

# Anticipating Conflict Facilitates Controlled Stimulus-response Selection

Ángel Correa<sup>1,2</sup>, Anling Rao<sup>2</sup>, and Anna C. Nobre<sup>2</sup>

## Abstract

■ Cognitive control can be triggered in reaction to previous conflict, as suggested by the finding of sequential effects in conflict tasks. Can control also be triggered proactively by presenting cues predicting conflict (“proactive control”)? We exploited the high temporal resolution of ERPs and controlled for sequential effects to ask whether proactive control based on anticipating conflict modulates neural activity related to cognitive control, as may be predicted from the conflict-monitoring model. ERPs associated with conflict detection (N2) were measured during a cued flanker task. Symbolic cues were either informative or neutral with respect to whether the target involved conflicting or congruent responses. Sequential effects were controlled by analyzing the congruency of the previous trial. The results showed that cueing conflict facilitated conflict resolution and reduced the N2 la-

tency. Other potentials (frontal N1 and P3) were also modulated by cueing conflict. Cueing effects were most evident after congruent than after incongruent trials. This interaction between cueing and sequential effects suggests neural overlap between the control networks triggered by proactive and reactive signals. This finding clarifies why previous neuroimaging studies, in which reactive sequential effects were not controlled, have rarely found anticipatory effects upon conflict-related activity. Finally, the high temporal resolution of ERPs was critical to reveal a temporal modulation of conflict detection by proactive control. This novel finding suggests that anticipating conflict speeds up conflict detection and resolution. Recent research suggests that this anticipatory mechanism may be mediated by preactivation of ACC during the preparatory interval. ■

## INTRODUCTION

Most classic models of cognition posit a control system that selects stimuli and responses according to task goals (e.g., Norman & Shallice, 1986; Kahneman, 1973; Atkinson & Shiffrin, 1968). This system is engaged under conflicting situations, such as when simultaneously occurring events call for multiple and incompatible responses (Eriksen & Eriksen, 1974), and after negative behavioral outcomes, such as errors (for a review, see Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). After conflict or errors, behavioral measures reveal a more deliberate mode of responding (“sequential effects,” Gratton, Coles, & Donchin, 1992; “posterror slowing,” Rabbitt, 1966).

Neural models of conflict monitoring (e.g., Botvinick, Braver, Barch, Carter, & Cohen, 2001) posit that the ACC detects conflict and recruits DLPF cortex to bias stimulus-response selection through top-down signals. Neuroimaging research has confirmed increased ACC activation by conflict, followed by decreased ACC activation on the subsequent conflicting trial, suggesting successful conflict reduction by an increased attentional set (Kerns et al., 2004; Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999).

Conflict monitoring is triggered in reaction to previous conflict (“reactive control”), as suggested by the finding of sequential effects in conflict tasks (Gratton et al., 1992). Gratton et al. (1992) used a flanker task (Eriksen & Eriksen, 1974) and found that the conflict effect (RT difference between incongruent and congruent conditions) was reduced when the previous trial was incongruent rather than congruent. However, top-down control is not only triggered reactively but also proactively (Logan & Zbrodoff, 1982). Logan and Zbrodoff (1982) induced “proactive control” by presenting cues predicting conflict in a Stroop (1935) task. The results showed that the anticipation of an incongruent trial reduced the conflict effect. Thus, proactive control is analogous to attentional orienting more generally, during which expectations about a particular event attribute trigger top-down biases of relevant stimulus/response representations (Kastner & Ungerleider, 2000).

Although the conflict-monitoring model was primarily based on reactive control (Botvinick et al., 2001), a clear prediction can also be made for proactive control. Behavioral facilitation of conflict resolution by proactive control should be associated with modulations of conflict-related neural activity (e.g., decreased ACC activity). However, neuroimaging studies using conflict-predicting cues have led to inconclusive results (Luks, Simpson, Dale, & Hough, 2007; Sohn, Albert, Jung, Carter, & Anderson,

<sup>1</sup>Universidad de Granada, Spain, <sup>2</sup>University of Oxford, UK

2007; Fassbender, Foxe, & Garavan, 2006; Luks, Simpson, Feiwell, & Miller, 2002). The scarce evidence showing that proactive control modulates conflict processing could be explained by two factors: (1) the limited temporal resolution of hemodynamic measures or (2) the masking of proactive control effects due to modulatory effects caused by reactive control. None of these studies manipulating proactive control considered the effect of previous trial congruency. If proactive and reactive control share neural mechanisms, then the effects of proactive control would be most evident when reactive control is low (i.e., after congruent rather than incongruent trials).

Here, we (1) exploited the high temporal resolution of ERPs and (2) controlled for the effects of reactive control in a cued flanker task to ask whether proactive control modulates neural activity related to conflict. Previous research using conflict tasks and ERPs has identified two frontal potentials related to cognitive control. A negative deflection is observed after stimuli that carry incongruent versus congruent response tendencies (N2; Kopp, Rist, & Mattler, 1996) as well as after an incorrect response (error-related negativity—"ERN" or "Ne"; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990). ACC is thought to constitute an important source to both the N2 and the ERN (Debener et al., 2005; van Veen & Carter, 2002), which, respectively, have been interpreted as indices of conflict detection (van Veen & Carter, 2002) and error detection (reviewed by Falkenstein, Hoormann, Christ, & Hohnsbein, 2000).

The central hypothesis of this study was that the anticipation of conflict should modulate control-related potentials. Two ERP analyses were conducted on control-related ERPs: stimulus locked and response locked. The stimulus-locked analysis tested whether conflict processing, as indexed by the N2 potential, was attenuated by cueing conflict. Additionally, this analysis allowed us to explore whether proactive and reactive control operate through a common attentional system, as suggested by their analogous behavioral effects (Gratton et al., 1992; Logan & Zbrodoff, 1982). We compared modulations of stimulus-locked potentials by cueing versus by previous conflict processing. If their neural mechanisms overlap, proactive and reactive control should interact in their modulation of conflict-related neural processing. Stronger effects of proactive control should hence be expected under low versus high reactive control. On the other hand, if proactive and reactive control involve independent mechanisms, we should observe dissociable modulations of conflict processing.

The response-locked analysis tested whether error-related processing indexed by the ERN and the ensuing "error positivity" (Pe; Falkenstein et al., 1990, 2000) was modulated by cueing conflict. High time pressure for responding was imposed to ensure reliable numbers of error trials for ERP analysis (e.g., Nieuwenhuis et al., 2006; Ullsperger, Bylsma, & Botvinick, 2005).

