Unraveling Early Error Processing

The Relationship Between the Ne/ERN, Conflict Monitoring, Error Awareness, and Autonomic Responses

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Julia Dumsky

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Name des Erstgutachters:	Prof. Dr. Marco Steinhauser
Name der Zweitgutachterin:	Jun.-Prof. Dr. Christina Pfeuffer

Zusammenfassung

Die Fehlerüberwachung ermöglicht es Menschen, Fehler in ihrer Leistung zu erkennen und ihr Verhalten entsprechend anzupassen. Ein wichtiger neuronaler Marker für diesen Prozess ist die Error negativity (Ne/ERN), eine negative Auslenkung an frontozentralen Elektroden kurz nach einem Fehler, die eine schnelle und automatische Fehlerverarbeitung widerspiegelt. Theoretische Erklärungen führen die Ne/ERN auf Konfliktüberwachung und Vorhersagefehler zurück, aber der genaue Mechanismus bleibt unklar. In drei Studien habe ich die Rolle der Ne/ERN bei der Fehlerüberwachung und ihre Beziehung zu anderen fehlerbezogenen Phänomenen untersucht. Studie 1 ergab, dass die Ne/ERN mit dem Grad des Post-Response-Konflikts variiert. Studie 2 zeigte, dass die bewusste Fehlererkennung auch ohne Ne/ERN stattfinden kann, was auf separate neuronale Grundlagen für die frühe Fehlerverarbeitung und das Fehlerbewusstsein hindeutet. Studie 3 untersuchte Zusammenhänge zwischen der Ne/ERN und autonomen Signalen und zeigte, dass die fehlerbedingte Pupillenweitung mit der Ne/ERN assoziiert ist, die Herzratenverlangsamung jedoch nicht. Diese Ergebnisse verdeutlichen die funktionelle Rolle der frühen Fehlerprozesse, wie sie sich in der Ne/ERN widerspiegeln, und ihre Beziehung zu anderen Aspekten der Fehlerverarbeitung.

Abstract

Error monitoring allows humans to detect errors in their performance and adapt behavior accordingly. A key neural marker of this process is the error negativity (Ne/ERN), a negative deflection at frontocentral electrodes shortly after an error, which reflects fast and automatic error processing. Theoretical accounts attributed the Ne/ERN to conflict monitoring and prediction error, but the exact mechanism remains unclear. In three studies, I investigated the Ne/ERN's role in error monitoring and its relationship with other error-related phenomena. *Study 1* found that the Ne/ERN varies with the level of post-response conflict. *Study 2* showed that conscious error detection can occur without the Ne/ERN, suggesting separate neural bases for early error processing and error awareness. *Study 3* explored links between the Ne/ERN and autonomic signals, showing that error-related pupil dilation is associated with the Ne/ERN while heart rate deceleration is not. These findings clarify the functional role of early error processes as reflected by the Ne/ERN and their relationship with other aspects of error processing.

Contents

Introduction

Our everyday lives are full of mistakes, big and small. For example, who has not written a critical email and used the wrong address form? However, when you realize you have made a mistake, you have already hit send. In the future, you might double-check the message before sending it, i.e., you optimize your behavior. This, or something similar, could be an everyday situation in which the brain's error-monitoring system is active. The system continuously monitors behavior and is linked to the metacognitive ability to detect and signal performance errors (Yeung & Summerfield, 2012). This monitoring process uses information from the external environment, such as performance outcomes, and internal states, such as conflicts or uncertainty, to adapt behavior to changing needs in the short and long term (Yeung & Summerfield, 2012). In this sense, this particular form of metacognition can be defined as the neural process that takes place after a decision has been made (Yeung & Summerfield, 2012). It includes early and automatic processes without any awareness of errors and late processes associated with explicit error awareness (Steinhauser & Yeung, 2010; Ullsperger, Fischer et al., 2014; reviewed by van der Veen & Carter, 2002b). In particular, the present thesis focuses on the interaction between early error processes and other error-related phenomena.

The error-monitoring system is a neural network located in the medial prefrontal cortex (MPFC). This system mainly consists of the pre-supplementary as well as supplementary motor area (pre-SMA and SMA) and the anterior cingulate cortex (ACC; Carter et al., 1998; Pezzetta et al., 2022; Ullsperger, Fischer et al., 2014; Wang et al., 2005; Magno et al., 2006; Ullsperger & von Cramon, 2004). It monitors for errors and conflicts during goal-directed behavior and adjusts behavior as needed (Ullsperger, 2017; Ullsperger, Danielmeier et al., 2014) by signalling the information to other brain areas such as prefrontal areas, sensory and motor areas, the anterior insular cortex (AIC) and the autonomic nervous system (ANS; Danielmeier et al.,

2011; King et al., 2010; Cavanagh et al., 2009; Kerns et al., 2004; Rothe et al., 2011; Ullsperger et al., 2010; Wessel et al., 2011).

Two key hubs often activated in parallel during error processing (Klein et al., 2007; Ridderinkhof, Ullsperger et al., 2004) are the ACC and AIC, which are associated with different functions during error monitoring. The AIC is thought to contribute to sensory, visceral, socio-emotional, interoceptive, and cognitive processing, such as the processing of salience by integrating various internal and external information (Craig, 2009, 2011; Harsay et al., 2012; Uddin et al., 2017). As the AIC is one of the most essential areas for interoceptive projections (e.g., Craig, 2002; Critchley & Harrison, 2013; Uddin, 2014), it also processes ANS signals such as pupil and heart rate changes. These parameters are associated with performance errors and are considered as additional evidence that an error has been made (Ullsperger et al., 2010). Moreover, it is generally associated with emotional awareness and awareness of the states of the body (Barrett & Simmons, 2015; Critchley et al., 2004; Gu et al., 2013; Klein et al., 2013; Medford & Critchley, 2010; Ullsperger et al., 2010). Therefore, the relation between the AIC activity and error awareness (Hester et al., 2009; Klein et al., 2007) is not surprising. The ACC is also associated with several functions. In addition to the perception of pain (Tracey & Mantyh, 2007) and the regulation of emotion (Etkin et al., 2011), the ACC is also assumed to be related to the prediction of error likelihood (Brown & Braver, 2005), response selection (Turken & Swick, 1999) and the regulation of arousal (Critchley et al., 2001; Paus, 2001). Additionally, it is activated during error detection (Coles et al., 2001; Scheffers et al., 1996) and conflict monitoring (Botvinick et al., 2001; Yeung et al., 2004; Senderecka, 2018). The activity of the ACC is generally related to the need for adapting behavior (Ullsperger, Fischer et al., 2014).

Notably, there is a substantial overlap between areas that are active during error processing and processing of salient events by the salience network, particularly with regard to the ACC and the AIC. The salience network is responsible for registering salient events important for various

functions, including cognitive control, even without goal-directed behavior (Menon & Uddin, 2010; Seeley et al., 2007). It is typically activated in contexts needing behavioral adaptations (Dosenbach et al., 2007). This is achieved by orienting response to internal or external salient stimuli and facilitating information processing (Harsay et al., 2012). The overlap mentioned above between the salience network and the error-monitoring system is unsurprising, given that errors can be interpreted as salient events (Notebaert et al., 2009; Ullsperger et al., 2010).

An often-used methodological way to study error processing is to use simple perceptual decision tasks. Classic paradigms are the Stroop task (Stroop, 1935), the Simon task (Simon & Small, 1969), and the Eriksen flanker task (Eriksen & Eriksen, 1974), which are all interference, i.e., conflict-inducing tasks. The interference is realized by task-irrelevant stimuli or stimuli features, which automatically trigger competing responses (Hübner & Töbel, 2019; Kunde et al., 2012). The Eriksen flanker task, e.g., consists of horizontally presented letters or symbols (e.g., H, S, or <,>). We differentiate the case of targets being surrounded by congruent flankers, i.e., the central target is the same as the flanker (e.g., HHHHH or <<<<<). In contrast, in the case of incongruent flankers, the central target varies from the flanker (e.g., HHSHH or <<><<). Participants have to respond to the central target. At the same time, the task-irrelevant flankers automatically activate either the same response (in the case of congruent stimuli) or a different response (in the case of incongruent stimuli), which then competes with the correct response (Eriksen & Eriksen, 1974; Ridderinkhof et al., 2021). There are typically the following response categories that can be distinguished. First, the correct response if the central target letter was responded to. Second, flanker errors, if a response was made to the flanker of an incongruent stimuli. Third, nonflanker errors, when there are more than two possible response options and neither the correct nor the flanker was responded to. While tasks like the Eriksen flanker task are relatively simple, they are usually performed under considerable time pressure, leading to frequent errors. However, participants are usually aware of these errors (error awareness), i.e.,

the errors are consciously detected and can be indicated, i.e., signaled, reliably (Yeung & Summerfield, 2012).

On the behavioral level, there are classic findings regarding error processing. To respond slower after error trials is referred to as post-error slowing (PES; Laming, 1968; Rabbitt, 1966) and is a robust finding in interference tasks (Cavanagh et al., 2009). Another typical finding is the congruency effect, which describes performance differences between congruent and incongruent trials (e.g., Dignath et al., 2022; Hübner & Töbel, 2019). These performance differences can refer to response times (RTs) and error rates, i.e., RTs and error rates are typically increased for incongruent trials (e.g., Dignath et al., 2022; Hübner & Töbel, 2019). In addition to these behavioral findings, there are also typical error-related patterns in peripheral physiology. For example, errors usually cause phasic pupil dilation (e.g., Braem et al., 2015; Murphy et al., 2016; Rondeel et al., 2015; Wessel et al., 2011), an increase in skin conductance (Crone et al., 2004; Hajcak et al., 2003) and a deceleration of the heart rate (e.g., Fiehler et al., 2004; Hajcak et al., 2003; Somsen et al., 2004; van der Veen et al., 2000; Wessel et al., 2011). Another way to study error processing is to investigate electrophysiological components of the electroencephalogram (EEG), i.e., the brain's electrical activity measured at the scalp and time-locked to events. However, before some components of this activity are explained in detail, the following section briefly introduces the history of the EEG and the method of event-related potentials (ERP).

In 1929, the neurologist Hans Berger published a set of studies introducing electroencephalography (EEG; Berger, 1929). With this technique, electrodes on the scalp can record human electrical brain activity (Berger, 1929). Since then, the usage of the EEG expanded and has become an established tool. However, in order to draw conclusions on how certain instances, such as responses to or processing of stimuli, are reflected by the electrical brain activity, the continuous brain waves must first be preprocessed into ERPs, i.e., electrical potentials, which can be related to internal or external events (Luck, 2012). Therefore, many

trials are averaged together so that nonspecific noise, which is not caused by the event of interest and randomly varies from trial to trial, is cancelled out while the specific activity remains (Luck, 2012). The result is an ERP component temporally linked to the event of interest, used to investigate, e.g., aspects of cognitive or affective processes (Luck, 2012). ERPs are often characterized descriptively as a deflection with a negative or positive polarity, maximally at a specific scalp position with a particular latency, and elicited under certain experimental conditions (e.g., Vidal et al., 2022). Biologically, they usually indicate postsynaptic potentials evoked by a large and equally aligned group of neurons simultaneously (Luck, 2012). The first ERP was probably detected in the 1930s (Luck, 2012) and over time, many ERP components were discovered and further differentiated. Some ERPs are modality-specific, e.g., elicited only after auditory stimuli; others are related to different functions such as language- or error- and conflict-processing.

The present thesis holds the potential to significantly advance our understanding of the metacognitive ability of error monitoring using the ERP method. By clarifying whether and how early error monitoring neural processes relate to typical phenomena associated with error processing, such as conflict, error awareness, pupil dilation, and heart rate deceleration, this research could pave the way for new insights and applications in neuroscience and psychology.

Neural signatures of error and conflict processing

ERP components, referring to error- and conflict-processing, have been widely studied over the last decades. In the present thesis, three of them are considered. First, the error negativity (Ne; Falkenstein et al., 1990) or error-related negativity (ERN; Gehring et al., 1993). The Ne/ERN is typically time-locked to the response (response-locked). It was discovered in the 1990s and occurs after incorrect responses when compared with correct ones (e.g., Falkenstein et al., 1991; Valt & Stürmer, 2021; Vidal et al., 2022). The Ne/ERN is an early negative deflection, which is elicited within 100 ms after errors with a peak around 50 ms (Gehring et al., 1993) and occurs

at frontocentral electrodes (Carter et al., 1998; Dehaene et al., 1994; Ullsperger & von Cramon, 2001). Depending on the corresponding account, it is, e.g., functionally associated with response conflict (Yeung et al., 2004) or reinforcement learning (Holroyd & Coles, 2002) and can also occur without or with little awareness (Endrass et al., 2007). In addition, the Ne/ERN is typically increased in some psychiatric disorders such as anxiety disorders and obsessive-compulsive disorders (Gehring et al., 2000; Hajcak, 2012; Hanna et al., 2016; Luu et al., 2000; Weinberg & Hajcak, 2010) and is also generally associated with emotional processing by some researchers (e.g., Aarts et al., 2013; Hajcak & Foti, 2008). The Ne/ERN is also modality independent, meaning it occurs after performance errors, regardless of whether the stimuli are visual, auditory, or tactile (Falkenstein et al., 2000; Gruendler et al., 2011). Recently, the Ne/ERN was associated with the frontocentral theta power, a special power band in oscillatory brain activity, which seems to be characterized by similar aspects as the Ne/ERN (e.g., Steinhauser et al., 2024; Murphy et al., 2015).

Vidal et al. (2000) differentiated between correct, completely incorrect, and partially incorrect responses by recording the motor activity of the participants' fingers using electromyography. In this sense, partially incorrect responses were characterized by the fact that a threshold in the motor activity of the wrong response finger was initially exceeded, but then the correct response finger was activated and thus, the correct response was given. They found that the Ne/ERN was also present for partially incorrect responses but smaller than for entirely incorrect ones where no correction was made. They also registered an even smaller negative deflection for correct trials, the so-called correct-related negativity (CRN, Ford, 1999; Vidal et al., 2000; Vidal et al., 2003; Nc, Yordanova et al., 2004), an Ne/ERN-like component on correct trials, but once again smaller than for partial errors. According to source localization and neuroimaging studies (e.g., Gruendler et al., 2011; Debener et al., 2005; Nieuwenhuis et al., 2003; van der Veen & Carter, 2002b) most researchers link the generator of the Ne/ERN to the posterior medial frontal cortex (pMFC), which consists of the presupplementary motor area (pre-SMA), the dorsomedial

prefrontal cortex (dmPFC) and especially the ACC. A few other researchers posit that the Ne/ERN is generated in the posterior cingulate cortex (PCC) and then conveyed to the ACC (e.g., Agam et al., 2011; Buzzell et al., 2017). However, in both cases, more than one brain region seems to generate the Ne/ERN.

Another ERP of interest is the error positivity (Pe; Falkenstein et al., 1991; Overbeek et al., 2005), a positive deflection that typically follows the Ne/ERN. It occurs at posterior electrodes between 200 ms and 400 ms after the error response (Falkenstein et al., 1990, 1991; Overbeek et al., 2005). The Pe is more distributed over the scalp and its neural generator is less well-known in contrast to the Ne/ERN (Wessel, 2012). However, the Pe and the ERN have in common that they are not modality-specific (Falkenstein et al., 1991; Falkenstein et al., 1995; Falkenstein et al., 2000). Functionally, the Pe is considered an indicator of error awareness or the accumulated evidence that an error has been made, which then may lead to error awareness (Desender et al., 2021; Steinhauser & Yeung, 2010; Ullsperger, Danielmeier et al., 2014).

The final ERP of interest is the N2. The N2 is a negative deflection, such as the Ne/ERN, and is likely generated in the same brain area (e.g., Debener et al., 2005; Nieuwenhuis et al., 2003; van der Veen & Carter, 2002b). It is also elicited at frontocentral electrodes but occurs in a time interval between 200 and 300 ms after stimulus onset and before an overt response (Jost et al., 2022; Yeung et al., 2004; Yeung & Cohen, 2006; Heil et al., 2000; Kopp et al., 1996; Liotti et al., 2000). Therefore, it is temporally linked to the stimulus (stimulus-locked). The N2 is also associated with different functions within specific frameworks, such as response inhibition and response conflict (Yeung et al., 2004; Falkenstein et al., 1999; Gajewski & Falkenstein, 2013; Jodo & Kayama, 1992; Kopp et al., 1996; Vuillier et al., 2016).

I focus on early error processes that occur in the first 100 ms after an erroneous response and, therefore, on the Ne/ERN. Nevertheless, the Pe and N2 are also considered, because these ERPs provide information on conflict processing and error awareness as well. The following section gives an overview of different theories on the functional role of all three ERPs. Regarding the

models of the Ne/ERN, all approaches have in common that error monitoring is based on processes that proceed after the decision (post-decision processing; Rabbitt, 1966; Rabbitt & Vyas, 1981).

Theories of the Ne/ERN and N2

Mismatch theory of the Ne/ERN. Initially, the Ne/ERN was assumed to be a signal that indicates error detection by registering a mismatch between the motor output, i.e., the given erroneous response and the representation of the correct response at the time point when the Ne/ERN occurred (Falkenstein et al., 1991; Gehring et al., 1993). This error-detection hypothesis is the basis of the mismatch model (Coles et al., 2001). Typical paradigms to investigate error monitoring used simple but speeded tasks so that most errors are caused by premature decisions, i.e., the stimulus processing was not fully completed (Kappenman et al., 2012; Scheffers & Coles, 2000; Vidal et al., 2022). This is why the stimulus processing proceeds after response onset. The correct and the actual response representations are then compared and whenever they differ, a comparative mechanism detects a mismatch or error signal. In more challenging conditions, such as the incongruent condition in the Eriksen flanker paradigm, the representation of the correct response is impaired and thus, the Ne/ERN is reduced (Pailing & Segalowitz, 2004b; Scheffers & Coles, 2000). There are slightly different perspectives, on which subprocesses of this error-detection system are represented in the Ne/ERN, i.e., the Ne/ERN could indicate the output of this comparative mechanism (Coles et al., 2001; Falkenstein et al., 1990) or the process itself (Falkenstein et al., 2000; Vidal et al., 2000). It is also possible that the Ne/ERN is the index of another process necessary for preventing or correcting errors or for behavioral adjustments, which therefore utilizes some information from the comparative mechanism (Gehring et al., 1993; Holroyd & Coles, 2002).

Conflict monitoring theory of the Ne/ERN and N2. The conflict monitoring theory is another influential framework for explaining the Ne/ERN in combination with the N2 and is an alternative to the mismatch theory (Botvinick et al., 2001; Yeung et al., 2004). While the mismatch model is based on comparing the representation of the correct response and the incorrect output, the conflict monitoring theory focuses more generally on the response conflict, which occurs when multiple competing response options are activated simultaneously. This can also account for ACC activity on correct trials, as has been observed in different fMRI studies (e.g., Carter et al., 1998; Kiehl et al., 2000; Milham & Banich, 2005), explaining the CRN. Based on those conflict detection processes, behavior is then adapted to prevent future conflicts by adjusting attentional control (Yeung et al., 2004). The Ne/ERN is assumed to indicate a post-response conflict. A post-response conflict arises when the correct response is activated after the response by further stimulus processing, which then competes with the incorrect response that is still active (Yeung & Cohen, 2006). Whenever the post-decisional activation of the representation of the correct response and thus the corrective tendency can develop more easily, the post-response conflict and, hence, the Ne/ERN is increased (Yeung & Cohen, 2006). This is why the Ne/ERN is typically higher following congruent or neutral compared to incongruent trials (e.g., Forster & Pavone, 2008; Maier et al., 2011; Scheffers & Coles, 2000; for simulated data, see Yeung et al., 2004).

While the Ne/ERN is seen as a post-response conflict on error trials, the N2 is suggested to reflect a pre-response conflict on correct trials (Yeung et al., 2004; Yeung & Cohen, 2006). The pre-response conflict arises because competing responses are activated simultaneously before making the correct response (Yeung & Cohen, 2006). This is why the N2 is typically higher following incongruent compared to congruent correct trials (e.g., Danielmeier et al., 2009; Folstein & van Petten, 2008; Forster & Pavone, 2008; Jost et al., 2022). Both components seem to indicate the same phenomenon, i.e., response conflict but are affected by characteristics of tasks in different ways and relate to different levels of conflict processing (Yeung & Cohen,

2006). This perspective is supported by studies that show that the dorsal part of the ACC (dACC) is thought to be the neural generator of both components (van der Veen & Carter, 2002b; Yeung, 2004). In addition, the N2 and the Ne/ERN have opposing patterns, i.e., aspects that enhance the Ne/ERN, reduce the N2, and vice versa. This pattern has been shown in several studies (Danielmeier et al., 2009; Pailing & Segalowitz, 2004a; Yeung et al., 2004). Therefore, the conflict monitoring theory offers the great advantage of explaining both components, the N2 as pre-response conflict and the Ne/ERN as post-response conflict.

Reinforcement learning theory of the Ne/ERN. The third major theory of the Ne/ERN is the reinforcement learning theory (Holroyd & Coles, 2002). Whenever the outcome of an action is worse than expected, a negative learning signal is conveyed from dopaminergic midbrain areas to the ACC, eliciting the Ne/ERN (Holroyd & Coles, 2002), e.g., when an error is detected. According to this theory, the ACC activity and thus the Ne/ERN are higher, especially in conditions in which errors are less frequent and therefore less expected, which is why those conditions with a higher Ne/ERN are also typically accompanied by lower error rates in this theory (Kappenman et al., 2012). The ACC is trained by these signals to optimize its control over motor programs, hence the ACC learns, which motor controller fits best for the respective task (Holroyd & Coles, 2002). This learning process depends on how many errors are registered from the error-monitoring system. Another framework that explains the Ne/ERN based on reinforcement learning is the predicted response outcome model (PRO-Model), developed by Alexander and Brown (2011). In contrast to the former reinforcement learning theory, this account assumes ACC activity to indicate unexpectedness, independent of whether the outcome is worse or better. In conditions with infrequent correct trials, the ACC activity should be stronger for correct trials than for incorrect trials, which should be reflected by a higher CRN. Indeed, Meckler et al. (2011) showed that the CRN effect for correct trials was enhanced when occurring infrequently.

Inhibition theory of the N2. The inhibition theory is an early model of the N2. While the N2 is associated with a pre-response conflict in the conflict monitoring theory, it is related to an inhibition process in the inhibition theory. It is therefore suggested that the N2 indicates the inhibition of irrelevant responses, activated by distractive stimuli, to make goal-directed behavior possible (Falkenstein et al., 1999; Gajewski & Falkenstein, 2013; Jodo & Kayama, 1992; Kopp et al., 1996; Vuillier et al., 2016). When the need for inhibition is high, the N2 is enhanced relative to situations where no or less inhibition is necessary. E.g., in a Go/No-Go paradigm, in which participants have to respond to Go- but withhold their responses for No-Go-stimuli, the N2 is enhanced, i.e., more negative for No-Go stimuli (Falkenstein et al., 1999; Jodo & Kayama, 1992; Kopp et al., 1996). Kopp et al. (1996) used an Eriksen flanker task with incongruent, congruent, and neutral flankers to study the inhibition process. They found that incongruent stimuli caused a higher N2, while it did not differ between the other two conditions (Kopp et al., 1996). The authors argued that a perceptual conflict between dissimilar targets and flankers could not explain these results because, in the neutral condition, flankers also differ from the target stimulus, and therefore, the N2 should also be affected (Kopp et al., 1996). In this vein, the authors conclude that the N2 reflects the inhibition of a response to an irrelevant but similar stimulus like the incongruent flankers. In the conflict monitoring theory framework, there should have been a significant difference between the neutral and congruent conditions as well, although to a lesser degree than between congruent or neutral trials and incongruent trials. However, it has to be noted that a response conflict could not occur because no response button was assigned to the neutral flanker, hence the results from Kopp et al. (1996) do not speak against the conflict monitoring theory.

Selection for action theory of the N2. Another alternative account for the N2 is the selection for action theory. While, e.g., the conflict monitoring account suggests that the ACC monitors behavior for conflicts and increases attention to diminish these conflicts if necessary (Yeung et al., 2004), the selection for action theory assumes that attentional adaptation serves to support

behavior directly (Allport, 1987). In this sense, the ACC contributes to the selection, preparation, and execution of actions (Turken & Swick, 1999) by coupling stimuli with their respective actions (Gajewski et al., 2008). When responses are simultaneously activated, e.g., via incongruent stimuli of an Eriksen flanker task, the dACC is suggested to select the response that is most appropriate to reach a performance goal (Pardo et al., 1991; Posner et al., 1988; Posner & Dehaene, 1994; Turken & Swick, 1999). Although the conflict monitoring theory is more suitable to explain the engagement of the dACC for different task requirements such as overriding prepotent responses or signaling error commission (Botvinick et al., 1999; Botvinick et al., 2001), the selection for action theory, which associates the dACC to a direct or indirect action selection process, is still popular (Akam et al., 2021; Banich, 2019; Holroyd & Yeung, 2012; Silton et al., 2010). The N2 was related to this selection process by Gajewski et al. (2008), who used incorrect and correct cues before the target stimulus occurred. The N2 was enhanced when incongruent cues were shown and even more enhanced when those cues were infrequent (Gajewski et al., 2008). In the authors' perspective, the enhanced N2 is based on the higher need for selection when a revision of the initial response becomes necessary by violating the expectancies of the incorrectly cued response (Gajewski et al., 2008).

Theories of the Pe

The functional significance of the Pe was discussed by Falkenstein et al. (2000), who concluded that the Pe reflects further error processes in addition to the Ne/ERN. In this vein, the question arises, which function this additional error process could serve. Three main hypotheses about this additional process suggest that it could be (1) the affective processing, which accompanies erroneous responses; (2) post-error adjustments; or (3) the conscious awareness of errors (Falkenstein et al., 2000). In the following sections, all three hypotheses are explained in further detail.

Affective processing hypothesis. In the affective processing hypothesis, the Pe indicates the emotional evaluation of an erroneous response (Falkenstein, 2003; Overbeek et al., 2005). This hypothesis was formulated by Falkenstein et al. (2000), who found lower Pe amplitudes for participants with many errors in relation to participants with fewer errors. The authors argued that those participants with many errors cared less than those with fewer errors (Falkenstein et al., 2000) and that the Pe effect was supposed to be driven by negative affect. However, these results could not be replicated by others (e.g., Hajcak et al., 2003; Herrmann et al., 2004) and Hajcak et al. (2004) even found the opposite, i.e., participants with high scores for negative affects showed a decreased Pe in contrast to those with low scores. In addition, some researchers tried to allocate the neural generator of the Pe to caudal (Herrmann et al., 2004) or, more importantly, to rostral (van Boxtel et al., 2005; van der Veen & Carter, 2002a) parts of the ACC using dipole source modelling. Because rostral parts of the ACC are also associated with the processing of emotions (e.g., Bush et al., 2000), the connection between Pe and emotional processing seems to be evident in this respect. However, there is no direct evidence that the Pe is related to affect, so there is little support for the affective processing hypothesis.

Behavior-adaption hypothesis. Another alternative to explain the Pe is the behavior-adaptation hypothesis, suggesting that the Pe reflects the degree of behavioral adaptations after erroneous responses indicated by PES (Hajcak et al., 2003; Nieuwenhuis et al., 2001). The correlation between PES and the Pe gave rise to this hypothesis (Hajcak et al., 2003; Nieuwenhuis et al., 2001), but other studies did not confirm this correlation (Overbeek et al., 2005). Overbeek et al. (2005) stated that the awareness of errors could also drive this correlation. In summary, the behavior-adaption hypothesis also receives little support, while the relation between error awareness and the Pe is the most robust finding.

Error-awareness hypothesis. Nieuwenhuis et al. (2001) demonstrated that the Pe is only elicited robustly when errors are consciously detected, while the Ne/ERN is unaffected by error awareness. The authors used an antisaccade paradigm to elucidate these relationships. In this

study, participants fixate on the center of the screen and are instructed to direct their gaze in the opposite direction of an appearing stimulus. Sudden stimuli often cause an automatic and minimal saccade to their direction (e.g., Theeuwes et al., 1998), which is why numerous erroneous saccades are made, remaining unaware. Nieuwenhuis et al. (2001) found a robust relationship between error awareness and the Pe, while the Ne/ERN was unaffected by error awareness. Afterward, this finding was corroborated by a majority of studies, which found enhanced Pe for aware relative to unaware errors, which is why the Pe is supposed to be a reliable electrophysiological marker of the metacognitive experience of error awareness (O'Connell et al., 2007; Boldt & Yeung, 2015; Dhar et al., 2011; Endrass et al., 2005, 2007; Hewig et al., 2011; Hughes & Yeung, 2011; Kirschner et al., 2021; Murphy et al., 2012; Overbeek et al., 2005; Shalgi et al., 2009; Steinhauser & Yeung, 2010; Wessel et al., 2011).

Mechanisms of error awareness

The evidence accumulation account and the Pe

Error awareness can be defined as the capacity to access error-related information in consciousness (Block, 2007). The ability to report errors is the main operationalization of access to consciousness. This means that the neural system can access representations of mental content and provide them for verbalization (Wessel, 2012). Anatomically, error awareness seems to be highly associated with the activation of the AIC, while the error-related activity in the ACC seems independent of awareness (Hester et al., 2005; Klein et al., 2007), although a contribution of the ACC cannot be excluded (Herrmann et al., 2004; van Boxtel et al., 2005; van der Veen & Carter, 2002b). The insula shows activation for different phenomena such as processing autonomic (Critchley et al., 2004, Critchley et al., 2005), visceral motor as well as sensory information (Augustine, 1996), and interoception (Craig, 2009, 2011). This diverse information could significantly contribute to error awareness because erroneous responses cause activation in different systems, such as the ANS. Therefore, one approach to explain the

Pe is that the Pe does not indicate error awareness per se but reflects the accumulated evidence that an erroneous response has just been made, which then leads to error awareness (Desender et al., 2021; Steinhauser & Yeung, 2010, 2012).

This postdecisional accumulation process of error evidence can be explained within the mathematical framework of the drift-diffusion model (DDM), which was developed by Ratcliff (1978) in order to analyze data from speeded response time tasks. Initially, the DDM describes the evidence accumulation process on the predecisional level (Ratcliff, 1978; Ratcliff & McKoon, 2008) and is one variant of a class of models in which binary decisions are explained by an accumulation-to-bound mechanism (Yeung & Summerfield, 2012). It means that evidence for one of two options in a choice task is accumulated until the bound, the criterion, of this corresponding option is reached and hence, the related decision is made (Ratcliff, 1978; Ratcliff & McKoon, 2008). These boundaries reflect the amount of evidence needed to make the related decision (Ratcliff, 1978; Ratcliff et al., 2016). From a starting point, evidence accumulation drifts with a specific rate towards one of the two boundaries. The better the quality of evidence of a given stimulus is, the faster a boundary is reached, that is, the faster the RT. However, the accumulated evidence is also contaminated with noise, which can lead to variable RTs for the same stimulus and incorrect decisions as well (Ratcliff & McKoon, 2008; Ratcliff et al., 2016). In the DDM, both drift rate and noise constitute the decision variable (DV), which is seen as the parameter that indicates the quality of the accumulated evidence (Ratcliff, 2014) and has a particular slope. Both the boundaries and the DV can be manipulated via the speed-accuracy trade-off (e.g., Ratcliff & McKoon, 2008; Pleskac & Busemeyer, 2010). When speed in decision task is emphasized over accuracy, e.g., the bound is lower, less evidence is needed to make the corresponding decision. However, the evidence is also more contaminated with noise, i.e., it reduces the accuracy of choice and is, therefore, associated with more errors. When accuracy is emphasized over speed, the bound is higher. Hence, more evidence is needed and it takes longer until the boundary is crossed, but the effect of noise also decreases.

As mentioned above, error monitoring has been thought to depend on postdecisional evidence accumulation processes since Rabbitt's initial work in the 1960s (Rabbitt, 1966; Rabbitt & Vyas, 1981). In Rabbitts' and Vyas' (1981) interpretation, some errors occur due to uncompleted information processing. After an error response is made, further information processing causes evidence accumulation in favor of the correct response, which is why the error then can become obvious. This postdecisional process can be expressed in terms of modern DDM-based theories. When the initial decision is correct, the DV further develops in the same direction as the original decision bound, that is, further evidence is accumulated in favor of this correct decision (Pleskac & Busemeyer, 2010). Instead, when an error has been made, the DV crosses the error decision boundary for the second time, exceeding another boundary, reflecting the change-of-mind point (Desender et al., 2021; Resulaj et al., 2009). Ideally, the DV then crosses the second boundary, reflecting the correct response bound (Steinhauser et al., 2008; Yeung & Summerfield, 2012), leading to a signaled error (Pleskac & Busemeyer, 2010). The time interval between recrossing the original error bound and crossing the actual correct bound is characterized by high uncertainty or conflict (Desender et al., 2021; Yeung & Summerfield, 2012).

Investigating those postdecisional error evidence accumulation processes in relation to error awareness, Steinhauser and Yeung (2010) successfully manipulated the level of the postdecisional boundaries (the internal criterion) by manipulating the incentives for specific responses. In the first condition, participants lost more money when they failed to signal an error, which led to a lower internal criterion (a lower bound), i.e., less evidence was needed. In the second condition, they lost more money when they falsely indicated a correct response as an error, causing a higher internal criterion (a higher bound). The amplitude of the Pe differed significantly, with a lower amplitude for the lower and a higher amplitude for the higher internal criterion. In contrast, the Ne/ERN was unaffected by this manipulation. These results strengthen the notion that the Pe refers to error awareness more than the Ne/ERN and also that the Pe seems to reflect evidence accumulation, leading to Steinhauser and Yeung (2010) formulating

the evidence accumulation account. Furthermore, these results were corroborated by another study by Steinhauser and Yeung (2012). The Pe, therefore, probably indicates not error awareness per se but rather the process leading to error awareness (Steinhauser & Yeung, 2012). Although this interpretation should be handled with caution because the Pe could reflect different cognitive processes (Desender et al., 2021; Luck, 2014), the Pe could, nevertheless, reflect the DV of evidence accumulation, which is driven by different sources such as cognitive, sensory and autonomic information (Ullsperger et al., 2010; Wessel et al., 2011).

The Pe can vary gradually as well, as was shown by Boldt and Yeung (2015), who investigated decision confidence by asking participants to indicate how confident they were about responding (in)correctly on a graded 6-point scale ranging from 'certainly correct' to 'certainly incorrect'. The more confident the participants were that they made an error, the higher the Pe amplitude was. Correspondingly, the Pe decreases monotonically the higher the confidence was that a correct response was made. Boldt and Yeung (2015) suggested that error detection as well as decision confidence, as two metacognitive judgments, have a shared neural basis, which was also demonstrated by other studies (Desender et al., 2019; Selimbeyoglu et al., 2012) and is consistent with the accumulation process after decisions. In summary, the Pe amplitude is likely to reflect a gradual indicator of accumulated evidence for erroneous responses (Steinhauser & Yeung, 2010; Di Gregorio et al., 2018) or the graded confidence for erroneous responses based on this accumulation process (Boldt & Yeung, 2015; Di Gregorio et al., 2018).

Error awareness and the Ne/ERN

Although the Pe is more strongly associated with error awareness, the Ne/ERN has also been discussed in relation to this phenomenon. Scheffers and Coles (2000) were the first, who addressed the relationship between the Ne/ERN and error awareness and found that the amplitude of the Ne/ERN got higher with increasing error awareness. Other studies also found these differences in the Ne/ERN between reported and nonreported errors (e.g., Maier et al.,

2008; Steinhauser & Yeung, 2010; Wessel et al., 2011; Woodman, 2010). However, some studies found no effects (e.g., Dhar et al., 2011; Endrass et al., 2007; Nieuwenhuis et al., 2001; Shalgi et al., 2009) or even the opposite effect, i.e., a negative relation between the Ne/ERN and error awareness (Di Gregorio et al., 2016; Maier et al., 2008). It was argued that these inconsistent findings could be caused by moderating or mediating variables such as attentional effects or response conflicts (Di Gregorio et al., 2016, Di Gregorio et al., 2018). However, a study conducted by Di Gregorio et al. (2022) on early error sensations showed that latencies of the Ne/ERN can serve as internal information that affects error awareness. Early error sensation is a phenomenon that reflects the subjective feeling of having made an error even before it occurs. The study asked participants to distinguish between early and late errors. The Pe amplitudes were more prominent for early errors, but there was no difference in their latencies. The Ne/ERN differed only in terms of its latencies, with an earlier onset for early errors than for late errors, but showed no difference in the amplitudes. (Di Gregorio et al., 2022). Di Gregorio et al. (2022) noted that error awareness may not necessarily occur at the time point of the Ne/ERN. Instead, error awareness may be dated back, allowing for coherence in conscious perception (see Di Gregorio et al., 2020; Libet et al., 1979; Libet et al., 1983). In summary, the Pe is commonly regarded as a reliable marker of error awareness. Nevertheless, the possibility that the Ne/ERN can contribute to error awareness is not excluded.

