTREC and is delegated to act for UTREC.

Approval is given for three years. Projects, which have not commenced within two years of original approval, must be re-submitted to your School Ethics Committee.

You must inform your School Ethics Committee when the research has been completed. If you are unable to complete your research within the 3 three year validation period, you will be required to write to your School Ethics Committee and to UTREC (where approval was given by UTREC) to request an extension or you will need to re-apply.

Any serious adverse events or significant change which occurs in connection with this study and/or which may alter its ethical consideration, must be reported immediately to the School Ethics Committee, and an Ethical Amendment Form submitted where appropriate.

Approval is given on the understanding that the 'Guidelines for Ethical Research Practice' (http://www.st-andrews.ac.uk/media/UTRECguidelines%20Feb%2008.pdf) are adhered to.

Yours sincerely

Convenor of the School Ethics Committee OR Convener of UTREC

Ccs Dr Ines Jentzsch (Supervisor)
School Ethics Committee

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