## METHODS

### Participants

Twenty participants from the University of Oxford (aged 19–49, nine women) took part voluntarily in the experiment. Data from one participant were rejected due to excessively low accuracy during task performance (58% correct). The experimental methods were noninvasive and had ethical approval from the University of Oxford.

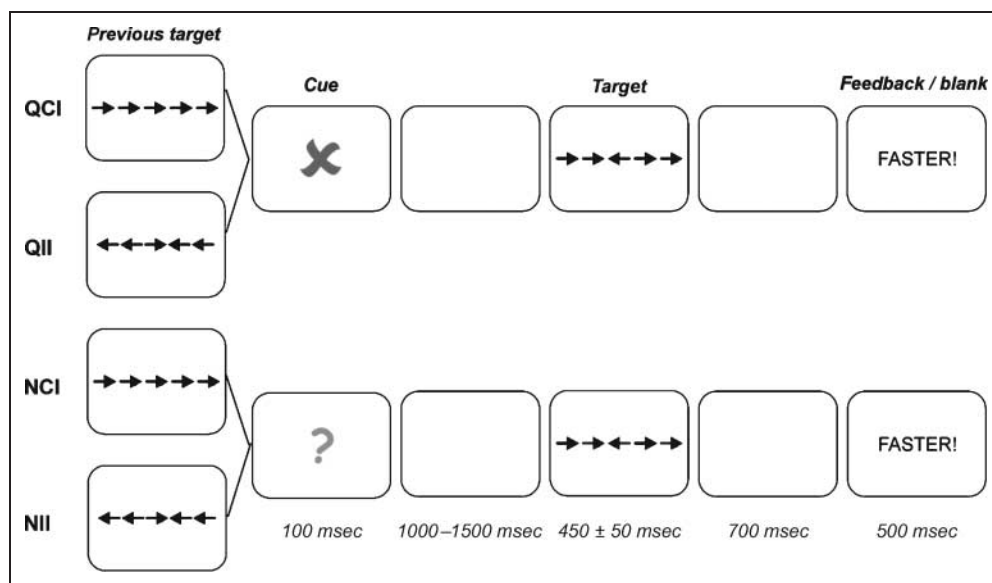
### Stimuli and Task

Participants completed a cued flanker task under high time pressure. They were to respond according to the direction pointed by a central arrow while ignoring the direction of the flanking arrows. The target was preceded by a cue stimulus, which was either informative or neutral with respect to the congruency between the central and flanking arrows. Informative cues were 100% predictive ("cued" condition). A green checkmark indicated that the central and the flanking arrows would be congruent. A red cross indicated they would be incongruent. A yellow question mark provided no prediction about congruency ("neutral" condition). A sufficiently long interval was used between the cue and the flanker array (1000–1500 msec) to enable the implementation of endogenously generated expectations about the upcoming stimulus congruency (e.g., see Monsell, 2003; Gratton et al., 1992; Müller & Rabbitt, 1989). The cue stimulus subtended  $1.2^\circ \times 1.2^\circ$ . Target arrays consisted of five white arrows ( $1.4^\circ \times 1^\circ$  each) appearing in a row and centered on the fixation point (a gray dot with  $0.3^\circ$  diameter). The direction of the central arrow (left or right) could be the same (congruent) or different (incongruent) from that of the flankers. All flankers pointed in the same direction.

Figure 1 illustrates the main events of the task for the four incongruent conditions used to compare proactive cueing and reactive sequential effects. These included cueing (cued and neutral) and congruency of the target on the previous trial ("previous congruency": congruent and incongruent).

Each trial began with a fixation point presented centrally for a random interval that ranged between 500 and 1000 msec. The cue was then presented centrally for 100 msec and followed by a blank display for a random interval of 1000–1500 msec. The target array then appeared and remained until the participant responded or a maximum deadline elapsed. The deadline was set at 450 msec and was adjusted across blocks to maintain a good level of performance that was below ceiling (around 70%). The deadline was increased by 50 msec if the error rate was above 35% in the preceding block or was decreased by 50 msec if it was below 25%. The participant had to press the "z" key with the left index finger or the "m" key with the right index finger, according to the direction pointed by the central arrow.

**Figure 1.** The main events of the task. The four conditions included in the stimulus-locked analysis are displayed. This analysis included current incongruent targets with the factors of cueing (cued and neutral) and previous congruency (congruent and incongruent). QCI: cued, previous congruent, and current incongruent target; QII: cued, previous incongruent, and incongruent; NCI: neutral, previous congruent, and incongruent; NII: neutral, previous incongruent, and incongruent.



After a timely response or the deadline, there was a blank display of 700 msec. If participants had failed to respond within the deadline, a visual feedback “faster!” then appeared for 500 msec. Following a timely response or the feedback, a blank display of 500 msec preceded the next trial.

The experiment included three practice blocks plus 42 experimental blocks. Each block consisted of 16 trials, including two trials of each type according to the type of cueing (cued, neutral), flanker (congruent, incongruent), and direction of the central arrow (left, right). The proportion of congruent and incongruent targets was 0.5. On average, half of current trials were preceded by congruent or incongruent trials.

### EEG Recording

The EEG recording was performed in an electrically shielded room, using Ag/AgCl electrodes mounted on an elastic cap and distributed along 34 scalp sites according to the 10-20 International system (AEEGS, 1991) using NuAmp amplifiers (Neuroscan, El Paso, TX). The montage included six midline sites (FZ, FCZ, CZ, CPZ, PZ, and OZ) and 14 sites over each hemisphere (FP1/FP2, F7/F8, F3/F4, FT7/FT8, FC3/FC4, T7/T8, C3/C4, TP7/TP8, CP3/CP4, P7/P8, P3/P4, PO7/PO8, PO3/PO4, and O1/O2). Additional electrodes were used as ground and reference sites and for recording the EOG. All electrodes were referenced to the right mastoid during the recording and were algebraically rereferenced off-line to calculate the average of the right and the left mastoids. Eye movements were monitored by horizontal and vertical EOG bipolar recordings with electrodes placed around the eyes. The EEG was amplified with a low-pass filter of 300 Hz and digitized at a sampling rate of 1000 Hz.

### ERP Analysis

The continuous EEG was filtered off-line with a 40-Hz low-pass filter. Separate epochs were constructed for targets (between  $-100$  and  $600$  msec relative to target onset) and responses (between  $-600$  and  $700$  msec relative to response onset). Their respective baselines consisted of a 100-ms epoch before target onset and a 50-ms epoch between  $-100$  and  $-50$  msec relative to response onset. Epochs in which an eye blink or eye movement occurred were rejected based on large deflections ( $\pm 50$   $\mu$ V) in the HEOG or the VEOG electrodes. Epochs with large signal drift were also removed based on large deflections ( $\pm 100$   $\mu$ V) in any channel.