One or two underlying mechanisms: How the Pe relates to the Ne/ERN

The Ne/ERN can occur without the Pe, especially when errors remain unaware (e.g., Nieuwenhuis et al., 2001; Wessel et al., 2011). Nevertheless, whether the later Pe can occur without the Ne/ERN was controversial until recently. There were two discussed possibilities: On the one hand, both ERPs could form a stage-like architecture, i.e., the Pe could be causally dependent on the earlier Ne/ERN (Scheffers & Coles, 2000; Ullsperger, Fischer, et al., 2014; Yeung et al., 2004). On the other hand, it could be possible that the Pe does not necessarily rely

on the Ne/ERN, i.e., there are different and independent mechanisms for both (Charles et al., 2013; Falkenstein et al., 2000; Maier et al., 2015). Di Gregorio et al. (2018) conducted a study to investigate these possibilities by using a target-masking paradigm. The main objective of this paradigm is to create errors that participants are aware of without knowing the correct response. This manipulation causes the absence of the Ne/ERN, which relies on the representation of the correct response according to major theories such as the mismatch model (Falkenstein et al., 1991; Gehring et al., 1993), the conflict monitoring theory (Botvinick et al., 2001; Yeung et al., 2004) and the reinforcement learning theory (Holroyd & Coles, 2002).

The target-masking paradigm by Di Gregorio et al. (2018) is an adapted version of the letter-based Eriksen flanker task (Eriksen & Eriksen, 1974), where a mask conceals the central target letter, while flanker letters remain visible. In the visible-target condition, the target letter is replaced by a mask after a specific time, making the correct response detectable. In the invisible-target condition, the target letter is replaced by the mask from the start, making the correct response invisible. However, in the invisible-target condition, responses to flankers (flanker errors) can still be perceived as errors because participants are instructed that flankers always differ from the central target letter. Corrects and nonflanker errors, in turn, cannot be distinguished in the invisible-target condition, which is why they are summarised as nonflanker guesses. Using the target-masking paradigm, Di Gregorio et al. (2018) demonstrated that the Ne/ERN disappears, while the Pe still occurs in the absence of the Ne/ERN, suggesting that the processes underlying the Ne/ERN and Pe are indeed independent and access different information. Moreover, by using a multivariate pattern analysis, Di Gregorio et al. (2018) demonstrated that the Pe in the invisible-target condition is based on the same activity as the Pe in the visible-target condition, which is why the positive deflection in the invisible-target condition can be interpreted as a Pe. Finally, the frontocentral theta power, another robust marker of error processing (e.g., Cavanagh et al., 2012; Murphy et al., 2015), showed the same

pattern as the Ne/ERN, indicating that both phenomena are based on similar mechanisms (Di Gregorio et al., 2018).

In summary, Di Gregorio et al. (2018) directly demonstrated for the first time that the Pe is not necessarily dependent on the Ne/ERN. Therefore, both ERPs appear to be based on two independent mechanisms. Because the Ne/ERN is absent for errors in the invisible-target condition, while the Pe remains unaffected, it can now be directly examined whether other post-decisional phenomena such as conscious error detection, error-related pupil dilation, and heart rate deceleration are associated with the early error processes that cause the Ne/ERN.

Pupil and heart rate-related responses during error processing

Error-related pupil dilation and heart rate deceleration enable additional insights into information processing. The locus-norepinephrine system (LC-NE system) seems to be an essential circuit for both parameters. The LC, a nucleus in the brainstem, is the primary source of norepinephrine to most parts of the brain (Moore & Bloom, 1979) and regulates functions of the central nervous system (CNS) as well as of the ANS (Berridge & Waterhouse, 2003; Joshi et al., 2016; Sved et al., 2002). Initially, the activity of the LC-NE system was linked to arousal (Berridge & Waterhouse, 2003), but this view was specified and extended to a more complex role of the system for neural processes (Aston-Jones et al., 1994; Aston-Jones et al., 1997; Clayton et al., 2004; Rajkowski et al., 2004) such as its contribution to attention and flexible behavior (Bouret & Sara, 2004). Overall, two different activity modes of LC neurons can be distinguished, a phasic and a tonic mode (Usher et al., 1999; Vazey et al., 2018). The tonic mode is characterized by a high baseline activity of the LC with continuous but irregular firing (Vazey et al., 2018; Murphy et al., 2011). During the tonic mode, task performance is poorer and the responsitivity to task-related stimuli is reduced (Usher et al., 1999). Instead, the phasic mode is characterized by short bursts of activation with higher frequencies (Vazey et al., 2018)

and intermediate tonic activation (Murphy et al., 2011). The phasic mode is associated with good performance and higher responsitivity to task-relevant stimuli (Usher et al., 1999).

One critical framework that tries to explain these two modes' functions is the adaptive gain theory (AGT) from Aston-Jones and Cohen (2005). Within the AGT, the phasic mode is supposed to support performance optimization by strengthening task-related behavior (exploitation). In turn, the system switches to the tonic mode whenever the current task loses its utility and reorientation to alternatives is needed (exploration), which is why the distractibility increases. Within these two modes, neurons' responsivity (gain) to task-related cognitive processes is modulated within local circuits. The phasic mode increases and the tonic mode decreases this gain. This is in line with the proposal that the phasic LC-NE mode improves behavior by enhancing responsiveness to input (Łukowska et al., 2018; Servan-Schreiber et al., 1990). Besides the high level of tonic baseline activation (tonic mode), leading to distractability and intermediate baseline levels (phasic mode), corresponding to good performance, a low baseline activity is possible as well, causing sleepiness (Aston-Jones & Cohen, 2005). These relations resemble the arousal curve from Yerkes-Dodson (Yerkes & Dodson, 1908). Thus, phasic activation of the LC and performance are best when the tonic activation has an intermediate level, whereas performance declines during high and low tonic levels (Aston-Jones & Cohen, 2005).

Finally, the LC-NE system is interconnected with the central autonomic network, which is functionally crucial for goal-directed behavior and behavioral adaptation by internally regulating and controlling visceral, neuroendocrine, and behavioral functions (Benarroch, 1993, 1997). This network encompasses the ACC, the AIC, and prefrontal brain areas and subcortical areas such as the amygdala, the hypothalamus, and nuclei of the brain stem (Thayer et al., 2009; Blessing, 1997). It, therefore, also overlaps with the salience network and the error-monitoring system.

Pupillometry

It is well known that pupils change their size due to light incidences, i.e., getting smaller when brightness increases and vice versa, which is known as the pupillary light reflex (Lowenstein & Loewenfeld, 1950; Toates, 1972). However, the pupil can even be affected by non-reflexive processes such as attentional states, perception, memory processes, and cognition (e.g., Granholm & Steinhauer, 2004; Hess & Polt, 1960, 1964; Kahneman & Beatty, 1966; Kuntz, 1936; Nassar et al., 2012). Pupil measurements have, therefore, become an essential and non-invasive tool to infer cognitive processes, such as decision-making and error monitoring (Ebitz & Moore, 2019; Critchley et al., 2005; Wessel et al., 2011).

Generally, the pupil's muscles are under the control of the sympathetic and parasympathetic branches of the ANS. Parasympathetic activity indirectly inhibits the sphincter pupillae via the Edinger-Westphal nucleus and the neurotransmitter acetylcholine (ACh), whereas sympathetic activity directly controls the dilator muscles through the neurotransmitter NE (Loewenfeld, 1999). Moreover, the LC-NE system is a relevant neural circuit that connects cognitive processes with pupil reactions (Joshi & Gold, 2020; Joshi et al., 2016; Larsen & Waters, 2018; van der Wel & van Steenbergen, 2018). The phasic or tonic NE release from the LC-NE system is highly related to pupil size, as was shown by different studies (e.g., Gilzenrat et al., 2010; Hou et al., 2005; Morad et al., 2000; Murphy et al., 2011, 2014; Phillips et al., 2000; Rajkowski et al., 1993). For example, there was a robust covariation between blood-oxygen-level-dependent (BOLD) activity in the LC and pupil diameter, measured in human participants in a functional magnetic resonance tomography (fMRI) study (Murphy et al., 2014). Joshi et al. (2016) directly demonstrated that changes in pupil diameter can be successfully predicted by electrical stimulation of the LC, which has been corroborated by other studies (Privitera et al., 2020; Reimer et al., 2016). However, the existence of a pathway that directly links the LC and the pupils is still under debate (Nieuwenhuis et al., 2011; Viglione et al., 2023). The LC-NE activity is, nevertheless, a good predictor of changes in pupil size, which is why effects on pupil

size reliably indicate regulative influences of the LC-NE system on other neural processes (Joshi et al., 2016; Viglione et al., 2023). Moreover, the LC-NE system is affected by information about cognitive processes such as conflict processing of the ACC, i.e., there could be an indirect contribution of the ACC to pupil dilations (Aston-Jones & Cohen, 2005; Ebitz & Platt, 2015; Hayden et al., 2011; Porrino & Goldman-Rakic, 1982). Next to the ACC, activity in the AIC is strongly associated with pupil dilations as well (Critchley et al., 2005). Because all three brain regions, the LC, ACC, and AIC, are supposed to be involved in error monitoring, pupillometry is a valid method for studying error processing in cognitive psychology.

Pupil dilation is generally more prominent after errors (Braem et al., 2015; Critchley et al., 2005; Murphy et al., 2016; Rondeel et al., 2015; Wessel et al., 2011). Transient pupil dilation, independent from light, can be seen as a marker of the orienting response to salient stimuli (Sokolov, 1963; Lynn, 1966). The orienting response is reflected by increased autonomic arousal after novel and unexpected events and, therefore, by physiological changes (Sokolov, 1963; e.g., reviewed by Graham & Clifton, 1966; Pribram & McGuinness, 1975; van der Molen et al., 1991). Functionally, the orienting response refers to a reallocation of attention to the corresponding event by an inhibition of ongoing behavior to improve perception, evaluation as well as selection of responses (Barry et al., 2011; MacDonald et al., 2012; Sokolov, 1963). Since errors can be interpreted as salient and unexpected events (Harsay et al., 2012; Wessel, 2018), error-related pupil dilation has been suggested to constitute an orienting response to errors (Murphy et al., 2016; Notebaert et al., 2009). In this sense, error-related pupil dilation is based on the fact that errors are rare occurrences (Wessel, 2018).

Another study showed that error significance could also affect pupil dilation, not just by error frequency differences, as stated in the orienting account (Maier et al., 2019). Error significance refers to the importance of errors for behavioral adaptations. Because cognitive resources for behavioral adaptation are limited and costly (Shenhav et al., 2017), these resources should be allocated mainly after highly significant errors (Maier et al., 2019). For resource allocation, the

LC-NE system is thought to play an important role (Aston-Jones & Cohen, 2005; Jepma & Nieuwenhuis, 2011). This is why pupil dilation should be influenced by differences in error significance, which was confirmed by Maier et al. (2019). They used an adapted Eriksen flanker task, in which flanker and nonflanker errors were possible. Comparing flanker and nonflanker errors, flanker errors are additionally characterized by a failure of selective attention to the target stimulus, which is why selective attention has to be improved in the subsequent trial. Flanker errors are, therefore, more significant for the neural system to optimize behavior. Indeed, pupil dilation was higher for flanker than nonflanker errors (Maier et al., 2019).

While the relation between phasic pupil dilation and erroneous responses is a robust finding (Braem et al., 2015; Critchley et al., 2005; Murphy et al., 2016; Rondeel et al., 2015; Wessel et al., 2011), the relation with the Ne/ERN and the Pe is still controversial. Only a few studies examined whether manipulating the Ne/ERN by different error types (Maier et al., 2019), memory demands as well as state anxiety (LoTemplio et al., 2023), or error awareness (Wessel et al., 2011) also influence pupil dilation. However, these studies yielded different results regarding the Ne/ERN. Also, regarding the Pe, there is no consistent pattern. Indeed, phasic pupil dilation appears to be positively related to error awareness, which aligns with most studies (Harsay et al., 2018; Quirins et al., 2018; Wessel et al., 2011). However, although the Pe robustly indicates error awareness, a positive relation between the Pe and pupil dilations cannot be derived unequivocally. An electrophysiological study investigating this relation, as well as the relation with the Ne/ERN, was conducted by Wessel et al. (2011). They asked whether and how both components and activity from the ANS, measured via heart rate and pupillometry, are affected by conscious error awareness. Despite the finding that both the Pe and the Ne/ERN were significantly enhanced by error awareness, pupil dilation was also increased for consciously perceived erroneous responses (Wessel et al., 2011). The authors suggested that different sources of error evidence, such as proprioception, sensory inputs, response conflict, and early autonomic reactions, serve as important information for the cognitive system to drive

error awareness (Wessel et al., 2011; Wessel, 2012). However, by comparing perceived and unperceived trials, they did not directly relate pupil dilation to the Ne/ERN or the Pe and they also found increased Ne/ERN amplitudes for perceived errors, so a clear relation between pupil dilation and error awareness cannot be deduced.

Moreover, the study mentioned above by Maier et al. (2019), investigating error significance, revealed another pattern between pupil dilation and error awareness. While the Ne/ERN perfectly mirrored the pupil dilation pattern, thus a higher activity for flanker than nonflanker errors, the Pe was more increased for nonflanker errors. Furthermore, nonflanker errors were also associated with increased error awareness. These results, i.e., greater pupil dilations and a greater Ne/ERN but a lower Pe and reduced error awareness for flanker errors, indicate that the error-related pupil dilation is not necessarily related to error awareness (Maier et al., 2019). Maier et al. (2019) argued that the ACC, the generator of the Ne/ERN, registers erroneous responses and determines their significance for behavioral adjustments. This signal is then relayed to the LC-NE system, amplifying autonomic arousal. The authors therefore assumed that the unconscious evaluation of error significance, indicated by the Ne/ERN, is sufficient for the increase in autonomic arousal (Maier et al., 2019).

In summary, cognitive processes can influence pupil size and are a valid indicator for the LC-NE system's phasic and tonic NE release. Pupil dilation can be interpreted as an orienting response to unexpected events, such as errors, to facilitate various cognitive processes. It is generally enhanced after errors, but the relationship between pupil dilation, the Ne/ERN, and the Pe remains unclear as the studies that have investigated this relationship yielded mixed results. Considering error awareness, there seems to be a link with pupil dilation. However, there is evidence that pupil dilation relates more to early error processes, as indicated by the Ne/ERN. For this reason, the present thesis investigates whether and how the pupil reacts in the absence of the Ne/ERN and, thus, whether pupil dilation is already related to early error processing.

Heart rate measurement

The heart can run without any external influence because of its pacemakers, the sinoatrial node, and the atrioventricular node (Shaffer et al., 2014). These pacemakers are mainly responsible for the heartbeat, which consists of a cardiac cycle with alternating contractions (systole) and relaxations (diastole; Monfredi et al., 2010; Shaffer et al., 2014). This heart activity constitutes an electrical conduction system and, therefore, can be recorded by an electrocardiogram (ECG; Shaffer et al., 2014). In the ECG, a non-pathological signature of a heartbeat consists of different waves, which typically have the same sequence and are abbreviated with the letters P, Q, R, S, and T (Ahmed et al., 2010; AlGhatrif & Lindsay, 2012; Shaffer et al., 2014). The P wave indicates the depolarization of both heart's atriums, while the QRS complex resembles the depolarization of both ventricles (systole; Ahmed et al., 2010; Shaffer et al., 2014). The T wave, in turn, represents the re-polarization of the ventricles and, following the T wave, the ventricles are relaxed again (diastole; Ahmed et al., 2010; Shaffer et al., 2014). The QRS complex with its R peak is the most prominent, which is why these R peaks are mainly used for calculations of heart-related measurements. One measurement is, therefore, the time interval between two R peaks, the so-called interbeat interval (IBI; Shaffer et al., 2014).

The number of heartbeats per minute (bpm) constitutes the heart rate, which is determined by the activation of the sinoatrial and atrioventricular nodes (Monfredi et al., 2010; Shaffer et al., 2014). At rest, the heart rate is around 60 to 90 bpm (Monfredi et al., 2010; Shaffer et al., 2014), but could be modulated by the ANS through its sympathetic and parasympathetic branches, which influence the sinoatrial and atrioventricular node with the neurotransmitter NE for sympathetic and ACh for parasympathetic innervation (Levy & Martin, 1984; Obrist, 1981; Shaffer et al., 2014; Somsen et al., 2004). In healthy, resting subjects, the parasympathetic influence is dominant over sympathetic innervations (Shaffer et al., 2014), while shifts in their balance allow them to adapt to relevant emotional, physiological, or environmental changes flexibly (Prokhorov et al., 2021). This balance can be modulated by input from cortical and

subcortical brain areas like the ACC, AIC, and LC-NE system, which then leads to heart rate changes (Critchley et al., 2000; Hajcak et al., 2003; Oppenheimer et al., 1992; Shaffer & Venner, 2013; Sved et al., 2002; Sved & Felsten, 1987). The AIC cortically integrates cardiovascular and other visceral afferent information (Craig, 2002; Strigo & Craig, 2016), whereas the ventral portion of the ACC is another structure that processes information from internal organs (Critchley & Harrison, 2013; Stevens et al., 2011). The LC, in turn, is interconnected with other nuclei in the brainstem, processing autonomic information and, therefore, indirectly with the AIC and ACC (Nieuwenhuis et al., 2011).

Typically, stress increases the sympathetic influence and heart rate, while the parasympathetic influence decreases the heart rate. Paradoxically, erroneous responses, which can be considered as stressors, decelerate heart rate, which has been found in several studies (e.g., Danev & de Winter 1971; Crone et al., 2003; Fiehler et al., 2004; Hajcak et al., 2003; Ullsperger et al., 2010; van der Veen, van der Molen et al., 2004; Bastin et al., 2017; Wessel et al., 2011). This paradoxical finding can be explained by Porges' (1992) two-component model. In this framework, a distinction is made between a reactive and sustained heart-related response. In the first milliseconds after an event, the reactive response dominates with its parasympathetic influence and causes an initial heart rate deceleration (Abercrombie et al., 2008). After that, the sustained response with its sympathetic influence of NE dominates and leads to a subsequent heart rate acceleration.

Heart rate deceleration, in general, is associated with the processing of negative emotional stimuli (Bradley et al., 1996; Bradley et al., 2001; Bradley & Lang, 2000; Brosschot & Thayer, 2003; Waldstein et al., 2000), leading van der Veen, van der Molen et al. (2004) to interpret the error-related heart rate deceleration as an affective signal as well. In addition to this interpretation, the prevailing view is that it is part of an orienting response, analogous to pupil dilation (Sokolov, 1963; e.g., reviewed by Graham & Clifton, 1966; Pribram & McGuinness, 1975; van der Molen et al., 1991). Because an orienting response is based on the novelty or

unexpectedness of an event, Ullsperger et al. (2010) argued that the conscious detection of this event should be necessary, which is in line with the study from Wessel et al. (2011), comparing perceived and unperceived error trials. They showed that heart rate deceleration was more prominent for perceived errors. The same pattern was found for the Ne/ERN and the Pe. However, as with pupil dilation, they did not directly relate heart rate deceleration with the Pe and the Ne/ERN. Also, other studies have not clarified either due to mixed results. These studies investigated different aspects such as experimentally induced stress (Rodeback et al., 2020), pitch errors in musicians (Bury et al., 2019), feedback processing (van der Veen, Nieuwenhuis et al., 2004), and negative affect (Hajcak et al., 2004). While some studies found an effect of early error processes or the Ne/ERN, respectively, on heart rate (Bury et al., 2019; Wessel et al., 2011), others did not (Hajcak et al., 2004; Rodeback et al., 2020; van der Veen, Nieuwenhuis et al., 2004). So far, there is no clear relationship between error-related heart rate deceleration and the Ne/ERN and the Pe. Recently, this was corroborated by a comprehensive review (Di Gregorio et al., 2024).

In summary, cognitive processes can influence cardiac activity, making the ECG a valid method to gain insight into information processing, such as error monitoring. An important heart-related measurement is the heart rate deceleration after errors. Heart rate deceleration can also be interpreted as an orienting response, like pupil dilation. In general, heart rate deceleration is higher after erroneous than correct responses, but the relationship with the Ne/ERN, indicating early error processes, and the Pe, indicating error awareness, is still up for debate. While some studies found a relationship between heart rate deceleration and both ERPs, others did not. Therefore, the present thesis addresses whether error-related heart rate deceleration can be linked to early error processes and, thus, to the Ne/ERN.

Outline of subsequent studies

All three subsequent studies center around the Ne/ERN. The Ne/ERN indicates early error detection without conscious error awareness and is associated with fast and automatic behavioral adaptations, such as error correction (Rabbitt, 1966; Rabbitt & Vyas, 1981; Steinhauser & Yeung, 2010; Ullsperger, Fischer et al., 2014). Despite these consistent findings, its functional role is still under debate. It is unclear what process the Ne/ERN is based on and how it contributes to other error-related phenomena. This is why the present thesis aims to clarify further the role of the Ne/ERN in error processing and the interaction between early error processes and later events. More specifically, I investigate whether the Ne/ERN is influenced by increasing response conflict and how conscious error detection, error-related pupil dilation, and heart rate deceleration are affected by the absence of the Ne/ERN. Previous studies have not yet been able to establish a clear relationship. Clarifying these aspects, thus, helps to advance the knowledge of the cognitive mechanisms of error processing. Moreover, it enables a better understanding of psychiatric disorders, such as obsessive-compulsive disorder or generalized anxiety disorder, which are associated with a larger Ne/ERN and autonomic symptoms as well (e.g., Alvares et al., 2016; Gehring et al., 2000; Hajcak, 2012; Hu et al., 2016; Weinberg & Hajcak, 2010; Hanna et al., 2016; Luu et al., 2000). Methodologically, behavioral, EEG, and physiological measures are collected to study conflict and error processing using adapted Eriksen flanker tasks.

Study 1. My investigation begins with the question of whether the Ne/ERN and the N2 are further decreased or increased when more than one competing incorrect response is activated. Computational models suggest that additional activated responses should enhance the degree of conflict (Botvinick et al., 2001; Yeung et al., 2004). Mathematically, this enhanced conflict level, or the Hopfield energy (Hopfield, 1982), can be derived from a formula defining conflict as a weighted sum over the number of response units. Each additional response should,

therefore, increase the overall amount of conflict. We used a color-based Eriksen flanker paradigm with only incongruent flankers to test this. These flankers activate one (same-flankers condition) or two additional competing responses (different-flankers condition) compared to the central target. My new approach allows me to empirically investigate the pattern of the N2 and Ne/ERN between the same- and different-flanker conditions when more than one competing response is activated. The processing of the target stimulus before and after the response should be more impaired in the different-flankers condition. The N2 should be increased in the different-flankers condition because the target stimulus competes with two instead of one additional response option, which enhances the pre-response conflict. The Ne/ERN, in turn, should be reduced because the activation of the correct response after the erroneous decision is more strongly impaired by two rather than just one additional flanker stimulus, which reduces the post-response conflict.

In *Study 2*, I addressed the question of whether conscious error detection is still possible in the absence of the Ne/ERN. Although Di Gregorio et al. (2018) showed that a Pe is possible without the Ne/ERN and the Pe is assumed to indicate error awareness (O'Connell et al., 2007; Dhar et al., 2011; Di Gregorio et al., 2018; Endrass et al., 2007; Hughes & Yeung, 2011; Murphy et al., 2012; Wessel et al., 2011), it cannot be concluded from this relation that conscious error detection is possible in the absence of the Ne/ERN. The Pe and error awareness can occur together but can be partially or entirely independent of each other. In *Study 2*, I used the target-masking paradigm of Di Gregorio et al. (2018), in which the Ne/ERN and the Pe can be dissociated. As mentioned above, in the invisible-target condition, the target was replaced by the mask from the beginning, so processing the correct response was impossible in this condition. However, errors could still be processed because participants knew that responding to flankers was always an error. After the primary task, in which participants responded to the central target letter, they had to evaluate their performance in a second task (correct, error, unsure).

In order to gain further insight into the role of ANS activity during error processing, two additional physiological parameters, heart rate deceleration, and pupil dilation, were investigated in *Study 3*. I aimed to clarify whether both parameters occur in the absence of the Ne/ERN and, thus, whether they relate to early or late error processes. In contrast to the Ne/ERN, error-related pupil dilation and heart rate deceleration need more time to develop, i.e., they occur after the time course of the Ne/ERN and the Pe. Using the same target-masking paradigm as in *Study 2*, I was able to examine whether and how both parameters are related to the Ne/ERN. In particular, I investigated whether error-related heart rate deceleration and pupil dilation can also occur in the absence of the Ne/ERN.

Study 1: Effects of the number of competing responses on neural signatures of pre- and post-response conflict

By Julia Dumsky, Martin Maier and Marco Steinhauser

Abstract

The optimization of human performance requires the continuous monitoring of behavioral conflicts. According to conflict monitoring theory, the dorsal anterior cingulate cortex registers response conflict which is reflected by two electrophysiological signatures, the N2 and the Ne/ERN. The theory assumes that, if a stimulus activates an incorrect response that competes with the correct response, pre-response conflict on correct trials (reflected by the N2) is enhanced but post-response conflict on error trials (reflected by the Ne/ERN) is reduced. Here, we asked whether response conflict depends on the number of competing incorrect responses activated by a stimulus, that is, whether the N2 is further enhanced and the Ne/ERN is further reduced if two incorrect responses are activated as compared to one. To this end, we used a modified flanker paradigm, in which the two flankers were associated either with the same incorrect response or with different incorrect responses. Our results indicate an increased N2 on correct trials and a reduced Ne/ERN on error trials in the latter as compared to the former condition. These results confirm central predictions of conflict monitoring theory and demonstrate that response conflict is directly related to the number of competing incorrect responses.

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Effects of the number of competing responses on neural signatures of pre- and post-response conflict☆

Julia Dumsky *, Martin E. Maier, Marco Steinhauser

Department of Psychology, Catholic University of Eichstätt-Ingolstadt, Ostenstraße 27, 85072 Eichstätt, Germany

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ABSTRACT

The optimization of human performance requires the continuous monitoring of behavioral conflicts. According to conflict monitoring theory, the dorsal anterior cingulate cortex registers response conflict which is reflected by two electrophysiological signatures, the N2 and the Ne/ERN. The theory assumes that, if a stimulus activates an incorrect response that competes with the correct response, pre-response conflict on correct trials (reflected by the N2) is enhanced but post-response conflict on error trials (reflected by the Ne/ERN) is reduced. Here, we asked whether response conflict depends on the number of competing incorrect responses activated by a stimulus, that is, whether the N2 is further enhanced and the Ne/ERN is further reduced if two incorrect responses are activated as compared to one. To this end, we used a modified flanker paradigm, in which the two flankers were associated either with the same incorrect response or with different incorrect responses. Our results indicate an increased N2 on correct trials and a reduced Ne/ERN on error trials in the latter as compared to the former condition. These results confirm central predictions of conflict monitoring theory and demonstrate that response conflict is directly related to the number of competing incorrect responses.

1. Introduction

Detecting conflicts in ongoing behavior is crucial for the optimization of human performance. It is frequently assumed that response conflict indicates the necessity to adjust cognitive control, and that conflict can occur at different stages of task processing, both before and after overt behavior (Botvinick et al., 2001; Yeung et al., 2004). To identify correlates of response conflict in neural activity, studies typically used conflict paradigms, such as the Eriksen flanker task (Eriksen & Eriksen, 1974), the Stroop task (Stroop, 1935) or the Simon task (Simon & Small, 1969). In these paradigms, a target or target feature has to be classified while a distractor or distractor feature is present that is also associated with a response. Correlates of response conflict can be measured by contrasting conditions in which the distractor activates either a competing incorrect response or the same correct response as the target. While this standard scenario defines response conflict as a competition between two responses, it is still unclear whether conflict can be further enhanced if a stimulus activates more than one incorrect response, and thus, whether response conflict varies with the number of activated competing responses. In the present study, we address this question by considering conflict measures in event-related potentials (ERPs) that have previously been identified as correlates of conflict processing.

Conflict monitoring theory is an influential framework that explains how conflict is registered in the brain and how this affects ongoing attention and behavior (Botvinick et al., 2001; Yeung et al., 2004). According to this theory, conflict arises when multiple competing responses are activated simultaneously. It assumes that the dorsal anterior cingulate cortex (dACC) detects this response conflict and generates a control signal which then allows for the adjustment of cognitive control in other brain areas. A frequently used conflict task to study response conflict is the Eriksen flanker task (Eriksen & Eriksen, 1974). The stimuli in this task consist of a central target (e.g., the letters H or S) that has to be classified by manual key presses and that is surrounded by irrelevant flankers. Target and flankers can activate the same response (congruent stimuli; e. g., *HHHHH*) or different responses (incongruent stimuli; e. g., *HHSHH*). Incongruent stimuli lead to response conflict, which leads to higher error rates and response times (RTs; e.g., Jost et al., 2022; Coles et al., 1985; Danielmeier et al., 2009; Gratton et al., 1988).

An ERP correlate of response conflict in the flanker task is the N2.

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* Correspondence to: Katholische Universität Eichstätt-Ingolstadt, Ostenstraße 27, 85072 Eichstätt, Germany.
E-mail address: julia.dumsky@stud.ku.de (J. Dumsky).

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The N2 is a negative deflection emerging between 200 and 300 ms after stimulus onset at frontocentral electrodes (Jost et al., 2022; Yeung et al., 2004; Yeung & Cohen, 2006; Heil et al., 2000; Kopp et al., 1996; Liotti et al., 2000). According to conflict monitoring theory, the N2 is a signature of a *pre-response conflict* (Yeung et al., 2004; Yeung and Cohen, 2006), i.e., a conflict that occurs because a competing incorrect response tendency is activated simultaneously with the correct response tendency prior to the response (Yeung and Cohen, 2006). Another ERP component related to response conflict is the error negativity (Ne; Falkenstein et al., 1990) or error-related negativity (ERN; Gehring et al., 1993). The Ne/ERN is a negative deflection that occurs within 100 ms after errors relative to correct responses. Similar to the N2, it emerges at frontocentral electrodes (Carter et al., 1998; Dehaene et al., 1994; Ullsperger & Von Cramon, 2001). While the N2 is assumed to reflect a pre-response conflict on correct trials, the Ne/ERN is viewed to be a signature of *post-response conflict* on error trials (Yeung et al., 2004; Yeung & Cohen, 2006). After errors, continued stimulus processing eventually leads to the activation of the correct response, even if no overt correction occurs (Beatty et al., 2021). As a consequence, a response conflict emerges between this corrective response tendency and other activated responses including the still active erroneous response (Yeung et al., 2004).

According to conflict monitoring theory, the N2 and the Ne/ERN are both considered to reflect the same phenomenon - response conflict, which receives support from studies showing that the neural generators of both ERP components lie in the dACC (Van Veen & Carter, 2002; Yeung et al., 2004). However, the two components reflect different stages of task processing and are affected by task characteristics in different ways (Yeung & Cohen, 2006). Whatever activates incorrect responses that compete with the correct response leads to higher levels of pre-response conflict on correct trials and therefore leads to a higher N2 (Yeung & Cohen, 2006). This explains why the N2 is typically higher on incongruent than congruent, correct trials (Danielmeier et al., 2009; Folstein & Van Petten, 2008; Forster & Pavone, 2008; Heil et al., 2000; Jost et al., 2022; Kopp et al., 1996; Liotti et al., 2000; Yeung et al., 2004), and this N2 conflict effect increases the more the processing of incongruent flankers is facilitated (e.g., Danielmeier et al., 2009). In contrast, whatever facilitates the activation of the correct response (and thus the corrective tendency after errors) increases the post-response conflict and leads to a higher Ne/ERN (Yeung & Cohen, 2006). This explains why the Ne/ERN is typically higher on congruent (or neutral) trials than on incongruent trials (e.g., Forster & Pavone, 2008; Maier et al., 2011; Scheffers & Coles, 2000; for simulated data, see Yeung et al., 2004).* This opposing pattern in the N2 and Ne/ERN – variables that increase the N2 typically lead to a reduced Ne/ERN and vice versa – is a hallmark of conflict monitoring theory and has been found across several studies (Danielmeier et al., 2009; Pailing & Segalowitz, 2004a; Yeung et al., 2004).

All the above-mentioned studies have in common that response conflict was manipulated across conditions in which the flanker activated either one competing incorrect response (incongruent trials) or no competing incorrect response (congruent or neutral trials). The question arises whether even higher levels of response conflict emerge if multiple competing incorrect responses are activated. The prediction that the level of response conflict increases with the number of activated response can be directly derived from computational models of conflict monitoring theory (Botvinick et al., 2001; Yeung et al., 2004). In these connectionist models, conflict is defined as the so-called Hopfield energy (Hopfield, 1982) over the set of response units,

$$E = -\sum_i \sum_j a_i a_j w_{ij},$$

where i and j are indexed over all response units, a represents the activation of a specific unit, and w indicates the (negative) weight connecting two units. With two responses, this formula simplifies to the product of the two response activations (multiplied with a constant value). With more than two responses, however, conflict can be characterized as the sum of the products of each possible pair of response activations. This implies that each additional response with a non-zero response activation adds additional components to the overall conflict, thus increasing the potentially observable conflict in a task.[2] Whether a higher level of response conflict is actually obtained depends on how strongly the responses are activated and on the dynamics of mutual inhibition. However, conflict monitoring theory predicts that increasing the number of competing responses leads to a *potentially* higher response conflict. While this constitutes a core feature of conflict in this theory, it has never been empirically tested in a conflict paradigm like the flanker task.

First evidence for a relation between the number of activated competing responses and response conflict comes from a study by Maier et al. (2010). In their study, an Eriksen flanker task with eight possible letters was used. In three groups of participants, these eight letters were mapped to either two responses (4:1 mapping), four responses (2:1 mapping), or eight responses (1:1 mapping). This manipulation led to a decreasing Ne/ERN with increasing response-set size. Although each single incongruent stimulus could still activate only one incorrect response, an increasing number of pre-activated responses might have led to a decreasing post-response conflict because more responses competed with the corrective tendency after an error. This is in line with conflict monitoring theory and points to the possibility that response conflict indeed varies with the number of competing incorrect responses.

While Maier et al. (2010) reported only Ne/ERN data, the N2 was in the focus of another study by Forster et al. (2011). These authors used a standard two-choice version of the Eriksen flanker task but manipulated conflict by varying the degree of incongruency parametrically, i.e., stepwise from completely congruent to completely incongruent. Stimuli consisted of a target and three flankers on each side, and incongruency was manipulated by varying the ratio of incongruent and congruent flankers within a single stimulus (e.g., on each side, a stimulus could contain two incongruent and one congruent flankers, HHSSSHH, or one incongruent and two congruent flankers, HSSSSSH). Forster et al. (2011) found that the N2 conflict effect increased with increasing incongruency, which again is in line with conflict monitoring theory. The higher the degree of incongruency, the higher the pre-response conflict and thus the N2 conflict effect. Although the experiment of Forster et al. (2011) involved only two responses, it demonstrates that response conflict is not an all-or-nothing phenomenon but gradually reflects to which extent competing responses are activated by the stimulus. Taken together, both Maier et al. (2010) and Forster et al. (2011) suggest that pre-response conflict as indexed by the N2 and post-response conflict as indexed by the Ne/ERN might depend on how many and how strongly competing responses are activated. However, Maier et al. (2010) considered only the Ne/ERN and manipulated the number of relevant responses instead of responses activated by the stimulus. In contrast, Forster et al. (2011) considered only the N2 and investigated conditions in which the same two responses were activated by the flankers to different degrees. Thus, it remains unclear whether response conflict can

[1] Congruent error trials are less frequent than incongruent error trials. A recent study has shown that peak-to-peak measures but not mean amplitude measures are biased towards larger Ne/ERNs with fewer error trials (Fischer et al., 2017). Accordingly, imbalanced trial numbers can account for Ne/ERN differences between congruent and incongruent trials when peak-to-peak measures are used (Scheffers & Coles, 2000) but not when mean amplitudes are used to quantify the Ne/ERN (Forster & Pavone, 2008; Maier et al., 2011).

[2] With two response units with activations a_1 and a_2, conflict is proportional to $a_1 a_2 w_{12}$; with three response units with activations a_1, a_2, and a_3, conflict is proportional to $a_1 a_2 w_{12} + a_2 a_3 w_{23} + a_1 a_3 w_{13}$; and so forth.

be further enhanced if the stimulus activates more than one competing incorrect response.

The present study aims to fill this gap by explicitly addressing the question whether activating further competing incorrect responses additionally increase pre-response conflict on correct trials and decrease post-response conflict on error trials as reflected by the N2 and Ne/ERN, respectively. If such a result were observed, this would confirm another central prediction of conflict monitoring theory and would validate its formal definition of conflict as Hopfield energy. To achieve this, we used a four-choice color flanker task, in which only incongruent trials could occur. Participants had to classify the color of a central square while ignoring two adjacent flanker squares. The number of activated competing responses was manipulated by introducing conditions in which both flankers were shown in the same incongruent color and thus activated the same incorrect response (e.g., red green red; *same-flankers condition*) or in which each flanker was shown in a different incongruent color and thus activated different incorrect responses (e.g., red green blue; *different-flankers condition*). Based on the assumptions of conflict monitoring theory, we expected the different-flankers condition to generate higher pre-response conflict on correct trials, and thus a higher N2, than the same-flankers condition. This results because the stimuli in the different-flankers condition activate more responses that compete with the correct response than the same-flanker condition. For the Ne/ERN, we expected the opposite pattern. We expected the different-flankers condition to produce lower post-response conflict on error trials, and thus a less pronounced Ne/ERN, than the same-flankers condition, because the corrective response tendency after errors should be more strongly impaired if the stimulus activates two competing incorrect responses as compared to one competing incorrect response. Finally, we expected slower RTs and higher error rates for the different-flankers condition, because higher degrees of pre-response conflict typically increase RTs and error rates.