An initial analysis compared ERPs triggered by congruent versus incongruent targets to replicate the well-established effects of conflict on the N2 potential. The analysis of main interest followed, which tested the effects of proactive control upon *conflict* processing and compared them with the effects of reactive control. Accordingly, only ERPs elicited by the conflict-carrying incongruent target conditions were included. Epochs were averaged according to the four conditions defined by cueing (cued and neutral) and previous congruency (congruent and incongruent). Only incongruent targets with correct responses were analyzed. Identifiable target-related potentials were analyzed at electrode locations and temporal windows where they were most evident according to the segmentation analysis performed with Cartool software (see Topographical analyses section). Target-locked waveforms averaged across participants are shown in Figure 3 (top). The first negative deflection peaked at 110 msec over frontal electrodes and was referred to as “frontal N1” (N1f). According to the segmentation analysis, the N1f occurred around 90–130 msec and extended from frontal to centroparietal electrodes with maximal intensity over the left frontal scalp (Figure 3,

bottom). The N1f was therefore analyzed during 90–130 msec over frontal to centroparietal electrode positions (F3/Z/4, FC3/Z/4, C3/Z/4, and CP3/Z/4). The P1 overlapped temporally with the N1f, peaking at 120 msec, and was maximal over occipital electrodes (O1/2, PO3/4, and PO7/8). N1 peaked at 170 msec and was also maximal over occipital electrodes (O1/2, PO3/4, and PO7/8). P1 and N1 were analyzed during 110–130 and 160–180 msec over three occipital positions (O1/2, PO3/4, and PO7/8). The N2 peaked at about 305 msec over frontal electrodes and was analyzed during 290–330 msec at F3/Z/4 and FC3/Z/4. The P3 peaked at 425 msec and was broadly distributed over the central scalp. The P3 was analyzed during 360–460 msec at FC3/Z/4, C3/Z/4, CP3/Z/4, and P3/Z/4.

An analysis of response-related ERPs focused on whether cueing conflict modulated *error* processing. The epochs were averaged separately for errors and correct responses during incongruent trials, according to the cueing conditions (cued and neutral). Responses to congruent targets led to insufficient numbers of observations on the error condition and hence were not analyzed. Likewise, conditions with previous congruent and incongruent targets were collapsed to gain statistical power.

Error analyses followed the same approach as for targets, using periods defined by topographical segmentation and electrodes showing the maximal distribution of potentials. Around the time of response, ERPs elicited by errors contained an ERN that peaked at about 20 msec after the response (see Figure 4, top)<sup>1</sup> and was focally distributed over frontocentral electrodes (Figure 4, bottom, Map 2). This was followed by positive-going potentials, similar to the Pe, which separated into two different topographical states (Maps 4 and 5). ERPs elicited by correct responses were characterized by a positive potential, the “correct positivity” (Pc), at the same time range as the ERN, which was distributed over central electrodes (Figure 4, bottom, “Map 3”). The Pc was then followed by continued positive potentials subdivided into two topographical states. The ERN was analyzed at 0–40 msec over frontal and frontocentral electrodes (F3/Z/4 and FC3/Z/4). The Pe was analyzed over frontal to central electrodes (F3/Z/4, FC3/Z/4, and C3/Z/4) at two epochs: early Pe (50–110 msec, Map 4) and late Pe (250–290 msec, Map 5) (van Veen & Carter, 2002). Analyses of correct responses included the Pc at 0–40 msec over frontal to central electrodes: F3/Z/4, FC3/Z/4, and C3/Z/4 (see Map 3).

Mean amplitude values were obtained for the temporal window of each potential and submitted to repeated measures ANOVAs. Target-related analyses consisted of one ANOVA, which tested the effects of cueing, previous congruency, electrode position, and electrode side. Response-related analyses included separate ANOVAs for error and correct conditions with the factors of cueing, electrode position, and electrode side. Significant effects of electrode position and electrode side were not reported, unless they involved interactions with either

cueing or previous congruency. Peak latencies of the main potentials of interest (N2 and ERN) and potentials showing amplitude modulation (N1f and P3) were also subjected to ANOVAs. The latency analyses included the same temporal windows as the mean amplitude analyses and focused on the electrode showing the highest activity: F3 electrode for the N2, N1f, and ERN potentials and Cz for the P3. The criterion to include data from a participant in either target or response-related analyses consisted of 20 artifact-free trials per condition. This criterion led to the exclusion of two participants from the target-related analyses and three participants from the response-related analyses.

### Topographical Analysis

The topographical analysis determined how the distribution of voltage over the scalp (topographical maps) evolved over time in each experimental condition and compared the distributions across conditions, using Cartool software (D. Brunet, Geneva, Switzerland; <http://brainmapping.unige.ch/Cartool.php>). The segmentation consisted of a spatiotemporal cluster analysis of the ERP group-averaged data normalized for global field power (with the constraints that each scalp topography should remain stable for at least 20 msec and that the correlation between different topographies should not exceed 90%). The choice of the optimal number of topographies that best explained the whole data was based on a cross-validation criterion (Pascual-Marqui, Michel, & Lehmann, 1995). The output of this segmentation analysis consists of a set of topographical maps that represent stable periods of electrical field patterns, which reflect dissociable functional states of the brain (see Figure 4, center and bottom). Different maps reflect different stages of information processing with different underlying brain sources (Lehmann, 1987). This procedure served to guide the selection of the optimal temporal windows and electrodes for the ERP analysis. Moreover, the segmentation analysis served to compare the topographical maps and their time courses across different experimental conditions. The comparison across conditions (e.g., Figure 4, Maps 2 and 3) was performed statistically at the single-subject level through a fitting procedure. The topographical maps identified for the group-average were fitted to the scalp topography of each participant and for each time point of data. During this procedure, data from each subject and condition were compared with the maps identified at the group-average level, using a spatial correlation. Each time point of the data of each participant was then labeled with the map with which it had the highest spatial correlation. For each participant, the number of milliseconds during which one specific map was present on each condition was computed. These values would indicate whether one map was dominant over another map for a given condition. These values were compared across conditions

with ANOVAs with the maps and the experimental conditions as factors. An interaction involving the factor map would indicate that a particular experimental condition was better explained by one map than another, and therefore that a different configuration of brain generators better explains the results.