2. Method

2.1. Participants

Twenty-seven participants (20 females and four left-handed) recruited at the Catholic University of Eichstätt-Ingolstadt participated in the study. This lies in the range of sample sizes used in previous studies that employed this task (Maier & Steinhauser, 2016; Maier et al., 2019). The participants had an age between 18 and 36 years ($M = 22.5$; $SD = 3.9$), had normal or corrected-to-normal vision, and either received course credits or 11 € per hour for their participation. The study was approved by the ethics committee of the university, and informed consent was obtained from all participants.

2.2. Task and stimuli

Stimulus presentation and response registration was controlled by Presentation software (Neurobehavioral Systems, Albany, CA). A 21-inch color monitor was used for stimulus presentation. The task was a four-choice color version of the Eriksen flanker paradigm (e.g., Maier et al., 2019). Participants had to respond to the color of a target while they had to ignore adjacent flanker stimuli. The stimuli consisted of three horizontally arranged squares, a centrally presented target square and a left and right flanker square. Each square was displayed in one of four colors (red, green, blue, yellow) and had a side length of 0.74° at a viewing distance of about 70 cm. As shown in Fig. 1, the target color always differed from the color of the two flankers. The two flankers had either the same color (same-flankers condition; e.g., blue yellow blue) or different colors (different-flankers condition; e.g., blue yellow red). All possible color combinations were realized for each condition, resulting in 12 possible stimuli in the same-flankers condition and 24 possible combinations in the different-flankers condition. Each of the four colors was assigned to one of four keys ('W', 'S', 'L', 'P') of a standard computer keyboard, which had to be pressed with the left middle finger, the left index finger, the right middle finger, or the right index finger, respectively. The mappings between color and response were fully counterbalanced across participants for the first 24 participants. In three additional participants, a randomly drawn color-response mapping was used.

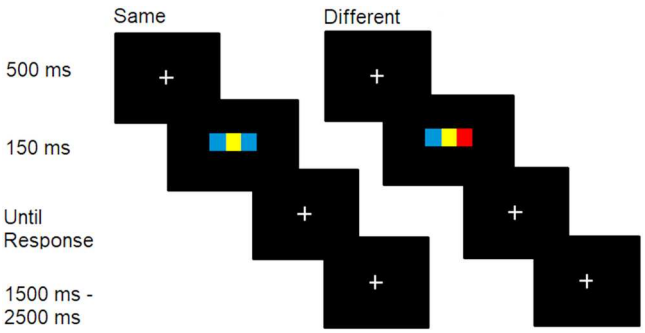

Fig. 1. Typical trial sequences, where participants had to respond to the color of centrally presented target squares and to ignore adjacent flanker squares. Left: Same-flankers condition. Right: Different-flankers condition.

At the beginning of each trial, a black screen with a central fixation cross was shown for 500 ms. The duration of the subsequent stimulus array was 150 ms. After a response key was pressed, an interval with a randomized duration between 1500 ms and 2500 ms was presented, and this interval was restarted whenever an additional response (e.g., a spontaneous error correction) was given before the interval was over. The fixation cross remained on the screen at all times and was turned off only during presentation of the stimulus.

Each block consisted of 72 randomized trials with 36 trials belonging to each condition. Whereas each of the same-flankers stimuli were presented three times within each block, different-flankers stimuli were selected according to the following procedure: The 24 stimuli in this condition were randomly divided into two subsamples, such that the two mirrored variants of each color combination were assigned to different subsamples (i.e., red-yellow-blue in one subsample, blue-yellow-red in the other subsample). The first subsample was assigned to odd block numbers, the second subsample was assigned to even block numbers. As for the same-flankers stimuli, the respective subsample for the different-flankers stimuli were presented three times in each block. This ensures that within each block, target colors, flanker colors as well as their combination occur equally frequent within each block. Three randomly drawn practice trials preceded the 72 trials of each block, but were later excluded from data analysis.

The experiment consisted of two sessions distributed across two days. On the first day, the participants practiced eight blocks of the flanker task. On the second day, they first performed one randomly drawn practice block before 12 test blocks (duration about 1–1.5 h) were administered resulting in 864 trials for data analysis. Participants were able to take breaks between blocks if they desired. To obtain an appropriate number of error trials for the analysis, an oral speed instruction between blocks was given depending on the number of errors in each block. Participants were instructed to respond faster if the error rate in the preceding block was below 15 % and were instructed to respond slower if the error rate was above 20 % Fig. 1.

2.3. Psychophysiological recording

The electroencephalogram (EEG) was recorded only for the test blocks using a BIOSEMI Active-Two system (BioSemi, Amsterdam, The Netherlands) with 64 Ag-AgCl electrodes from channels Fp1, AF7, AF3, F1, F3, F5, F7, F17, FC5, FC3, FC1, C1, C3, C5, T7, TP7, CP5, CP3, CP1, P1, P3, P5, P7, P9, PO7, PO3, O1, Iz, Oz, POz, Pz, CPz, Fpz, Fp2, AF8, AF4, AFz, Fz, F2, F4, F6, F8, FT8, FC6, FC4, FC2, FCz, Cz, C2, C4, C6, T8,

TP8, CP6, CP4, CP2, P2, P4, P6, P8, P10, PO8, PO4, O2. Furthermore, the left and right mastoids were additional channels. As reference and ground electrodes, the CMS (Common Mode Sense) and DRL were used. The vertical and horizontal electrooculogram (EOG) was recorded with electrodes above and below the right eye as well as on the outer canthi of both eyes. The sampling rate of EEG and EOG data was 512 Hz, and EEG data were referenced to the average of both mastoids and band-pass filtered between 0.1 and 40 Hz.

2.4. Data analysis

2.4.1. Behavioral data

Trials with RTs deviating more than three standard deviations from the mean of each condition were excluded from RT analyses (1.1 %). RTs were analyzed using a repeated-measurement ANOVA with the variables flanker condition (same-flankers vs. different-flankers) and correctness (error vs. correct). Error rates were arcsine-transformed prior to statistical analysis (Winer et al., 1971), and were compared between flanker conditions using a paired t-test.

2.4.2. ERP data

ERP data were analyzed with custom routines based on EEGLAB v14.1.1 (Delorme & Makeig, 2004) and implemented in MATLAB R2017b (The Mathworks, Natic, MA, USA). Response-locked ERP data were derived by extracting epochs from 400 ms before and 1000 ms after the response, and baseline-corrected using a baseline from 150 ms to 50 ms before the response (taking into account that the Ne/ERN typically starts before the response; e.g., Steinhauser & Yeung, 2010). Stimulus-locked ERP data were obtained by extracting epochs from 200 ms before and 800 ms after the stimulus, and a baseline between 100 ms and 0 ms relative to the stimulus was applied. Electrodes were interpolated using spherical spline interpolation if the joint probability criterion (threshold 5) or the kurtosis criterion (threshold 5) was met in EEGLAB's channel rejection routine (pop_rejchan). To remove artifacts, response- as well as stimulus-locked epochs were excluded whenever a) activity exceeded a threshold of ± 200 μV, or b) whose joint probability deviated more than 5 SDs from the epoch mean in EEGLAB's joint probability function (pop_jointprob). Artifact rejection was based on all channels except AF1, Fp1, Fpz, Fp2, and AF8 to ignore blink artifacts which were corrected at a later stage. In this way, an average of 19.4 % of trials (SE = 3.8%) of the response-locked data and an average of 14.5% of trials (SE = 3.5%) of the stimulus-locked data were rejected. Next, any trial that contained a second response (e.g., because of spontaneous error corrections) were removed, because the preparation of further responses can distort the post-response ERP waveform. ERP data were then subjected to a temporal independent component analysis (ICA; Makeig et al., 1996) applying the infomax algorithm (Bell & Sejnowski, 1995). Using CORRMAP v1.02 (Viola et al., 2009), independent components (ICs) representing blink artifacts were automatically identified and removed from the data by means of an inverse matrix multiplication. Finally, averaged ERP data were calculated for error and correct trials in both conditions, separately for each participant. For the response-locked ERP data, an average of 736.5 trials (SE = 25.5) were entered into the ERP analysis for each participant (correct, same-flankers: 293.1, SE = 13.1; error, same-flankers: 57.0, SE = 4.0; correct, different-flankers: 291.1, SE = 13.7; error-different-flankers: 55.3, SE = 3.3). For the stimulus-locked data, an average of 828.6 trials (SE = 3.4) were entered into the ERP analysis for each participant (correct, same-flankers: 309.3, SE = 11.7; error, same-flankers: 61.3, SE = 3.6; correct, different-flankers: 308.3, SE = 12.2; error, different-flankers: 59.6, SE = 3.1).

The N2 for correct trials and error trials was quantified as the mean amplitude in the time interval between 200 ms and 300 ms (e.g., Jost et al., 2022; Kopp et al., 1996) after stimulus onset at electrode FCz. Same-flankers and different-flankers conditions were compared using a paired t-test. To investigate whether the conditions also differ with respect to peak amplitude and peak latency, we additionally determined the N2 peak as the lowest amplitude in the interval between 250 and 350 ms of each condition and analyzed the resulting amplitude and latency. This interval was based on the approximate time point of the peak identified through visual inspection. The Ne/ERN was quantified as the mean amplitude in the time interval between 0 ms and 100 ms after the response (e.g., Di Gregorio et al., 2018; Maier et al., 2008) at electrode FCz, and subjected to a two-way repeated measures ANOVA with the variables flanker condition (same-flankers vs. different-flankers) and correctness (error vs. correct). Paired t-tests were then applied to test the difference between errors and correct responses separately for each condition.

3. Results

3.1. Behavioral data

Table 1 shows error rates and mean RTs in all conditions. In the error rates, there was no significant difference between the same-flankers condition and the different-flankers condition, $t(26) = 0.15$, $p = .885$, $d = 0.028$. In mean RTs, the ANOVA revealed no significant main effects, neither for flanker condition, $F(1, 26) = 0.021$, $p = .887$, $\eta_p^2 = 0.001$, nor for correctness, $F(1, 26) = 1.375$, $p = .252$, $\eta_p^2 = 0.05$, and no significant interaction, $F(1, 26) = 0.005$, $p = .944$, $\eta_p^2 = 0.000$.

3.2. ERP data

N2. Fig. 2AB shows the stimulus-locked waveforms and the difference wave between the same-flankers and different-flankers conditions together with the corresponding topographies for correct trials (Figs. 2EF). The difference wave shows the expected larger negativity for the different-flankers condition and the statistical analysis confirms that the mean amplitudes are significantly larger in the different-flankers condition ($M = -4.94$ μV, $SE = 0.17$ μV) than in the same-flankers condition ($M = -4.17$ μV, $SE = 0.17$ μV) between 200 and 300 ms, $t(26) = 3.25$, $p = .003$, $d = 0.625$. However, although the conflict effect in this time interval is in accordance with the literature (Jost et al., 2022; Yeung et al., 2004; Yeung & Cohen, 2006; Heil et al., 2000; Kopp et al., 1996; Liotti et al., 2000), Fig. 2A shows that the peak of the N2 occurs later at around 300 ms. When increasing the time window for the mean amplitude analysis to 200–350 ms, we still obtain a significant difference, $t(26) = 2.276$, $p = .031$, $d = 0.438$. However, to investigate whether the conflict effect also occurs at the N2 peak, we additionally analyzed peak amplitudes and latencies. The peak amplitude revealed no significant difference (same-flankers: −9.49 μV, $SE = 0.25$ μV; different-flankers: −9.9 μV, $SE = 0.25$ μV), $t(26) = 1.18$, $p = .247$, $d = 0.227$. The same holds for the peak latencies (same-flankers: 300 ms, SE = 2 ms; different-flankers: 294 ms, SE = 2 ms), $t(26) = 1.7$, $p = .101$, $d = 0.327$. In sum, we obtained a N2 that was larger in the different-flankers condition than in the same-flankers condition when quantified as mean amplitude in the typical time interval between 200 and 300 ms, whereas the N2 peak amplitudes and latencies did not differ significantly between both conditions.

For completeness, we additionally investigated whether this effect is

Table 1
Behavioral results.

	Error rates in %	
	Same-flankers	Different-flankers
	11.5 (± 1.5)	11.5 (± 1.1)
	Response times in ms	
	Same-flankers	Different-flankers
Correct	534 (± 11)	532 (± 11)
Error	560 (± 33)	559 (± 23)

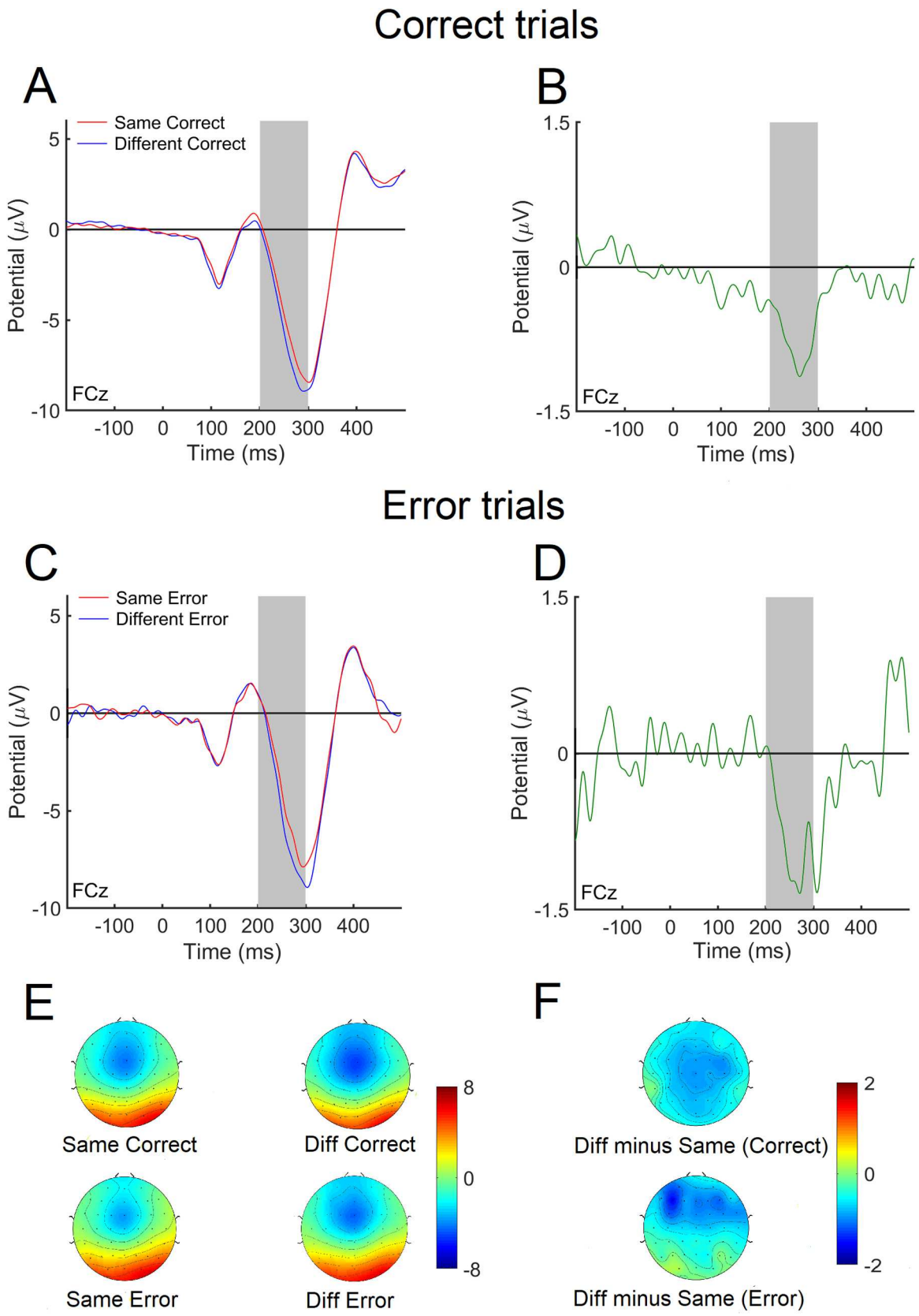

Fig. 2. A: Stimulus-locked waveforms for correct trials in the same-flankers and different-flankers conditions at electrode FCz. B: Difference wave for correct trials representing the different-flankers condition minus the same-flankers condition. CD: Stimulus-locked waveforms and difference wave for error trials. E: Topographies of each condition from the time window between 200 and 300 ms. F: Topographies of the difference waves. The time point zero indicates the stimulus onset. Gray areas indicate the time range in which mean amplitudes were analyzed.

also present on error trials. Although the stimulus-locked waveforms and the difference wave in Figure 2CD reveal a very similar pattern as for correct trials with a more negative mean amplitude for the different-flanker condition (−4.25 μV, $SE = 0.24$ μV) than for the same-flanker condition (−3.50 μV, $SE = 0.24$ μV), this difference failed to reach significance, $t(26) = 1.824$, $p = .080$, $d = 0.351$ (and the same holds for the larger time window between 200 and 350 ms, $t(26) = 1.753$, $p = .090$, $d = 0.337$). This failure to find a similarly robust effect for error trials might be due to the smaller trial numbers for errors but could also reflect that the dynamics of response activation differ between correct trials and errors.

Ne/ERN. Fig. 3 shows the response-locked waveforms and the difference waves between the correct and error trials (Fig. 3AB) together with their topography (Figs. 3CD) separately for the same-flankers and different-flankers conditions. The difference waves show a typical Ne/ERN which is more negative in the same-flankers condition than in the different-flankers condition. The corresponding ANOVA revealed no main effect of flanker condition, $F(1, 26) = 2.39$, $p = .134$, $\eta_p^2 = 0.084$, but a significant main effect of correctness, $F(1, 26) = 84.6$, $p < .001$, $\eta_p^2 = 0.765$, and a significant interaction, $F(1, 26) = 7.32$, $p = .012$, $\eta_p^2 = 0.22$. The difference between correct and error trials was more negative for the same-flankers condition ($M = -7.26$ μV, $SE = 0.57$ μV), $t(26) = 8.98$, $p < .001$, $d = 1.728$, than for the different-flankers condition ($M = -5.85$ μV, $SE = 0.5$ μV), $t(26) = 8.29$, $p < .001$, $d = 1.595$. In sum, we observed the expected increased Ne/ERN for the same-flankers condition.

4. Discussion

Previous research has shown that pre-response conflict on correct trials is increased and post-response conflict on error trials is decreased if a stimulus activates a competing incorrect response (in addition to the correct response) as compared to if only the correct response is activated (e.g., Yeung et al., 2004). Here, we aimed to investigate whether these effects become more pronounced if two competing incorrect responses are activated. Such a prediction can be derived from the formal definition of conflict as Hopfield energy in conflict monitoring theory, which implies that the potential strength of conflict increases with the number of activated competing responses. Our approach was to compare a same-flankers condition in which the two incongruent flankers activated the same incorrect response with a different-flankers condition in which the flankers activated two different incorrect responses. The N2 and Ne/ERN were used as measures of pre-response conflict on correct trials and post-response conflict on error trials, respectively.

As expected, the N2 on correct trials was significantly enhanced in the different-flankers condition relative to the same-flankers condition, which can be explained in terms of conflict monitoring theory (Botvinick et al., 2001; Yeung et al., 2004). Two different incongruent flankers imply that two activated incorrect responses compete with the correct response. This leads to a higher pre-response conflict as compared to the same-flankers condition, in which only one incongruent response is activated. The difference between the different-flankers and the same-flankers condition (i.e., the conflict effect) occurred prior to the N2 peak, whereas neither the peak amplitude nor the peak latency of the N2 differed between the two conditions. This is consistent with previous

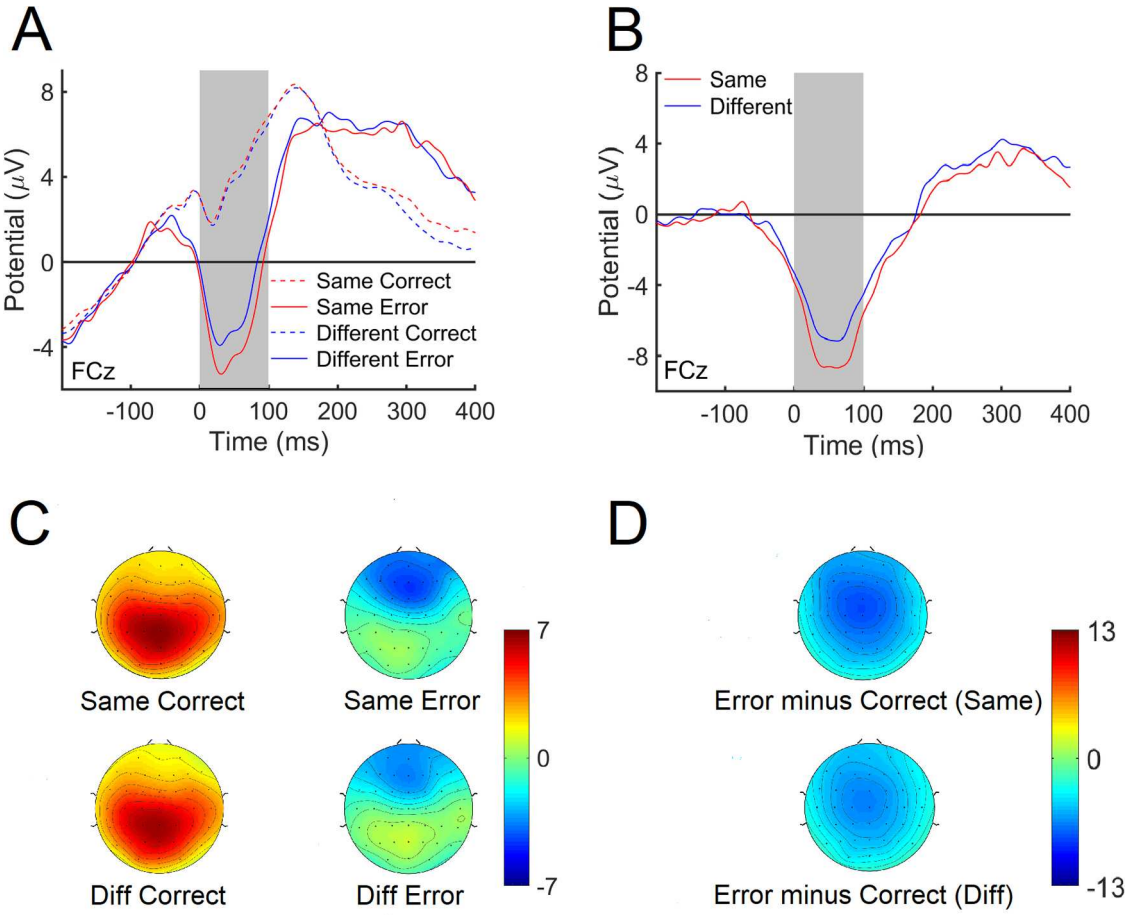

Fig. 3. A: Response-locked waveforms for correct and error trials in the same-flankers and different-flankers conditions at electrode FCz. B: Difference waves for both conditions representing errors minus correct trials. C: Topographies of each condition from the window between 200 and 300 ms. D: Topographies of the difference waves. The time point zero indicates the response. Gray areas indicate the time range in which mean amplitudes were analyzed.

studies in which effects due to conflict manipulations extended before or after the N2 peak (Fong et al., 2018; Grützmann et al., 2014; Van Veen & Carter, 2002), and might indicate that the N2 and these conflict effects reflect distinct components of the stimulus-locked ERP.

Also the Ne/ERN showed the expected pattern with a smaller Ne/ERN in the different-flankers condition as compared to the same-flankers condition. According to conflict monitoring theory, this indicates that the corrective tendency after errors is impaired if the correct response competes with two incorrect responses as compared to if it competes with one incorrect response only. The resulting reduced post-response conflict on error trials in the different-flankers condition manifests in a smaller Ne/ERN. In sum, these data are fully in line with conflict monitoring theory and demonstrate that pre- and post-response conflict vary with the number of competing responses. Our results also show the frequently observed opposing relationship between pre- and post-response conflict, and thus, N2 and Ne/ERN (e.g., Danielmeier et al., 2009; Yeung et al., 2004).

Surprisingly, task performance was unaffected by our conflict manipulation and did not differ between the same-flankers and different-flankers conditions. Typically, RTs and error rates increase with higher levels of pre-response conflict as shown in many previous studies (e.g., Asanowicz et al., 2021; Danielmeier et al., 2009; Folstein & Van Petten, 2008; Forster & Pavone, 2008; Gajewski et al., 2008; Heil et al., 2000; Jost et al., 2022; Yeung et al., 2004). The absence of a behavioral conflict effect in our study could reflect that conflict detection (as indexed by the N2) could have led to the recruitment of control processes which prevented that conflict led to slower or more error-prone responding. Indeed, Scherbaum et al. (2011) demonstrated conflict resolution associated with stronger target enhancement and distractor suppression within a trial in which conflict occurred (see also Ridderinkhof, 2002; Ridderinkhof et al., 2004). Control processes like these might have been even more involved in our paradigm in which only incongruent stimuli were used. Previous studies found decreased error rates and RTs on incongruent trials if the frequency of incongruent stimuli was high (Bartholow et al., 2005; Corballis & Gratton, 2003; Grützmann et al., 2014; Wendt et al., 2008; Wendt & Luna-Rodriguez, 2009). It is thus conceivable that conflict resolution in our paradigm was strong enough to suppress the conflict effect on RTs and error rates, whereas neural correlates of pre- and post-response conflict were sensitive enough to detect differences between conflict levels associated with our manipulation.

4.1. Alternative accounts of the N2 and Ne/ERN

Conflict monitoring theory attributes the N2 and the Ne/ERN to a common underlying mechanism. However, each component alone has been explained by alternative accounts. Although it was not the goal of this study to empirically test between alternative theoretical explanations, we discuss our results in the light of some of these alternative accounts. The earliest account of the Ne/ERN assumes that this component is directly related to error detection and that its amplitude reflects a mismatch between the executed response and the correct response (Bernstein et al., 1995; Falkenstein et al., 1990). Within this framework, differences in the Ne/ERN amplitude are often explained in terms of differences in the representation of the correct response. Any uncertainty about which response is correct (e.g., by increasing task difficulty) directly leads to reduced Ne/ERN amplitudes (Pailing & Segalowitz, 2004b; Scheffers & Coles, 2000). Because conflict monitoring also assumes that correct response activation after the error determines post-response conflict, and thus the Ne/ERN, both theories often make very similar predictions. For instance, the described error-detection theory could explain the present results by assuming that the representation of the correct response at the time of the response is impaired in the different-flankers condition because it previously received inhibition from two responses instead of one. However, the theory would have to explain why this is the case despite performance in the two conditions being similar.

Another class of theories has attributed the Ne/ERN to error expectancy. While reinforcement learning theory (Holroyd & Coles, 2002) suggests that the Ne/ERN reflects a reward prediction error conveyed by the dopaminergic system, the PRO model (predicted response-outcome model; Alexander & Brown, 2011) assumes that the Ne/ERN reflects a surprise signal. According to both frameworks, higher Ne/ERN amplitudes are elicited in conditions with less expected errors driven by lower experienced error rates. This negative relationship between Ne/ERN amplitudes and error rates has indeed been demonstrated by several studies (e.g., Falkenstein et al., 1995; Gehring et al., 1993; Jessup et al., 2010; Oliveira et al., 2007), while others failed to find such an effect or even showed the opposite (e.g., Maier & Steinhauser, 2013; Maier & Steinhauser, 2016). In an attempt to directly test between conflict monitoring and error expectancy as the origin of the Ne/ERN, Hughes and Yeung (2011) created two conditions with similar error rates but different levels of post-response conflict on error trials. They found that the Ne/ERN varied with post-response conflict under these conditions, thus contradicting the predictions of error expectancy accounts. A similar scenario was observed in the present study. Although we did not actively control for error rates, similar error rates but different Ne/ERN amplitudes were obtained in the same-flankers and different-flankers conditions. These results are in line with conflict monitoring theory but contradict the idea that the Ne/ERN reflects error expectancy. However, whereas the mentioned theoretical accounts (e.g., Holroyd & Coles, 2002) assume that outcome expectancy is learned from previously experienced error rates, it is also conceivable that error expectancy is influenced by the subjective difficulty of a task or condition. It is still an ongoing debate to which extent top-down effects based on expectancies can influence prediction errors in the dopaminergic system (e.g., Di Gregorio et al., 2019; Walsh & Anderson, 2011). Moreover, these expectations must vary from trial to trial depending on the nature of the stimulus to explain the present results.

Alternative accounts have also been proposed for the N2 conflict effect. One alternative theory is the selection-for-action account. Within this framework, the dACC is assumed to select the most appropriate response from multiple simultaneously activated response options to achieve goal-directed performance (Pardo et al., 1991; Posner et al., 1988; Posner & Dehaene, 1994; Turken & Swick, 1999). While early work demonstrated that conflict monitoring can better account for specific aspects of dACC function (Botvinick et al., 1999; Botvinick et al., 2001), the idea that the dACC is directly or indirectly involved in action selection is still popular (Akam et al., 2021; Banich, 2019; Holroyd & Yeung, 2012; Silton et al., 2010). Gajewski et al. (2008) linked the N2 conflict effect to such a selection process. They found that cueing an incorrect response increases the N2, and this effect becomes stronger if these invalid cues are infrequent. They assumed that selection becomes strengthened if the initially cued response plan must be revised because of violated response expectancies. This account is also in line with an inhibition account which explains the N2 conflict effect in terms of an inhibition process that suppresses inappropriate responses activated by a distractor (Falkenstein et al., 1999; Gajewski & Falkenstein, 2013; Jodo & Kayama, 1992; Kopp et al., 1996; Vuillier et al., 2016). Unfortunately, conflict monitoring and selection-for-action accounts often make very similar predictions (but see Donkers & Van Boxtel, 2004). N2 results like that of Gajewski et al. (2008) can often be interpreted in terms of both accounts. The finding that activating more competing responses implies a larger N2 as in the present study could reflect either the increased conflict or the increased need for selection due to this conflict. Recently, Asanowicz et al. (2021) argued that the N2 reflects conflict monitoring whereas the later occurring conflict-related theta is related to selection, a perspective that would integrate the two accounts.

Whereas many of the mentioned accounts can explain either the N2 or Ne/ERN results in the present study, the advantage of conflict monitoring theory is that it accounts for both the N2 and the Ne/ERN results, thus providing a parsimonious explanation for the full pattern in

our data. However, it is also possible that these accounts are not mutually exclusive and that either or both of these components reflect multiple mechanisms. A core assumption of conflict monitoring is that both components share a neural generator in the dACC (Yeung et al., 2004). In contrast, a source modeling study by Buzzell et al. (2017) revealed multiple sources for the Ne/ERN including dorsal structures like the dACC and more ventral structures like the orbitofrontal cortex. Crucially, the contribution of dorsal and ventral structures to the Ne/ERN showed a differential developmental course in adolescents, suggesting that they form dissociable networks with different functions, such as conflict monitoring and outcome valuation. Likewise, Beldzik et al. (2022) used combined EEG and fMRI to investigate the neural sources of theta oscillations related to errors and (pre-response) conflict. They found that error-related theta correlated with activity in the medial frontal cortex whereas conflict-related theta showed characteristics of an inhibitory mechanism. Although the N2 and conflict-related theta do not necessarily reflect the same mechanism (Asanowicz et al., 2021), it is possible that both are manifestations of a network that serves both, conflict monitoring and inhibition.

While our results are in accordance with predictions of conflict monitoring theory, there are still aspects in our study design that should be addressed in future studies to increase the generalizability of the present results. First, to obtain enough errors for the Ne/ERN analysis, participants were encouraged to respond more quickly whenever less than 15 % error were made in a block. The average error rate (11.5 %) was smaller than this criterion which shows that participants had difficulties to adhere to this instruction. Typical N2 studies analyzed only correct trials and thus do not use strong speed pressure, and it is known that speed pressure alters attentional orienting (Van der Lubbe et al., 2001) and conflict processing (Asanowicz et al., 2019). Future studies should thus replicate our N2 results without such an instruction. Second, we omitted congruent trials in our study to increase the trial numbers available for the Ne/ERN analysis. As already discussed, this might have led to the enhanced recruitment of control processes which possibly suppressed a behavioral effect in our data. Furthermore, congruent trials could serve as a valuable baseline against which the two incongruent conditions could be compared. Accordingly, it would be desirable to also include congruent trials in future studies.

5. Conclusion

The present study showed that pre-response conflict and post-response conflict, as measured by the N2 and Ne/ERN, vary with the number of activated competing responses. Stimuli activating more competing incorrect responses led to enhanced pre-response conflict on correct trials but reduced post-response conflict on error trials, which confirms core predictions of conflict monitoring theory and again demonstrates the close link between frontocentral ERPs and response conflict at different stages of task processing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

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Study 2: Error awareness can occur in the absence of an error-related negativity

By Julia Dumsky, Martin Maier, Francesco Di Gregorio and Marco Steinhauser

Abstract

Errors in choice tasks lead to a cascade of error-related brain activity in event-related potentials. While the error-related negativity (Ne/ERN) reflects an early error signal, the error positivity (Pe) has been attributed to the later emergence of error awareness. Previous work has shown that these two components can be dissociated using a target-masking paradigm. In this modified flanker paradigm, an invisible-target condition is realized in which errors are detectable even if the correct response is unknown. These errors have been shown to elicit a Pe without an Ne/ERN demonstrating the independence of the two underlying systems. Here, we employed this paradigm to ask whether also error awareness can emerge without an Ne/ERN. While performing the target-masking paradigm, participants provided metacognitive judgments to indicate whether an error has occurred on each trial (i.e., error signaling). The majority of participants were able to report detectable errors in the invisible-target condition. Crucially, this error signaling as well as a Pe was observable in the absence of an Ne/ERN. Our findings demonstrate that both error awareness (as indicated by successful error signaling) and the Pe do not depend on the early error signal reflected by the Ne/ERN and thus confirm the existence of two independent systems of error monitoring.

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Error Awareness Can Occur in the Absence of an Error-Related Negativity

Julia Dumsky[1] | Martin E. Maier[1] | Francesco Di Gregorio[2] | Marco Steinhauser[1]

[1]Department of Psychology, Catholic University of Eichstätt-Ingolstadt, Eichstätt, Germany | [2]Center for Studies and Research in Cognitive Neuroscience, Department of Psychology, Alma Mater Studiorum – University of Bologna, Cesena, Italy

Correspondence: Julia Dumsky (julia.dumsky@stud.ku.de)

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ABSTRACT

Errors in choice tasks lead to a cascade of error-related brain activity in event-related potentials. While the error-related negativity (Ne/ERN) reflects an early error signal, the error positivity (Pe) has been attributed to the later emergence of error awareness. Previous work has shown that these two components can be dissociated using a target-masking paradigm. In this modified flanker paradigm, an invisible-target condition is realized in which errors are detectable even if the correct response is unknown. These errors have been shown to elicit a Pe without a Ne/ERN, demonstrating the independence of the two underlying systems. Here, we employed this paradigm to ask whether error awareness can emerge without a Ne/ERN. While performing the target-masking paradigm, participants provided metacognitive judgments to indicate whether an error has occurred on each trial (i.e., error signaling). The majority of participants were able to report detectable errors in the invisible-target condition. Crucially, this error signaling as well as a Pe was observable in the absence of a Ne/ERN. Our findings demonstrate that both error awareness (as indicated by successful error signaling) and the Pe do not depend on the early error signal reflected by the Ne/ERN and thus confirm the existence of two independent systems of error monitoring.

1 | Introduction

Detecting errors is a crucial precondition for adaptive behavior. It has long been known that the human brain is equipped with an error monitoring system that registers errors and then initiates adjustments to attention and behavior to prevent errors in the future (Gehring et al. 2012; Ullsperger, Danielmeier, and Jocham 2014; Ullsperger, Fischer, et al. 2014). The most prominent neural correlate of this system is the error-related negativity (ERN; Gehring et al. 1993) or error negativity (Ne; Falkenstein et al. 1991), reflecting an early error signal that is elicited almost immediately after an error. Another important aspect of error processing is the ability to become consciously aware of errors (Wessel et al. 2011). Decades ago, it has been shown that when humans commit errors in speeded perceptual choice tasks, they can indicate these errors with high reliability within the fraction of a second (Rabbitt 1966). Although research on the functional architecture of error awareness has made considerable progress in recent years, many fundamental questions are still unanswered. In the present study, we investigate to which extent error awareness relies on early error processing reflected by the Ne/ERN. More specifically, we ask whether error awareness can occur in errors in which the Ne/ERN is absent.

The Ne/ERN is a negativity with a maximum around 50 ms after an erroneous response at frontocentral electrodes and has its origin in the medial-frontal cortex (Debener et al. 2005; Iannaccone et al. 2015; Ullsperger and von Cramon 2001). It has initially been suggested to represent a signal which indicates a mismatch between the actual and correct response (Fu et al. 2023; Scheffers and Coles 2000); whereas later theoretical accounts proposed that it is a representation of a postresponse

conflict (Yeung et al. 2004) or a prediction error (Holroyd and Coles 2002). In all these accounts, this early component of error processing relies on a discrepancy between the expected correct response and the actual response and hence requires that a representation of this correct response is available. A second component, the error positivity (Pe; Falkenstein et al. 1991; Overbeek et al. 2005), occurs between 200 ms and 500 ms after the error and has a more posterior distribution on the scalp. The Pe has frequently been related to error awareness (Steinhauser and Yeung 2010; Ullsperger, Danielmeier, and Jocham 2014; Ullsperger, Fischer, et al. 2014) or the confidence about the correctness of a response (Boldt and Yeung 2015).