## RESULTS

### Behavioral Results

Error rates and mean RTs from 19 participants were submitted to separate repeated measures ANOVAs with the factors of cueing (cued and neutral), previous congruency (congruent and incongruent), and congruency (congruent and incongruent). Error rates constituted the main dependent variable of interest because RTs were highly constrained by our time-pressure procedure. Table 1 shows detailed data for all conditions.

The analysis of the error rates only considered responses given within the deadline (18% rejected). The ANOVA showed a main effect of congruency (i.e., the conflict effect),  $F(1,18) = 80.07, p < .001$ , so that incongruent targets induced higher error rates than congruent targets (Figure 2, top left).

A significant main effect of previous congruency,  $F(1,18) = 6.19, p = .023$ , revealed that fewer errors were committed after an incongruent trial (18%) than after a congruent trial (20%). These two effects also interacted significantly leading to sequential effects<sup>2</sup> (previous congruency by congruency),  $F(1,18) = 8.25, p = .01$ . Specifically, the conflict effect was smaller when the previous trial was incongruent (20%) rather than congruent (24%). Cueing also modulated the conflict effect (cueing by congruency),  $F(1,18) = 12.75, p = .002$ , which was smaller on cued (19%) compared with neutral (25%) conditions. Follow-up analysis of incongruent targets showed that cueing conflict tended to reduce error rates, especially when the previous trial was congruent,  $F(1,18) = 3.49, p = .078$  (see Figure 2, bottom left).

The RT analysis only considered responses that were slower than 100 msec and within the deadline (14% rejected). The main effect of congruency was significant,

$F(1,18) = 53.1, p < .001$ , revealing slower responses for incongruent (296 msec) compared with congruent (261 msec) trials. The main effect of cueing was also significant,  $F(1,18) = 26.41, p < .001$ ; responses were faster in valid versus neutral targets. The interaction between previous congruency and congruency (sequential effects) was only marginally significant,  $F(1,18) = 2.92, p = .1$ , which may be due to the strong constraints placed upon speeded responses. The interaction between cueing and congruency was significant,  $F(1,18) = 15.1, p = .001$ , showing larger cueing effects on congruent versus incongruent trials (see also Gratton et al., 1992, Experiment 3). Most relevant, follow-up analysis of RTs in the incongruent condition revealed a significant effect of cueing, with faster RTs in cued (294 msec) versus neutral (299 msec) conflicting targets,  $F(1,18) = 5.21, p = .035$ .

Combined, the analyses of error rates and RTs show that the anticipation of conflict can speed up and improve the performance during conflict resolution. Figure 2 (bottom) shows cueing benefits on both errors and RT to incongruent targets. The neural correlates of this attentional improvement in conflict resolution were studied by ERPs to the incongruent condition.

### Electrophysiological Results

#### Target-locked ERPs

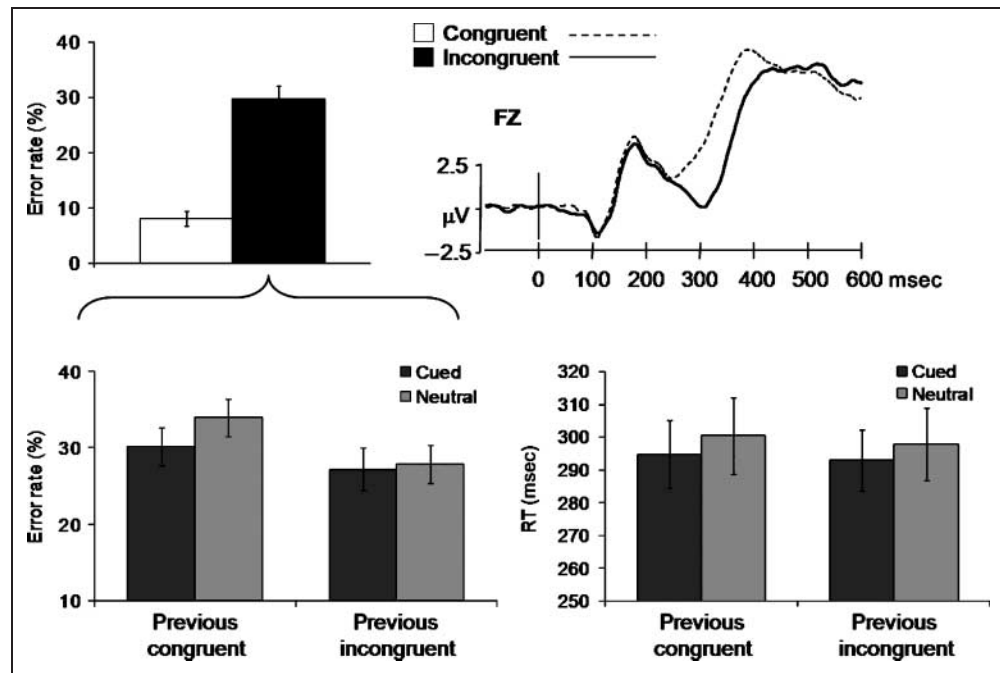
Figure 2 (top right) shows that congruent and incongruent targets were processed differently. Consistent with previous research (Kopp et al., 1996), the fronto-central N2 was sensitive to different degrees of conflict between congruent and incongruent conditions despite being matched for stimulus frequency (50%). The conflict effect was evident as a larger and later N2 for incongruent compared with congruent targets,  $F(1,16) = 7.93, p = .01$ .<sup>3</sup> This electrophysiological correlate of conflict processing was consistent with the conflict effect observed on behavior. Once the conflict effect was validated, the ERP analyses focused on incongruent targets yielding correct responses. These analyses tested whether processing of a conflicting stimulus was modulated by cueing when sequential effects were controlled.

**Table 1.** Mean RTs and Error Rates from 19 Participants Broken Down by Cueing (Cued and Neutral), Previous Congruency (Congruent and Incongruent), and Congruency (Congruent and Incongruent)

Cued				Neutral			
Previous Congruent		Previous Incongruent		Previous Congruent		Previous Incongruent	
Congruent	Incongruent	Congruent	Incongruent	Congruent	Incongruent	Congruent	Incongruent
252 (6.8)	295 (10.5)	253 (6.8)	293 (9.4)	267 (7.6)	300 (11.8)	271 (8.2)	298 (11)
11 (1.8)	30 (2.5)	10 (1.7)	27 (2.8)	6 (1.1)	34 (2.4)	6 (1.1)	28 (2.5)

Standard errors are shown in parentheses.

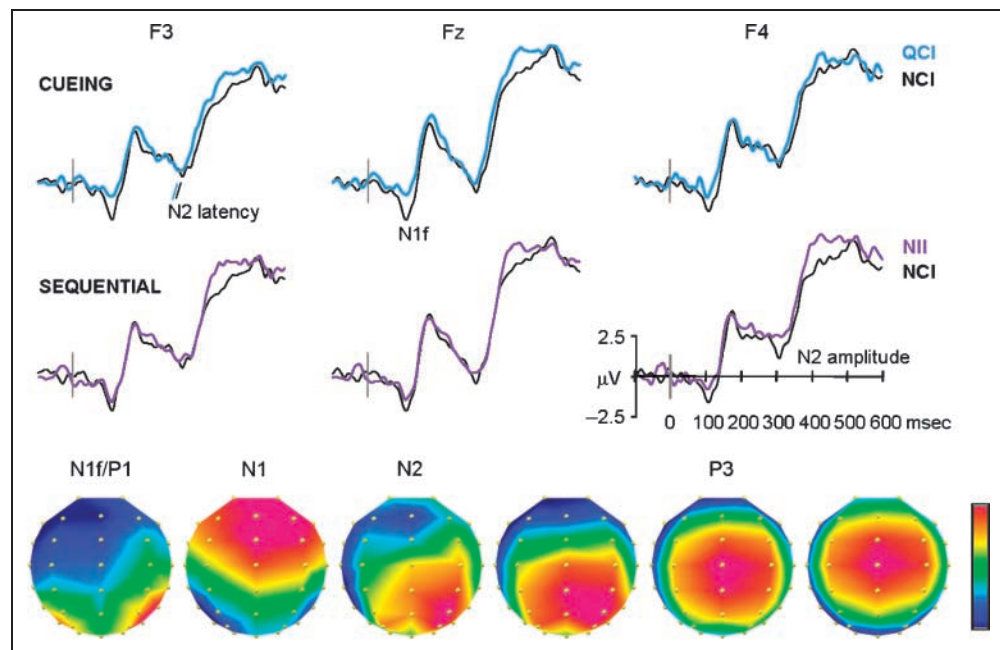
**Figure 2.** Top: Behavioral (left) and electrophysiological (right) correlates of the conflict effect. Target-locked ERP waveforms averaged across 17 participants for congruent and incongruent trials. The frontal N2 potential was larger and peaked later for incongruent versus congruent conditions. Bottom: Mean error rates (left) and RTs (right) from the incongruent condition as a function of cueing (cued and neutral) and previous congruency (congruent and incongruent). Vertical bars represent standard errors.



The ANOVA performed on the mean amplitudes of the N2 showed an interaction between cueing and electrode side,  $F(2,32) = 3.57, p = .04$ , and a further three-way interaction between cueing, previous congruency, and electrode side,  $F(2,32) = 3.30, p = .05$ . Together, these effects demonstrated that previous incongruent targets reliably

attenuated the N2 over right frontal electrodes (F4 and FC4),  $F(1,16) = 4.32, p = .05$ , but only in the absence of predictive cueing (i.e., on neutral conditions; see Figure 3, NII vs. NCI). The N2 appeared to be attenuated by cueing over left frontal electrodes, but this did not reach statistical significance (F3 and FC3),  $F(1,16) = 1.37, p = .26$ .

**Figure 3.** Top: Stimulus-locked ERP waveforms averaged across 17 participants showing the effects of cueing (top row) and sequential effects (bottom row) over the frontal electrode sites [F3 (left), Fz (midline), and F4 (right)] where the N2 potential was largest. To illustrate the proactive, cueing effects, waveforms elicited by *cued* incongruent targets preceded by congruent targets (QCI, thick blue line) are compared with neutral incongruent targets preceded by congruent targets (NCI, thin black line). To illustrate the reactive, sequential effects, waveforms elicited by neutral incongruent targets preceded by incongruent targets (NII, thick purple line) are compared with neutral incongruent targets preceded by congruent targets (NCI, thin black line). The significant effects on the N1f and N2 potentials are labeled where they were strongest. The P3 effects occurred more posteriorly and are not shown in the figure. Bottom: Topographical representation of the voltage distribution over the scalp averaged during each segmentation map. Positive voltage is plotted in red, and negative voltage is plotted in blue. Voltage was normalized during each period shown, according to the scale provided on the right. Yellow dots show the electrode montage.



To illustrate the reactive, sequential effects, waveforms elicited by neutral incongruent targets preceded by incongruent targets (NII, thick purple line) are compared with neutral incongruent targets preceded by congruent targets (NCI, thin black line). The significant effects on the N1f and N2 potentials are labeled where they were strongest. The P3 effects occurred more posteriorly and are not shown in the figure. Bottom: Topographical representation of the voltage distribution over the scalp averaged during each segmentation map. Positive voltage is plotted in red, and negative voltage is plotted in blue. Voltage was normalized during each period shown, according to the scale provided on the right. Yellow dots show the electrode montage.

Crucially, the analysis of the N2 latency revealed a reliable main effect of cueing,  $F(1,16) = 5.15, p = .04$ , such that the N2 peaked earlier for cued (303 msec) compared with neutral conditions (310 msec). Figure 3 (top) shows that the N2 peak and the subsequent rising occurred earlier for cued (QCI) versus neutral (NCI) conditions.<sup>4</sup> In contrast to cueing, previous congruency did not modulate the N2 latency ( $F < 1$ ).

The analysis of the early N1f revealed a significant attenuation by cueing,  $F(1,16) = 4.42, p = .05$ . This main effect was modulated by the congruency of the previous trial (cueing by previous congruency),  $F(1,16) = 4.98, p = .04$ . When the previous trial was congruent, the N1f was clearly attenuated by the cued condition (QCI) as compared with the neutral condition (NCI),  $F(1,16) = 8.27, p = .01$  (Figure 3). When the previous trial was incongruent, the cueing effect was far from significant ( $F < 1$ ). In contrast to cueing, previous congruency did not exert a main effect upon the N1f,  $F(1,16) < 1$ , in either cued (QII vs. NCI),  $F(1,16) = 1.66, p = .2$ , or neutral conditions (NII vs. NCI),  $F(1,16) = 1.08, p = .3$ . No significant main effects of cueing, previous congruency, or interactions were observed for the latency of the N1f potential (all  $F$ s  $< 1$ ).