Error awareness can be measured by a metacognitive judgment called error signaling. After the primary task response, participants press a key to indicate whether an error has occurred or not. A frequent finding is that the Pe is larger on signaled errors than on unsignaled errors (e.g., Endrass et al. 2007; Kirschner et al. 2021; Nieuwenhuis et al. 2001; O'Connell et al. 2007; Porth et al. 2022; Steinhauser and Yeung 2010) or is observed only on signaled errors (e.g., Murphy et al. 2012). Moreover, the Pe has been shown to be generally larger if error signaling is required than if not (Grützmann et al. 2014). Based on findings like these, the idea has been formulated that the Pe amplitude represents the accumulated evidence for an error that eventually leads to error awareness as measured by error signaling (Desender et al. 2021; Steinhauser and Yeung 2010; Ullsperger, Danielmeier, and Jocham 2014; Ullsperger, Fischer, et al. 2014), and several studies provided direct evidence for this hypothesis (e.g., Steinhauser and Yeung 2010, 2012).

However, it remains unclear which role is played by the Ne/ERN for the emergence of error awareness and thus the ability to signal errors. A plausible assumption is that the early error signal represented by the Ne/ERN provides the evidence that is fed into evidence accumulation represented by the Pe and thus error awareness. For instance, Yeung et al. (2004) proposed that the Ne/ERN represents response conflict and that an error is signaled whenever this accumulated response conflict exceeds a criterion (see also Steinhauser et al. 2008). However, several findings speak against the idea that the Ne/ERN is the sole source of evidence for error awareness. First, the link between the Ne/ERN and error awareness is much weaker than that between the Pe and error awareness (for a review, see Wessel 2012). Whereas some studies reported a larger Ne/ERN for signaled errors than for unsignaled errors (e.g., Scheffers and Coles 2000; Wessel et al. 2011), others did not find such a relation (e.g., Endrass et al. 2007; Hughes and Yeung 2011; Nieuwenhuis et al. 2001; Porth et al. 2022). Second, many studies demonstrated that experimental manipulations had different effects on the Ne/ERN and Pe (e.g., Charles et al. 2013; Hughes and Yeung 2011; Maier et al. 2015; Steinhauser and Yeung 2010). These findings do not rule out that error awareness can be based on early error signals reflected by the Ne/ERN. But they imply that the Ne/ERN is not the only source of evidence that drives error awareness. In this vein, Wessel et al. (2011) proposed that error awareness might rely on evidence provided by perceptual, cognitive, and autonomous processes.

While the link between the Ne/ERN and error awareness appears to be weaker than initially assumed, results from another study even raise the possibility that error awareness can emerge without an Ne/ERN at all. In the study of Di Gregorio et al. (2018), participants performed a three-choice flanker task, in which participants had to classify a central target letter while ignoring two laterally presented identical flanker letters. The target was always associated with a different response than the flankers, and participants were explicitly instructed that a response to the flanker letter is always an error. Crucially, the target letter was masked with variable stimulus-mask intervals (SMI). In most trials, the SMI was sufficiently long for the target to be easily visible. But in one third of trials, only the mask was presented, and no target was visible at all. This invisible-target condition created a scenario in which an error was detectable (whenever participants accidentally responded to the flankers) but the correct response remained unknown (because the target was fully masked). In this condition, Di Gregorio et al. (2018) observed a Pe in the absence of an Ne/ERN. This demonstrated that the Ne/ERN and Pe represent independent and dissociable systems of error monitoring: a system associated with the Ne/ERN that elicits early error signals and relies on a representation of the correct response and another system associated with the Pe that forms the basis of error awareness and can rely on other types of evidence (such as the knowledge that a response to the flanker must be an error).

Demonstrating that a Pe can occur in the absence of an Ne/ERN (Di Gregorio et al. 2018) has fundamental implications for the functional architecture of error monitoring. It shows that the Ne/ERN and Pe are not two stages of a single process (e.g., Yeung et al. 2004; Ullsperger, Fischer, et al. 2014) but rather form two independent systems (Charles et al. 2013; Maier et al. 2015). Crucially, as the Pe has been suggested to represent the evidence accumulation process that leads to error awareness (Steinhauser and Yeung 2010), this result further suggests that error awareness can also emerge in the absence of an Ne/ERN. However, direct evidence for this conjecture has not been provided because the study of Di Gregorio et al. (2018) did not include an error signaling task. In the present study, we combined the target-masking paradigm from Di Gregorio et al. (2018) with an error signaling task. Participants had to indicate after each response whether this response was an error, a correct response, or whether they do not know. The latter category was introduced because some errors in this paradigm are objectively undetectable. Our main goal was to test whether objectively detectable and signaled errors in the invisible-target condition lead to a Pe in the absence of an Ne/ERN. This would demonstrate that error awareness without an Ne/ERN is possible and would replicate the findings of Di Gregorio et al. (2018) regarding the dissociation of the Pe and Ne/ERN.

2 | Method

2.1 | Participants

We aimed to obtain the same sample size ($n = 20$) as Di Gregorio et al. (2018). This sample size implies a power of $> 90\%$ to detect within-subject effects with a Cohen's $d > 0.79$, which was the smallest effect size of a significant contrast (correct vs. error) reflecting an Ne/ERN or Pe in Di Gregorio et al. (2018). However, because not all participants showed

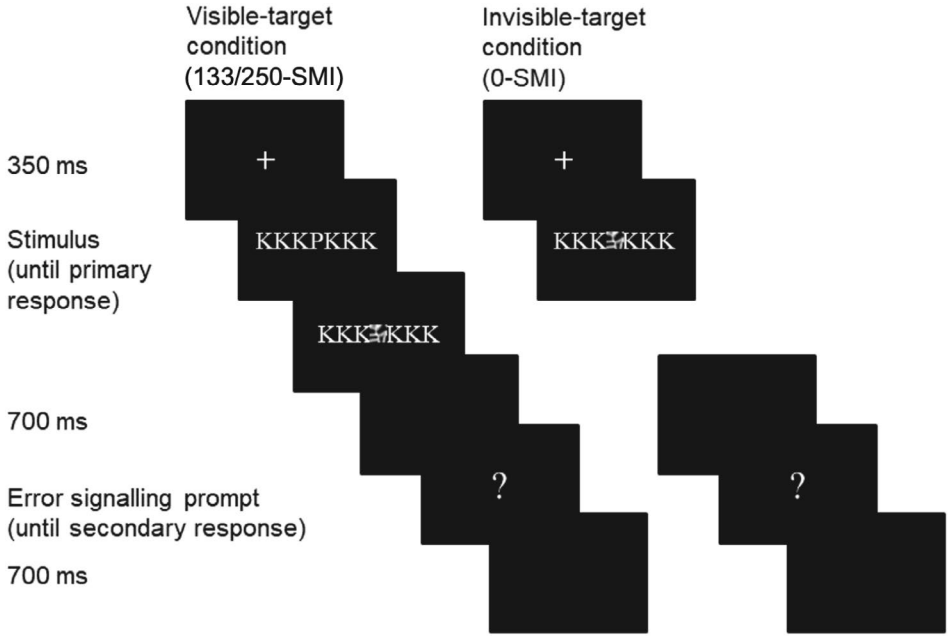

FIGURE 1 | Typical trial sequence. Participants had to respond to the target letter while flanker letters had to be ignored. In the visible-target condition, the target letter was masked after 133 or 250 ms. In the invisible-target condition, only the mask was presented but no target. After the response, a prompt appeared, and participants had to indicate whether the response was correct, incorrect, or whether they do not know. SMI = stimulus-mask interval.

a sufficient detection performance in the invisible-target condition, we had to collect more data to obtain at least 20 participants for the main analysis. Eventually, thirty-three participants participated in the study (self-reported gender: 25 female, 7 male, 1 nonbinary). All participants were recruited at the Catholic University of Eichstätt-Ingolstadt, had normal or corrected-to-normal vision, had an age between 19 and 38 years ($M = 20.9$, SD = 2.57) and received either course credits or 8 Euro per hour. The study was approved by the ethics committee of the university, and informed consent was obtained from all participants.

2.2 | Task and Stimuli

Stimuli were presented using Presentation software (Neurobehavioral Systems, Albany, CA) on a 21-in. screen with a resolution of 1280 × 1024 and a refresh rate of 60 Hz. Viewing distance was approximately 70 cm. Each trial comprised a primary task and a secondary task. The *primary task* required participants to classify a target letter by pressing the 'A', 'S', or 'D' key of a standard computer keyboard with the ring finger, middle finger, and index finger, respectively, of their left hand. The primary task stimuli were horizontal strings of seven white letters presented on a black background. They consisted of a central target letter and three identical flanker letters on each side of the central target. The letters P, W, M, V, X, or K were used, each with a height of 0.41° visual angle and a width of 0.25° visual angle. The same pairs of letters ('PW', 'MV' and 'XK') were assigned to one of the response buttons, for example, 'P' to the button 'A', 'MV' to the button 'S', and 'XK' to the button 'D'. The assignment of these pairs of letters to the three response buttons was additionally varied across participants. This resulted in six different combinations of the stimulus–response mappings. All stimuli were incongruent, that is, target and flanker letters were always assigned to different responses. This resulted in 24 possible stimuli. Six different feature masks of the same size as a letter were created by randomly rearranging features of the original letter stimuli. The *secondary task* required participants to judge the correctness of their primary task response by pressing one of three keys with their right hand: (a) 'H' key with the index finger if the previous response was correct, (b) the 'J' key with their middle finger if the previous response was an error, or (c) the 'K' key with the ring finger if they did not know whether the previous response was correct or not ("unsure").

An exemplary trial is depicted in Figure 1. At the beginning of each trial, a fixation cross was presented for 350 ms, followed by the primary task stimulus. One of the six masks replaced the central target after a stimulus-masking interval (SMI) of 0 ms, 133.3 ms, or 250 ms. Whereas participants could see a target in the 133-SMI and 250-SMI trials (the *visible-target condition*), no target was presented at all in the 0-SMI condition (the *invisible-target condition*, Figure 1B). Mask and flankers were presented on the screen until the participant provided the primary task response. After the response, a black screen was presented for 700 ms followed by a white question mark, which remained on the screen until the secondary task response was made. At the end of each trial, another black screen was presented for 700 ms again.

Each block consisted of 72 trials, in which each of the possible 24 stimuli was combined with each of the three possible SMIs. Stimuli and conditions were presented in pseudo-randomized order (without any constraints) within blocks. The experiment was conducted over a two-day period, with a maximal interval of 48 h in between. The first day was used for practicing the

primary as well as the secondary task. In the first two practice blocks, the participants practiced only the primary task. Then, the secondary task was introduced, and the participants had to complete ten blocks with both tasks. During the whole practice session, the participants could look at a paper sheet displaying the rules for the primary and secondary task for support. During all practice blocks, a deadline was implemented to train the participants to provide their primary response within a certain time interval and in this way to commit a sufficient number of erroneous trials. If this deadline was exceeded, automatic feedback about the latency was presented at the center of the screen, which urged the participant to respond faster ("SCHNELLER"). Each participant started with the same deadline of 500 ms. Afterwards, it was adapted from trial to trial in the following way (see Steinhauser and Yeung 2010, for a similar procedure): if the primary task response in the previous trial was incorrect, the deadline in the current trial was increased by 100 ms. If the response was correct, the deadline was decreased by 20 ms (but could never fall below 100 ms). A 5:1 ratio was applied to achieve an approximate error rate of 15% (Kaernbach 1991). This trial-to-trial adaptation was only applied on 133-SMI and 250-SMI trials, while the deadline remained unchanged (i.e., the same as after the previous trial) on 0-SMI trials. Moreover, if the previous trial was a 0-SMI trial, the deadline was adapted based on the last 133-SMI or 250-SMI trial.

On the second day, the test session was conducted. The participants first completed two practice blocks with 48 trials each (including the deadline procedure). Then, twelve test blocks were conducted in which no feedback was provided. Instead, the participants were instructed at the beginning of a block to respond faster whenever the number of errors in the visible-target condition fell below eight (16.7%). At the beginning of each session, participants were instructed about the tasks. Specifically, they received the information that (a) they should guess if the target letter could not be identified as sometimes the target would be very difficult to see, and (b) responding to the flankers would always be an error. The whole test session lasted approximately 1.5 h, and the participant could take short breaks between blocks whenever necessary.

2.3 | EEG Data Acquisition

Only during the test session, the electroencephalogram was recorded using a BIOSEMI Active-Two system (BioSemi, Amsterdam, The Netherlands) with 64 Ag-AgCl electrodes in a 10–10 montage from channels Fp1, AF7, AF3, F1, F3, F5, F7, F17, FC5, FC3, FC1, C1, C3, C5, T7, TP7, CP5, CP3, CP1, P1, P3, P5, P7, P9, PO7, PO3, O1, Iz, Oz, POz, Pz, CPz, Fpz, Fp2, AF8, AF4, AFz, Fz, F2, F4, F6, F8, FT8, FC6, FC4, FC2, FCz, Cz, C2, C4, C6, T8, TP8, CP6, CP4, CP2, P2, P4, P6, P8, P10, PO8, PO4, O2, as well as the left and right mastoid. The CMS (Common Mode Sense) and DRL (Driven Right Leg) electrodes were used as reference and ground electrodes. The vertical and horizontal electrooculogram (EOG) was recorded with BioSemi FLAT Active electrodes positioned over and under the right eye as well as on the outer canthi of both eyes. EEG and EOG were continuously recorded at a sampling rate of 512 Hz. No online filtering was applied, and all electrodes were off-line re-referenced to the average reference.

2.4 | Data Analysis

Only data from the test blocks were analyzed; the first three trials in each block were removed from all analyses. A Greenhouse–Geisser correction was conducted (Greenhouse and Geisser 1959) where necessary, and corrected *p*-values but uncorrected degrees of freedom were reported in this case.

2.5 | Response Categories

Each trial was assigned to a response category depending on the primary response (for an example, see Figure 1). In the visible-target condition (133/250-SMI), there were three possible response categories: (1) A *correct response* resulted if the response matched the target response. (2) A *flanker error* resulted if the response matched the flanker response. (3) A *nonflanker error* resulted if the response matched neither the target nor the flanker response. In the invisible-target condition (0-SMI), there were two possible response categories: (1) A *flanker error* resulted if the response matched the flanker response. (2) A *nonflanker guess* resulted if the response did not match the flanker response. Depending on the secondary response, trials were further classified as *"correct"*, *"error"*, or *"unsure"* (note that quotation marks are used to indicate the response categories of the secondary response).

2.6 | Behavioral Data

The statistical analyses of the behavioral data were conducted with R Studio version 4.0.3 for Windows (R Core Team, released 2020, Vienna, Austria). The time between stimulus onset and the primary response was defined as response time (RT). If the RT deviated more than three standard deviations from the condition mean, the corresponding trial was excluded from the analyses. Overall, 0.29% of trials (SE = 0.004%) were excluded in this way. For frequency data, an arcsine transformation was performed to stabilize variances before statistical testing (Winer et al. 1971).

We first analyzed behavioral data to investigate whether reliable task performance and error signaling was possible in our paradigm. For the *visible-target condition*, paired *t*-tests were used to compare error rates and proportions of flanker errors (among all errors) between 133-SMI and 250-SMI conditions. Mean RTs were subjected to a two-way repeated-measures ANOVA with the variables SMI (133-SMI, 250-SMI) and correctness (correct, errors). Mean error RTs were further analyzed using a two-way repeated-measures ANOVA with the variables SMI and error type (flanker error, nonflanker error). To investigate whether reliable error signaling was achieved in the visible-target condition, the rate of detected errors and the rate of errors that were classified as "unsure" was subjected to a two-way ANOVA with the variables SMI and error type. The rate of false alarms and the rate of correct trials classified as unsure were analyzed using paired *t*-tests to compare between 133-SMI and 250-SMI conditions. For the *invisible-target condition*, primary task performance was analyzed by comparing mean RTs between flanker errors and nonflanker guesses using a paired *t*-test. To see whether participants were able to

detect errors by taking the flanker into account, we compared the rate of detected errors and the rate of errors that were classified as "unsure" between the two trial types (flanker errors, nonflanker guesses).

2.7 | EEG Data

EEG data were analyzed using custom routines based on EEGLab (Delorme and Makeig 2004) implemented in MatLab R2013b (The Mathworks, Natick, MA). Preprocessing was the same as in Di Gregorio et al. (2018) unless otherwise stated. EEG data were first low-pass filtered (40 Hz) with a least-square FIR filter (−6 dB cutoff) and then high-pass filtered (0.1 Hz). Epochs were extracted from 200 ms before and 600 ms after the primary response. Baseline correction was applied based on the interval [−100 ms; 0 ms], which was a different baseline than in Di Gregorio et al. (2018) (in which [−150 ms; −50 ms] was used). This was done to properly align correct and error trials at the onset of the Ne/ERN to prevent overestimation or underestimation of error-related activity. Channels were interpolated using spherical spline interpolation whenever they met the joint probability criterion (threshold 5) or the kurtosis criterion (threshold 10) in EEGLAB's pop_rejchan function. In this way, 4.22 (SE = 0.21) channels were interpolated on average. Epochs were excluded if the amplitude exceeded a threshold of ±200 μV at any electrode (except at the frontal electrodes as blinks were removed at a later stage) or if the joint probability deviated more than 5 standard deviations from the epoch mean (according to EEGLAB's pop_jointprob function). On average, 4.63% of trials (SE = 0.24%) were rejected in this way. In the condition with the smallest trial number (nonflanker errors in the 250-SMI condition), an average of 20 trials (SE = 3.0) remained in the analysis. An independent component analysis (Delorme and Makeig 2004) was computed, and independent components with blink artifacts were removed using CORRMAP (Campos Viola et al. 2009). Finally, epochs were averaged separately for each condition and participant. The Ne/ERN was quantified as mean amplitude in the time window [0 ms; 100 ms] at a frontocentral electrode cluster including electrodes Cz, FC1, FCz, FC2, and Fz. The Pe was quantified as mean amplitude in the time window [200 ms; 500 ms] at a posterior electrode cluster including electrodes POz, P1, Pz, P2, and CPz.

Because waveforms from the frontocentral cluster suggested that there might be an earlier Ne/ERN peak with a more variable latency in the invisible-target condition, we additionally ran a control analysis with an alternative quantification method. The so-called adaptive mean measure (Clayson et al. 2013) requires, first, identifying the largest negative peak in the difference wave between errors and correct trials separately for each participant, and second, analyzing mean amplitudes around these individual peak latencies. This quantification method is more sensitive than standard methods if peak latencies vary across participants. It works particularly well if a large Ne/ERN is present and thus the largest negative peak is indeed due to an Ne/ERN. However, in a scenario like ours in which an Ne/ERN is possibly absent and differences between errors and corrects might reflect noise, then the method could artificially induce an Ne/ERN-like negativity because there is necessarily always a negative peak in noisy data. We therefore modified this method to make it applicable to a scenario in which the goal of the analysis is to decide whether an Ne/ERN is present at all: Instead of identifying the largest negative peak, we identified the largest absolute peak (irrespective of whether it is positive or negative). If there is no Ne/ERN, then the average of participants with positive and negative maxima should be zero (which is the null hypothesis). If an Ne/ERN is present, then this mean should be significantly negative. As the data suggest that an Ne/ERN peak in the invisible-target condition might occur earlier than the original time window, we used a larger and earlier time window (−50 ms to 100 ms) and consequently an earlier baseline (−150 to −50 ms) for this analysis. Mean amplitudes were analyzed in a time window of 100 ms centered around the respective peaks.

In all reported analyses, we included only correctly classified errors and correct trials as well as nonflanker guesses for which participants indicated to be unsure. We report only analyses of the good detector group, as only these participants had sufficient numbers of detected errors (see Supporting Information). In the *visible-target condition*, the Ne/ERN and Pe (defined as the amplitude difference between correct responses and errors in the respective time window) were subjected to a two-way repeated-measures ANOVA with the variables SMI (133-SMI, 250-SMI) and error type (flanker error, nonflanker error). Paired *t*-tests were used for follow-up analyses. While these analyses should demonstrate how an Ne/ERN and Pe look like if the target is visible, analyses of the *invisible target condition* serve to determine whether an Ne/ERN and/or Pe is obtained if the target is invisible and the correct response is unknown. Only for the Ne/ERN analyses in this part, we additionally used the modified adaptive mean measure described above. We first tested for the presence of an Ne/ERN and Pe by directly comparing flanker errors and nonflanker guesses against the correct baseline from the 133-SMI condition using paired *t*-tests (see results section for a discussion of the choice of this baseline). Moreover, paired *t*-tests were also used to test for differences between flanker errors and nonflanker guesses. In a final set of analyses, the Ne/ERN and Pe for flanker errors were first subjected to separate one-way repeated-measures ANOVAs with the variable SMI (0-SMI, 133-SMI, 250-SMI). Then the pattern of Ne/ERNs and Pes on flanker errors was directly compared using a two-way repeated-measures ANOVA with the variables component (Ne/ERN, Pe) and SMI (0, 133, 250) to statistically establish the dissociation between the two components.

3 | Results

A crucial goal of this study was to investigate whether the Ne/ERN was absent for detected flanker errors in the invisible-target condition, and thus, whether error signaling as a behavioral correlate of error awareness is possible without an Ne/ERN. However, participants turned out to differ with respect to whether they consistently detected these errors, and visual inspection of the distribution of the rate of detected flanker errors suggested a bimodal distribution (see Supporting Information for more information). We therefore split our

TABLE 1 | Primary task performance.

Conditions	Error rates (%)	Prop. FE (%)	RT correct (ms)	RT FE (ms)	RT NFE/G (ms)
250-SMI	16.0 (±1.7)	57.2 (±2.3)	537 (±17)	531 (±33)	523 (±31)
133-SMI	22.5 (±1.6)	56.8 (±1.5)	564 (±18)	551 (±34)	537 (±26)
0-SMI	—	31.4 (±1.4)	—	513 (±17)	549 (±23)

Note: NFE/G refers to NFE in the 250-SMI and 133-SMI conditions but NFG in the 0-SMI condition.
Within-participants standard errors of the mean are provided in parentheses.
Abbreviations: FE, flanker error; ms, milliseconds; NFE, nonflanker error; NFG, nonflanker guess; Prop, proportion; RT, response time; SMI, stimulus-masking interval.

sample into two subgroups: a group of good detectors ($n = 21$) with a detection rate of more than 40% and a group of bad detectors ($n = 12$) with a detection rate of less than 40%. The 40% criterion was selected as it (a) separated the two groups based on their detection rate, and (b) ensured a sufficient number of detected flanker errors for EEG analysis for each participant in this group (>= 35 trials, see Supporting Information). In the following, only the results for the good detectors group are presented; however, a comparison of the two groups is provided in the Supporting Information.

3.1 | Behavioral Data

3.1.1 | Visible-Target Condition (133/250-SMI)

We first analyzed the behavioral data for the visible-target condition (133-SMI, 250-SMI) to investigate whether participants reliably detected errors in this condition. Primary task performance (see Table 1) showed differences between trials with SMIs of 133 ms and 250 ms. Error rates were higher for the 133-SMI condition than for the 250-SMI condition; $t(20) = 3.56$, $p = 0.002$, $d = 0.778$. Also, RTs were higher in the 133-SMI than in the 250-SMI condition; $F(1, 20) = 13.7$, $p = 0.001$, $\eta_p^2 = 0.407$, and were higher for correct than for error trials; $F(1, 20) = 11.0$, $p = 0.002$, $\eta_p^2 = 0.355$. Next, we differentiated between flanker errors and nonflanker errors. The relative frequency of flanker errors among all errors was 57% (SE = 1.9%) and did not differ between SMI conditions; $t(20) = 0.22$, $p = 0.832$, $d = 0.048$. Regarding error RTs, there was neither a significant difference between error types; $F(1, 20) = 0.61$, $p = 0.445$, $\eta_p^2 = 0.030$, nor between SMI conditions; $F(1, 20) = 2.35$, $p = 0.141$, $\eta_p^2 = 0.105$.

The detection rates revealed that participants were able to reliably detect errors in the visible-target condition. 85% (SE = 3.3%) of errors were detected, which is comparable with previous findings with unmasked stimuli (Steinhauser et al. 2008). The rate of detected errors did not differ between error types, $F(1, 20) = 2.38$, $p = 0.138$, $\eta_p^2 = 0.106$, but was higher in the 250-SMI condition (89%, SE = 3.1%) than in the 133-SMI condition (81.1%, SE = 3.4%), $F(1, 20) = 14.5$, $p = 0.001$, $\eta_p^2 = 0.420$. In 8.7% (SE = 1.5%) of errors, participants were unsure whether an error had occurred or not, and this rate was higher for the 133-SMI condition ($M = 11.2\%$, SE = 2.1%) than for the 250-SMI condition ($M = 6.3\%$, SE = 1.9%), $F(1, 20) = 8.43$, $p = 0.009$, $\eta_p^2 = 0.297$, but did not differ across error types, $F(1, 20) = 0.11$, $p = 0.745$, $\eta_p^2 = 0.006$. The rate of correct trials that were falsely judged as "error" (false alarm rate) was low (0.53%, SE = 0.21%) and did not differ between SMI conditions, $t(20) = 1.10$, $p = 0.285$, $d = 0.250$, and the same held for the rate of correct trials judged as "unsure" ($M = 0.71\%$, SE = 0.22%), $t(20) = 1.43$, $p = 0.167$, $d = 0.312$.

Taken together, we obtained considerable effects of the masking interval in almost all behavioral parameters. The 133-SMI condition was associated not only with impaired error awareness but also with impaired primary task performance; an effect that was not found in our previous study (Di Gregorio et al. 2018).

3.1.2 | Invisible-Target Condition (0-SMI)

A central goal of the present study was to investigate whether error awareness is possible for flanker errors in the invisible-target condition, that is, for errors for which we expected a Pe but no Ne/ERN. Because a correct response was not possible in this condition, participants could either commit a flanker error (because responses to the flankers were always errors) or a nonflanker guess. The rate of flanker errors (among all responses) in the invisible-target condition was 31.4% (SE = 1.4%), and flanker errors had a lower RT than nonflanker guesses (Table 1); $t(20) = 5.73$, $p < 0.001$, $d = 1.250$.

Crucially, a substantial proportion of flanker errors in the invisible-target condition was detected (81.6%, SE = 4.3%). This detection rate was higher than that for nonflanker guesses (2.9%, SE = 0.7) for which errors were not objectively detectable; $t(20) = 16.1$, $p < 0.001$, $d = 3.513$, but was not significantly different from the averaged detection rate for flanker errors in the visible-target conditions (84.0%, SE = 3.1%); $t(20) = 0.44$, $p = 0.667$, $d = 0.095$. As one would expect, the rate of trials classified as "unsure" in turn was higher for nonflanker guesses (93.0%, SE = 1.7%) than for flanker errors (17.7%, SE = 4.2%); $t(20) = 13.4$, $p < 0.001$, $d = 2.928$. Finally, for the rate of trials classified as "correct", no significant difference between flanker errors (0.8%, SE = 0.3%) and nonflanker guesses (4.2%, SE = 1.6%) was observed; $t(20) = 1.98$, $p = 0.062$, $d = 0.095$.

Taken together, the participants showed a pattern of error awareness that roughly corresponded to the objective probability of errors in the two trial types. Flanker errors were classified as "error" with a similar rate as flanker errors in the visible-target condition. In contrast, nonflanker guesses were predominantly classified as "unsure". Please note that this pattern differs from that in the bad detectors group; a detailed comparison of the two groups is provided in the Supporting Information.

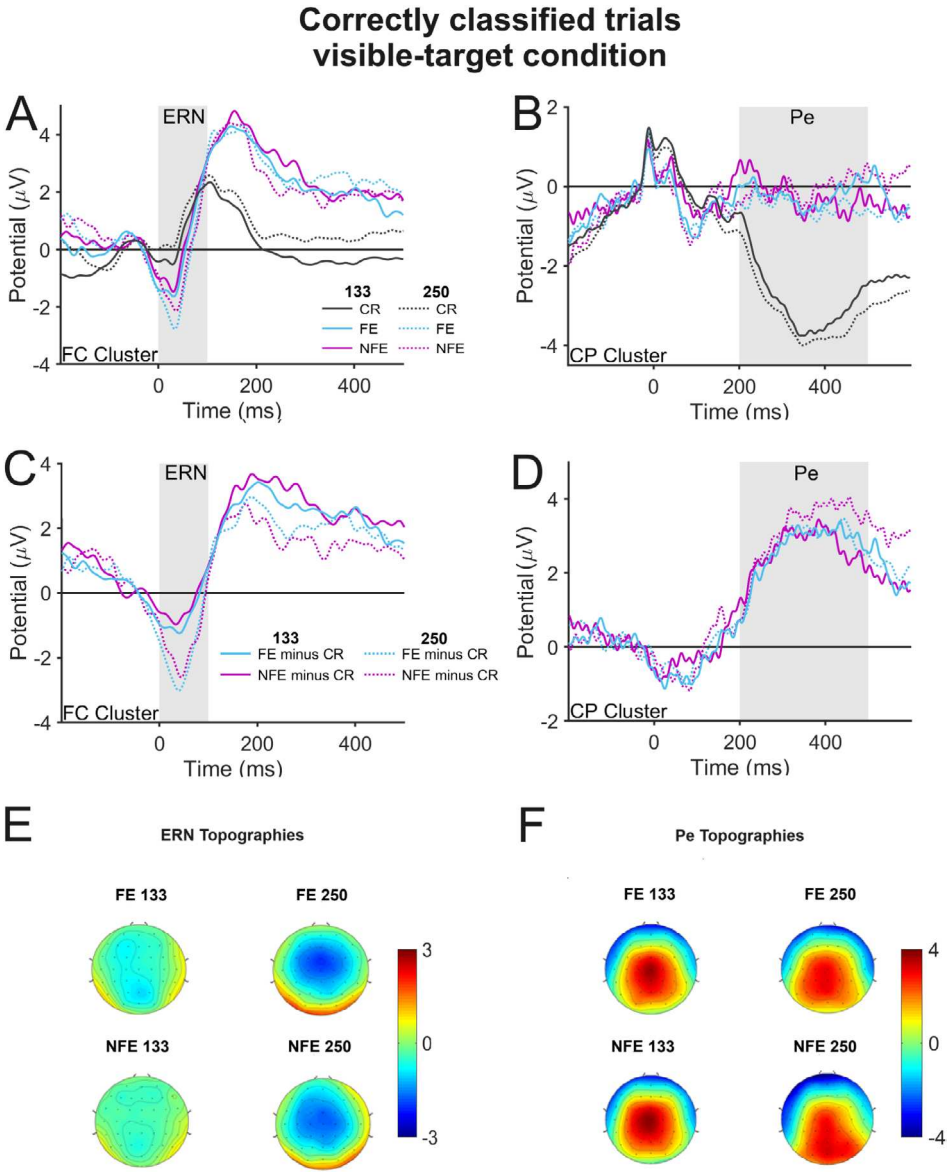

FIGURE 2 | Error-related negativity (Ne/ERN; frontocentral cluster in left column) and error positivity (Pe; centroparietal cluster in right column) in the visible-target (133/250-SMI) conditions. Only correctly classified trials of the good detectors group were considered. (A, B) Waveforms from all response types. (C, D) Difference waves for flanker errors (minus corrects) and nonflanker errors (minus corrects). (E, F) Topographies of the difference waves in the time range of the Ne/ERN and Pe. Gray areas indicated the time range of the Ne/ERN and Pe in each graph. CP, centroparietal; CR, correct response; FC, frontocentral; FE, flanker error; NFE, nonflanker error; SMI, stimulus-mask interval.

3.2 | ERP Data

We next analyzed ERP data to investigate whether the results from Di Gregorio et al. (2018) can be replicated for detected errors in the invisible-target condition; and thus, whether error awareness can occur in the absence of a Ne/ERN. In all analyses, we included only correctly classified errors and correct trials as well as nonflanker guesses for which participants indicated to be unsure.

3.2.1 | Visible-Target Conditions (133/250 SMI) in Good Detectors

We first analyzed the 250-SMI and 133-SMI conditions to demonstrate that a typical Ne/ERN and Pe can be shown in our paradigm. To visualize the Ne/ERN, waveforms for all possible response types (correct, flanker error, nonflanker error) for the frontocentral electrode cluster are shown in Figure 2A, and waveforms and topographies of the difference between errors and corrects are depicted in Figure 2C,E, respectively. Both error types show negative deflections relative to correct trials in both SMI conditions, and these negativities were maximal at frontocentral electrodes, thus demonstrating a typical Ne/ERN. The ANOVA on the difference between errors and correct trials revealed a significant effect of SMI, $F(1, 20) = 9.15$, $p = 0.007$, $\eta_p^2 = 0.314$, indicating a larger Ne/ERN for the 250-SMI condition ($M = -1.75\,\mu V$, $SE = 0.89\,\mu V$) than for the 133-SMI condition ($M = -0.55\,\mu V$, $SE = 0.62\,\mu V$). However, there was no main effect for error type, $F(1, 20) = 1.3$, $p = 0.268$, $\eta_p^2 = 0.061$, and no significant interaction, $F(1, 20) < 0.01$, $p > 0.999$,

$\eta_p^2 < 0.001$. Collapsed across SMIs, the difference between errors and corrects in the time window of the Ne/ERN reached significance for flanker errors ($M = -1.25\,\mu V$, $SE = 0.19\,\mu V$), $t(20) = 6.55$, $p < 0.001$, $d = 1.429$, as well as for nonflanker errors ($M = -1.08\,\mu V$, $SE = 0.26\,\mu V$), $t(20) = 4.13$, $p < 0.001$, $d = 0.901$. For correct trials, the waveform in the time range of the Ne/ERN showed a more negative mean amplitude for the 133-SMI condition ($M = 0.62\,\mu V$, $SE = 0.18\,\mu V$) than for the 250-SMI condition ($M = 1.09\,\mu V$, $SE = 0.18\,\mu V$), which, however, reached only marginal significance, $t(20) = 1.84$, $p = 0.080$, $d = 0.402$.

Figure 2B,D,F show the same waveforms, difference waves, and topographies for the centroparietal electrode cluster to visualize the Pe. The amplitudes of both error types for both SMIs are clearly more positive than those of the corresponding correct trials, and these positivities have a centroparietal distribution, thus indicating a Pe. The ANOVA on the difference between errors and corrects revealed no significant main effects of error type, $F(1, 20) = 0.68$, $p = 0.419$, $\eta_p^2 = 0.033$, and SMI, $F(1, 20) = 0.60$, $p = 0.449$, $\eta_p^2 = 0.029$, and no significant interaction, $F(1, 20) = 0.79$, $p = 0.385$, $\eta_p^2 = 0.038$. Collapsed across SMIs, this difference reached significance for flanker errors ($M = 2.75\,\mu V$, $SE = 0.34\,\mu V$, $t(20) = 8.05$, $p < 0.001$, $d = 1.757$) as well as for the nonflanker errors ($M = 2.92\,\mu V$, $SE = 0.42\,\mu V$, $t(20) = 4.13$, $p < 0.001$, $d = 0.901$). Moreover, there was no significant difference in the time range of the Pe between correct trials in the two SMI conditions, $t(20) = 1.35$, $p = 0.193$, $d = 0.295$.

In sum, there was a highly significant Ne/ERN and Pe for both error types and SMI conditions. However, as for the behavioral data, we obtained differences between the two SMI conditions that were not observed in our previous study (Di Gregorio et al. 2018). There was a reduced Ne/ERN for the 133-SMI condition as well as a trend toward different amplitudes for correct waveforms in the time range of the Ne/ERN. It appears that the shorter masking interval impaired error processing relative to the longer masking interval, and that the mask had also an effect on correct trials. While effects like these have previously been reported (e.g., Charles et al. 2013), they imply that we cannot collapse across the two conditions in our analyses.

3.2.2 | Invisible-Target Condition (0-SMI) in Good Detectors

Di Gregorio et al. (2018) found that errors in the invisible-target condition were associated with a Pe even though no Ne/ERN was observed. Here, we asked whether we can replicate this result when only correctly classified trials (i.e., flanker errors classified as "errors" and nonflanker guesses classified as "unsure") are included, thus demonstrating that error awareness is possible in the absence of an Ne/ERN. As in our previous study, we had to deal with the problem that no correct trials are available in the invisible-target condition that can be used as a baseline for quantifying the Ne/ERN and Pe. Again, we applied two strategies to determine whether an Ne/ERN or Pe was present: First, we used correct trials from the visible-target condition as a baseline. Because the 133-SMI and 250-SMI conditions differed in several aspects of the data including the waveform of correct trials, we decided to use only the 133-SMI condition for which the masking interval was closer to that of the invisible-target condition. Second, we compared flanker errors and nonflanker guesses in the invisible-target condition. For flanker errors, the stimulus provides more objective information that an error has occurred, which should lead to stronger error signals and thus a larger Pe and also a larger Ne/ERN if our hypothesis is incorrect and an Ne/ERN is observed in this condition.

To investigate whether a Ne/ERN is observable in the invisible-target condition, we inspected waveforms and topographies from the frontocentral electrode cluster in Figure 3A,C. It reveals almost identical waveforms for all trial types in the time range of the Ne/ERN, suggesting that there is neither a negativity relative to the correct baseline nor a larger negativity for flanker errors than for nonflanker guesses. Also, the scalp topographies (Figure 3E) provided no evidence for a frontocentral negativity that could be interpreted as a Ne/ERN. These observations received support from statistical analyses. Neither the difference between flanker errors and correct trials ($M = -0.08\,\mu V$, $SE = 0.24\,\mu V$) reached significance; $t(20) = 0.33$, $p = 0.749$, $d = 0.072$, nor the difference between nonflanker guesses and correct trials ($M = -0.07\,\mu V$, $SE = 0.27\,\mu V$), $t(20) = 0.24$, $p = 0.814$, $d = 0.052$, and also waveforms for flanker errors and nonflanker guesses did not differ significantly; $t(20) = 0.10$, $p = 0.923$, $d = 0.022$. For completeness, we repeated this analysis with correct trials of the 250-SMI condition. Correct trials in this condition differed significantly from flanker errors ($M = -0.55\,\mu V$, $SE = 0.20\,\mu V$), $t(20) = 2.79$, $p = 0.011$, $d = 0.609$, as well as nonflanker guesses ($M = -0.53\,\mu V$, $SE = 0.21\,\mu V$), $t(20) = 2.52$, $p = 0.020$, $d = 0.550$. However, these significant differences are most likely due to the difference between correct trials of the 133-SMI and 250-SMI reported above.

A closer inspection of Figure 3C shows that the difference wave peaks earlier in the invisible-target condition than in the visible-target condition. Moreover, Figure 3A suggests that the peak in the averaged waveforms of errors is not clearly identifiable, which could point to variability in the latency of the alleged Ne/ERN across participants. We therefore ran a control analysis in which we used a modified adaptive mean measure (Clayson et al. 2013) that can deal with variable peak latencies and applied this to a larger and earlier time window (−50 to 100 ms) and an earlier baseline period (−150 to −50 ms). But again, there was neither a significant difference between flanker errors and correct trials ($M = -0.46\,\mu V$, $SE = 0.43\,\mu V$), $t(20) = 1.06$, $p = 0.303$, $d = 0.231$, nor between nonflanker guesses and correct trials ($M = -1.09\,\mu V$, $SE = 0.68\,\mu V$), $t(20) = 1.60$, $p = 0.125$, $d = 0.349$, or between flanker errors and nonflanker guesses ($M = -0.68\,\mu V$, $SE = 0.70\,\mu V$), $t(20) = 0.96$, $p = 0.348$, $d = 0.209$.

Next, we considered the Pe in the waveforms from the centroparietal electrode cluster (Figure 3B,D). There was a clear positivity in flanker errors and nonflanker guesses relative to the correct baseline in the time range of the Pe.[1] Moreover, this Pe is larger for flanker errors than for nonflanker guesses. Figure 3F shows a clear posterior distribution resembling that in the visible-target condition. Corroborating these observations, the statistical analyses revealed a significant positivity for flanker errors ($M = 2.43\,\mu V$, $SE = 0.40\,\mu V$); $t(20) = 4.29$, $p < 0.001$, $d = 0.936$, as well as for nonflanker guesses ($M = 1.54\,\mu V$, $SE = 0.3\,\mu V$); $t(20) = 3.68$, $p < 0.001$, $d = 0.803$, relative to the correct baseline. Furthermore, this Pe was larger for flanker errors than for nonflanker guesses; $t(20) = 2.40$, $p = 0.026$, $d = 0.524$.

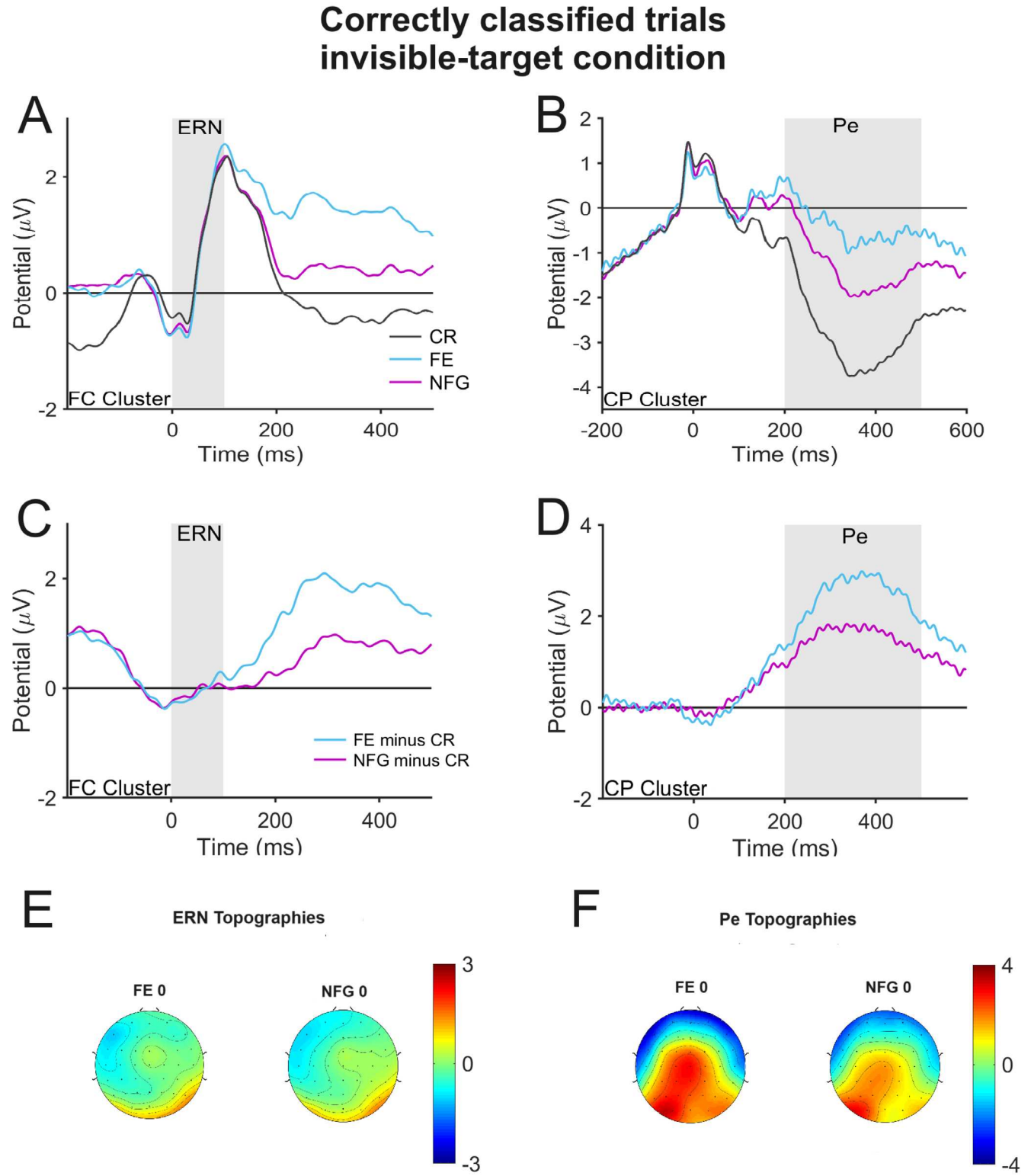

FIGURE 3 | Error-related negativity (Ne/ERN; frontocentral cluster in left column) and error positivity (Pe; centroparietal cluster in right column) in the invisible-target (0-SMI) condition. Only correctly classified trials of the good detectors group were considered. Data for correct responses were taken from the 133-SMI condition. (A, B) Waveforms from all response types. (C, D) Difference waves for flanker errors (minus corrects) and nonflanker guesses (minus corrects). (E, F) Topographies of the difference waves in the time range of the Ne/ERN and Pe. Gray areas indicated the time range of the Ne/ERN and Pe in each graph. CP, centroparietal; CR, correct response; FC, frontocentral; FE, flanker error; NFG, nonflanker guess; SMI, stimulus-mask interval.

3.2.3 | Visible-Target Condition Versus Invisible-Target Condition

Finally, we compared Ne/ERN and Pe amplitudes across all conditions in a combined analysis. Only flanker errors were included as only these error types could occur in all conditions. Ne/ERN amplitudes varied significantly across the three SMI conditions, $F(2, 40) = 10.7$, $p < 0.001$, $\eta_p^2 = 0.349$, reflecting that the invisible-target condition ($M = -0.33\,\mu V$, $SE = 0.21\,\mu V$) showed smaller Ne/ERNs than the 250-SMI ($M = -1.88\,\mu V$, $SE = 0.29\,\mu V$), $t(20) = 4.26$, $p < 0.001$, $d = 0.930$, and the 133-SMI condition ($M = -0.69\,\mu V$, $SE = 0.24\,\mu V$), $t(20) = 1.28$, $p = 0.214$, $d = 0.279$. In contrast, Pe amplitudes did not vary significantly across conditions, $F(2, 40) = 0.01$, $p = 0.988$, $\eta_p^2 < 0.001$. When including both components in a two-way ANOVA with component (Ne/ERN, Pe) as an additional variable, we obtained significant main effects of SMI condition, $F(2, 40) = 5.49$, $p = 0.008$, $\eta_p^2 = 0.215$, but not for component, $F(1, 20) = 2.35$, $p = 0.141$, $\eta_p^2 = 0.105$. The interaction between SMI condition and component only reached marginal significance, $F(2, 40) = 3.14$, $p = 0.054$, $\eta_p^2 = 0.136$.

4 | Discussion

Although the Ne/ERN, an early error signal, has been widely studied over the last decades, it is far from clear whether and how the Ne/ERN relates to error awareness. Whereas Di Gregorio et al. (2018) showed that an Ne/ERN is not necessary for the emergence of a Pe, it is still unclear whether this holds for error awareness as well. The main objective of the present study was thus to investigate whether errors can be consciously detected in the absence of an Ne/ERN. To test this, we added an error signaling task to the target-masking paradigm of Di Gregorio et al. (2018). This paradigm allows for the creation of errors that are detectable without knowing the correct response. As the Ne/ERN is assumed to rely on the representation of a correct response, these error trials should therefore lead to error awareness but not to an Ne/ERN.

Confirming this prediction, our study was able to show that error signaling (and thus error awareness) can occur without an Ne/ERN. Most participants (the good detectors group) detected flanker errors in the invisible-target condition very reliably, and these detected flanker errors showed no Ne/ERN. This is plausible as the Ne/ERN requires a representation of the correct response, and participants could not know what the correct response was in this condition. This demonstrates that an Ne/ERN is not a necessary precondition for error awareness to emerge. Moreover, these errors were associated with a Pe, which replicates the core result of Di Gregorio et al. (2018) and again confirms that Ne/ERN and Pe are dissociable. This again demonstrates that the two components do not represent stages of a unitary process (e.g., Yeung et al. 2004; Ullsperger, Fischer, et al. 2014) but can be viewed as two independent mechanisms of performance monitoring (Charles et al. 2013; Maier et al. 2015).

Our results can be interpreted within the framework of the evidence accumulation account of error awareness (Desender et al. 2021; Steinhauser and Yeung 2010; Ullsperger et al. 2010), which states that error awareness emerges when the accumulated evidence for an error exceeds a criterion, and that the Pe reflects this accumulated evidence. Whereas the early error signal reflected by the Ne/ERN could form one source of evidence, it is probably not the only one and—as shown by the present data—also not a necessary source of evidence. It is rather conceivable that there are various internal and external sources of evidence such as sensory, proprioceptive, and interoceptive signals that contribute to error awareness (Wessel et al. 2012; Wessel et al. 2011; Ullsperger et al. 2010). In the present paradigm, error detection is an inference-based process that relies on the explicit knowledge that a response to the flankers must be an error. Other sources of evidence can be interoceptive signals including error-related changes in heart rate, pupil response, and skin conductance, for which it has already been shown that they can correlate with error awareness (Di Gregorio et al. 2024; Hajcak et al. 2003; Harsay et al. 2018; Quirins et al. 2018; Wessel et al. 2011; but see Maier et al. 2019). Despite the absence of the Ne/ERN, these interoceptive signals could have contributed to error awareness in our study, for instance, by mediating between inference and error awareness.

As in our previous study, we also obtained a Pe for nonflanker guesses; but this Pe was smaller than that for flanker errors in the invisible-target condition. This can be interpreted by assuming that the Pe amplitude scales with the conditional probability that a response is an error, which is 100% for flanker errors and 50% for nonflanker guesses in the invisible-target condition (Di Gregorio et al. 2018). This relationship might be a consequence of the fact that the accumulated evidence for an error varies with these probabilities across error types. However, there are also alternative ways to account for this result. It is possible that the reduced Pe for nonflanker guesses reflects an averaging artifact indicating that nonflanker guesses are perceived as correct by some participants but as errors by others. Inspection of the single-subject data did not reveal a bimodal pattern of the differences between flanker errors and nonflanker guesses; but such a pattern is generally difficult to identify in noisy physiological data. Alternatively, it is also possible that, within each participant, we averaged across single nonflanker guesses which were perceived as an error (full Pe) or as a correct response (no Pe). As the Pe difference between flanker errors and nonflanker guesses appears to be an interesting window into the mechanisms underlying the Pe, future studies would be desirable that shed more light on the origin of this effect.

Results from previous studies were inconsistent with regard to the relationship between error awareness and the Ne/ERN. Some found that signaled errors were associated with a larger Ne/ERN (e.g., Scheffers and Coles 2000; Wessel et al. 2011; Woodman 2010), while others did not find this relationship (e.g., Dhar et al. 2011; Endrass et al. 2007; Nieuwenhuis et al. 2001; Shalgi et al. 2009). It has previously been argued that the occasional observation of such a relationship could reflect a correlative rather than a causal link between Ne/ERN and error awareness, and that this correlation is mediated by features of task performance (e.g., Steinhauser and Yeung 2010; Di Gregorio et al. 2016). For instance, in some tasks, aware errors could be associated with higher postresponse conflict than unaware errors, and this leads to a higher Ne/ERN for the former (Di Gregorio et al. 2016). In the invisible-target condition, we largely deconfounded conflict and error awareness by preventing the correct response from being activated, and thus, preventing

postresponse conflict from occurring (although the presence of residual conflict, e.g., between flanker and nonflanker responses cannot be fully excluded). Accordingly, our results are compatible with the idea that the relationship between Ne/ERN and error awareness in previous studies is mediated by conflict or other variables related to the activation of the correct response. However, our results are also compatible with a second explanation for the inconsistent relationship between Ne/ERN and error awareness. While showing that an Ne/ERN is not necessary for error awareness to emerge, we cannot exclude the possibility that the Ne/ERN still contributes to the evidence for an error under some conditions. Therefore, it is possible that whether the Ne/ERN correlates with error awareness in a specific task depends on whether error awareness in this specific task relies on the Ne/ERN or other sources of evidence.

Unlike in our previous study (Di Gregorio et al. 2018), we observed a pronounced SMI-effect on performance as well as the EEG. In the good detectors group, the shorter SMI of 133 ms led to impaired primary task performance and error detection, but also a reduced Ne/ERN and a smaller amplitude for correct trials in the Ne/ERN time range (a so-called CRN; Vidal et al. 2000). This might be due to the inclusion of an error signaling procedure, which was not used in Di Gregorio et al. (2018). Grützmann et al. (2014) showed that error signaling leads to an increased Ne/ERN and CRN and argued that this reflects an enhanced attention to one's accuracy. In the present study, this enhanced attention to one's accuracy could have slightly reduced attention to external stimuli, which might have affected performance and the Ne/ERN particularly for the more difficult 133-SMI condition.

Another unexpected result of this study is the pronounced individual differences in signaling flanker errors in the invisible-target condition. Whereas 64% of participants reliably reported these flanker errors (the good detectors group), the remaining participants showed little or no error awareness in this condition (the bad detectors group). The fact that more than one third of the participants had to be excluded for the main analyses is a limitation of the present study as it potentially reduces the generalizability of the results. It is therefore crucial to explain the difficulty of detecting flanker errors in the invisible-target condition in so many participants. One explanation is that bad detectors applied a higher detection criterion. According to Steinhauser and Yeung (2010), a higher detection criterion implies that fewer errors are detected; however, the averaged evidence across all error trials (detected and undetected) and thus their Pe amplitude remains the same. However, our data seem to contradict this. In the Supporting Information, we compared Pe amplitudes in the invisible-target condition between good and bad detectors groups. The Pe for flanker errors was actually lower in the bad detectors group. This speaks against the idea of a higher detection criterion in the bad detectors group but rather suggests that the low detection rate in this group relies on an impaired accumulation of evidence for an error. Possibly, these participants either failed to notice that a response to the flanker had occurred or ignored the information that these flanker responses are always errors. Instead, error awareness could have relied more on the activation of the correct response, and thus the Ne/ERN, which was absent in the invisible-target condition. This receives support from the observation that overall detection rates in the visible-target condition (in which an Ne/ERN was generated) were similar in bad detectors and good detectors. The two groups did not differ in their general ability to detect errors but rather in collecting evidence for an error in the invisible-target condition. One has to note that the group differences in the present study have to be treated with caution due to the small sample size in the bad detectors group.[2]

The observed individual differences in the ability to utilize information for metacognition appear to be an interesting field for future research. Interindividual abilities in introspection have previously been found to rely on neuroanatomical differences (e.g., Fleming et al. 2010; Sinanaj et al. 2015). In our specific task, these differences might also be related to differences in working memory capacity, which has been demonstrated to impact error monitoring (Miller et al. 2012). In this vein, other studies have shown that both the Ne/ERN and Pe are affected by working memory load induced by dual-tasking (Klawohn et al. 2016; Moser et al. 2013; Steinhauser and Steinhauser 2021; Watanabe et al. 2024; Weißbecker-Klaus et al. 2016) although these results do not always follow a consistent pattern (for a discussion, see Schuch et al. 2019). Interestingly, one study suggests that particularly the identification of flanker errors might be difficult under high working memory load (Maier and Steinhauser 2017). As one could argue that our paradigm places high demands on working memory due to the use of a three-choice task and the need to signal errors, future studies could measure working memory capacity to account for inter-individual differences in this paradigm and to investigate whether participants with low working memory capacity rely more on the Ne/ERN rather than task rules to signal errors in our task.

5 | Conclusion

Taken together, the present study demonstrated that the ability to become aware of an error does not rely on early error processing as reflected by the Ne/ERN, but rather relates to late error processing represented by the Pe. This does not exclude the possibility that error awareness can be influenced by the Ne/ERN but shows that an Ne/ERN is not necessary for error awareness to emerge. These results support the notion that the Ne/ERN and Pe constitute independent systems of error monitoring.

Author Contributions

Julia Dumsky: conceptualization, writing – original draft, formal analysis, visualization, investigation. **Martin E. Maier:** writing – review and editing, conceptualization. **Francesco Di Gregorio:** writing – review and editing, conceptualization. **Marco Steinhauser:** conceptualization, writing – review and editing, supervision, funding acquisition.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Endnotes

[1] Figure 3 shows that the centroparietal cluster (Figs. 4BD) is associated with more noise than the frontocentral cluster (Figs. 4AC). A closer inspection of the data revealed that this is due to an enhanced noise level at electrode P2 in five participants, which however was not strong enough to meet our interpolation criterion. Importantly, re-analysis of the data without this electrode did not change the results.

[2] If bad detectors do not take into account whether a response to the flanker has occurred, they should assign an error probability of 66% to either of the three responses (irrespective of whether it is a flanker error or nonflanker guess). As this is higher than the 50% error probability assigned to nonflanker guesses by good detectors, one would expect that the Pe on nonflanker guesses is higher for good detectors than for bad detectors. This was not observed which might reflect the restricted power to find such a small effect given the small sample sizes or to the fact that error awareness in bad detectors relied more on early error processing as discussed.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Data S1:** psyp70128-sup-0001-Supinfo01.docx. **Figure S1:** Distribution of detection frequencies of flanker errors in the invisible-target condition. Participants with detection frequencies of 40% or above were included in the group of good detectors. **Figure S2:** Error-related negativity (Ne/ERN; frontocentral cluster in left column) and error positivity (Pe; centroparietal cluster in right column) in the visible-target (133/250-SMI) conditions of the good detectors group. All trials were considered irrespective of whether they were correctly classified or not. (A, B) Waveforms from all response types. (C, D) Difference waves for flanker errors (minus corrects) and nonflanker errors (minus corrects). (E, F) Topographies of the difference waves in the time range of the Ne/ERN and Pe. Gray areas indicated the time range of the Ne/ERN and Pe in each graph. CP = centroparietal, CR = correct response, FC = frontocentral, FE = flanker error, NFE = nonflanker error, SMI = stimulus-mask interval. **Figure S3:** Error-related negativity (Ne/ERN; frontocentral cluster in left column) and error positivity (Pe; centroparietal cluster in right column) in the visible-target (133/250-SMI) conditions of the bad detectors group. All trials were considered irrespective of whether they were correctly classified or not. (A, B) Waveforms from all response types. (C, D) Difference waves for flanker errors (minus corrects) and nonflanker errors (minus corrects). (E, F) Topographies of the difference waves in the time range of the Ne/ERN and Pe. Gray areas indicated the time range of the Ne/ERN and Pe in each graph. CP = centroparietal, CR = correct response, FC = frontocentral, FE = flanker error, NFE = nonflanker error, SMI = stimulus-mask interval. **Figure S4:** Error-related negativity (Ne/ERN; frontocentral cluster in left column) and error positivity (Pe; centroparietal cluster in right column) in the invisible-target (0-SMI) condition of the good detectors group. All trials were considered irrespective of whether they were correctly classified or not. Data for correct responses were taken from the 133-SMI condition. (A, B) Waveforms from all response types. (C, D) Difference waves for flanker errors (minus corrects) and nonflanker guesses (minus corrects). (E, F) Topographies of the difference waves in the time range of the Ne/ERN and Pe. Gray areas indicated the time range of the Ne/ERN and Pe in each graph. CP = centroparietal, CR = correct response, FC = frontocentral, FE = flanker error, NFG = nonflanker guess, SMI = stimulus-mask interval. **Figure S5:** Error-related negativity (Ne/ERN; frontocentral cluster in left column) and error positivity (Pe; centroparietal cluster in right column) in the invisible-target (0-SMI) condition of the bad detectors group. All trials were considered irrespective of whether they were correctly classified or not. Data for correct responses were taken from the 133-SMI condition. (A, B) Waveforms from all response types. (C, D) Difference waves for flanker errors (minus corrects) and nonflanker guesses (minus corrects). (E, F) Topographies of the difference waves in the time range of the Ne/ERN and Pe. Gray areas indicated the time range of the Ne/ERN and Pe in each graph. CP = centroparietal, CR = correct response, FC = frontocentral, FE = flanker error, NFG = nonflanker guess, SMI = stimulus-mask interval. **Table S1:** Primary task Performance. **Table S2:** Frequencies of detection types and trial numbers for flanker errors and nonflanker errors in the visible-target condition for both subgroups. **Table S3:** Frequencies of detection types and trial numbers for flanker errors and nonflanker guesses in the invisible-target condition for both subgroups.

Supporting information

To investigate whether error awareness can occur without an Ne/ERN, we analyzed detected flanker errors in the invisible-target condition. However, this was possible only in 21 of the 33 participants as only these participants (good detectors group) had a sufficient detection rate for these errors. The remaining 12 participants (bad detectors group) could not be analyzed in this way as the number of detected flanker errors in the invisible-target condition was too low. In the following, we compare the two groups with respect to behavioral and EEG data and describe how the two groups were formed.

Behavioral data

The results of the primary task performance are presented in Table S1, and the detection rates in the visible-target and invisible-target conditions are shown in Table S2 and S3, respectively. The latter tables include mean trial numbers for each condition. For these behavioral data, we repeated all analyses with the additional variable group (good detectors, bad detectors). Only significant effects involving this additional variable are reported. If an interaction with group was significant, groups were analyzed separately. The analyses are reported in the same order as in the main text. The criteria for creating the two groups are discussed when describing the results from the invisible-target condition.

Visible-target condition (133/250-SMI). No significant effects involving the variable group were obtained for RTs, error rates, or the proportion of flanker errors in the visible-target condition. However, group differences emerged for the rates of error detection. Submitting the rate of detected errors to a three-way mixed ANOVA with the variables error type, SMI and group revealed a significant interaction between SMI and group, $F(1, 31) = 5.05$, $p = .032$, $\eta_p^2 = .140$. The rate of detected errors was similar across SMIs for bad detectors, $t(11) = 0.37$, $p = .717$, $d = 0.108$, but was significantly lower for the 133-SMI condition ($M = 81.1\%$, $SE = 2.4\%$)

than for the 250-SMI condition (M = 89.0%, SE = 2.2%) for the good detectors, $t(20) = 3.81$, $p = .001$, $d = 0.831$. A different pattern was obtained when considering the rate of errors classified as "unsure". The same ANOVA as above again revealed a significant two-way interaction between SMI and group, $F(1, 31) = 5.28$, $p = .029$, $\eta_p^2 = .146$. The rate of errors classified as "unsure" was similar across SMIs for bad detectors, $t(11) = 0.56$, $p = .586$, d =0 .092, but was significantly higher for the 133-SMI condition (M = 11.2%, SE = 1.5%) than for the 250-SMI condition (M = 6.3%, SE = 1.3%) for good detectors, $t(20) = 2.90$, $p = .009$, d =0 .633. No significant effects involving group were obtained for correct trials that were classified as "error" or correct trials that were classified as "unsure". Taken together, the SMI had a detrimental effect on error signaling in the good detectors group but not in the bad detectors group.

Invisible-target condition (0-SMI). The rate of flanker errors was higher for bad detectors (M = 38.9%, SE = 3.3%) than for good detectors (M = 31.4%, SE = 1.4%), $t(31) = 2.48$, $p = .019$, $d = 0.438$. Moreover, subjecting RTs to a two-way mixed ANOVA with the variables error type and group revealed a significant interaction, $F(1, 31) = 8.06$, $p = .008$, $\eta_p^2 = .206$. Whereas similar RTs were obtained for flanker errors and nonflanker guesses in the bad detectors group, $t(12) = 1.02$, $p = .330$, $d = 0.283$, flanker errors showed lower RTs than nonflanker guesses in the good detectors group, $t(20) = 5.73$, $p < .001$, $d = 1.250$.

Before we compare the two groups with respect to secondary task performance in the invisible-target condition, we discuss how the two groups were formed. Detected flanker errors in the invisible-target condition are a crucial condition for the present study, but a closer look at the data indicates that the detection rate for this condition differs strongly across participants. Figure S1 reveals a bimodal distribution of these detection rates across participants. Whereas most participants detected flanker errors with a medium to high frequency, others almost completely failed to detect these errors. Because we were ultimately interested in error-related brain activity in detected errors, which were sufficiently available only in the former

participants, we had to split our sample into the two mentioned subgroups. The 21 participants with a detection rate of more than 40% were assigned to the good detectors group whereas the remaining 12 participants were included in the bad detectors group. Table S3 shows that only good detectors have sufficient trial numbers for detected flanker errors (72.8 trials on average; range: 38 – 111 trials) to conduct analyses of Ne/ERN and Pe amplitudes in the invisible target condition, while bad detectors lack the necessary trial numbers in this condition (4.8 trials on average).

Table S3 characterizes the two groups in terms of secondary task performance and trial numbers. A two-way mixed ANOVA on the detection rates (i.e., proportion of trials classified as "errors") with group (good detectors, bad detectors) and trial type (flanker error, nonflanker guesses) revealed a significant interaction, $F(1, 31) = 129.4$, $p < .001$, $\eta_p^2 = .807$. While the detection rate was higher for flanker errors than for non-flanker guesses in good detectors, $t(20) = 18.6$, $p < .001$, $d = 4.059$, it was comparably low in both trial types for bad detectors, $t(11) = 0.174$, $p = .998$, $d = 0.050$. The same ANOVA applied to the rate of trials classified as "unsure" also showed a significant interaction, $F(1, 31) = 90.0$, $p < .001$, $\eta_p^2 = .744$. The rate was higher for nonflanker guesses than for flanker errors in good detectors, $t(20) = 15.8$, $p < .001$, $d = 3.448$, but was comparably high for both trial types in bad detectors, $t(11) = 0.07$, $p = .999$, $d = 0.020$. Finally, for the rate of trials classified as "correct", no significant main effect or interaction involving group was observed.

ERP data

For the group analysis of ERP data, we included all trials irrespective of whether trials were correctly classified by the participants or not. Because this implies that the results could differ from the original analyses in the main text, we first conducted each analysis without the variable group and report significant effects. We then added the variable group and report whether

significant effects involving this variable were obtained. In this case, separate analyses for each group are provided.

Visible-target conditions (133/250 SMI). Waveforms for all response types (correct, flanker error, nonflanker error) from the visible-target condition together with difference waves and topographies are provided in Figure S2 for the good detectors group and in Figure S3 for the bad detectors group. The two-way ANOVA on the difference between errors and correct trials in the time range of the Ne/ERN revealed a significant effect of SMI, $F(1,32)= 7.70$, $p = .009$, $\eta_p^2 = .194$, with a larger Ne/ERN for the 250-SMI condition ($M = -1.52$ µV, $SE = 0.96$ µV) than for the 133-SMI condition ($M = -0.71$ µV, $SE = 0.63$ µV), but no main effect of error type, $F(1,32)= 0.04$, $p = .844$, $\eta_p^2 = .001$, and no interaction, $F(1,32)= 0.02$, $p = .894$, $\eta_p^2 < .001$. Again, when only waveforms for correct trials were considered, a larger amplitude was found for the 250-SMI condition ($M = 1.03$ µV, $SE = 0.32$ µV) than for the 133-SMI condition ($M = 0.51$ µV, $SE = 0.28$ µV), $t(32) = 2.64$, $p = .013$, $d = 0.460$. With the additional variable group (good detectors, bad detectors), no significant interactions with group were found in any of these analyses.

The two-way ANOVA on the difference between errors and correct trials in the time range of the Pe now revealed a significant effect of SMI, $F(1,32) = 5.84$, $p = .022$, $\eta_p^2 = .154$, with a larger Pe for the 250-SMI condition ($M = 3.51$ µV, $SE = 0.31$ µV) than for the 133-SMI condition ($M = 2.35$ µV, $SE = 0.30$ µV). No main effect of error type, $F(1,32) = 1.09$, $p = .304$, $\eta_p^2 = .033$, and no interaction was obtained, $F(1,32) = 2.28$, $p = .141$, $\eta_p^2 = .067$. When including group as additional variable in this analysis, the two-way interaction between SMI and group became significant, $F(1,31) = 5.03$, $p = .032$, $\eta_p^2 = .140$. We therefore conducted this two-way ANOVA separately for each group. As in the main analysis, the good detectors showed no significant effects of SMI, $F(1, 20) = 0.08$, $p = .774$, $\eta_p^2 = .004$, or error type, $F(1, 20) = 0.60$, $p = .446$, $\eta_p^2 = .029$, and no significant interaction, $F(1, 20) = 0.42$, $p = .522$, $\eta_p^2 = .021$ (Fig.

S2BD). Please note that the exact results slightly differ from the main analysis, because here, we included both correctly and incorrectly classified trials, whereas in the main analysis, we included only correctly classified trials. The bad detectors showed no significant effect of error type, $F(1, 11) = 0.29$, $p = .600$, $\eta_p^2 = .026$, but a significant effect of SMI, $F(1, 11) = 4.99$, $p = .047$, $\eta_p^2 = .312$, with a higher Pe for the 250-SMI condition than for the 133-SMI condition (Fig. S3BD). The interaction was not significant, $F(1, 11) = 2.11$, $p = .174$, $\eta_p^2 = .161$. Again, we analyzed only correct waveforms in the time range of the Pe, which revealed a significantly larger amplitude for the 133-SMI condition (M = -2.79 μV, SE = 0.35 μV) than for the 250-SMI condition (M = -3.65 μV, SE = 0.41 μV), $t(32) = 3.44$, $p = .002$, $d = 0.599$. Analyzing correct waveforms in a two-way ANOVA with the variables SMI and group revealed a significant interaction, $F(1,31) = 5.58$, $p = .025$, $\eta_p^2 < .153$, indicating a significant effect of SMI for the bad detector group (Fig. S3B), $t(11) = 3.18$, $p = .009$, $d = 0.918$, but not for the good detector group (Fig. S2B), $t(20) = 0.77$, $p = .452$, $d = 0.168$.

Taken together, in the visible-target condition the two groups showed similar effects in the time range of the Ne/ERN but differed with respect to the Pe. Regarding the latter, while no effects of SMI were obtained in the good detectors group, the bad detectors group showed a higher Pe in the 250-SMI condition which was mainly due to a higher amplitude on correct waveforms in the 133-SMI condition.

Invisible-target condition (0 SMI). As in the main analysis, we used the correct trials of the 133-SMI condition as baseline for analyzing the invisible-target condition. Waveforms for all respective response types (correct, flanker errors, nonflanker guesses) together with difference waves and topographies are provided in Figure S4 for the good detectors group and in Figure S5 for the bad detectors group. In the time range of the Ne/ERN, neither the difference between flanker errors and correct trials (M = -0.01 μV, SE = 0.18 μV) reached significance, $t(32) = 0.06$, $p = .949$, $d = 0.010$, nor the difference between nonflanker guesses and correct trials (M

= -0.2 μV, *SE* = 0.16 μV), *t*(32) = 0.36, *p* = .722, *d* = 0.063. There was also no significant difference between flanker errors and non-flanker guesses, *t*(32) = 0.89, *p* = .378, *d* = 0.155. The variable group was included by subjecting the difference wave between each response type and the respective correct baseline to a two-way ANOVA with the variables response type and group. The interaction between both variables did not reach significance, *F*(1, 31) = 3.82, *p* = .060, η_p^2 = .110.

In the time range of the Pe, the analyses revealed significant differences between flanker errors and corrects (*M* = 1.71 μV, *SE* = 0.38 μV), *t*(32) = 4.55, *p* < .001, *d* = 0.792, as well as between nonflanker guesses and corrects (*M* = 1.31 μV, *SE* = 0.30 μV), *t*(32) = 4.38, *p* < .001, *d* = 0.762. The difference between flanker errors and nonflanker guesses was not significant, *t*(32) = 1.77, *p* = .086, *d* = 0.308. However, the two-way ANOVA showed an interaction between response type and group, *F*(1,31) = 7.04, *p* = .013, η_p^2 = 0.185. The Pe was higher for flanker errors than for nonflanker guesses in the good detectors group (Fig. S4BD), *t*(20) = 2.87, *p* = .009, *d* = 0.626, but not in the bad detectors group (Fig. S5BD), *t*(11) = 1.58, *p* = .142, *d* = 0.456. Moreover, the Pe for flanker errors was higher in the good-detectors group than in the bad detectors group, *t*(31) = 2.33, *p* = .027, *d* = 0.412, whereas the Pe for nonflanker guesses was similar in both groups, *t*(31) = 0.99, *p* = .331, *d* = 0.175.

To summarize, as the visible-target condition, the invisible-target condition shows no group differences in the time range of the Ne/ERN. Regarding the Pe, however, the bad detectors group did not show the pattern of an increased Pe for flanker errors relative to nonflanker guesses that we obtained for the good detectors group.

Visible-target condition vs. invisible-target condition. In a final step, we compared flanker errors across all three SMI conditions as in the main analysis. For the Ne/ERN, the one-way ANOVA with the variable SMI (250-SMI, 133-SMI, 0-SMI) revealed a significant effect, *F*(2, 64) = 11.5, *p* < .001, η_p^2 = .264, indicating a lower Ne/ERN in the 0-SMI condition (*M* = -0.01

μV, *SE* = 0.22 μV) than in the 250-SMI condition (*M* = -1.52 μV, *SE* = 0.27 μV), $t(32) = 4.06$, $p < .001$, $d = 0.707$, and in the 133-SMI condition (*M* = -0.73 μV, *SE* = 0.17 μV), $t(32) = 2.44$, $p = .020$, $d = 0.425$. In this analysis, the 250-SMI condition also showed a higher Ne/ERN than the 133-SMI condition, $t(32) = 2.44$, $p = .020$, $d = 0.425$. When adding the variable group, no significant interaction was obtained, $F(2, 62) = 1.03$, $p = .362$, $\eta_p^2 = .032$. For the Pe, we now obtained a significant main effect of SMI, $F(2, 62) = 5.76$, $p = .005$, $\eta_p^2 = .157$, and SMI interacted significantly with group when the latter variable was added, $F(2, 62) = 9.50$, $p < .001$, $\eta_p^2 = .235$. Separate analyses for each group revealed that the Pe did not differ significantly across SMIs for the good detectors group, $F(2, 40) = 0.01$, $p = .992$, $\eta_p^2 < .001$, but for the bad detectors group, $F(2, 22) = 11.7$, $p < .001$, $\eta_p^2 = .515$. In the latter, the Pe was smaller for the 0-SMI condition than for the 250-SMI condition, $t(11) = 4.00$, $p = .002$, $d = 1.152$, and the 133-SMI condition, $t(11) = 5.85$, $p < .001$, $d = 1.689$.