The P1 potential showed a significant interaction between cueing, previous congruency, and electrode position,  $F(2,32) = 3.81, p = .03$ . When effects were tested at the separate electrode locations, however, the interaction only approached a trend over electrodes PO3/4,  $F(1,16) = 2.83, p = .11$ , but remained clearly nonsignificant over O1/2 and PO7/8 ( $p$ s  $> .2$ ). The N1 analysis showed no significant effects of cueing or previous congruency ( $F$ s  $< 1$ ) or interactions ( $p$ s  $> .1$ ).

The P3 analysis also showed an interaction between cueing and previous congruency,  $F(1,16) = 9.20, p = .01$ . When the previous trial was congruent, the P3 was enhanced by cued rather than neutral conditions,  $F(1,16) = 8.01, p = .01$ . When the previous trial was incongruent, the cueing effect was far from significant ( $F < 1$ ). Analogously, previous congruency was only significant on neutral conditions,  $F(1,16) = 7.53, p = .01$ , rather than on cued conditions ( $F < 1$ ). Both cueing and previous congruency effects consisted of an enhancement on the P3 amplitude. Cueing also interacted with electrode position and side,  $F(6,96) = 3.02, p = .01$ , revealing the largest cueing effect over the central left electrode (C3),  $F(1,16) = 4.83, p = .04$ . The latency of the P3 potential was not modulated by cueing,  $F(1,16) = 1.37, p = .26$ , or previous congruency ( $F < 1$ ).

### Response-locked ERPs

Figure 4 (top) shows that errors elicited an ERN peaking shortly after the response (Map 2). The ANOVA showed no effect of cueing on either ERN amplitude,  $F(1,15) < 1$ , or latency,  $F(1,15) < 1$ . The ERN was fol-

lowed by two positive peaks (early and late Pe, Maps 4 and 5). The ANOVAs showed no effects of cueing on the amplitudes of either early or late Pe,  $F(1,15) = 1.39, p = .26$  and  $F(1,15) < 1$ , respectively.

Correct responses were associated with the Pc potential (Pc, Map 3), which was observed in the same time range as the ERN on error conditions. The amplitude of the Pc was enhanced by cueing,  $F(1,15) = 14.28, p = .002$ . Following the Pc, the topographical segmentation (Figure 4, center) suggested that waveforms elicited by correct responses contained the same two positive potentials observed on error conditions but greatly anticipated in time (100–150 msec earlier than in error conditions).

### Topographical Analysis of Response-locked ERPs

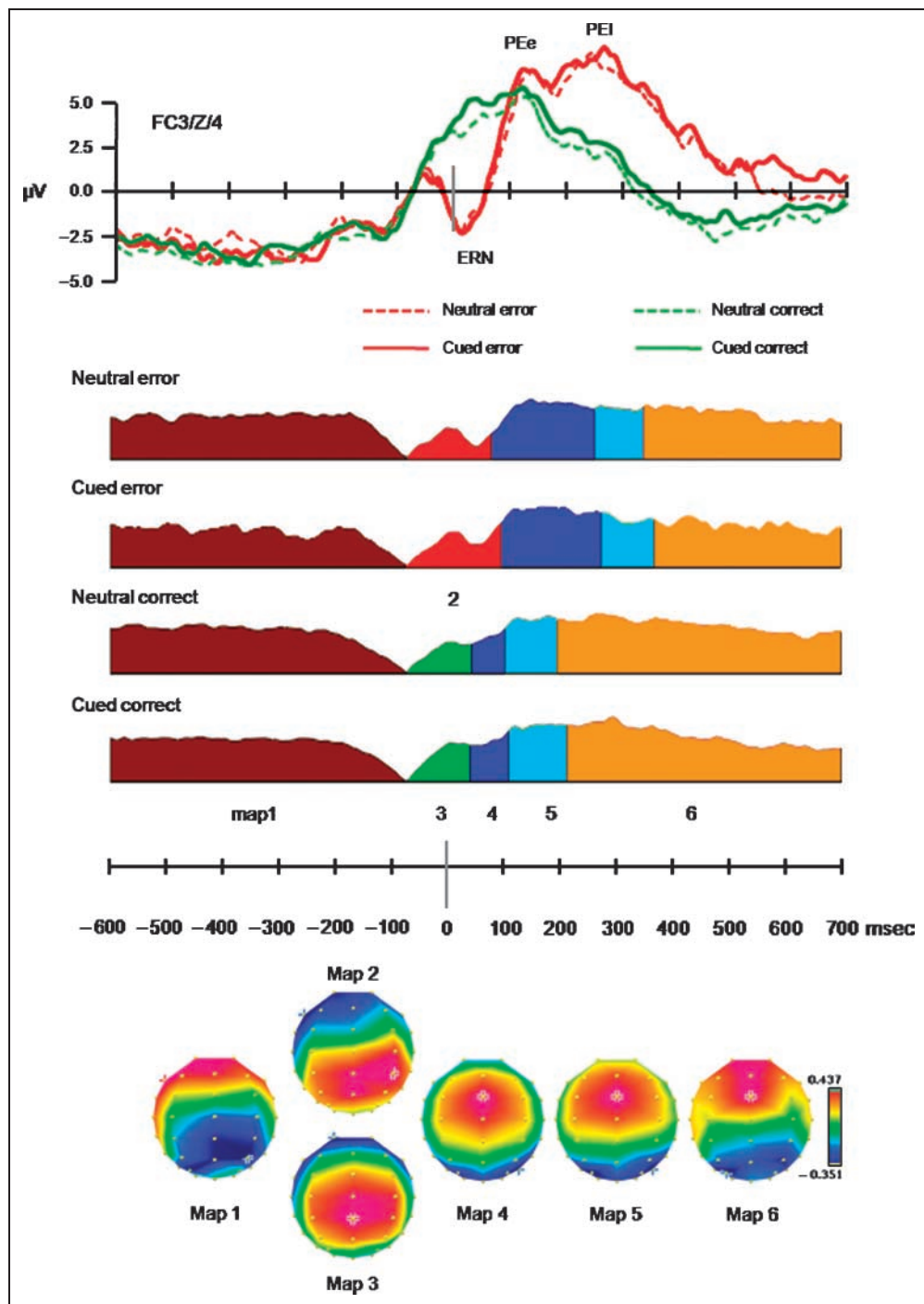
Topographical analyses were performed to test the following two observations based on the morphology of the waveforms: (a) whether the ERN and the Pc had different topographies associated to errors and correct responses, respectively, and (b) whether the similarities in the two positive peaks after the ERN and the Pc for errors and correct responses, respectively, corresponded to similar topographical distributions that were offset in time.

Figure 4 (center) shows the time course of the segmentation maps corresponding to stable voltage topographies, colored on the global field power waveform, for each of the four conditions. The progression of maps was equivalent for all the conditions except during the period of the ERN and the Pc (Maps 2 and 3). Map 2 was only present on error conditions, whereas Map 3 was only present on correct responses. The topographical representation of these segments (Figure 4, bottom) showed a frontal negativity that was specific to Map 2 but not Map 3, which was slightly lateralized to the left hemisphere. In the group-averaged waveforms (Figure 4, top), this negativity corresponds to the ERN potential that was specifically observed on error conditions.

An ANOVA testing the preponderance of Maps 2 and 3 across conditions between  $-100$  and  $+60$  msec confirmed the difference in distribution for error versus correct trials. The interaction between response and topographical map was significant,  $F(1,15) = 26.12, p < .001$ . For errors, the ERN topography (Map 2) was significantly dominant over Map 3 (65% of the entire 160-ms temporal window vs. 35%),  $t(15) = 2.1, p = .027$ , one-tailed. In contrast, for correct responses, Map 3 was significantly dominant over Map 2 (63% vs. 37%),  $t(15) = 1.77, p = .048$ , one-tailed.

The maps after the ERN map (Maps 4–6) followed identical sequences for both error and correct responses. The main difference consisted of Map 4 (early Pe) being present for a longer duration on errors than on correct responses. This visual impression was tested by ANOVAs

**Figure 4.** Top: Response-locked ERP waveforms averaged across 16 participants for errors (red) and correct responses (green) as a function of cueing (cued, solid line; neutral, dashed line). ERPs recorded at frontocentral electrodes are shown (left, center, and right electrode sides are collapsed in this figure). The main error-related potentials are labeled (PEe/PEl, early/late Pe). On errors, the ERN and the Pe did not differ for cued versus neutral conditions. On correct responses, the first positive potential was enhanced by cueing. Center: Segmentation maps of the group-averaged data for the four conditions during the interval between  $-600$  and  $+700$  msec (zero indicates response execution, keypressing). Map 2 was present on errors, whereas Map 3 was present on correct responses. Maps 4, 5, and 6 followed the same sequence for both errors and correct responses. Map 4 was longer after errors compared with correct responses. Bottom: Topographical representation of the voltage distribution over the scalp averaged across each segmentation map. Map 2 shows the ERN distribution as a negative voltage (blue) over the frontal part of the head.



testing the preponderance of Maps 4 and 5 for errors and correct responses over three consecutive 80-ms bins from 0 to 240 msec. The interaction between response, topographical map, and time bin was significant,  $F(2,30) = 7.58, p = .002$ . For errors, Map 4 was significantly dominant over Map 5 during the first time bin (77% vs. 23%),  $t(15) = 3.05, p = .004$ , one-tailed, and the second bin (68% vs. 32%),  $t(15) = 2.04, p = .029$ , one-tailed, but not at the third bin (56% vs. 44%),  $t(15) = 0.59, p = .28$ , one-tailed. In contrast, for correct re-

sponses, Map 4 was dominant over Map 5 during the first time bin only (80% vs. 20%),  $t(15) = 3.91, p < .001$ , one-tailed, but not at the second bin (60% vs. 40%),  $t(15) = 1, p = .18$ , one-tailed. At the third bin, the pattern reversed significantly, so that Map 5 was now dominant over Map 4 (75% vs. 25%),  $t(15) = 2.24, p = .02$ , one-tailed, suggesting that the processing stage indexed by topographical Map 4 was resolved at that time for correct responses. In brief, Map 4 remained for longer after errors (0–160 msec) than after correct responses (0–80 msec).



## DISCUSSION

### Modulation of Conflict Processing by Proactive and Reactive Control

The analysis of behavioral performance confirmed that both proactive and reactive control effectively facilitated conflict resolution. This facilitation was also reflected in ERP measures. The main finding showed that proactive cueing altered the time course of conflict processing.

We expected proactive anticipation of conflict to engage increased top-down control, hence to facilitate conflict detection, as indexed by the N2 potential. The high temporal resolution of ERPs was critical to uncover that proactive control enhanced on-line conflict processing by reducing the latency of the N2. This novel finding reveals the ability to prepare for conflict based on predictive information. More generally, this finding confirms that top-down attention is not only constrained to cued physical attributes of forthcoming stimuli and responses (for a review, see Nobre, 2004). Rather, in the present study, cueing prompted the anticipation of conflict and the need to engage the control system for cautious stimulus-response selection. As a result, conflict was detected and resolved more rapidly. The rapid detection of conflict was particularly useful under the task parameters of the current experiment, which imposed very high temporal pressure for responding. When more deliberate processing is possible under normal conditions of time pressure (being all the remaining task parameters identical to the current experiment), the effects of proactive cueing of conflict may become less conspicuous (A. Correa, A. Rao, J. Lupiáñez, and A. C. Nobre, unpublished observations).

The temporal modulation of conflict detection by proactive control had remained unnoticed so far. Hemodynamic neuroimaging studies have provided unclear results, probably because the technique is highly insensitive to short-lived or latency effects. During the anticipatory interval, increments in ACC activation have been observed in some studies (Sohn et al., 2007; Luks et al., 2002) but not others (Luks et al., 2007; Fassbender et al., 2006; MacDonald, Cohen, Stenger, & Carter, 2000). During conflict processing, decrements in ACC activation selectively related to cued conditions have been observed in some studies (Luks et al., 2007; Sohn et al., 2007) but not others (Fassbender et al., 2006; Luks et al., 2002). The inconsistency of the fMRI results has then precluded establishing strong conclusions about the role of ACC in anticipatory conflict monitoring. Importantly, however, the present ERP results strongly suggest that conflict processing can be anticipated. As recently suggested, this anticipatory mechanism may be mediated by preactivation of ACC during the preparatory interval (Sohn et al., 2007).

An additional explanation for the absence of proactive modulations upon ACC activity considers that the previous studies cueing conflict did not control for se-

quential effects. It is possible that proactive cueing could not attenuate ACC activity further when it was already attenuated by reactive control. Therefore, this possibility assumes that proactive and reactive control involve common or interacting neural mechanisms. To our knowledge, the current study is the first to test this assumption by comparing neural modulation resulting from both types of control directly. The temporally rich dependent variables provided by ERPs were able to identify particular stages during which proactive and reactive control interact. From early through late neural target processing, proactive control was most effective under low reactive control. Specifically, the attenuation of the early N1f by cueing was stronger after congruent versus incongruent trials and the amplification of the late P3 was only significant after congruent trials. Analogously, reactive control was only effective in the absence of proactive control: N2 attenuation and P3 amplification by previous conflict were only significant on neutral versus cued conditions. These interactions suggest neural overlap or crosstalk between the control networks triggered by proactive and reactive signals.