Next, we included the Ne/ERN and Pe for flanker errors from each SMI-condition in a two-way ANOVA with the variables SMI and component (Ne/ERN, Pe). We obtained a significant main effect for component, $F(1, 32) = 71.8$, $p < .001$, $\eta_p^2 = .692$, but not for SMI, $F(2, 64) = 0.20$, $p = .817$, $\eta_p^2 = .006$. Most important, the interaction between SMI and component reached significance, $F(2, 64) = 12.7$, $p < .001$, $\eta_p^2 = .284$, which shows that the Ne/ERN and Pe varied differently across conditions. When additionally adding the variable group, the corresponding three-way ANOVA resulted in a significant two-way interaction between SMI and group, $F(2, 62) = 8.04$, $p < .001$, $\eta_p^2 = .206$, and a significant three-way interaction, $F(2, 62) = 5.96$, $p = .004$, $\eta_p^2 = .161$. Separate analyses of each group revealed interactions between SMI and component for the good detectors group, $F(2, 40) = 4.02$, $p = .026$, $\eta_p^2 = .167$, as well as the bad detectors group, $F(2, 22) = 11.1$, $p < .001$, $\eta_p^2 = .502$.

To summarize, the analyses of all trials and participants replicated the dissociation of the Ne/ERN and Pe that was found in Di Gregorio et al. (2018) and in the analyses of correctly

classified trials in the good detectors group in the main text. However, we also found that the bad detectors group differed in some respects from the good detectors group. Most notably, the bad detectors group did not show the pattern of Pe amplitudes across response types that characterized the good detectors group. In good detectors, the Pe was larger for flanker errors than for nonflanker guesses mirroring that the probability of an error was 100% in the former but only 50% in the latter. In contrast, bad detectors did not show this difference. The Pe for flanker errors was similar as that for nonflanker guesses and smaller than the Pe in the visible-target condition.

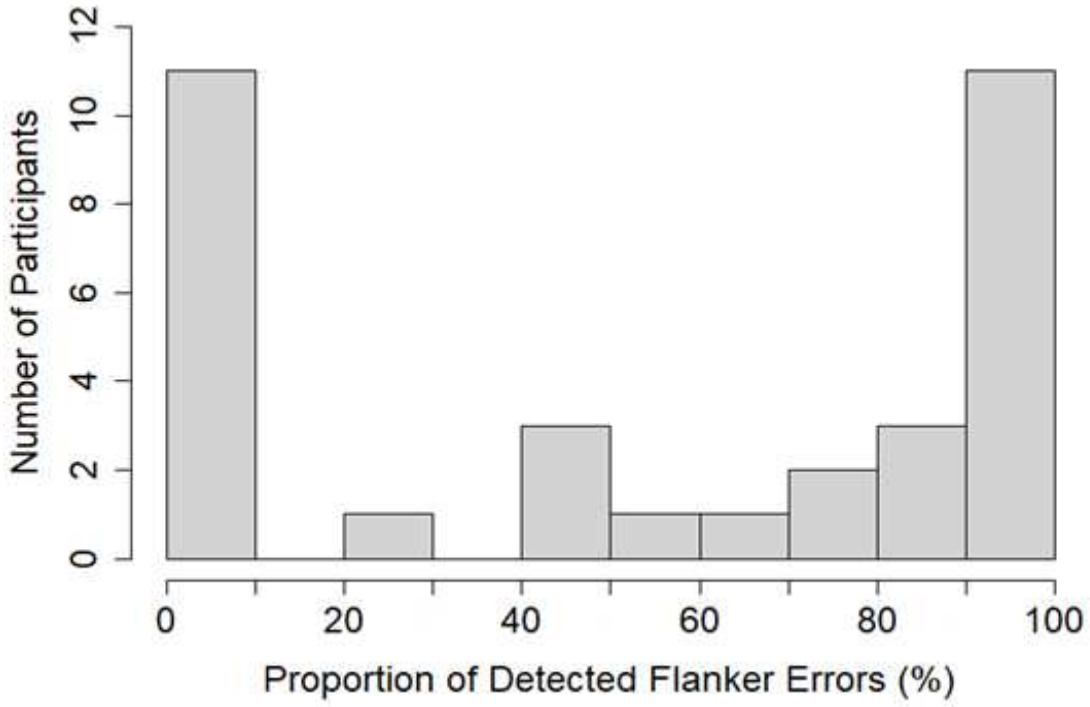

Figure S1. Distribution of detection frequencies of flanker errors in the invisible-target condition. Participants with detection frequencies of 40% or above were included in the group of good detectors.

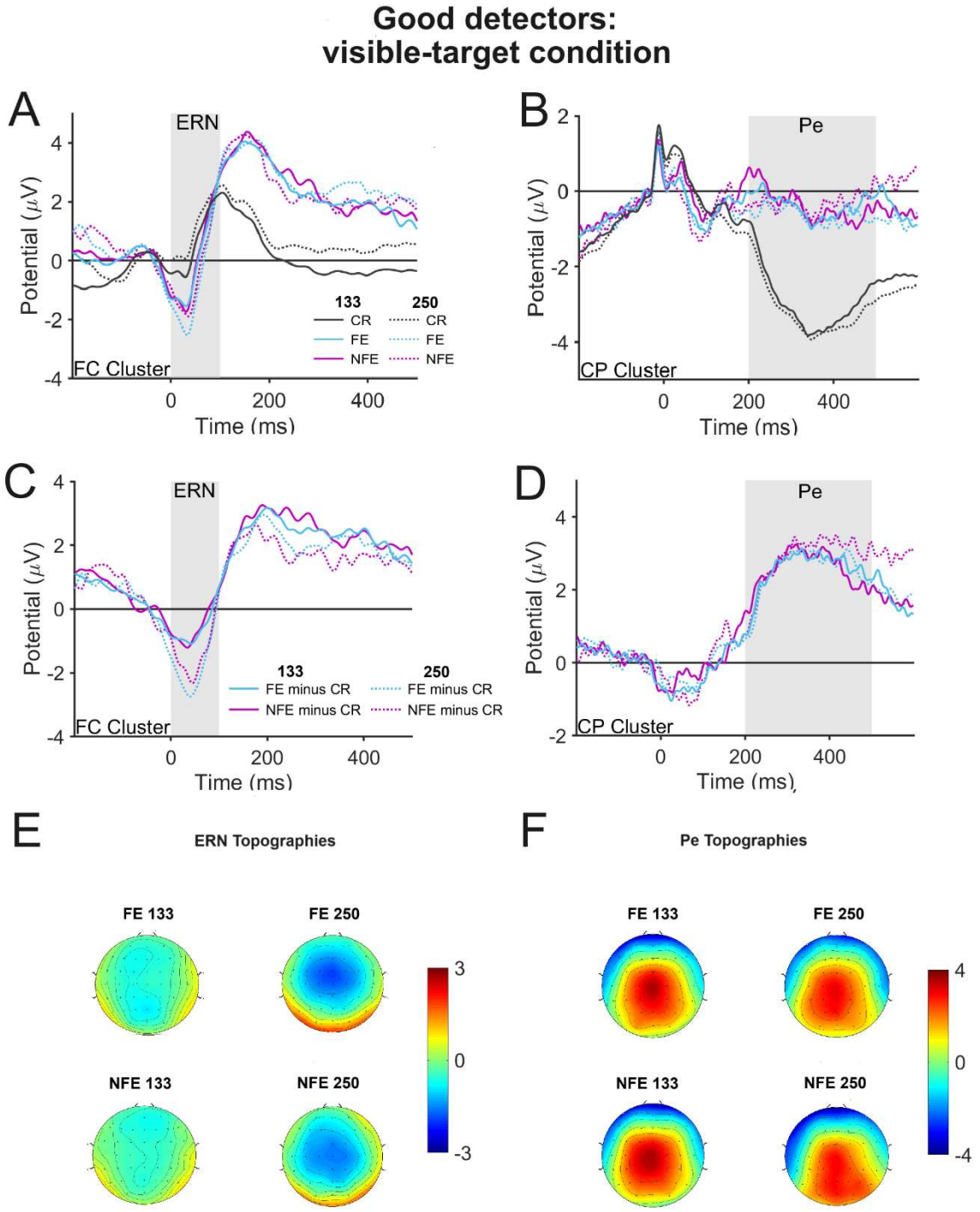

Figure S2. Error-related negativity (Ne/ERN; frontocentral cluster in left column) and error positivity (Pe; centroparietal cluster in right column) in the visible-target (133/250-SMI) conditions of the good detectors group. All trials were considered irrespective of whether they were correctly classified or not. AB: Waveforms from all response types. CD: Difference waves for flanker errors (minus corrects) and nonflanker errors (minus corrects). EF: Topographies of the difference waves in the time range of the Ne/ERN and Pe. Grey areas indicated the time range of the Ne/ERN and Pe in each graph. SMI = stimulus-mask interval, CR = correct response, FE = flanker error, NFE = nonflanker error, FC = frontocentral, CP = centroparietal.

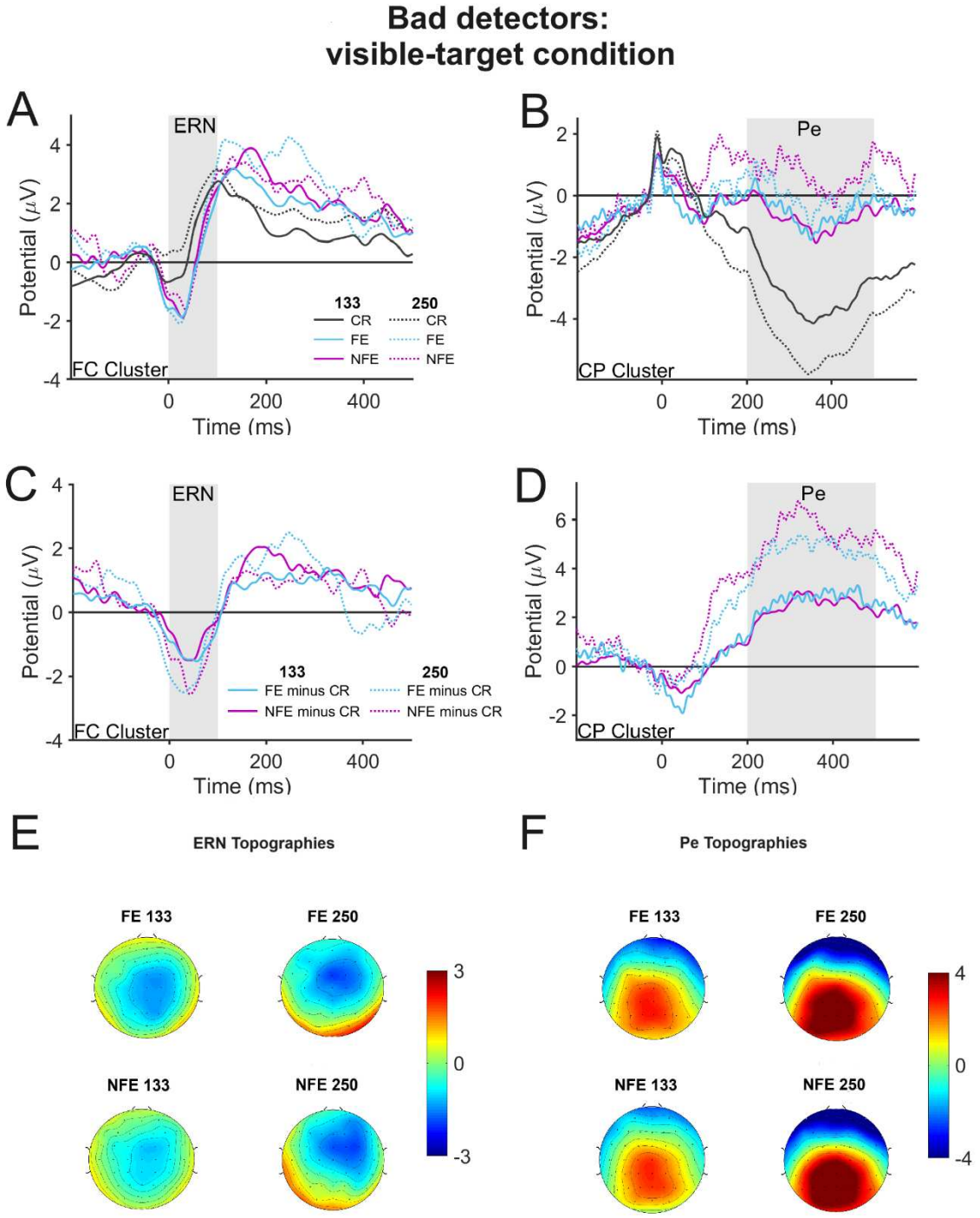

Figure S3. Error-related negativity (Ne/ERN; frontocentral cluster in left column) and error positivity (Pe; centroparietal cluster in right column) in the visible-target (133/250-SMI) conditions of the bad detectors group. All trials were considered irrespective of whether they were correctly classified or not. AB: Waveforms from all response types. CD: Difference waves for flanker errors (minus corrects) and nonflanker errors (minus corrects). EF: Topographies of the difference waves in the time range of the Ne/ERN and Pe. Grey areas indicated the time range of the Ne/ERN and Pe in each graph. SMI = stimulus-mask interval, CR = correct response, FE = flanker error, NFE = nonflanker error, FC = frontocentral, CP = centroparietal.

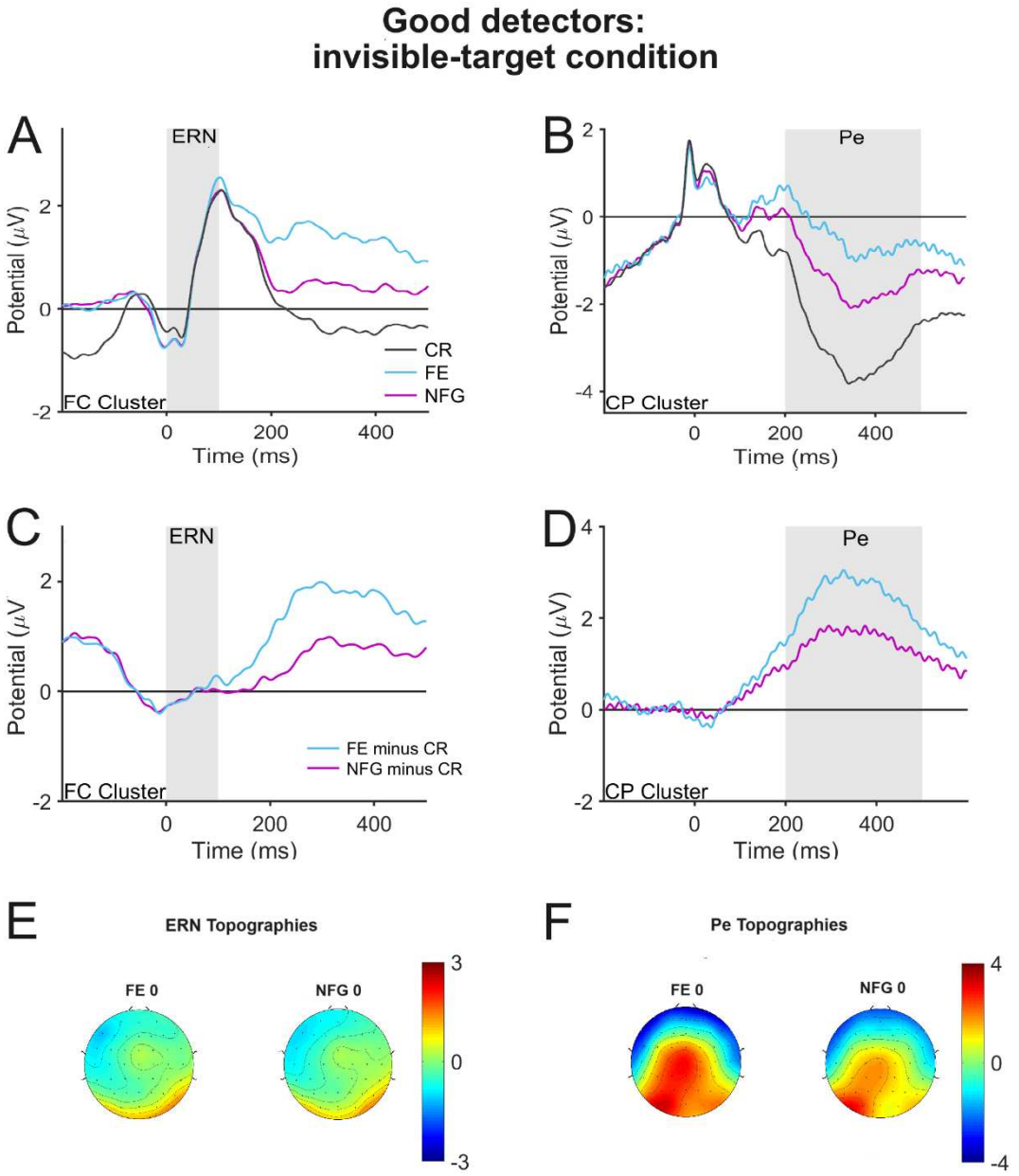

Figure S4. Error-related negativity (Ne/ERN; frontocentral cluster in left column) and error positivity (Pe; centroparietal cluster in right column) in the invisible-target (0-SMI) condition of the good detectors group. All trials were considered irrespective of whether they were correctly classified or not. Data for correct responses were taken from the 133-SMI condition. AB: Waveforms from all response types. CD: Difference waves for flanker errors (minus corrects) and nonflanker guesses (minus corrects). EF: Topographies of the difference waves in the time range of the Ne/ERN and Pe. Grey areas indicated the time range of the Ne/ERN and Pe in each graph. SMI = stimulus-mask interval, CR = correct response, FE = flanker error, NFG = nonflanker guess, FC = frontocentral, CP = centroparietal.

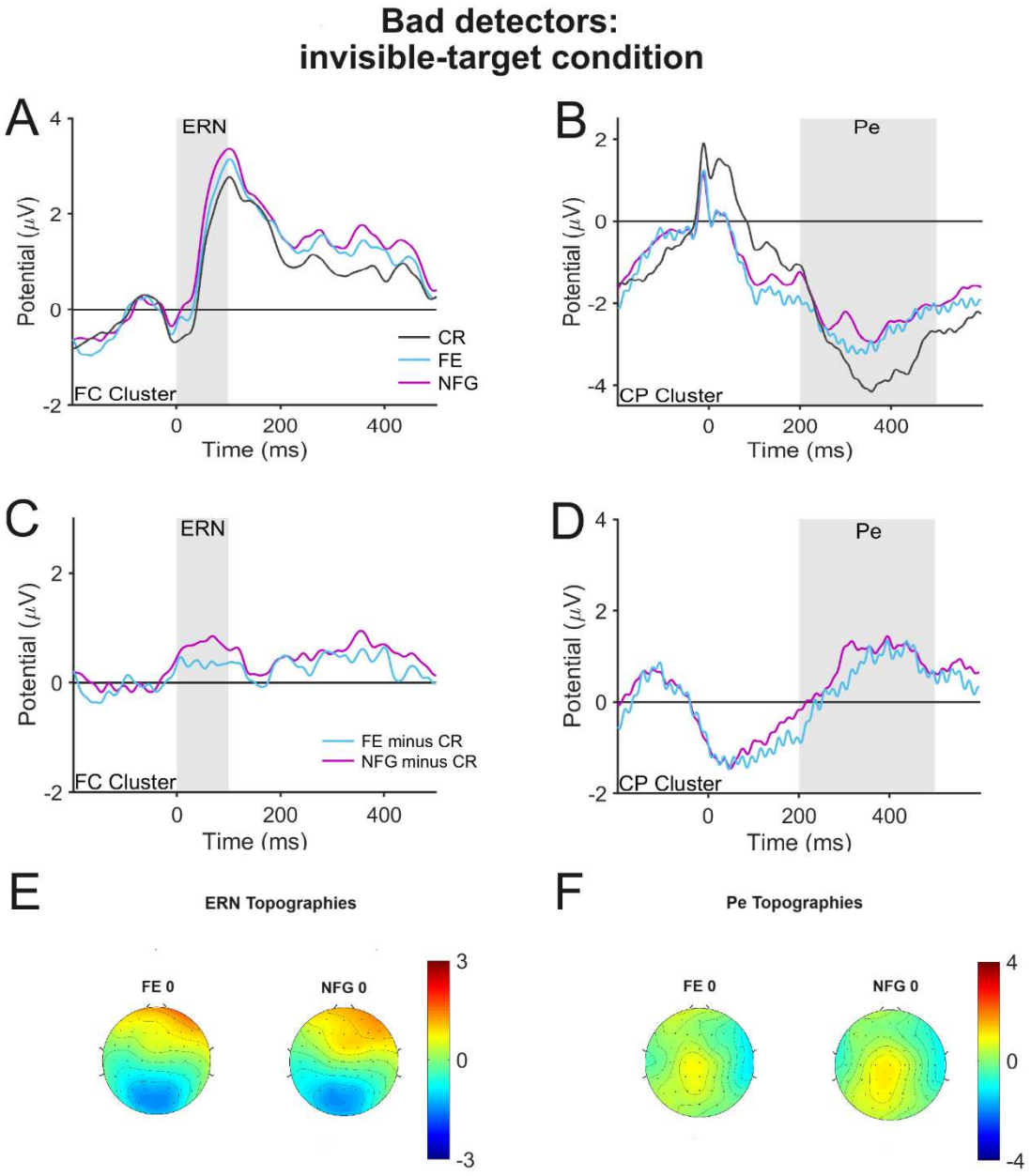

Figure S5. Error-related negativity (Ne/ERN; frontocentral cluster in left column) and error positivity (Pe; centroparietal cluster in right column) in the invisible-target (0-SMI) condition of the bad detectors group. All trials were considered irrespective of whether they were correctly classified or not. Data for correct responses were taken from the 133-SMI condition. AB: Waveforms from all response types. CD: Difference waves for flanker errors (minus corrects) and nonflanker guesses (minus corrects). EF: Topographies of the difference waves in the time range of the Ne/ERN and Pe. Grey areas indicated the time range of the Ne/ERN and Pe in each graph. SMI = stimulus-mask interval, CR = correct response, FE = flanker error, NFG = nonflanker guess, FC = frontocentral, CP = centroparietal.

Table S1. Primary Task Performance

Conditions	Error Rates (%)	Prop. FE (%)	RT Correct (ms)	RT FE (ms)	RT NFE/G (ms)
Good Detectors					
250-SMI	16.0 (±1.7)	57.2 (±2.3)	537 (±17)	531 (±33)	523 (±31)
133-SMI	22.5 (±1.6)	56.8 (±1.5)	563 (±18)	551 (±34)	537 (±27)
0-SMI	-	31.4 (±1.4)	-	551 (±22)	603 (±28)
Bad Detectors					
250-SMI	16.4 (±3.1)	53.9 (±2.3)	499 (±15)	492 (±35)	501 (±48)
133-SMI	26.9 (±2.7)	54.9 (±1.4)	512 (±17)	479 (±23)	490 (±25)
0-SMI	-	38.9 (±3.3)	-	445 (±16)	456 (±25)

Note. NFE/G refers to NFE in the 250-SMI and 133-SMI conditions but NFG in the 0-SMI condition. SMI = stimulus-masking interval, ms = milliseconds, RT = response time, FE = flanker error, NFE = nonflanker error, NFG = nonflanker guess, Prop. = proportion. Within-participants standard errors of the mean are provided in parentheses.

Table S2. Frequencies of Detection Types and Trial Numbers for Flanker Errors and Nonflanker Errors in the Visible-Target Condition for Both Subgroups

Conditions	**Frequencies of Detection Types in % and Averaged Trial Numbers**			
	Good Detectors	**Trials**	**Bad Detectors**	**Trials**
250-SMI				
Flanker Errors				
„correct“	4.3 (± 2.1)	1.2	3.2 (± 1.3)	0.6
„error“	88.6 (± 3.1)	22.2	90.8 (± 2.2)	24.3
„unsure“	7.1 (± 1.8)	1.7	6 (± 2.1)	1.3
Nonflanker Errors				
„correct“	5.2 (± 2)	1.3	1.5 (± 0.9)	0.4
„error“	89.4 (± 3.2)	18.4	89.8 (± 3.6)	19
„unsure“	5.4 (± 2)	1.2	8.6 (± 3.3)	1.6
	Good Detectors	**Trials**	**Bad Detectors**	**Trials**
133-SMI				
Flanker Errors				
„correct“	10.1 (± 2.8)	3.4	4 (± 1)	1.8
„error“	79.4 (± 3.6)	29.7	89.7 (± 2.5)	37.7
„unsure“	10.5 (± 2)	4	6.2 (± 2)	2.9
Nonflanker Errors				
„correct“	5.3 (± 1.3)	1.5	3.4 (± 1.1)	1.3
„error“	82.9 (± 3.2)	23	90.4 (± 2.3)	31.8
„unsure“	11.9 (± 2.3)	3.4	6.2 (± 1.9)	2.3

Note. SMI = stimulus-masking interval. Within-participants standard errors of the mean are provided in parentheses.

Table S3. Frequencies of Detection Types and Trial Numbers for Flanker Errors and Nonflanker Guesses in the Invisible-Target Condition for Both Subgroups

Conditions	Frequencies of Detection Types in % and Averaged Trial Numbers			
	Good Detectors	**Trials**	**Bad Detectors**	**Trials**
Flanker Errors				
„correct“	0.8 (±0.3)	0.8	11.3 (±7.6)	20.3
„error“	81.6 (±4.3)	72.8	3.9 (±2.3)	4.8
„unsure“	17.7 (±4.2)	16.8	84.9 (±8.4)	86.8
Nonflanker Guesses				
„correct“	4.2 (±1.6)	8.2	11.2 (±5.9)	16.2
„error“	2.9 (±0.7)	5.5	5.2 (±3.5)	5.8
„unsure“	93.0 (±1.7)	184	83.6 (±8.2)	154.2

Note. Within-participants standard errors of the mean are provided in parentheses.

Study 3: Error-related pupil dilation but not heart rate deceleration is linked to the error-related negativity

By Julia Dumsky, Martin Maier, and Marco Steinhauser

Abstract

The central and autonomic nervous systems interact to optimize human performance. While the error-related negativity (Ne/ERN) indicates an early error process related to automatic error detection, the error-related positivity (Pe) likely indicates conscious error detection. Performance errors also cause typical autonomic reactions such as heart rate deceleration, and pupil dilation. Because both autonomic parameters develop over a more extended period than the Ne/ERN, it is still an open question whether heart rate deceleration, and pupil dilation are associated with early error processes. In the present study, we, therefore, investigated whether both autonomic parameters occur in the absence of the Ne/ERN by using the target-masking paradigm from Di Gregorio et al. (2018) that permits the occurrence of the Pe without an Ne/ERN. Interestingly, while error-related pupil dilation vanished in the absence of the Ne/ERN, error-related heart rate deceleration was still present. These findings demonstrated for the first time that both autonomic parameters seem to be based on different mechanisms. Error-related pupil dilation may indicate an orienting response, while error-related heart rate deceleration is more associated with the affective processing of errors.

This paper is currently in preparation.

Introduction

Error monitoring is pivotal in our daily lives because it optimizes our behavior. A coordinated interaction between the central, and autonomic nervous systems appears necessary for error-related adjustments. Error monitoring comprises two systems of error detection, indicated by the early error negativity (Ne; Falkenstein, 1990; ERN; Gehring et al., 1993), and the late error positivity (Pe; Falkenstein et al., 1991; Overbeek et al., 2005). While the Ne/ERN is an automatic and rapid marker of error detection, the Pe indicates the conscious evaluation of errors. In addition, there are typical error-related autonomic changes such as phasic pupil dilations (e.g., Braem et al., 2015; Murphy et al., 2016; Rondeel et al., 2015; Wessel et al., 2011), and a decreased heart rate (e.g., Fiehler et al., 2004; Hajcak et al., 2003; Somsen et al., 2004; van der Veen et al., 2000; Wessel et al., 2011). However, the few empirical studies (Wessel et al., 2011; Maier et al., 2019; LoTemplio et al., 2023; Hajcak et al., 2004; Rodeback et al., 2020; van der Veen, Nieuwenhuis et al., 2004; Bury et al., 2019) that investigate these autonomic phenomena together with the Ne/ERN, do not allow any clear conclusions to be drawn about their mutual relationship. The present study, therefore, examines the behavior of error-related pupil dilation, and heart rate deceleration in a paradigm in which the Ne/ERN was absent. Thus, the study addresses whether they are associated with the process that causes the Ne/ERN.

The Ne/ERN is a negative deflection recorded at frontocentral electrodes, typically occurring within the first 100 ms after erroneous responses, peaking at around 50 ms (Gehring et al., 1993). The Pe, a positive deflection following the Ne/ERN, emerges at posterior electrodes between 200 ms and 500 ms after errors (Falkenstein et al., 1990, 1991; Overbeek et al., 2005). Major theories posit the Ne/ERN as a marker of the discrepancy between the correct and the given response (Scheffers & Coles, 2000), as a prediction error (Holroyd & Coles, 2002), or as a post-response conflict (Yeung et al., 2004), signifying an early stage of error processing.

Regardless of its specific function, these theories converge on the idea that the Ne/ERN requires the representation of the correct response. Moreover, the Ne/ERN was additionally associated with affective processing (Dignath et al., 2020) because the Ne/ERN was increased for individuals showing enhanced levels of negative emotions, respectively anxiety (e.g., Suzuki et al., 2020; Aarts & Pourtois, 2010; Aarts et al., 2013; Hajcak et al., 2004; Moser et al., 2013; Nieuwenhuis et al., 2005). This would also be consistent with the idea that errors could have a negative valence (Aarts et al., 2012, 2013; Aarts & Pourtois, 2010; Koban & Pourtois, 2014), especially as they indicate that behavior has failed. The Pe as a marker of later error processes, in turn, is thought to be correlated with error awareness, probably indicating an accumulation process of error occurrence (Steinhauser & Yeung, 2010; Ullsperger, Fischer et al., 2014; Nieuwenhuis et al., 2001).

Pupil dilation, unfolding within the initial two seconds after an event (Steinhauer et al., 2022), could be interpreted as an orienting response to salient stimuli that is based on the unexpectedness of an event (Lynn, 1966; Sokolov, 1963), with the locus coeruleus – norepinephrine (LC-NE) system playing a pivotal role (Abercrombie et al., 2008; Aston-Jones & Bloom, 1981). Typically, error monitoring processes are accompanied by pupil dilation (Braem et al., 2015; Critchley et al., 2005; Murphy et al., 2016; Rondeel et al., 2015; Wessel et al., 2011). It is possible that this error-related pupil dilation also constitutes an orienting response (Murphy et al., 2016; Notebaert et al., 2009) because errors can be interpreted as salient stimuli (Harsay et al., 2012). However, while the impact of NE on pupil dilations is well-established (e.g., Joshi et al., 2016; Preuschoff et al., 2011; Samuels & Szabadi, 2008), the Ne/ERN is purportedly more influenced by dopamine (Kenemans & Kähkönen, 2011). This contradicts the idea that pupil dilation is related to early error processing. Indeed, a robust relationship cannot be conclusively derived from prior studies. Only a handful of studies have investigated whether manipulation of the Ne/ERN by error awareness (Wessel et al., 2011), different error types (Maier et al., 2019), or memory demands as well as state anxiety

(LoTemplio et al., 2023) also affect pupil dilation. While the manipulation of error awareness, and error types resulted in an effect on the Ne/ERN, and pupil dilation (Wessel et al., 2011; Maier et al., 2019), the manipulation of working memory load, and state anxiety only influenced pupil dilation, not the Ne/ERN (LoTemplio et al., 2023). Additionally, LoTemplio et al. (2023), and Wessel et al. (2011) did not directly relate both parameters. Finally, in the study by Maier et al. (2019), the previously observed positive relation between both parameters vanished when analyzed at a single-trial level. To sum up, the empirical findings do not provide a clear relationship between pupil dilation and the Ne/ERN.

The same holds for heart rate deceleration. Heart rate deceleration develops within the initial second following an event and was also interpreted as an orienting response (Sokolov, 1963; e.g., reviewed by Graham & Clifton, 1966; Pribram & McGuinness, 1975; van der Molen et al., 1991). As with pupil dilation, heart rate deceleration typically occurs after error responses (Fiehler et al., 2004; Hajcak et al., 2003; Somsen et al., 2004; van der Veen et al., 2000; Wessel et al., 2011) which has also been interpreted as an orienting response (Łukowska et al., 2018; Notebaert et al., 2009). Another commonality with pupil dilations is that the relationship between the Ne/ERN and heart rate deceleration is still unclear, as recently demonstrated by a comprehensive review (Di Gregorio et al., 2024). Investigations into various aspects such as negative affect (Hajcak et al., 2004), experimentally induced stress (Rodeback et al., 2020), feedback processing (van der Veen, Nieuwenhuis et al., 2004), pitch errors in musicians (Bury et al., 2019), and error awareness (Wessel et al., 2011) have revealed diverse relationships between the Ne/ERN amplitudes and heart rate. While some studies observed no relationship (Hajcak et al., 2004; Rodeback et al., 2020; van der Veen, Nieuwenhuis et al., 2004), others demonstrated that manipulations of early error processes influenced heart rate (Bury et al., 2019; Wessel et al., 2011). However, as already mentioned, Wessel et al. (2011) did not analyze the correlation between Ne/ERN and heart rate. To summarise, neither the relationship between

the Ne/ERN and error-related heart rate deceleration nor between the Ne/ERN and error-related pupil dilation has been empirically clarified.

The current study aims to shed more light on the relationships between the Ne/ERN and both autonomic parameters using the target-masking paradigm from Di Gregorio et al. (2018). The core rationale of this paradigm is to create errors for which participants know that they made an error without knowing the correct response. This makes the Ne/ERN disappear, as it relies on the representation of the correct response according to the major theories. The target-masking paradigm is an adapted version of a letter-based Eriksen-flanker task (Eriksen & Eriksen, 1974). In an invisible-target condition, the central target letter is hidden by a mask, while flanker letters always remain visible. In the visible-target condition, the target letter is masked after a certain amount of time, the correct response is, therefore, still detectable. With the invisible target condition, the target letter is masked from the start, so the correct answer is not visible at all. Nevertheless, in the invisible-target condition, responses to flankers (flanker errors) can still be perceived as errors because participants are instructed that flankers always differ from the central target letter. Using this target-masking paradigm, Di Gregorio et al. (2018) showed that the Ne/ERN disappears in the invisible-target condition while the subsequent Pe can still occur in the absence of the Ne/ERN, which is why they were able to show that the processes underlying the Ne/ERN, and Pe are independent. With this approach, it is also possible to investigate the association between the Ne/ERN and other error-related phenomena, such as pupil dilation, and heart rate deceleration. Different potential outcomes would offer intriguing insights. If error-related pupil dilation, and heart rate deceleration are not observed in the invisible-target condition, hence in the absence of the Ne/ERN, it strongly suggests that error-related pupil dilation, and heart rate deceleration are indeed associated with an early stage of error processing. Otherwise, the relation between both parameters and later error processes, as indicated by the Pe, is more likely to be assumed, which means that heart rate deceleration, and pupil dilation are more related to error awareness.

Method

Participants

Thirty-four participants (21 females) recruited at the Catholic University of Eichstätt-Ingolstadt participated in the study. They were between 18 and 38 years old (M = 23; SD = 4.4), and their vision was normal or corrected to normal. For their participation, they either received course credits or 11 € per hour. The study received approval from the university's ethics committee, and all participants provided informed consent.

Task and Stimuli

The Presentation software (Neurobehavioral Systems, Albany, CA) was used for stimuli presentation on a 21-inch screen with a refresh rate of 80 Hz, and a resolution of 1280 x 1024. The viewing distance was approximately 70 cm. In each trial, participants were required to classify a target letter by pressing the 'A,' 'S,' or 'D' keys on a standard computer keyboard using their left hand's index, middle, and ring fingers, respectively. Each stimulus consisted of seven white letters in Arial font, presented on a black background. The central letter represented the target, surrounded by three identical flanker letters on each side. The letters P, W, M, V, X, or K were used, each 3 mm wide and 5 mm high. Two of the six possible letters were assigned to each response, and the target-response mapping was counterbalanced across participants. Target and flanker letters were always assigned to different responses, which is why all stimuli were incongruent, resulting in 24 possible stimuli. Six different masks, each the same size as a letter, were created by randomly rearranging the features of the original letter stimuli.

In Figure 1, a typical trial is depicted. Each trial began with a fixation cross, presented for 350 milliseconds, followed by the task stimulus. The central target letter was replaced by one of the six masks after a stimulus-masking interval (SMI) of 0 ms (invisible-target condition) or 120 ms (visible-target condition). We, therefore, did not use two different SMI conditions in the

visible-target condition as in the study by Di Gregorio et al. (2018). Instead, to prevent a prediction of the masking onset, we randomly varied the 120 SMI between 101 ms and 150 ms, resulting in an average of 120 ms. In the visible-target condition, the target letter remains visible. In contrast, no target was presented at all in the invisible-target condition (see Fig. 1B). Mask and flankers remained on the screen until the participant responded. After the response, a fixation cross was presented again for a randomly drawn time interval between 1500 ms and 2000 ms.

Each block comprised 72 randomly presented trials, with each of the 24 possible stimuli presented twice in the visible target condition and once in the invisible target condition. One-third of these trials, therefore, belonged to the invisible-target condition and two-thirds to the visible-target condition. The experiment was conducted over two days with a maximum of 48 hours in between. The first day was used to practice the task in twelve blocks, each containing the visible and invisible-target conditions. Throughout the practice session, participants had access to a paper sheet displaying the task rules to provide support. Throughout all practice blocks, a deadline was implemented to train participants to provide their responses within a specific time interval. This was done to obtain a sufficient number of erroneous trials. Automatic feedback about the latency was presented at the center of the screen whenever this deadline was exceeded, urging the participants to respond faster ("SCHNELLER"). Each participant started with the same deadline of 500 ms, which was then adapted on a trial-by-trial basis in the following way (see Steinhauser & Yeung, 2010, for a similar procedure): If the previous trial's response was incorrect, the current trial's deadline was increased by 100 ms. If the previous trial's response was correct, the current trial's deadline was decreased by 20 ms (but could never fall below 100 ms). Applying this 5:1 ratio was used to achieve an approximate error rate of 15% (Kaernbach, 1991). This adaptation on a trial-by-trial basis was only applied in the visible-target condition, while in the invisible-target condition, the deadline remained

unchanged. In addition, if the previous trial was in the invisible-target condition, the deadline in the current trial was adapted based on the last trial in the visible-target condition.

On the second day, the test session was conducted. Initially, two practice blocks consisting of 48 trials were performed, with the deadline procedure included. Subsequently, the participants completed twelve test blocks without automatic feedback, resulting in 576 trials for the visible-target condition and 288 trials for the invisible-target condition. Instead, the participants were instructed to respond faster at the beginning of a block when the average number of errors fell below 32%. Participants received instructions about the tasks at the beginning of each session. They especially received instructions to guess if they could not identify the target letter, as sometimes it may be difficult to see the target. Additionally, they were informed that responding to the flankers would always be an error. The whole test session lasted approximately 1.5 hours, and participants were given short breaks between blocks as required.

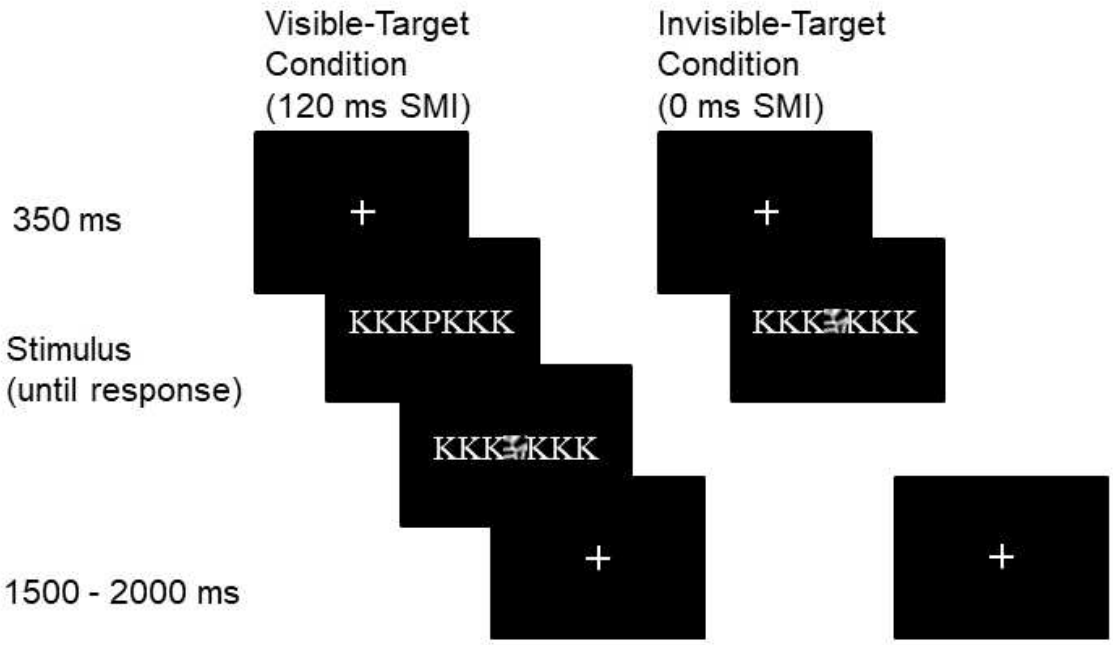

Figure 1. Typical trial sequence. Participants had to respond to the target letter, while flanker letters had to be ignored. The target letter was masked after 120 ms on average in the visible-target condition. Only the mask was presented in the invisible-target condition, but no target. See text for details. SMI = Stimulus mask interval

Psychophysiological recording

Throughout the experiment, the electroencephalogram was recorded using a BIOSEMI Active-Two system (BioSemi, Amsterdam, The Netherlands) with 64 Ag-AgCl electrodes from channels Fp1, AF7, AF3, F1, F3, F5, F7, F17, FC5, FC3, FC1, C1, C3, C5, T7, TP7, CP5, CP3, CP1, P1, P3, P5, P7, P9, PO7, PO3, O1, Iz, Oz, POz, Pz, CPz, Fpz, Fp2, AF8, AF4, AFz, Fz, F2, F4, F6, F8, FT8, FC6, FC4, FC2, FCz, Cz, C2, C4, C6, T8, TP8, CP6, CP4, CP2, P2, P4, P6, P8, P10, PO8, PO4, O2, as well as the left and right mastoid. The CMS (Common Mode Sense), and DRL (Driven Right Leg) electrodes were used as reference and ground electrodes. The vertical and horizontal electrooculogram (EOG) data came from electrodes positioned over and under the right eye and on the outer canthi of both eyes. All electrodes were off-line re-referenced to the right mastoid. For recording the electrocardiogram (ECG), an extra electrode was placed on the right side of the participant's neck to receive heart rate data. EEG and EOG were continuously recorded at a sampling rate of 512 Hz. The eye-tracker was calibrated with a nine-point fixation display for each participant. During the test session, both eyes' horizontal and vertical pupil diameters were collected in arbitrary units (a.u.) with an iView X Red250 mobile system. The sampling rate was 60 Hz.

Data analysis

The analysis was only conducted with the data from the test blocks, and the first three trials from each block were removed from all analyses. Whenever necessary, a Greenhous-Geisser correction (Greenhouse & Geisser, 1959) was conducted, and corrected p-values but uncorrected degrees of freedom were reported in this case.

Response categories. Each trial was categorized based on the response (see Fig. 1 for an example). Three response categories were possible in the visible-target condition: 1) A *correct response* resulted if the given response and target response matched; 2) A *flanker error* resulted if the given response and the flanker response matched; 3) A *nonflanker error* resulted if the

given response matched neither the target nor the flanker response. Two response categories were possible in the invisible-target condition: 1) A *flanker error* resulted if the given response and the flanker response matched. 2) A *nonflanker guess* resulted if the given response and flanker response did not match.

Behavioral data. The behavioral data was analyzed statistically with R Studios version 4.0.3 for Windows (R Core Team, released 2020, Vienna, Austria). The interval between stimulus onset and response was defined as response time (RT). Whenever the RTs deviated more than three standard deviations from the mean of each condition, the corresponding trials were not used for RT analysis. On average, 1.3% of trials ($SE = 0.17\%$) were excluded in this way. Before frequency data was statistically analyzed, an arcsine transformation (Winer, 1971) was performed to stabilize variances, but untransformed data were reported for descriptive statistics. In the visible-target condition, the proportion of error rates and flanker errors (among all errors) were computed. Mean RTs for correct and error trials collapsed across both error types and were subjected to a paired t-test. Mean error RTs were further analyzed using paired t-tests to compare flanker and nonflanker errors. In the invisible-target condition, task performance was analyzed by computing the proportion of flanker errors (among all response types). Mean RTs were analyzed using paired t-tests to compare flanker errors and nonflanker guesses.

ERP data. The analysis of the ERP and other physiological data was conducted with custom routines based on EEGLab v14.1.1 (Delorme & Makeig, 2004) implemented in MatLab R2017b (The Mathworks, Natick, MA). Preprocessing was the same as in Di Gregorio et al. (2018) unless otherwise stated. A bandpass filter was applied to exclude activity below 0.5 Hz and above 40 Hz. Epochs were extracted 200 ms before and 600 ms after response. Baseline correction was applied based on the interval between 150 ms and 50 ms before response because the Ne/ERN typically begins slightly before response execution (e.g., Steinhauser & Yeung, 2010). Epochs were excluded if the amplitude exceeded a threshold of ± 200 μV at any electrode (except at the frontal electrodes, as blinks were later removed) or if the joint

probability deviated more than 5 standard deviations from the epoch mean (according to EEGLAB's pop_jointprob function). In this way, 9.3% of trials (*SE* = 0.06%) were rejected on average. For the analysis, an average of 58 trials (*SE* = 4.5) remained in the condition with the smallest trial number (nonflanker error, visible-target condition). An independent component analysis (ICA; Delorme & Makeig, 2004) was computed, and independent components with blink artifacts were removed using CORRMAP (Campos Viola et al., 2009). In the final step, ERP data was averaged separately for both conditions and per participant. The Ne/ERN was quantified in a time window between 0 ms and 100 ms at a frontocentral electrode cluster around the FCz electrode (Cz, FC1, FCz, FC2, Fz), with more negative values indicating a greater Ne/ERN. The Pe was quantified in a time window between 200 ms and 500 ms at a posterior electrode cluster around the Pz electrode (POz, P1, Pz, P2, CPz), with more positive values indicating a higher Pe. All analyses were done with paired t-tests unless otherwise stated. First, the Ne/ERN and Pe in the visible-target condition were tested for flanker errors and nonflanker errors by comparing error waveforms with correct waveforms. In a further analysis, the error types were directly compared. In the invisible-target condition, we investigated if the Ne/ERN and Pe were present by directly comparing flanker errors and nonflanker guesses against the correct baseline from the visible-target condition. Furthermore, we tested for differences between flanker errors and nonflanker guesses. We then analyzed the ERN and Pe only for flanker errors across both conditions. Finally, we subjected them to a two-way repeated measures ANOVA with the variable SMI (visible-target condition, invisible-target condition) and component (Ne/ERN, Pe) to compare them directly.

Pupillometric data. The first step was to average the binocular horizontal and vertical pupil diameters. Linear interpolation, limited to periods of 1 s or less, was then used to correct for blinks in the pupil data. Epochs were extracted 400 ms before and 2000 ms after the response, and baseline correction was applied based on the interval between 100 ms and 0 ms before the response. Residual artifacts after linear interpolation were rejected. For this rejecting routine,

individual thresholds were used with a minimum of -1/1 a.u. and a maximum of -5/5 a.u. These thresholds were individually adapted for each participant after visual inspection. In this way, 33.9% of all trials (*SE* = 11.6%) were rejected on average. For the analysis, an average of 38.9 trials (*SE* = 3.4) remained in the condition with the smallest trial number (nonflanker error, visible-target condition). In the final step, pupillometric data was averaged separately for both conditions and per participant. Pupil dilation was quantified as the maximum difference between errors and corrects in a time window between 0 ms and 2000 ms, with more positive values indicating a greater dilation. All analyses were done with paired t-tests. First, pupil dilation was tested in the visible-target condition. First, pupil dilations were analyzed for flanker errors and nonflanker errors by comparing them to the corrects. In a further analysis, the error types were directly compared. In the invisible-target condition, we investigated if pupil dilation was present by directly comparing flanker errors and nonflanker guesses against the correct baseline from the visible-target condition. Furthermore, we tested for differences between flanker errors and nonflanker guesses.

Heart rate analysis. To improve the quality of the electrocardiogram and remove EEG and other signal sources, we first ran an ICA on the ECG channel and all EEG channels and visually identified the independent component that best reflected the ECG. Data from this component were then filtered with an 8 Hz high-pass filter and a 20 Hz low-pass filter. R peaks were detected using the FMRIB toolbox (Niazy et al., 2005). In this step, R peaks were removed if their distance to the preceding R peak was smaller than 200 ms to exclude misidentified peaks. The inter-beat interval was calculated as the interval between two successive R peaks and was transformed into a continuous heart rate (beats per minute; bpm) by linearly interpolating values in the inter-beat interval. Epochs were extracted from 500 before to 2000 ms after the response, and baseline correction was applied based on the interval between 100 ms and 0 ms before the response. Finally, epochs were excluded if the standard deviation of the heart rate exceeded a threshold, which was individually determined for each participant after visual inspection.

Finally, epochs were averaged separately for each condition and participant. The heart rate response was quantified as the mean heart rate in the interval between 1000 ms and 2000 ms after the response. Paired t-tests were used to, first, compare errors against corrects for each error type and condition, and second, comparing errors with each other. Again, nonflanker guesses were compared against the correct baseline from the visible-target condition.

Results

Behavioral data

Visible-target condition. First, we analyzed behavioral data in the visible-target condition. The summary of the behavioral results is shown in Table 1. Overall, the error rate was 10.8% (*SE* = 0.9%), with a proportion of flanker errors of 53.9% (*SE* = 0.9%). RTs of correct (*M* = 612 ms, *SE* = 17 ms) and erroneous trials (*M* = 592 ms, *SE* = 24 ms), collapsed across both error types, did not differ significantly, $t(33) = 1.78$, $p = .085$, $d = 0.305$. The difference of RTs between flanker errors (*M* = 583 ms, *SE* = 23 ms) and nonflanker errors (*M* =599 ms, *SE* = 26 ms) also did not reach significance, $t(33) = 2.01$, $p = .052$, $d = 0.345$.

Invisible-target condition. In the second step, we analyzed behavioral data in the invisible-target condition. Since no correct response was available in the invisible-target condition, participants could either commit a flanker error (since responses to flankers were always errors) or a nonflanker guess. In the invisible-target condition, the proportion of flanker errors (among all responses) was 34.5% (*SE* = 1.3%). Furthermore, RTs were higher for nonflanker guesses than for flanker errors, $t(33) = 2.74$, $p = .01$, $d = 0.470$.

Taken together, the main behavioral results were similar to the study conducted by Di Gregorio et al. (2018). In the visible- and invisible-target conditions, a similar proportion of flanker errors were obtained. Next, RTs between flanker errors and nonflanker errors and between flanker errors and nonflanker guesses showed a similar relationship. Nevertheless, in contrast to Di

Gregorio's study, the overall error rate in the visible-target condition was much lower. Moreover, RTs between corrects and errors in the visible-target condition did not differ in our study.

Table 1. Task performance

Conditions	Error rates (%)	Prop. FE (%)	RT CR (ms)	RT FE (ms)	RT NFE (ms)
120 SMI	10.8 (±0.9)	53.9 (±0.9)	612 (±17)	583 (±23)	599 (±26)
				RT FE (ms)	RT NFG (ms)
0 SMI	-	34.5 (±1.3)	-	554 (±16)	572 (±19)

*Note***.** SMI = stimulus-masking interval, ms = milliseconds, RT = response time, CR = correct response, FE = flanker error, NFE = nonflanker error, NFG = nonflanker guess

ERP data

We next analyzed the ERP data to investigate whether the results from Di Gregorio et al. (2018) can be replicated and thus whether the Ne/ERN, but not the Pe is absent in the invisible-target condition.

Visible-target condition. At first, we analyzed the visible-target condition. This was done to demonstrate a typical Ne/ERN and Pe. The Ne/ERN is visualized in Figures 2A and 2C by its waveforms for all possible response types (correct, flanker error, nonflanker error) and difference waves (error minus correct) of both error types and for the frontocentral cluster. In Figure 2E the corresponding topographies of the difference between correct and erroneous responses are shown. In contrast to correct trials, both error types show more negative deflections (Fig. 2AC). These negativities have frontocentral distribution (Fig. 2E), referring to a typical Ne/ERN. The analysis of the difference between erroneous and correct trials revealed a significant Ne/ERN for flanker errors (M = -3.53 μV, SE = 0.71 μV), $t(33) = 4.98$, $p < .001$, d = 0.854, and nonflanker errors (M = -3.46 μV, SE = 0.69 μV), $t(33) = 5.04$, $p < .001$, d =

0.864. Comparing both error types, there was no significant difference, $t(33) = 0.15$, $p = .886$, $d = 0.026$.

In Figures 2B and 2D the Pe is visualized by its waveforms, difference waves, and topographies for the centroparietal cluster. In contrast to correct trials, both error types clearly show more positive deflections (Fig. 2BD) and these positive deflections are maximal at posterior electrodes (Fig. 2F). The analysis of the difference between erroneous and correct trials thus revealed a significant Pe for flanker errors ($M = 4.28$ µV, $SE = 0.6$ µV), $t(33) = 7.14$, $p < .001$, $d = 1.224$) and nonflanker errors ($M = 4.58$ µV, $SE = 0.62$ µV), $t(33) = 7.45$, $p < .001$, $d = 1.278$. The difference between both error types did not reach significance, $t(33) = 0.87$, $p = .389$, $d = 0.149$.

Invisible-target condition. The same analyses were done for the invisible-target condition. We had to deal with the problem of having no correct trials in the invisible target condition to use as a baseline for quantifying Ne/ERN and Pe. As in our previous studies, two strategies were considered in determining whether an Ne/ERN or Pe was present: First, since no correct response was available, correct trials of the visible-target condition were used for the analyses. Second, flanker errors and nonflanker guesses were compared in the invisible-target condition. Since flanker errors provide more objective information about the occurrence of errors, a stronger error signal and, thus, larger Ne/ERN and Pe amplitudes should result. To examine, whether an Ne/ERN is present in the invisible-target condition we considered the waveforms as well as difference waves for all possible response types (flanker error, nonflanker guess) for the frontocentral cluster and topographies of the difference between correct and erroneous responses (Fig. 3ACE). The difference between correct trials and both error types is less pronounced than in the visible-target condition and shows no difference between both error types (Fig. AC). No evidence of frontocentral negativity interpreted as Ne/ERN can be found in the topographies, supported by the statistical analyses. The analysis of the difference between erroneous and correct trials neither revealed a significant Ne/ERN for flanker errors ($M = -0.97$

μV, *SE* = 0.75 μV), *t*(33) = 1.29, *p* = .206, *d* = 0.221, nor for nonflanker guesses (*M* = -0.76 μV, *SE* = 0.58 μV), *t*(33) = 1.32, *p* = .197, *d* = 0.226. Also, both response types did not differ significantly, *t*(33) = 0.71, *p* = .481, *d* = 0.122.

Next, we considered the waveforms and difference waves from the centroparietal electrode cluster (Fig. 3BD), indicating the Pe. In relation to the correct baseline, a clear positivity is shown for flanker errors and nonflanker guesses. Moreover, the Pe for nonflanker guesses is smaller than for flanker errors. These observations were corroborated by the statistical analyses, showing a significant positivity for flanker errors (*M* = 3.14 μV, *SE* = 0.61 μV), *t*(33) = 5.14, *p* < .001, *d* = 0.882, and for nonflanker guesses (*M* = 1.96 μV, *SE* = 0.54 μV), *t*(33) = 3.65, *p* < .001, *d* = 0.626. In addition, the difference between both error types also reached significance, *t*(33) = 3.45, *p* = .002, *d* = 0.592, with a higher Pe amplitude for flanker errors than for nonflanker guesses.

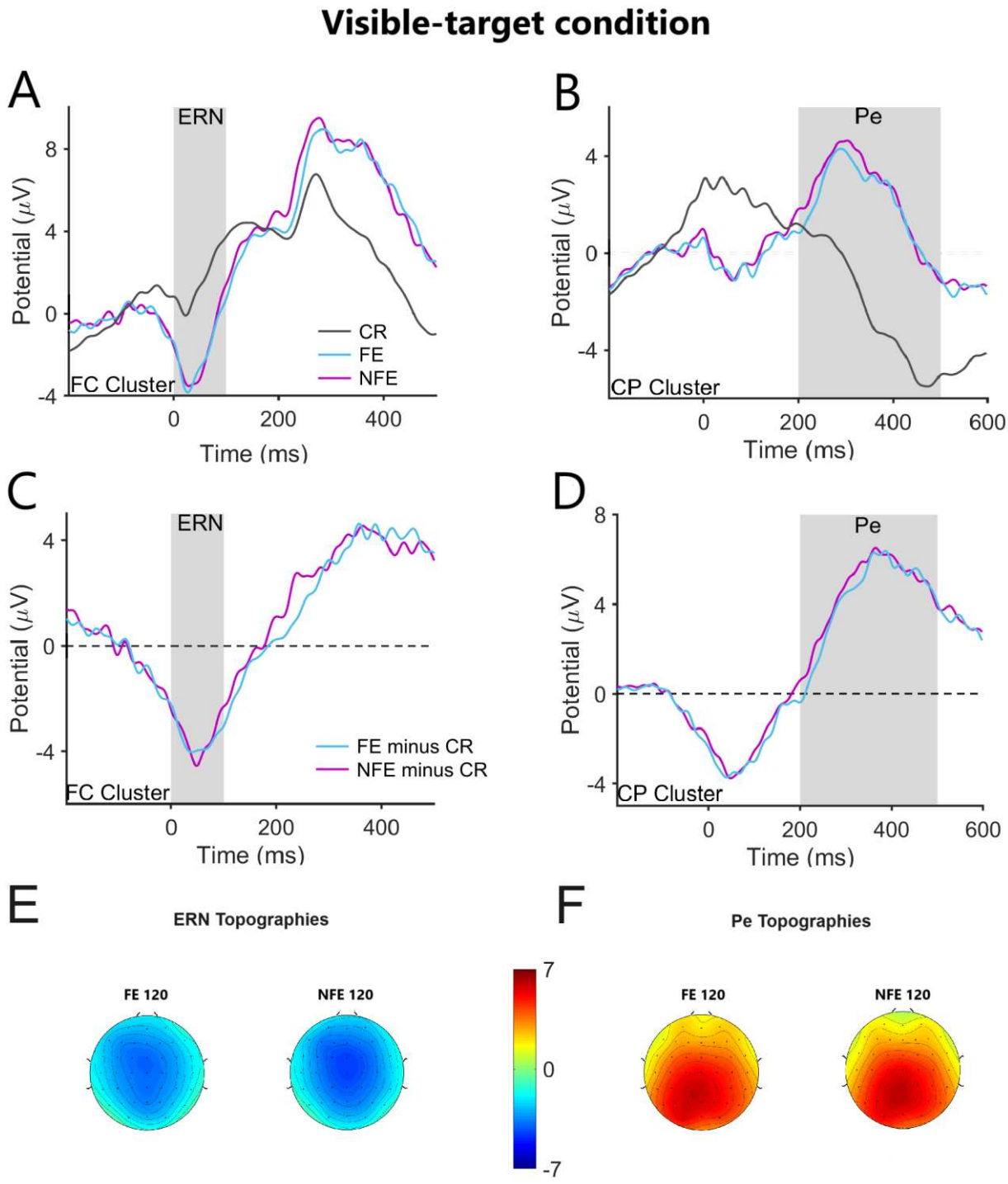

Figure 2. Error-related negativity (Ne/ERN; frontocentral cluster in left column), and error positivity (Pe; centroparietal cluster in right column) in the visible-target (120 SMI) condition. AB: Waveforms from all response types. CD: Difference waves for flanker errors (minus corrects), and nonflanker errors (minus corrects). EF: Topographies of the difference waves in the time range of the Ne/ERN and Pe. Grey areas indicated the time range of the Ne/ERN and Pe in each graph. SMI = stimulus-mask interval, CR = correct response, FE = flanker error, NFE = nonflanker error, FC = frontocentral, CP = centroparietal

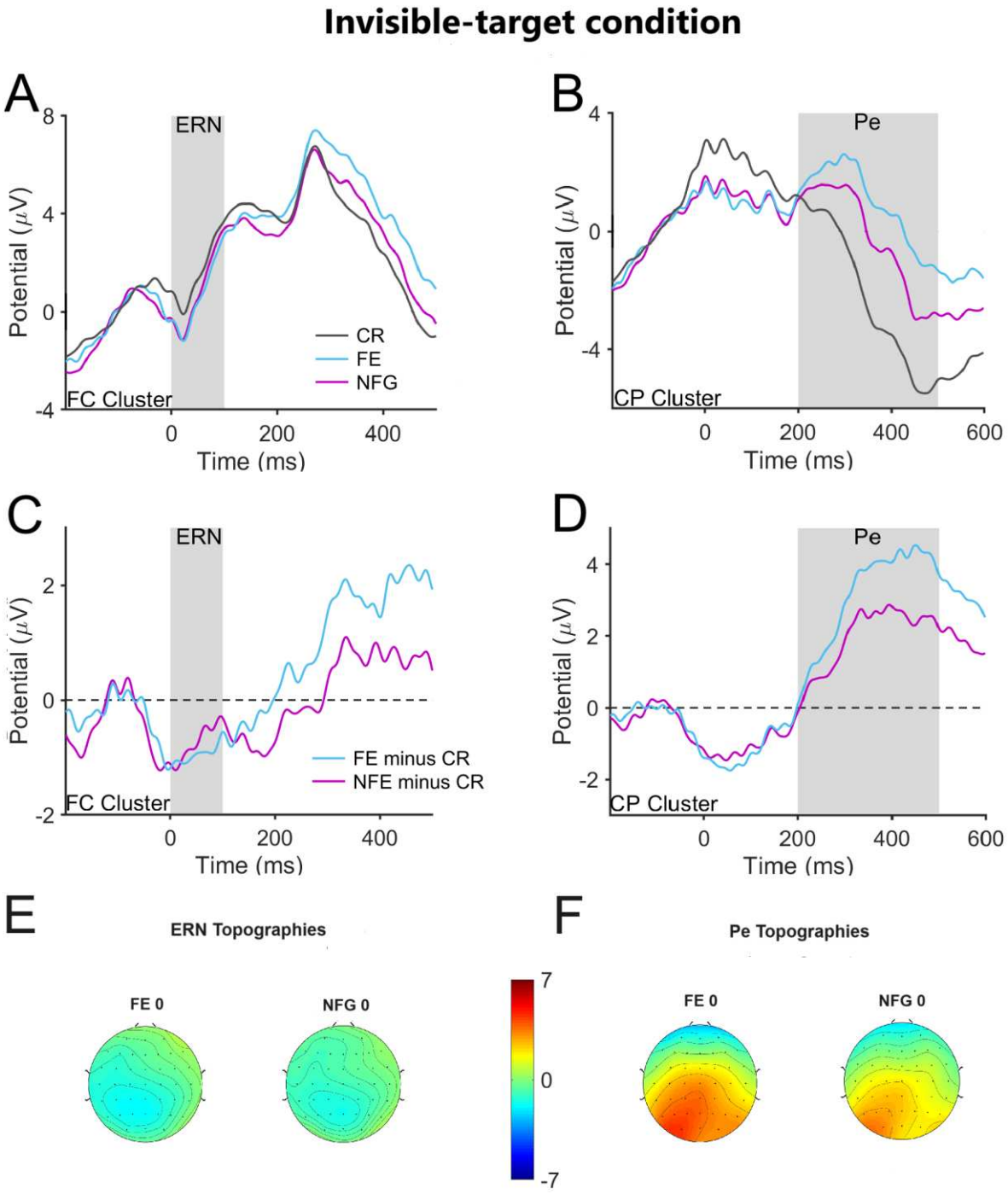

Figure 3. Error-related negativity (Ne/ERN; frontocentral cluster in left column) and error positivity (Pe; centroparietal cluster in right column) in the invisible-target (0 SMI) condition. AB: Waveforms from all response types. CD: Difference waves for flanker errors (minus corrects) and nonflanker guesses (minus corrects). EF: Topographies of the difference waves in the time range of the Ne/ERN and Pe. Grey areas indicated the time range of the Ne/ERN and Pe in each graph. CR = correct response, FE = flanker error, NFG = nonflanker guess, FC = frontocentral, CP = centroparietal

In a final step, Ne/ERN and Pe amplitudes were compared across conditions. Because flanker errors were available in the invisible-target and the visible-target condition, only this error type was included in the analyses. The Ne/ERN amplitudes of flanker errors significantly differed between both conditions, $t(33) = 4.7$, $p < .001$, $d = 0.807$, with higher amplitudes for flanker errors in the visible-target condition (M = -3.53 μV, SE = 0.71 μV) than in the invisible-target condition (M = -0.97 μV, SE = 0.75 μV). Also, Pe amplitudes for flanker errors showed a significant difference between both conditions, $t(33) = 2.45$, $p = .02$, $d = 0.420$, with a higher Pe for flanker errors in the visible-target condition (M = 4.28 μV, SE = 0.6 μV) than in the invisible-target condition (M = 3.14 μV, SE = 0.61 μV). The two-way ANOVA with the variables visibility (visible-target and invisible-target) and components (Ne/ERN and Pe) revealed a significant effect for visibility, $F(1, 33) = 4.9$, $p = .034$, $\eta_p^2 = 0.129$, as well as for the variable component, $F(1, 33) = 65.7$, $p < .001$, $\eta_p^2 = 0.666$. Most important, the interaction between visibility and component became significant, $F(1, 33) = 22$, $p < .001$, $\eta_p^2 = 0.95$, revealing that both components varied differently across conditions.

In sum, the Ne/ERN and the Pe were highly significant for both error types in the visible-target condition, while the Ne/ERN, but not the Pe, was absent in the invisible-target condition. Moreover, the Pe for both error types differed significantly in the invisible-target condition, while there was no significant difference in the visible-target condition. Further analyses corroborated that both components show a different pattern across conditions. Overall, these results align with Di Gregorio et al. (2018).

Pupillometric data

One main goal of the current study was to examine pupil responses in the absence of the Ne/ERN to investigate whether they relate to early error processes.

Visible-target condition. First, we analyzed the visible-target condition to show that typical error-related pupil dilation occurs. Pupil dilations are visualized in Figures 4A and 4C by their

waveforms for all possible response types (correct, flanker error, nonflanker error) and difference waves (error minus correct) for both error types. In contrast to correct trials, both error types show more positive deflections after 500 ms post-response (Fig. 4AC). The analysis on the maximum between erroneous and correct trials revealed a significant pupil dilation for flanker errors (M = 0.38 a.u., SE = 0.05 a.u.), $t(33) = 2.08$, $p = .045$, $d = 0.357$) and nonflanker errors (M = 0.4 a.u., SE = 0.05 a.u.; $t(33) = 2.8$, $p = .009$, $d = 0.48$). Comparing both error types, there was no significant difference, $t(33) = 0.3$, $p = .77$, $d = 0.051$.

Invisible-target condition. The same analyses were done for the invisible-target condition, using correct trials from the visible-target condition as the baseline. In Figures 4B and 4D, pupil dilations are visualized by their waveforms and difference waves for all possible response types (flanker error, nonflanker guess). In contrast to correct trials, both error types show smaller dilations starting at around 500 ms post-response (Fig. 4BD). The analysis on the maximum of pupil dilation did not reach significance for flanker errors (M = 0.29 a.u., SE = 0.04 a.u.), $t(33) = 0.72$, $p = .48$, $d = 0.122$), but for nonflanker guesses, $t(33) = 2.56$, $p = .015$, $d = 0.438$) with smaller pupil dilations for nonflanker guesses (M = 0.26 a.u., SE = 0.03 a.u.) than corrects (M = 0.31 a.u., SE = 0.04 a.u.). The difference between flanker errors and nonflanker guesses did not reach significance, $t(33) = 1.07$, $p = .294$, $d = 0.183$.

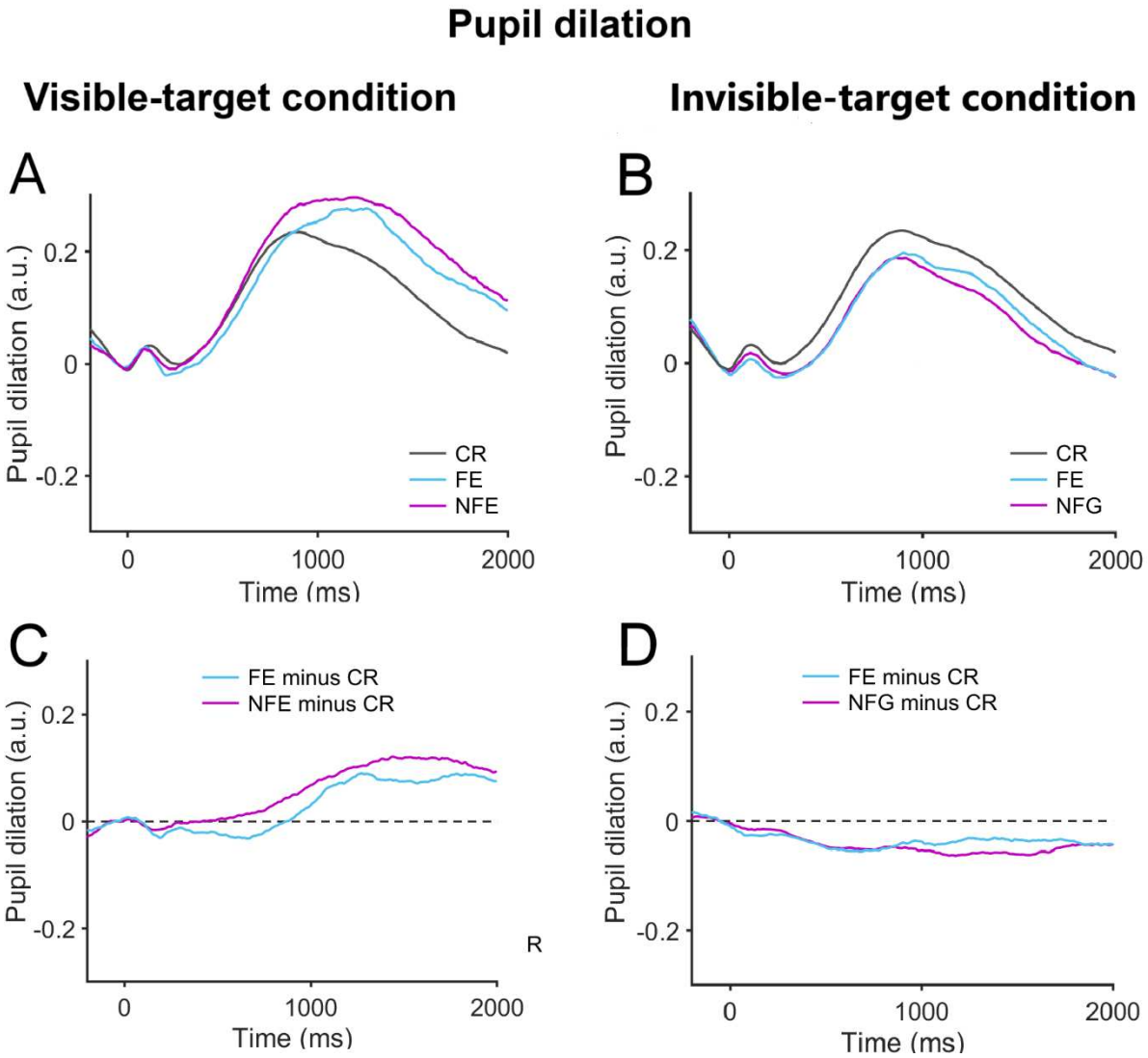

Figure 4. Pupil dilation data in the visible-target (120 SMI) condition (left column) and invisible-target (0 SMI) condition (right column). AB: Waveforms of both conditions and of the corresponding response types. CD: Difference waves of both conditions and the corresponding response types. CR = correct response, FE = flanker error, NFG = nonflanker guess.

Heart rate data

The second main goal of the current study was to examine heart rate in the absence of the Ne/ERN. We investigated whether heart rate after errors is related to early error processes.

Visible-target condition. In the first step, the visible-target condition was analyzed to demonstrate that the typical error-related heart rate occurs. Heart rate data is visualized in Figures 5A and 5C by its waveforms for all possible response types (correct, flanker error, nonflanker error) and difference waves (error minus correct) for both error types. In contrast to correct trials, both error types show an increased deceleration starting at around 500 ms post-response (Fig. 5AC). The analysis of the difference between errors and corrects revealed a heart rate deceleration for flanker errors (M = -0.22 bpm, SE = 0.15 bpm), $t(31) = 3.8$, $p < .001$, d = 0.672) and nonflanker errors (M = -0.17 bpm, SE = 0.15 bpm); $t(31) = 4.21$, $p < .001$, $d = 0.744$). Comparing both error types, there was no significant difference, $t(31) = 0.05$, $p = .82$, $d = 0.009$.

Invisible-target condition. The same analyses were done for the invisible-target condition, using correct trials from the visible-target condition as the baseline. In Figures 5B and 5D, heart rate is visualized by its waveforms and difference waves for all possible response types (flanker error, nonflanker guess). In contrast to correct trials, both error types show a stronger deceleration starting at around 500 ms post-response (Fig. 5BD). The analysis on the difference between flanker errors and corrects reached significance for flanker errors, $t(31) = 3.75$, $p <$ .001, $d = 0.663$), with a stronger deceleration for flanker errors (M = -0.004 bpm, SE = 0.11 bpm) than corrects (M = 0.35 bpm, SE = 0.10 bpm). The same analysis on the difference between nonflanker guesses and corrects did not reach significance, $t(31) = 1.93$, $p = .063$, d = 0.341 (M = 0.15 bpm, SE = 0.10 bpm). The difference between flanker errors and nonflanker guesses also did not reach significance, $t(31) = 1.32$, $p = .197$, $d = 0.233$.