The overlap between proactive and reactive control was only partial, however: Each modulated conflict processing with distinct time courses and spatial distributions. The anticipatory effects of proactive control upon conflict processing started early (frontal N1 at 110 msec)<sup>5</sup> and later became lateralized to the left hemisphere (P3). In contrast, the effects of reactive control started later (N2) and were lateralized to the right. Moreover, the N2 latency was selectively reduced by cueing, thus illustrating the proactive nature of this type of control over more reactive sequential effects. Overall, these findings would suggest multiple rather than a single control mechanism, although such differences could be specific to our experimental procedure. For example, signals for reactive versus proactive control took place at different times, which could have led to differential modulations of processing. Further research will be useful to clarify whether neural mechanisms subserving reactive and proactive control can be more clearly dissociated.

Our results regarding reactive control are consistent with neuroimaging research showing that increments in ACC activity on incongruent conditions correlate with subsequent reductions of ACC activity and behavioral conflict (for a review, see Botvinick, Cohen, & Carter, 2004; Durston et al., 2003), even when repetition priming effects are controlled (Notebaert, Gevers, Verbruggen, & Liefvooghe, 2006; Kerns et al., 2004; see also Ullsperger et al., 2005; but see Nieuwenhuis et al., 2006; Mayr, Awh, & Laurey, 2003). Previous ERP studies controlling for priming effects have provided mixed results. In one case, the sequential modulation of conflict-related processing survived (Scerif, Worden, Davidson, Seiger, & Casey, 2006), but in another case it was abolished (Wendt, Heldmann, Munte, & Kluwe, 2007). Our behavioral analysis showed that priming did not account for

the reductions in conflict effect exerted by either proactive or reactive control. Nevertheless, because we had insufficient trials to confirm our ERP results after controlling for stimulus-response repetitions, these must be taken with caution.

### Electrophysiological Correlates of Error Processing

Assuming that the ERN reflects ACC activity related to response-related conflict (Botvinick et al., 2004) or error detection (Debener et al., 2005), we hypothesized that the anticipation of error in conflict trials would modulate the ERN. Unexpectedly, we observed no modulation of the ERN. In light of the strong interactions between cueing and sequential effects we observed in the analysis of target-related processing, it is possible that modulation of the ERN would be detectable in conditions of low reactive control. However, due to an insufficient number of error trials in the different conditions of previous congruency, this possibility could not be tested here.

The analysis of map topographies revealed that the ERN was specific to trials with incorrect responses, replicating earlier studies (Gehring et al., 1993; Falkenstein et al., 1990). The absence of the typical ERN topography on “correct” conditions replicates previous research (e.g., Ullsperger, von Cramon, & Müller, 2002) but contrasts with reports of the so-called “correct response negativity” (Bartholow et al., 2005; Falkenstein et al., 2000; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). The current topographical analysis showed that the ERPs associated with errors and correct responses were generated by differing configuration of neural sources rather than differing only in the overall strength of their activation.

Rather surprisingly, the topographical analysis further revealed that, following the ERN, the stages of neural processing in error trials were equivalent to those in correct trials, although protracted in time. This finding questions the specificity of the Pe potential to error processing (Falkenstein et al., 1990, 2000). Our findings suggest instead that a common late stage of post-response processing is achieved in both correct and error trials (e.g., contextual updating), but that the conflict and/or error detection delays the onset of this stage in error trials.

In summary, proactive control based on the anticipation of conflict significantly modulated subsequent target processing and did so through neural mechanisms that interacted with reactive control based on sequential effects. The effects of proactive control were maximal when controlled stimulus-response selection was not already engaged by reactive signals coming from conflict in the previous trial. The present findings suggest that anticipating conflict enhances cognitive control by speeding up conflict detection and resolution.

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Reprint requests should be sent to Ángel Correa, Departamento de Psicología Experimental y Fisiología del Comportamiento, Campus Universitario de Cartuja s/n, 18011, Granada, Spain, or via e-mail: act@ugr.es.

### Notes

1. The use of a standard keyboard as response device may have delayed (and jittered) the registration of the response event, which might explain the unusually short apparent latencies (and amplitudes) observed for the ERN.
2. Given the controversy on whether sequential effects reflect top-down conflict monitoring (Notebaert et al., 2006; Ullsperger et al., 2005; Kerns et al., 2004) or bottom-up priming effects due to stimulus-response repetitions (Nieuwenhuis et al., 2006; Mayr et al., 2003), the influence of priming was tested by including “repetition” as a within-subjects factor (e.g., Mayr et al., 2003). Repetition exerted no influence on either the Cueing  $\times$  Congruency interaction ( $F < 1$ ) or the Previous congruency  $\times$  Congruency interaction,  $F(1,18) = 1.27$ ,  $p = .27$ . In light of this null effect and because of restricted trial numbers, this variable was not analyzed further in the ERP analyses.
3. Note that Figure 2 shows the N2 peaking at about 250 msec for congruent targets, thus falling outside the time window used in the analysis. In any case, the conflict effect was clearly reliable for a broader time window of 240–300 msec including both incongruent and congruent N2 peaks [mean amplitude effect:  $F(1,16) = 11.05$ ,  $p = .004$ ].
4. It could be argued that this latency difference may have determined the mean amplitude results. A peak amplitude analysis was performed on the N2, which replicated the mean amplitude data. This analysis replicated the significant interaction between cueing and electrode side,  $F(2,32) = 4.06$ ,  $p = .027$ , and the three-way interaction between cueing, previous congruency, and electrode side,  $F(2,32) = 3.19$ ,  $p = .055$ , which showed that sequential effects attenuated the N2 peak amplitude over right frontal electrodes under neutral cueing conditions (mean peak amplitudes: 1.33  $\mu\text{V}$  for previous congruent vs. 2.61  $\mu\text{V}$  for previous incongruent),  $F(1,16) = 5.10$ ,  $p = .038$ .
5. The early N1f potential has not been specifically associated with conflict processing. However, the attentional modulation observed here is reminiscent of ERP studies on attention and emotion, which reported attenuation of the anterior N1 when fearful faces were attended (e.g., Holmes, Vuilleumier, & Eimer, 2003). Hence, it might be that our N1f was reflecting emotional processing in reaction to conflict and/or the prediction of error.

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