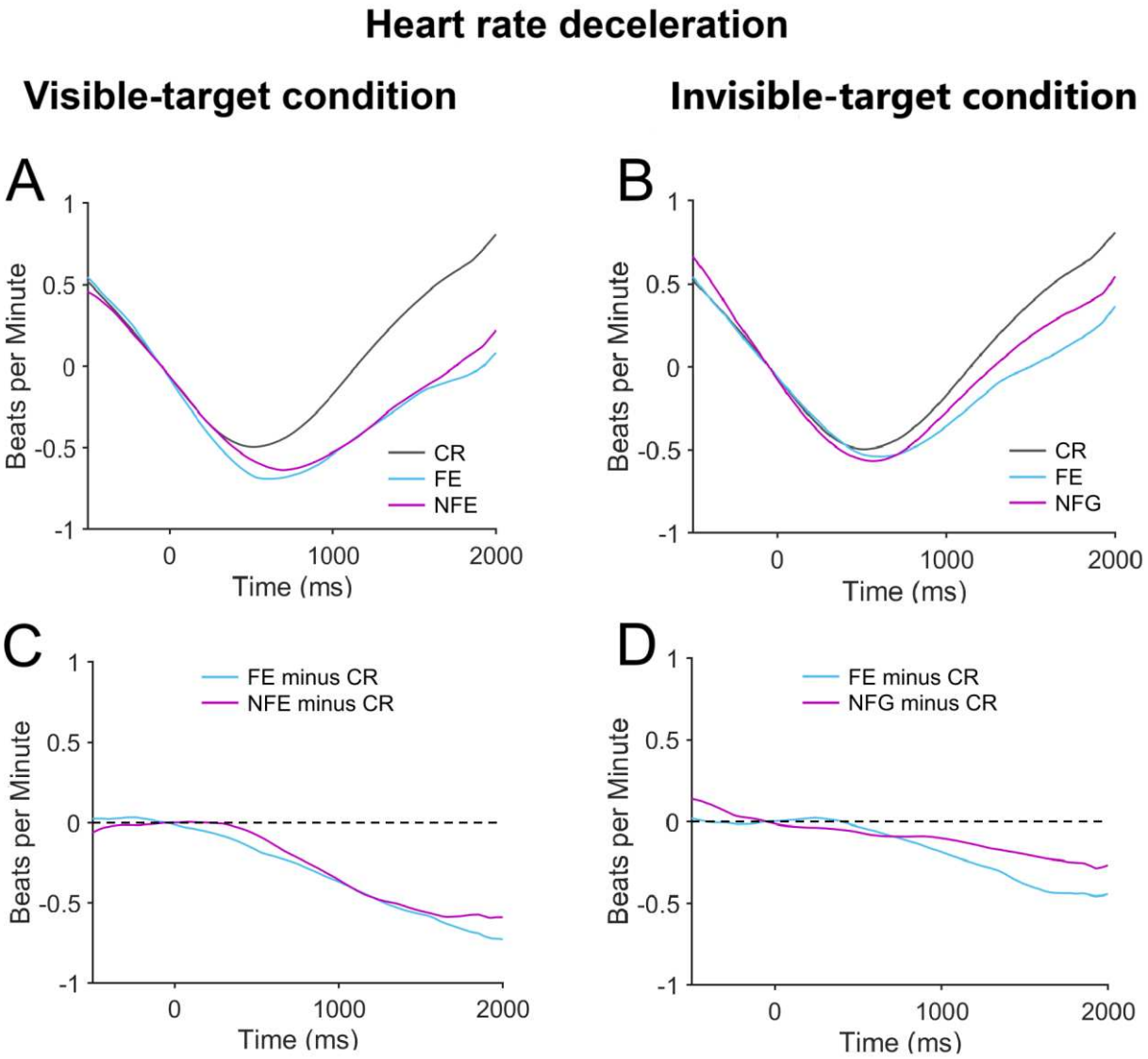

Figure 5. Heart rate deceleration data in the visible-target condition (left column) and invisible-target condition (right column). AB: Waveforms of both conditions and the corresponding response types. CD: Difference waves of both conditions and the corresponding response types. CR = correct response, FE = flanker error, NFG = nonflanker guess

Finally, we also compared flanker errors across both conditions. Because flanker errors were available in the invisible-target and the visible-target condition, only this error type was included in the analyses. The analyses revealed no significant difference, $t(31) = 1.58, p = .125$, $d = 0.279$. Overall, we found typical error-related heart rate deceleration in the visible-target condition and flanker errors in the invisible-target condition. Furthermore, heart rate decelerations for flanker errors did not differ significantly across conditions.

Discussion

The present study investigated whether error-related pupil dilation and heart rate deceleration are associated with the Ne/ERN. Results from previous studies are inconsistent and do not allow a clear conclusion on the relationship between the Ne/ERN and the two autonomic signals. To directly address this relationship, we used the target-masking paradigm from Di Gregorio et al. (2018). With this paradigm, participants can detect errors without knowing the correct response. Consequently, the Ne/ERN, relying on the representation of the correct response, disappears, while the Pe remains unaffected. This dissociation between the Ne/ERN and the Pe enables us to study the relationship between error-related signals such as pupil dilation as well as heart rate deceleration and early error processes, as indicated by the Ne/ERN.

Our results show an interesting pattern. While error-related pupil dilation vanished in the absence of the Ne/ERN, error-related heart rate deceleration was unaffected, i.e., our study suggests a more complex interplay between cognitive and autonomic processes after errors. As shown by Di Gregorio et al. (2018), there are two independent systems. One system underlies the Ne/ERN, and a second system underlies the Pe. Concerning our results, error-related pupil dilation seems to be associated with the Ne/ERN and, therefore, with early error processes. Pupil dilation after errors is interpreted as part of an orienting response, i.e., a physiological increase in arousal due to infrequent or unexpected events such as errors (e.g., Critchley et al., 2005; Danev & de Winter, 1971; Hajcak et al., 2003; Wessel et al., 2011). Indeed, Braem et al. (2015) and Alamia et al. (2019) confirm the relationship between pupil dilation and unexpectedness.

However, considering the Ne/ERN as a conflict signal, it is not plausible that this conflict signal could already include information about the violation of expectations for the outcome at this early stage. Nevertheless, because error-related pupil dilation is related to the Ne/ERN, the Ne/ERN must comprise at least two different processes, a perspective that was corroborated by

Buzzell et al. (2017). Hypothetically, a post-response conflict may be detected first, leading to calculating the expectancy violation of the outcome in a second step. If the orienting response is indeed the basis of error-related pupil dilations and unexpected events cause the orienting response, it is conceivable that pupil dilation could resemble this second step. Because in the invisible-target condition of our third study, no correct response was provided, no post-response conflict could occur and, thus, the calculation of the expectancy violation was not possible. This could be why error-related pupil dilation vanished in the absence of the Ne/ERN.

An alternative explanation would be that the Ne/ERN already indicates the violation of an expected outcome, as stated, for example, by Alexander & Brown (2011) and Holroyd & Coles (2002). This explanation posits that the Ne/ERN is higher in conditions where error responses are less probable or, in other words, where correct responses are more expected. Because in the invisible-target condition, no correct response was available, correct responses could not be expected. This could explain why there was no Ne/ERN and pupil dilation. However, flanker errors were the only responses where participants could be sure about its outcome, while they did not know the actual outcome in the case of nonflanker guesses. In this vein, flanker errors violated the expected outcome of correct responses more clearly than nonflanker guesses, which is why there should have been an effect on the Ne/ERN or pupil dilation, respectively. Although the question cannot be fully answered within this study's scope, it could be possible that the Ne/ERN indicates a postresponse-conflict in the first step.

The absence of pupil dilation in the presence of a Pe in the invisible-target condition speaks against the notion that the Pe is linked to an orienting response or is a P3-like component. The orienting response is based on an NE release into the cortical target areas, which causes an interruption of ongoing behavior and a reorientation to the unexpected event (Bouret & Sara, 2005; Sara & Bouret, 2012). Wessel et al. (2011) and Wessel (2012) suggested that the orienting response is associated with error awareness and thus with the Pe, which is thought to be a P3-like component (e.g., Overbeek et al., 2005; Ridderinkhof et al., 2009). The P3, in turn, is

considered by some researchers to be an index of the orienting response (Nieuwenhuis et al., 2011; Ritter et al., 1968). However, the relationship between the P3 and the LC-NE system is less clear (e.g., Kamp & Donchin, 2015; LoTemplio et al., 2021; Lu et al., 2023; Nieuwenhuis et al., 2001) than the relationship between pupil dilation and the LC-NE system (e.g., Gilzenrat et al., 2010; Hou et al., 2005; Morad et al., 2000; Murphy et al., 2011; Murphy et al., 2014; Phillips et al., 2000; Rajkowski et al., 1993). Riba et al. (2005) again showed an effect of a highly selective NE antagonist on the Ne/ERN, which would be consistent with our results. Although the interplay between NE and other neurotransmitter systems is too complex to attribute neural phenomena exclusively to specific neurotransmitters (see also Warren et al., 2023), our finding speaks against the relationship between the orienting response, very likely indicated by error-related pupil dilation and the Pe. However, in this case, the orienting response or error-related pupil dilation could still be one source of error evidence. Still, it seems not to be necessary for the Pe, as Wessel (2018) stated.

In contrast to pupil dilation, the error-related heart rate deceleration was not associated with the Ne/ERN; i.e., in our study, there was a significant heart rate deceleration in the absence of the Ne/ERN. That also means that the generation of the Pe was sufficient to elicit the error-related heart rate deceleration. In this vein, the lack of unexpected errors in the invisible-target condition cannot be the basis for the error-related heart rate deceleration. However, because errors were still detectable, this autonomic signal could be the affective response to these errors associated with error awareness. Indeed, in previous studies, negative emotional stimuli or negative external feedback affected heart rate deceleration (Bradley et al., 1996; Bradley et al., 2001; Bradley & Lang, 2000) or negative external feedback (Somsen et al., 2000). In turn, Hajcak et al. (2003) could extend these results by demonstrating that internal processes such as error processing also led to a significant heart rate deceleration. Heart rate deceleration in the context of error processing could be interpreted in terms of valence (negative and positive outcomes; van der Veen et al., 2000, van der Veen, Nieuwenhuis et al., 2004). Typically, errors

have a negative valence (Aarts et al., 2012; Aarts et al., 2013; Aarts & Pourtois, 2010; Koban & Pourtois, 2014) because they indicate that a performance failed. So, the presence of a heart rate deceleration without an Ne/ERN in our study could be explained in terms of the affective processing of errors, which were still detectable.

Moreover, error-related heart rate deceleration in our study cannot be associated with conflict processing because no response conflict occurred in the invisible-target condition.

So, if heart rate deceleration reflects affective processing, it cannot be based on the negative valence of a conflict. This is why error-related heart rate deceleration cannot be explained in the context of the affective-signaling hypothesis, an extension of the conflict monitoring theory (Dignath et al., 2020; Saunders et al., 2017). According to the affective-signaling hypothesis, response conflicts cause negative affect, and this affective signal is seen as the actual basis of the monitoring process as indexed by the Ne/ERN, leading to adaptations in cognitive control (Dignath et al., 2020). However, in our study, response conflict and the affective processing of errors were deconfounded, as indicated by the significant heart rate deceleration in the absence of the Ne/ERN. Our results would also align with Hajcak et al. (2003), who found no influence of different degrees of conflict on heart rate deceleration. Thus, our results show that heart rate deceleration is associated with error but not conflict processing. Instead, the presence of the Pe was sufficient for the error-related heart rate deceleration. It is, therefore, possible that error awareness, as indicated by the Pe, impacts the affective processing of errors (Ullsperger et al., 2010). If this is the case, our results suggest that heart rate deceleration follows rather than precedes error awareness.

So, pupil dilation and heart rate deceleration showed different patterns. This is why the same aspect of error monitoring cannot elicit both autonomic signals. While error-related pupil dilation seems to be based on the unexpectedness of errors and thus indicates an orienting response, error-related heart rate deceleration likely reflects the affective processing of an error. Although error-related heart rate deceleration was also interpreted as an orienting response

(e.g., Critchley et al., 2005; Danev & de Winter, 1971; Hajcak et al., 2003; Wessel et al., 2011), at least the unexpectedness does not play a crucial role. Heart rate deceleration may be indeed a later part of the orienting response. Considering the neurotransmitters involved in error-related pupil dilation and heart rate deceleration, this provides further evidence that the two signals are based on different mechanisms. As mentioned above, there is a tight link between pupil dilation and NE (e.g., Gilzenrat et al., 2010; Hou et al., 2005; Morad et al., 2000; Murphy et al., 2011; Murphy et al., 2014; Phillips et al., 2000; Rajkowski et al., 1993). While NE accelerates the heart rate, acetylcholine, in turn, decelerates it via the parasympathetic nervous system (Berntson et al., 2016).

Additionally, the dissociation between the Ne/ERN and Pe from Di Gregorio et al. (2018) could be replicated. While there was a significant Ne/ERN and Pe in the visible-target condition, the Ne/ERN vanished while the Pe still occurred in the invisible-target condition. This result corroborates that the Ne/ERN is indeed based on the representation of the correct response, which aligns with the major theories. Instead, this representation is not necessary for the Pe, respectively error awareness. Also, the difference between the Pe amplitudes of the flanker errors and nonflanker guesses in the invisible-target condition was replicated. As Di Gregorio et al. (2018) already discussed, the difference between flanker errors and nonflanker guesses is that flanker errors obtained the objective information that an error was made, while correct responses and nonflanker errors could not be distinguished by participants, resulting in nonflanker guesses. This confirms that the Pe is a gradual component and can indicate different levels of error evidence (Steinhauser & Yeung, 2010) or error confidence (Boldt & Yeung, 2015).

Our study showed for the first time that error-related pupil dilation and error-related heart rate deceleration reflect autonomic signals, which are associated differently with the Ne/ERN and Pe. The error-related pupil dilation is related to the Ne/ERN and, thus, to early error monitoring processes. Pupil dilation likely resembles an orienting response. On the other hand, the error-

related deceleration is not associated with the Ne/ERN but likely with the Pe. It could represent the conscious processing of the affective content of an error. In sum, both autonomic signals could be successfully dissociated.

General discussion

The focus of the present thesis was to further elucidate the metacognitive aspects of error monitoring, especially the relationship between early error processes as indicated by the Ne/ERN and other error-related phenomena such as conflict, error awareness, pupil dilation, and heart rate deceleration. The following discussion begins by outlining all three studies and the corresponding findings. Afterward, the functional role of the Ne/ERN will be discussed in light of these results before discussing the relationship between error awareness and the Ne/ERN, as well as the findings according to the error-related pupil dilation and heart rate deceleration. Finally, a proposal is made for a possible temporal sequence of the corresponding processes.

Overview of studies

In *Study 1*, we employed a color-based Eriksen flanker task (Eriksen & Eriksen, 1974) to examine whether pre-response conflict, as reflected by the N2, is further amplified and post-response conflict, as indicated by the Ne/ERN, is diminished if two incorrect responses compete with the correct response as compared to one incorrect response. Specifically, we only used incongruent stimuli with either one additional (same-flankers condition) or two additional competing flanker responses (different-flankers condition). Indeed, results show that two additional competing flanker responses further increased the N2 for correct trials and further decreased the Ne/ERN for error trials (Dumsky et al., 2023), which is in line with central predictions of the conflict-monitoring theory, a computational model of conflict processing (Botvinick et al., 2001; Yeung et al., 2004) as well as the mathematical formula, derived from the Hopfield energy (Hopfield, 1982). This formula states that each additional competing response should enhance the potential overall conflict in a task. Indeed, my study demonstrates

that the pre- and post-response conflicts are directly linked to the number of competing responses.

In *Study 2*, the target-masking paradigm from Di Gregorio et al. (2018) was used and extended by an error-signalling task. This target-masking paradigm allows the creation of flanker errors that participants can notice without knowing the correct response. Specifically, while the target was replaced by a mask after a specific time, flankers always remained visible. In the visible-target condition, the target was still perceivable despite masking. In turn, in the invisible-target condition, the target was unavailable. This manipulation resulted in the absence of the Ne/ERN because it is based on representing the correct response according to major theories. Although Di Gregorio et al. (2018) demonstrated that the Pe can occur without the Ne/ERN, it was still unclear whether error awareness is possible in this condition, which is why participants were additionally instructed to evaluate their responses in my study. Indeed, my results show that errors can be consciously detected even in the absence of an Ne/ERN, i.e., early error processes are not necessary for error awareness (Dumsky et al., 2025). However, a subgroup of participants could not detect flanker errors without the Ne/ERN, although they had no problem when it was present. This indicates that there could be interindividual differences in the ability to consciously detect errors in the absence of the Ne/ERN.

In *Study 3*, the target-masking paradigm by Di Gregorio et al. (2018) was applied to investigate how error-related pupil dilation and heart rate deceleration relate to early error processes or the Ne/ERN, respectively. Results demonstrate that the interplay between early error processes and these autonomic signals is very complex. So, while error-related pupil dilations also vanished in the absence of the Ne/ERN, heart rate deceleration was not affected by this manipulation. In this vein, I successfully dissociated heart rate deceleration and pupil dilation.

The functional role of the Ne/ERN further elucidated

Given these results, the functional role of the Ne/ERN becomes clearer. Two major classes of theories suggest that the Ne/ERN either reflects the violation of an outcome expectation (e.g., reinforcement learning theory, Holroyd & Coles, 2002; PRO-Model, Alexander & Brown, 2011) or a post-response conflict (conflict-monitoring theory, Botvinick et al., 2001; Yeung et al., 2004). Theories, which are based on the violation of an expected outcome, a higher Ne/ERN occurs due to less expected errors. This means that experimental conditions that are characterized by different error rates should also differ for Ne/ERN amplitudes, as demonstrated in previous studies (e.g., Falkenstein et al., 1995; Gehring et al., 1993; Jessup et al., 2010; Oliveira et al., 2007; but see e.g., Maier & Steinhauser, 2013; Maier & Steinhauser, 2016).

The findings of *Study 1* showed that differently strong response conflicts affect the N2 as pre-response conflict and the Ne/ERN as post-response conflict (Dumsky et al., 2023). Specifically, the N2 was further enhanced and the Ne/ERN was further reduced with higher conflict levels, while error rates did not differ between conditions, although we did not control for it. While theories based on the violation of expectancies have difficulties explaining these ERP effects without differences in error rates, these results align with the conflict-monitoring theory. Generally, higher conflicts are associated with higher error rates (e.g., Asanowicz et al., 2021; Jost et al., 2022; Yeung et al., 2004; Gajewski et al., 2008). However, it is possible that our paradigm, which only utilized incongruent trials, led to a general enhancement of control processes, which suppressed this effect. Other studies also showed that a high frequency of incongruent stimuli reduced error rates (Bartholow et al., 2005; Corballis & Gratton, 2003; Grützmann, Riesel et al., 2014; Wendt et al., 2008; Wendt & Luna-Rodriguez, 2009). Therefore, it can be assumed that in *Study 1*, the N2 and Ne/ERN were exclusively driven by different degrees of post-response conflict instead of the unexpectedness of errors.

Study 3 demonstrated that error-related pupil dilation is not linked to the Pe, but to the Ne/ERN, i.e., to early error processes, because pupil dilation vanished in the absence of the Ne/ERN. Interpreting the Ne/ERN as post-response conflict (Botvinick et al., 2001; Yeung et al., 2004) means that pupil dilation must be related to conflict processing and not to error awareness, as indicated by the Pe. Error-related pupil dilations, in turn, were interpreted as an orienting response (Murphy et al., 2016; Notebaert et al., 2009), which is reflected by enhanced physiological arousal after salient and unexpected events (Sokolov, 1963; e.g., reviewed by Graham & Clifton, 1966; Pribram & McGuinness, 1975; van der Molen et al., 1991). This association between unexpectedness and pupil dilation was recently corroborated by Alamia et al. (2019).

Although the specific temporal dynamic between the Ne/ERN and error-related pupil dilation cannot be inferred from my results, it can be speculated that there are two processes that develop in succession. First, there is the Ne/ERN, which is based on conflict detection and indicates that an error has occurred but is unlikely to already contain information about the violation of the expected outcome. Hence, this has to be calculated in a second step. Assuming that error-related pupil dilation expresses an orienting response and the orienting response is indeed based on the unexpectedness of an event, it can be suggested that error-related pupil dilation indicates this second step. In the invisible-target condition of *Study 3*, in which no correct response was available, no post-response conflict and, thus, no error-related pupil dilation would occur, as it is based on an error's unexpectedness. This could be why there was no pupil dilation in the absence of the Ne/ERN.

Alternatively, the Ne/ERN already indicates the violation of an expected outcome, or error unexpectancy, as suggested, for example, by Alexander & Brown (2011) and Holroyd & Coles (2002). According to this theory, the Ne/ERN is enhanced in conditions where correct responses are more expected or, in other words, where errors are less frequent and thus less expected. As a result, conditions with a higher Ne/ERN are typically associated with lower error rates

compared to conditions with a lower Ne/ERN (Kappenman et al., 2012). This poses the question of whether the absence of the Ne/ERN in the invisible-target condition in *Study 3* could be explained by this theory. On the one hand, flanker errors were the only responses where participants could be certain about the outcome because participants knew that flankers always differed from the masked target. The other two response options (correct and nonflanker errors) were not distinguishable in the invisible-target condition and were considered nonflanker guesses. In this sense, flanker errors indicate a clearer violation of the expected outcome than nonflanker guesses. This should lead to an impact on the Ne/ERN and pupil dilation, which was not evident. On the other hand, correct responses could not be expected at all because they were not available in the invisible-target condition, which would explain why there was no Ne/ERN and pupil dilation. Nevertheless, results from *Study 1* speak against the notion that the Ne/ERN is based on, or at least exclusively based on, the violation of an expected outcome or error unexpectancy. While this thesis cannot provide a complete answer to this question, it is possible that the Ne/ERN comprises more than one underlying process.

Conflict detection and the indication of the violation of an expected outcome can indeed be related to the Ne/ERN. It is conceivable that the Ne/ERN initially indicates a conflict, on which basis error expectancy is calculated in a second step, and that the Ne/ERN conveys both of these pieces of information. This possibility would reconcile empirical findings in favor of either conflict processing (e.g., Botvinick et al., 2001; Yeung & Cohen, 2004; Danielmeier et al., 2009; Hughes & Yeung, 2011; but see Masaki et al., 2007; Frank et al., 2005; Steinhauser et al., 2008) or error expectancy (e.g., Holroyd & Coles, 2002; Holroyd et al., 2009; Hajcak et al., 2007; 2008; Meckler et al., 2011; but see Maier et al., 2016; 2012; Hajcak et al., 2005).

The results of the study conducted by Buzzell et al. (2017) also corroborate the perspective that the Ne/ERN comprises different signals. The researchers investigated age-related alterations in the error monitoring system through EEG source localization. Their findings suggest that the Ne/ERN is underpinned by a core system of error monitoring within the cingulate cortex (the

dorsal network) that remains unaltered over time and by more ventral structures (the ventral network) that exhibit increasing activity during adolescence. The ventral structures comprise the orbitofrontal cortex (OFC) and AIC, which are associated with the processing of salience and affective responses following errors, as well as the processing of reward and interoceptive information, including ANS signals (Craig, 2002; 2009; Buzzell et al., 2017; Aston-Jones & Cohen, 2005). Therefore, it may be posited that these two processes, the activity of the dorsal and ventral network, are represented in the Ne/ERN. While the dorsal network could be associated with conflict processing, the ventral one could, in turn, be related to error-related pupil dilation. These considerations would also mean that the activity in the ACC as a part of the dorsal network comes prior to the activity in the AIC as part of the ventral network.

The question of whether the activity of the AIC follows, precedes, or runs parallel to that of the ACC remains a topic of ongoing scientific debate (Ullsperger et al., 2010). An earlier study employing an intracranial EEG offers the first evidence that the activity of the AIC precedes that of the ACC (Bastin et al., 2017). However, the AIC in humans has bidirectional connections to other brain areas, including the ACC (Deen et al., 2011). Furthermore, ACC and AIC have different functions (e.g., Barrett & Simmons, 2015; Critchley et al., 2001; Critchley & Harrison, 2013; Etkin et al., 2011; Hester et al., 2009; Uddin, 2014; Ullsperger, Fischer, et al., 2014; Yeung et al., 2004), i.e., the direction of information processing can vary depending on the specific task context. The direction of information processing observed in the study by Bastin et al. (2017) is, therefore, not necessarily the only possible one. Thus, the following temporal dynamic for error processing remains conceivable: First, a conflict is detected by the cingulate cortex, which signals that an error has occurred. Then error-related pupil dilation, which is likely based on the information about error expectancy, is caused by the activation of the AIC.

How error awareness relates to pupil dilation and heart rate deceleration

Study 2 demonstrated that conscious error detection is possible without early error processes, as indicated by the Ne/ERN (Dumsky et al., 2025). This corroborates the notion that conscious error detection is associated with the Pe. Error awareness is highly correlated with AIC activity (Hester et al., 2005; Klein et al., 2007) and pupil dilation (Wessel et al., 2011; Harsay et al., 2018). Wessel et al. (2011) and Harsay et al. (2018) suggested that error awareness is necessary for ANS responses, such as pupil dilation, which should indicate the recruitment of mental and physical effort after errors (Ridderinkhof, 2014; Ullsperger et al., 2010). However, whether error-related ANS responses cause error awareness or whether error awareness leads to these ANS responses is still being debated (Harsay et al., 2018; Ullsperger et al., 2010; Wessel et al., 2011). In *Study* 3, pupil dilations vanished together with the Ne/ERN, while the Pe was still present. This indicates that pupil dilations after error responses are caused by mechanisms that occur prior to the Pe. So, it is unlikely that pupil dilation is caused by error awareness.

Interestingly, the heart rate-related results of *Study 3* showed the opposite pattern of the pupil dilation results, i.e., error-related heart rate deceleration was still present in the absence of the Ne/ERN. This demonstrates that error-related ANS responses cannot be uniformly associated with the exact underlying mechanism. Error-related heart rate deceleration is independent of early error processes and, therefore, seems to be related to later ones. As with pupil dilations, error-related heart rate deceleration was also interpreted as a corollary of an orienting response (Sokolov, 1963; e.g., reviewed by Graham & Clifton, 1966; Pribram & McGuinness, 1975; van der Molen et al., 1991). However, the heart rate results from *Study 3* show that error-related heart rate deceleration must actually indicate a different mechanism than error-related pupil dilation. At least, error-related heart rate deceleration cannot be based on the error expectancy because regardless of the (un)expectedness of an error in the visible- or invisible-target condition, heart rate deceleration was present. Instead, it is likely associated with error awareness, as indicated by the Pe.

Wessel et al. (2011) provided the first evidence that consciously perceived errors are related to stronger heart rate decelerations and suggested that error awareness mediates the relationship between the brain and heart during error processing. Besides the interpretation as a part of an orienting response (Sokolov, 1963; e.g., reviewed by Graham & Clifton, 1966; Pribram & McGuinness, 1975; van der Molen et al., 1991), it was also hypothesized to reflect affective processing following errors (van der Veen, van der Molen et al., 2004). Indeed, heart rate deceleration, in general, was already associated with emotional arousal, especially with negative emotional stimuli (Bradley et al., 1996, 2001; Bradley & Lang, 2000; Brosschot & Thayer, 2003; Waldstein et al., 2000) and with negative external feedback (Somsen et al., 2000). Given that errors typically are negative events (Aarts et al., 2012; Aarts et al., 2013; Aarts & Pourtois, 2010; Koban & Pourtois, 2014), error-related heart rate deceleration can be interpreted to indicate affective processing. Previous studies, however, found mixed results regarding the relationship between error-related heart rate deceleration and affective processing (Hajcak et al., 2003, 2004; Rodeback et al., 2020). Nevertheless, these inconsistent results can be explained by the fact that these studies did not consider error awareness as a potential mediating variable (Di Gregorio et al., 2024). It is, therefore, possible that heart rate deceleration following errors indicate the affective processing of errors coupled with error awareness.

Error awareness, which is thought to be based on evidence accumulation, is tightly linked to the Pe amplitude (Di Gregorio et al., 2016, 2018; Steinhauser & Yeung, 2010, 2012; Wessel et al., 2011) and my study demonstrated that the underlying mechanism that gives rise to the Pe is sufficient to elicit heart rate deceleration. However, there is an ongoing debate about whether the output of the Pes underlying accumulation process is either directly reflected by the Pe amplitude or it only leads to an affective signal that provides this evidence (Steinhauser & Yeung, 2012; Wessel et al., 2011). Hence, error-related heart rate deceleration, possibly representing an affective signal, could occur just before the Pe or shortly after or error

awareness, independent of earlier error processes. My studies cannot answer this question conclusively. Theoretically, both directions are plausible.

Koban and Pourtois (2014) proposed two pathways for performance monitoring. One pathway is associated with the activation of the ACC that monitors, e.g., conflicts and prediction errors (e.g., Alexander & Brown, 2011; Botvinick et al., 2001; Holroyd & Coles, 2002; Shenhav et al., 2013). Moreover, this first pathway also automatically determines the emotional valence of actions such as errors, i.e., whether an action has a negative or positive value. This would also align with Dignath et al. (2020), who stated that the post-response conflict causes an affective signal, which is then monitored and indicated by the Ne/ERN. The second pathway integrates this information with more profound information, for example, about the emotional value through activating the AIC. This process may then lead to the Pe and/or error awareness, as stated by Koban and Pourtois (2014). The assumption that the second pathway is based on the first and considering that the Pe can emerge without the Ne/ERN means that the second pathway would also be absent in my *Study 3*. In turn, error-related heart rate deceleration cannot occur before the Pe but must be caused by the Pe.

In the context of my *Study 3*, it is therefore plausible that error awareness leads to an affective signal (Hajcak et al., 2003; Ullsperger et al., 2010) as indicated by heart rate deceleration, because flanker errors were still consciously detectable, even in the absence of the post-response conflict. Nevertheless, this does not mean that affective processing prior to error awareness is impossible. It is plausible that there are preconscious and conscious mechanisms of affective processing (see e.g., Öhman & Soares, 1994), but preconscious affective properties are likely only coarsely processed (Pessoa, 2005). This could explain why the Ne/ERN (Aarts et al., 2013; Dignath et al., 2020; Hajcak et al., 2003, 2004; Hajcak & Olvet, 2008; Koban & Pourtois, 2014; Luu et al., 2000; but see Härpfer et al., 2020) and pupil dilation (Leuchs et al., 2017; Paulus et al., 2015; Tamietto et al., 2015), are associated with affective processes as well.

To sum up, error-related pupil dilation that is coupled to early error processes or the Ne/ERN, likely contributes to error awareness as part of an orienting response and thus is based on the unexpectedness of an event. In turn, error-related heart rate deceleration, which is independent of early error processes, could represent affective processing that follows error awareness. Pupil dilation must, therefore, be caused earlier than heart rate deceleration during error processing. As both are highly correlated with activity in the AIC (Critchley et al., 2005; Harsay et al., 2018), the brain area that is thought to integrate autonomic arousal and is highly associated with error awareness (Craig, 2002; Critchley et al., 2002, 2004), my results address the question of whether the AIC monitors or actively regulates error-related responses (see Ullsperger et al., 2010).

The two possible functions of the AIC are not necessarily mutually exclusive because there is mounting evidence that the AIC receives and processes interoceptive information and modulates ANS responses due to its rich bidirectional connections with the ANS (Cechetto, 2014; Craig, 2009; Uddin et al., 2017). In this vein, it is interesting that there is a significant overlap of brain areas that are involved in the salience network, the central autonomic network, and the error monitoring system, such as the AIC and ACC (Ferraro et al., 2022; Klein et al., 2007; Ridderinkhof, Ullsperger et al., 2004; Seeley et al., 2007). The salience network registers salient and, therefore, unexpected events and is associated with an automatic orienting response (Critchley, 2005; Critchley et al., 2004; Harsay et al., 2012; Sara & Bouret, 2012). The central autonomic network mainly regulates the ANS (Cardinali, 2018), while the error monitoring system monitors for errors and conflicts in behavior (Ullsperger, 2017; Ullsperger, Fischer, et al., 2014).

The above-mentioned temporal dynamics for error processing could, therefore, be realized in the following way: After an error has been made, a conflict is registered by the error monitoring system, located in the ACC. This signal is used to calculate error expectancy when necessary. Both processes could be represented in the Ne/ERN. When errors are unexpected, pupil dilation

is elicited, which very likely represents an orienting response and includes the activity of the salience network. This orienting response then causes a reallocation of attention by interrupting the actual behavior to reorientate to the error (Barry et al., 2011; MacDonald et al., 2012; Sokolov, 1963). However, my results show that they do not appear to be necessary for error awareness per se, as errors can also be consciously detected without them. Finally, error awareness leads to affective processing of the error and triggers the central autonomic network through the AIC, which is reflected in heart rate deceleration.

Final Considerations

The goal of my doctoral thesis was to gain further insight into the interplay between early error processes and conflict processing, error awareness, and error-related ANS responses. I achieved this by conducting three EEG studies that focused on conflict processing (*Study 1*), error awareness (*Study 2*), and error-related pupil dilations and heart rate deceleration (*Study 3*). The findings of these three studies demonstrated that the interactions between these processes are more intricate than assumed, thereby offering significant insights that advance the field.

First, I showed that the pre- and postresponse conflict can be further enhanced when two incorrect responses compete with the correct response compared to one incorrect response (Dumsky et al., 2023). The decreased Ne/ERN cannot be explained by differences in error expectancies between the same-flankers and different-flankers condition, as there were no differences in error rates between both conditions. Therefore, *Study 1* demonstrated that the Ne/ERN actually indicates conflict processing, supporting the conflict monitoring theory (Botvinick et al., 2001; Yeung et al., 2004). However, it is still possible that the Ne/ERN comprises more than one mechanism, as described above. In *Study 2,* I showed that flanker errors can be consciously detected without the Ne/ERN (Dumsky et al., 2025). This result further proves that the Pe is linked to error awareness (Gehring et al., 2012) and that early error processes are unnecessary for conscious error detection. The most unexpected result was found

in *Study 3.* The error-related pupil dilation vanished without the Ne/ERN, while heart rate deceleration was unaffected. This indicates that pupil dilation and heart rate deceleration are based on different and independent underlying mechanisms. Pupil dilation was associated with the Ne/ERN and, thus, early error processes. In contrast, heart rate deceleration was linked to later error processes, which the Pe indicates. Although both autonomic parameters are supposed to be part of an orienting response (Lynn, 1966; Sokolov, 1963), according to *Study 3,* they must be functionally distinct. I outlined one possible underlying process, including the temporal dynamic, which accounts for all these findings. In this regard, my thesis significantly contributes to the existing body of research on how early error processes relate to conflict processing, error awareness, and accompanied phenomena, such as error-related pupil dilation and heart rate deceleration.

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Appendix

Authors' contributions

The authors' contributions to the stages of each study are listed in the order of their names, reflecting the extent of each author's contribution.

Study	Idea/ conception	Planning/ conduction	Data analysis	Writing
Dumsky, J., Maier, M. E., & Steinhauser, M. (2023). Effects of the number of competing responses on neural signatures of pre- and post-response conflict. *Biological Psychology*, *182*, 108643.	JD, MS	JD	JD	JD, MS, MM
Dumsky, J., Maier, M. E., Di Gregorio, F., & Steinhauser, M. (2025). Error awareness can occur in the absence of an error-related negativity, *Psychophysiology,* 62, e70128. (in press)	JD, MS	JD	JD, FDG	JD, MS, MM, FDG
Dumsky, J., Maier, M. E., & Steinhauser, M.. Error-related pupil dilation, but not heart rate deceleration, is linked to the error negativity. (in preparation)	JD, MS, MM	JD	JD, MM, MS	JD, MS, MM

Note. Abbreviations: MS = Marco Steinhauser, JD = Julia Dumsky, MM = Martin E. Maier, FDG = Francesco Di Gregorio

My journey began with many obstacles. Due to my severe disability, it was not expected that I would attend a mainstream school. So, primarily, I would like to thank my mother, who always believed in my abilities and campaigned for me to be enrolled in a mainstream school. Fortunately, the majority of the school community welcomed my enrolment. Contrary to all expectations, I graduated with the high school diploma in 2009. After some introspection and a regain of my self-confidence, I decided to study psychology. My genuine interest in neuroscience increased during this period and I made up my mind to my decision that I want to do my doctorate in this astonishing research field.

During my Master's program, I got to know Prof. Dr. Marco Steinhauser, an established researcher in cognitive neuroscience, as a highly intelligent but still down-to-earth person, something that I really appreciate about him. It was clear from the beginning that I wanted him as my supervisor for my doctorate, and he was immediately willing to support me on my academic journey. I would like to thank him for his very understanding accompaniment on the way to my successful thesis, an accompaniment that every student wishes for. My colleagues also deserve a big thank you, who were always willing to listen to my questions. Although I was rarely on the campus in Eichstätt, I always felt like an integrative part of the core team when we got together. I will definitely miss them a lot.

Another opportunity to enrich my academic life at the Catholic University of Eichstätt-Ingolstadt was the Forschungskolleg Naturwissenschaften - Mensch und Natur, which offered me interdisciplinary exchange between doctoral students in the natural sciences. This program enabled me to practice academic presentation and to discuss research topics in other fields of natural science and, therefore, to exchange ideas and perspectiveswith other doctoral students. I'm very glad that I could attend this multifaceted program that helped me to overcome different challenges during my doctorate.

Finally, I would like to express my special thanks to my family and friends, who have supported me mentally and encouraged me to take breaks from time to time. They have also accompanied me to important events such as academic conferences, enabling me to participate. I am grateful that you are always by my side.